EQUINE SURGERY

FOURTH EDITION

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EQUINE SURGERY, FOURTH EDITION ISBN: 978-1-4377-0867-7

Library of Congress Cataloging-in-Publication Data

Equine surgery / [edited by] Jörg A. Auer, John A. Stick.—4th ed.
p. ; cm.
Includes bibliographical references and index.
ISBN 978-1-4377-0867-7 (hardcover : alk. paper)
I. Auer, Jörg A. II. Stick, John A.
[DNLM: 1. Horse Diseases—surgery. 2. Horses—surgery. 3. Surgery, Veterinary—methods. SF 951]
LC-classification not assigned
636.1′0897—dc23

2011034886

Vice President and Publisher: Linda Duncan
Publisher: Penny Rudolph
Associate Developmental Editor: Brandi Graham
Publishing Services Manager: Julie Eddy
Senior Project Manager: Laura Loveall
Designer: Paula Catalano

Printed in the United States

Last digit is the print number: 9 8 7 6 5 4 3 2 1

The Fourth Edition is dedicated to:

Our colleagues and fellow diplomates in the European and American Colleges of Veterinary Surgeons, without whom this book would never have been realized.

To the horses we so value, which inspire us to improve our craft.

To Anita and Claudette, our loving wives, who support us with great appreciation of our chosen profession.

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Preface

Our goal for the fourth edition of *Equine Surgery* was to maintain the high standard of the last edition and continue its position as the leading worldwide clinical reference and teaching textbook for clinicians, practitioners, surgery residents, and students. We, the editors, continued the format of the last edition by taking direct responsibility for inviting authors in the sections for which we were in charge; however, we exchanged the assignment of some sections between us. Prior to embarking on the detailed planning of the fourth edition, we asked a group of senior surgeons, ACVS and ECVS diplomates who recently passed their board examinations, and residents preparing for the certifying examination to evaluate the third edition for omissions that would improve the textbook as well as for chapters that could be excluded to make room for new material. The results of these evaluations were passed on to each contributor to this edition, and it greatly improved the content of the book. We thank Dr. Gary Baxter from Colorado State University, Dr. Larry Galupo from the University of California, Davis, Dr. John Peroni from the University of Georgia, Dr. Kimberly Johnston from Michigan State University, Dr. Rich Redding from North Carolina State University, Dr. Gabor Bodo from Budapest, and Dr. Jan Kümmerle from the University of Zurich in Switzerland for their service in review of the third edition.

We have continued to focus on the clinically relevant aspects of equine surgery, presenting information in a concise, understandable, and logical format. Extensive use of figures, tables, cross-referencing within and among sections, and a comprehensive index help make the fourth edition of *Equine Surgery* a quick and easy-to-use reference textbook.

ORGANIZATION

The book contains twelve sections, starting with surgical biology, surgical techniques, and recent advances in anesthesia, and followed by sections pertaining to all organ systems with one new section on diagnostic imaging. Each section is logically structured and supported extensively by photographs and tables. A comprehensive list of references completes each chapter. Additionally, we prepared appendixes that list drugs and products, their American and European manufacturers (where applicable), and the chapters where they were mentioned throughout the text.

KEY FEATURES OF THE FOURTH EDITION

We have retained all of the features that were popular in the first three editions and have significantly updated all chapters in the fourth edition. We continued to select known and novel contributors who are recognized as experts in their fields to author the chapters in this edition.

New Features

The new features include:

- Thoroughly revised and updated content with expanded coverage on current and new topics throughout the textbook
- Expanded use of the expertise of more ECVS authors to acquire additional international representation
- The addition Chapter 8, Regenerative Medicine, responding to the current trend in equine therapeutic medicine
- Expansion of the new science and expertise in diagnostic imaging, which was compiled into a section of its own—Section XI, Diagnostic Imaging Examination
- Reorganized and updated Section VIII, Eye and Adnexa
- Added Chapter 101, Temporomandibular Joint Disorders, and Chapter 103, Postoperative Physiotherapy for the Orthopedic Patient

Acknowledgments

We are very proud that we were able to produce this textbook in a timely fashion so that the content will continue to represent current "state-of-the-art" surgical procedures and techniques. To our contributors, once again, thank you for the marvelous work.

We would like to extend our sincerest thanks to Penny Rudolph, our motivating, joyful, and very competent publisher at Elsevier. Our thanks also go to Brandi Graham, who kept us patiently on track, helped us to stay on time, and did it all in a very gracious manner. We also thank Laura Loveall, our Project Manager, who oversaw copyediting, made sure all the authors turned in their edited version to us on time, and aided the process of continuity in the book. A special thank you goes to Matthias Haab of Zurich, Switzerland, who continued to do a marvelous job in preparing all of the new artwork. Not only is the quality of the artwork outstanding, he did this work with great efficiency, which made the final product first-rate.

CHAPTER 1

Shock: Pathophysiology, Diagnosis, Treatment, and Physiologic Response to Trauma

Elizabeth A. Carr

DEFINITION OF SHOCK

In 1872 the trauma surgeon Samuel D. Gross defined shock as "the rude unhinging of the machinery of life." Shock is the progression of a cascade of events that begins when cells or tissues are deprived of an adequate energy source (oxygen). Shock occurs as a result of inadequate tissue perfusion; the lack of an adequate energy supply leads to the buildup of waste products and failure of energy-dependent functions, release of cellular enzymes, and accumulation of calcium and reactive oxygen species (ROS) resulting in cellular injury and ultimately cellular death. Activation of the inflammatory, coagulation, and complement cascades result in further cellular injury and microvascular thrombosis. The amplification of these processes coupled with increased absorption of endotoxin and bacteria (due to liver and gastrointestinal dysfunction) lead to the systemic inflammatory response syndrome (SIRS) (see Chapter 2), and multiorgan dysfunction and if uncontrolled, ultimately death.

Classifications of Shock

Tissue perfusion is dependent on blood flow. The three major factors affecting blood flow are the circulating volume, cardiac pump function, and the vasomotor tone or peripheral vascular resistance. The interplay of these three factors can be seen in the formula for cardiac output (CO):

$$\text{Cardiac output (CO)} = \text{Stroke volume} \times \text{Heart rate}$$

CO ultimately determines the blood flow to tissues and is regulated, in part, by the stroke volume. Stroke volume is affected by the preload (amount of blood returning from the body and entering the heart), the cardiac contractility (muscle function), and the afterload or arterial blood pressure the heart must overcome to push blood through the aortic and pulmonic valves. Preload is directly affected by the circulating blood volume or amount of blood returning to the heart. Causes of decreased preload include loss of volume, hypovolemia, decreases in vasomotor tone, and vasodilation, which results in pooling of blood in capacitance vessels and decreased return to the heart. In this situation, although the total volume of blood remains unchanged, the effective circulating volume decreases. Afterload, the third component of CO, is directly affected by vasomotor tone or peripheral vascular resistance. If vascular resistance or tone increases, afterload also rises (hypertension) with a resultant fall in CO and perfusion. The opposite extreme is a severe fall in vascular resistance, which results in pooling of blood in capacitance vessels and a fall in blood pressure and preload, and it ultimately results in inadequate perfusion and shock. CO or flow can, therefore, also be described by the equation:

$$\text{CO} = \text{Blood pressure}/\text{Total peripheral vascular resistance}$$

Shock most commonly occurs because of one of three primary disturbances and can be classified accordingly. *Hypovolemic* shock is the result of a volume deficit, either because of blood loss (e.g., resulting from profound hemorrhage), third space sequestration (e.g., occurring with a large colon volvulus), or severe dehydration. *Cardiogenic* shock or pump failure occurs when the cardiac muscle cannot pump out adequate stroke volume to maintain perfusion. *Distributive* shock or microcirculatory failure occurs when vasomotor tone is lost. Loss of vascular tone can result in dramatic fall in both blood pressure and venous return. Although the drop in blood pressure will initially decrease afterload (which will improve CO), the pooling of blood and loss of venous return results in a severe decrease in preload and consequently, decreased CO and perfusion.

Common causes of distributive shock include neurogenic shock, septic shock, and anaphylactic shock. Because distributive shock is a loss in effective circulating volume, fluid therapy is indicated to help restore perfusion. In contrast, cardiogenic shock is the result of pump failure, and fluid therapy may actually worsen clinical signs. Less commonly, shock can develop when increased metabolic demand results in relative perfusion deficits or when oxygen uptake is impaired because of mitochondrial failure, sometimes termed relative hypoxia or dysoxia.

It is important to recognize that although the inciting cause may differ, as shock progresses, there is often failure of other areas as well. For example, untreated hypovolemic shock can result in microcirculatory failure (loss of vasomotor tone) as oxygen debt causes muscle dysfunction and relaxation. Alternatively, hypovolemic shock can result in myocardial failure as perfusion deficits affect energy supply to the

myocardium (coronary artery blood flow), resulting in decreased cardiac contractility. Consequently, as shock progresses, treatment may require addressing all three disturbances.

An additional category called obstructive shock is described where the mechanism underlying shock is the obstruction of ventilation, or CO. This process is most commonly caused by tension pneumothorax (resulting in decreased venous return) or pericardiac tamponade, resulting in inadequate ventricular filling and stroke volume. Over time as aortic blood pressure falls, coronary artery blood flow is reduced, and myocardial ischemia and finally myocardial failure may develop. Because obstructive shock is ultimately a combination of the other three categories and rarely occurs in large animals, we will not discuss it further.

PATHOPHYSIOLOGY OF SHOCK

A blood loss or hypovolemic model of shock will be used to describe the pathophysiology of shock.

Shock is usually defined by the stage or its severity. Compensated shock represents an early or mild shock, during which the body's response mechanisms are able to restore homeostasis. As blood volume is depleted, pressure within the vessels falls. Baroreceptors and stretch receptors located in the carotid sinus, right atrium, and aortic arch sense this fall in pressure. These receptor responses act to decrease inhibition of sympathetic tone while increasing inhibition of vagal activity and decreasing the release of atrial natriuretic peptide (ANP) by cardiac myocytes. The increase in sympathetic tone and fall in ANP results in vasoconstriction, which increases total peripheral resistance and thereby increases blood pressure. Increased sympathetic activity at the heart increases heart rate and contractility, hence increasing stroke volume (SV) and CO. In addition, peripheral chemoreceptors stimulated by local hypoxia respond by enhancing this vasoconstrictive response. In mild to moderate hypovolemia these responses are sufficient to restore perfusion. Because these compensatory responses result in tachycardia, increased SV (increased pulse pressure), and shortened capillary refill time (CRT), the term hyperdynamic is often used to describe this stage of shock.

The vasoconstrictive response will vary between organ systems, with the greatest response occurring in the viscera, integument, and kidney. Cerebral and cardiac flow is preferentially maintained in mild to moderate hypovolemia. Although this response improves blood pressure and flow, it also decreases perfusion to individual microvascular beds, worsening local hypoxia. Consequently, as volume depletion worsens, certain tissues and organs will become ischemic more rapidly than others.

Other compensatory responses help to restore blood volume. An increase in precapillary sphincter tone results in a drop in capillary hydrostatic pressure, which favors movement of fluid into the capillary bed from the interstitium. This transcapillary fluid movement helps restore circulating volume by creating an interstitial fluid deficit. Transcapillary fill is sufficient to restore circulating volume with blood loss of 15% or less.

In addition to transcapillary fill, a decrease in renal perfusion results in secretion of renin from juxtoglomerular cells located in the wall of the afferent arteriole. Renin stimulates production of angiotensin I, which, after conversion to angiotensin II, increases sympathetic tone on peripheral vasculature and promotes aldosterone release from the adrenal cortex. Aldosterone restores circulating volume by increasing renal tubular sodium and water reabsorption. Vasopressin, released from the posterior pituitary gland in response to decreased plasma volume and increased plasma osmolality, is a potent vasoconstrictor and stimulates increased water reabsorption in the renal collecting ducts. Finally, an increase in thirst and a craving for salt is mediated by both the renin-angiotensin system and a fall in ANP (Figure 1-1).

With more severe blood loss (15% or more), compensatory mechanisms become insufficient to maintain arterial blood pressure and perfusion of vital organs. This stage is termed uncompensated or hypodynamic shock. Ischemia to more vital organs including the brain and myocardium begins to develop. Blood pressure may be maintained, but clinical signs including resting tachycardia, tachypnea, poor peripheral pulses, and cool extremities are present. Mild anxiety may be apparent as well as sweating from increased sympathetic activity. Urine output and central venous filling pressure will drop. As blood loss progresses, compensatory mechanisms are no longer capable of maintaining arterial blood pressure and perfusion to tissues. Severe vasoconstriction further worsens the ischemia such that energy supplies are inadequate and cellular functions (including the vasoconstriction responses) begin to fail. In addition, accumulations of waste products of metabolism (lactate and CO_2) cause progressive acidosis and further cellular dysfunction.

At the cellular level the combination of decreased oxygen delivery and increased accumulation of waste products results in loss of critical energy-dependent functions, including enzymatic activities, membrane pumps, and mitochondrial activity, leading to cell swelling and release of intracellular calcium stores. Cytotoxic lipids, enzymes, and ROS released from damaged cells further damage cells, triggering inflammation. Inflammatory cell influx, activation of the arachidonic acid cascade, the complement cascade, and the release of enzymes and ROS cause further cellular injury. Mitochondrial failure, calcium release, and reperfusion, if present, further increase production (and decrease scavenging) of ROS. Endothelial cell damage and exposure of subendothelial tissue factor further activate the coagulation and complement cascades. Formation of microthrombi coupled with coagulopathy impedes blood flow to the local tissues, worsening the already deteriorating situation. The lack of energy supplies coupled with accumulation of toxic metabolites, microthrombi formation, and the inflammatory injury ultimately result in vascular smooth muscle failure and vasodilation. The end results of decompensated shock are a pooling of blood and additional decreases in blood pressure, venous return, CO, and perfusion, ultimately resulting in organ failure (Figure 1-2). Failure of the gastrointestinal tract manifests itself as loss of mucosal barrier integrity resulting in endotoxin absorption and bacterial translocation. Renal ischemia leads to renal tubular necrosis and the inability to reabsorb solutes and water and excrete waste products. At the cardiac level, the continued fall in blood pressure and venous return decreases coronary blood flow. Cardiac muscle ischemia leads to decreased contractility and CO and ultimately to further deterioration of coronary artery blood flow. Acidosis and ischemia accentuate the depression of cardiac muscle function. These changes in combination with decreased venous return worsen hypotension and tissue perfusion (Figure 1-3).

As the situation deteriorates, compensatory mechanisms designed to continue to perfuse more vital organs like the heart

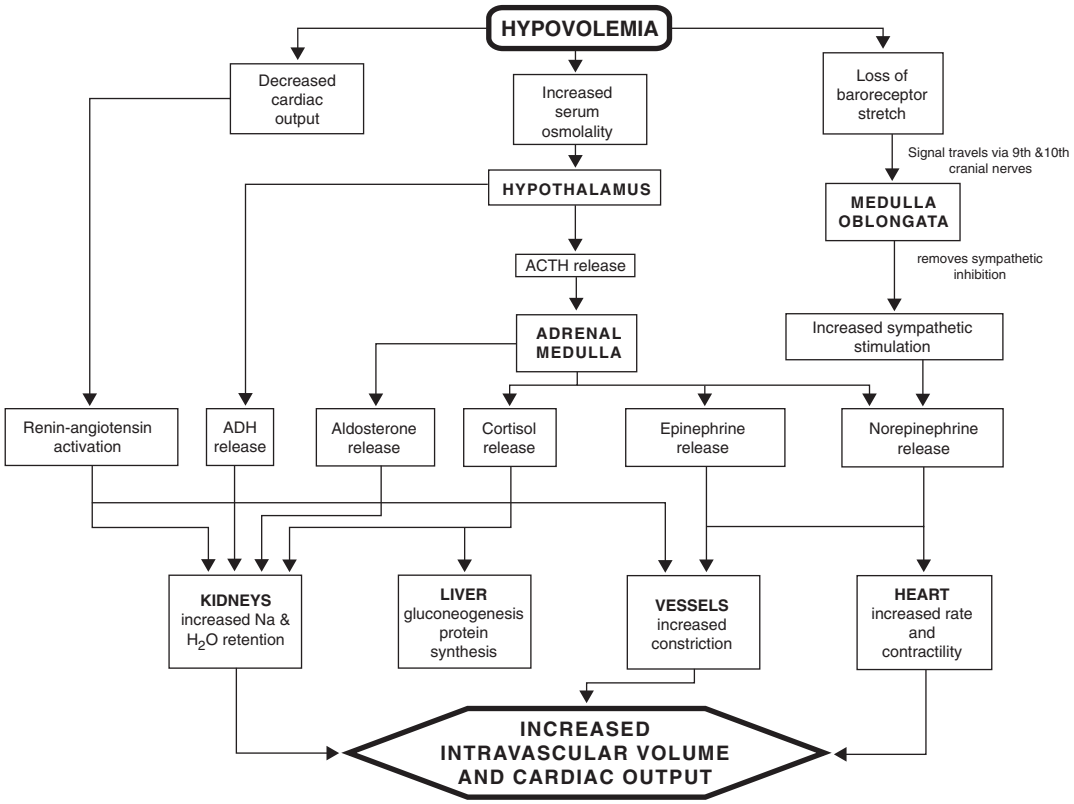

Figure 1-1. Physiologic compensatory responses to hypovolemia. *ACTH,* Adrenocorticotropic hormone; *ADH,* antidiuretic hormone. (From Rudloff E, Kirby R: Vet Clin North Am Small Anim Pract 24:1016, 1994.)

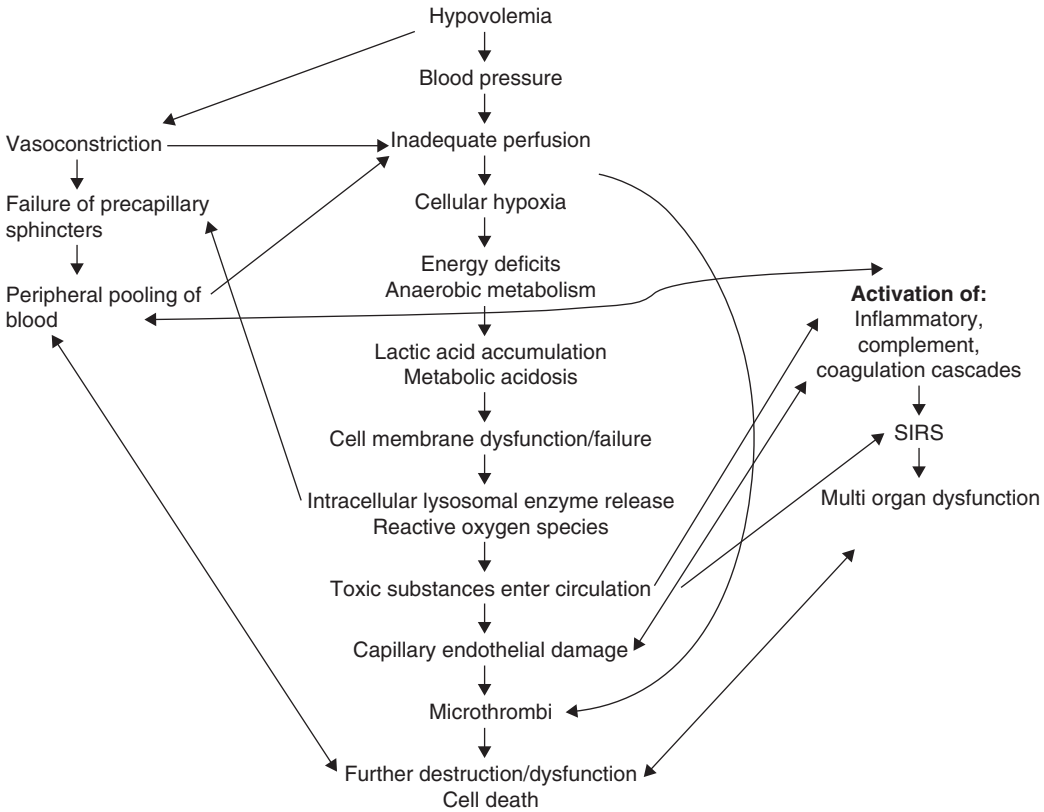

Figure 1-2. Cellular cascade of events that occur as the result of hypovolemia, poor perfusion and decreased oxygen delivery. *SIRS,* Systemic inflammatory response system.

and brain will continue to limit flow to other organs. This response results in the sparing of one organ with irreversible damage to another. Consequently, an individual may recover with aggressive intervention only to succumb later because of failure of these "less vital" organs. If blood flow is restored, these activated cellular and immunochemical cascades are washed into the venous circulation and lead to SIRS, multiple organ failures, and death (see Chapter 2). Intervention can no longer stop the cascade of events, and cellular, tissue, and organ damage is too severe for survival.

CLINICAL SIGNS OF SHOCK

Clinical signs of shock depend on the severity and persistence of blood loss. The American College of Surgeons advanced trauma life support guidelines divide shock into four categories with progressive blood loss.

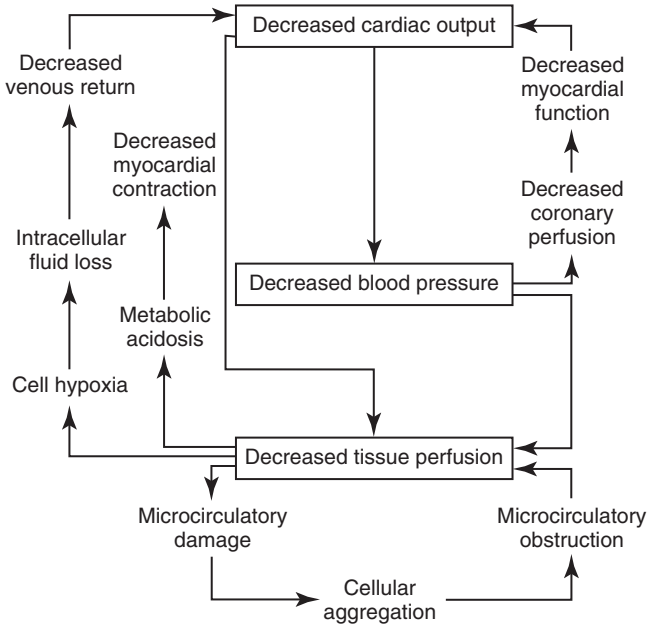

Figure 1-3. Viscious cycle of cellular and organ failure in shock.

With mild blood loss of less than 15% blood volume (Class I), the body is capable of restoring volume deficits via compensatory responses and there may be little to no change in physical parameters other than a fall in urine output. Blood pressure is maintained. Clinical signs typically become apparent when blood loss exceeds 15%. Early Class II blood loss (15% to 30%) is defined as the onset of hyperdynamic shock. Clinical signs include tachycardia, tachypnea, and bounding pulses (increased CO and peripheral vascular resistance). Mental agitation or anxiety is present, and increased sympathetic output results in pupil dilation and sweating. Although these compensatory mechanisms can normalize blood pressure, perfusion deficits will persist and can be detected by blood gas analysis (increased lactate and an anion gap metabolic acidosis). If blood loss continues, or if hypovolemia persists, compensatory mechanisms can become insufficient to restore circulating volume and decompensatory shock begins (Class III or moderate hypovolemic shock). At this time profound tachycardia and tachypnea, anxiety, and agitation are present. Urine output may desist, jugular filling and CRT are prolonged, pulse pressure is weak, and extremity temperatures are decreased. If blood gases are collected, a high anion gap acidosis and significant hyperlactatemia will be present (Table 1-1). Blood pressure will fall despite increases in heart rate, cardiac contractility, and total peripheral resistance. Without intervention, continued cellular hypoxia and acidosis result in failure of compensatory mechanisms, causing peripheral vasodilation and decreased cardiac contractility. A vicious cycle ensues with decreased coronary artery perfusion causing decreased cardiac function, resulting in decreased CO and a further fall in perfusion (see Figure 1-3). If uncontrolled, clinical signs will progress from tachycardia and anxiety to bradycardia, obtundation, anuria, profound hypotension, and circulatory collapse.

TREATMENT

Fluid Administration

Regardless of the underlying etiology of shock (cardiac failure, blood loss, or distributive problems), the greatest need is to restore perfusion and oxygen delivery to the tissues. Delivery of oxygen is determined by the concentration of oxygen in the blood as well as the amount of blood perfusing the tissue. The

TABLE 1-1. Clinical Assessment of the Different Stages or Progression of Shock

Parameter	Mild Compensated Shock Class I	Moderate Hypotension/ Shock Class II-III	Severe Hypotension/Shock Class III-IV
Extremity temperature	May be normal or cool	Cool	Cool to cold
Mentation	Normal to anxious	Agitation to lethargy	Obtunded
Urine output	Decreased	Decreased	Anuria possible
CRT	Normal to prolonged	Prolonged	End stage shock may be shortened because of blood pooling in peripheral tissues
Heart rate	Normal to tachycardia	Tachycardia	Severe tachycardia; bradycardia at end stage
Respiratory rate	Normal to tachypnea	Tachypnea	Tachypnea; bradypnea possible at end stage
Blood pressure	Normal	Normal to decreased	Decreased
Oxygen extraction ratio	May be normal	Increased	Increased
PvO_2	May be normal	Decreased	Decreased
Blood lactate	Mild increase	Increased	Markedly increased
Arterial pH	Normal to acidotic	Normal to acidotic	Acidotic
Central venous pressure	Normal to low	Low	Low

CRT, Capillary refill time.

concentration of oxygen per volume of blood is determined by the amount of hemoglobin or red cell mass and the saturation of that hemoglobin. It is important to assess both the hemoglobin concentration and the oxygen saturation because these will affect oxygen delivery. Decreased oxygen delivery is most commonly the result of decreased perfusion, not decreased oxygen content, but it is critical to evaluate all contributing factors when planning a treatment protocol for an individual in shock. Because hypovolemia is the most common cause of shock in the adult horse, fluid therapy is usually vital to restoring oxygen delivery. Extensive research efforts have addressed the determination of the ideal types and volumes of fluid for treating hypovolemic shock.

In the past, recommendations have been to rapidly infuse large volumes of isotonic crystalloids to replace circulating volume (shock dose). Because of their accessibility and low viscosity, crystalloids can be rapidly given and quickly restore volume. However, approximately 80% of the volume of administered crystalloids will diffuse out of the vascular space into the interstitial and intercellular space. Consequently, when using crystalloids, replacement volumes must be 4 to 5 times greater than the volume lost in order to restore the intravascular volume. In acute blood loss or hypovolemic states, this approach will result in excess total body water and extreme excesses of sodium and other electrolytes. This movement of fluid out of the vascular space is further exacerbated if the underlying disease process causes vascular leak syndrome, because intravascular colloid oncotic pressure will fall, favoring greater fluid movement out of the vascular space. In addition, if the electrolyte constituents of isotonic crystalloids differ from those in the intracellular space, cellular swelling will ensue. Cellular swelling affects the activity of various protein kinases; increases intracellular calcium concentrations; alters ion pump activity, membrane potential, and cytoskeletal structure; and activates phospholipase A_2.[1] Consequently, crystalloids can trigger or potentiate an inflammatory response and have a negative impact in the face of ischemia and reperfusion. Furthermore, large-volume infusions can result in significant complications including abdominal compartment syndrome, acute respiratory distress syndrome, congestive heart failure, gastrointestinal motility disturbances, and dilutional coagulopathy.[2]

Clinical trials have questioned the need for complete and rapid restoration of volume to maximize survival. In multiple hemorrhagic shock models, aggressive fluid therapy before hemorrhage control was associated with more severe blood loss, poorer oxygen delivery, and a higher mortality rate compared to more controlled, limited fluid therapy.[3,4] In a porcine model of uncontrolled hemorrhage, researchers studied the effects of three resuscitation regimens designed to mimic triage in the field before admission to a trauma center. One group received aggressive fluid resuscitation using crystalloids to restore CO to original levels, the second group received limited fluid therapy to restore CO to 60% of baseline, and the third group received no prehospital fluid therapy. Compared to aggressive fluid therapy, the limited resuscitation group lost less blood overall and had increased oxygen delivery (although survival was similar in all groups). In a prospective randomized clinical trial, subjects presented to a major trauma center with penetrating torso injuries and hypotension were assigned to either an immediate resuscitation or a delayed resuscitation group. Patients in the immediate resuscitation group received standard care including placement of bilateral IV catheters and rapid infusion of crystalloids during transport and triage at the emergency room. The latter group did not receive resuscitative fluid therapy until emergency surgery was begun. A total of 598 adults were included in the study; survival was 70% in the delayed resuscitation group compared to 62% ($p = 0.04$) in the immediate resuscitation group. In addition, patients in the immediate resuscitation group had more in-hospital complications (30%) including acute respiratory distress syndrome, sepsis, acute renal failure, coagulopathy, wound infection, and pneumonia compared to the delayed resuscitation group (23%). Although later studies have contradicted these results, the study does call into question the use of rapid, large-volume crystalloid fluid therapy for all hypovolemic shock cases.[5,6]

Clearly there are pros and cons to immediate, large-volume fluid resuscitation in the treatment of hypovolemic shock. Perfusion deficits need to be addressed, but the goal of therapy may need to be considered in light of the potential negative effects of infusing a large volume of fluids. The original idea of supranormal resuscitation (i.e., a shock dose of fluids) was based on the theory that tissue injury results in additional losses and sequestration of fluid into a third space, as well as the recognition that the majority of isotonic fluid infused into the vascular space will shift to the extravascular compartments. However, this additional third space loss has not been proved, and there may be negative consequences to supranormal resuscitation protocols. Large-volume fluid therapy has also been associated with cardiac and pulmonary complications[7,8] in both healthy human patients undergoing elective surgery and patients with risk factors for cardiopulmonary disease. Large-volume fluid therapy in patients with underlying SIRS or patients that have a low colloid oncotic pressure can result in significant edema, which can negatively affect gut motility and gut barrier function[9] and affect the function of other organ systems.

Despite this discrepancy in the literature, the reality is that shock is a manifestation of perfusion deficits, and the goal of therapy should be to restore perfusion and improve oxygen delivery. Prompt fluid therapy is indicated in the emergency situation to increase vascular volume, restore CO and blood pressure, and ultimately perfusion to the tissues. The amount and type of fluids should be determined by the individual needs of each patient. Careful, frequent monitoring to assess responses and prevent overload is recommended.

Types of Fluids

ISOTONIC CRYSTALLOIDS

Commercially available isotonic crystalloids for large animal medicine are designed to be replacement fluids, not maintenance fluids, meaning that the electrolyte composition is designed to closely approximate the electrolyte composition of the extracellular fluid and not the daily replacement needs. The isotonic crystalloids available to horses include lactated Ringer solution, Plasma-Lyte, and Normosol-R and are principally composed of sodium and chloride with varying amounts of calcium, potassium, and magnesium. Physiologic saline solution (0.9%) differs in that it contains only sodium and chloride and no other electrolytes.

These solutions are very useful in restoring fluid deficits in simple dehydration. Because the electrolytes are freely diffusible, approximately 80% of these fluids will diffuse into the interstitial and intracellular space from the extravascular space. This means that approximately 2 L of a 10 L fluid

bolus will remain in the vascular space. Consequently, a larger volume is needed to restore and maintain effective circulating volume.

The recommended method of administering isotonic crystalloids for hypovolemic shock is to calculate the fluid deficit and initially infuse fluids in doses of 10 to 20 mL/kg. Because the assessment of deficits is inexact, it is important to monitor the response during infusion and not to simply infuse the calculated amount. In cases of blood loss, infusion of crystalloids alone will cause dilutional anemia and hypoproteinemia. Depending on the severity of blood loss and amount of crystalloids infused, dilutional coagulopathy resulting from thrombocytopenia and dilution of clotting factors can occur, leading to further bleeding and deterioration. These patients may require subsequent plasma or whole blood transfusions to improve coagulation, oncotic pressure, and oxygen content of blood. Patients with endotoxemia or SIRS often have underlying coagulopathies as part of their disease process, leaving them at particular risk for further problems with aggressive crystalloid therapy.[10-12]

HYPERTONIC CRYSTALLOIDS

Hypertonic saline solution (HSS) is available in several concentrations, with 7.2% and 7.5% being the most commonly used formulations. The osmolarity of this concentration range is approximately 8 times the tonicity of plasma. An intravenous infusion of hypertonic saline will expand the intravascular space 2 to 4 times the amount infused, pulling fluid from the intracellular and interstitial spaces. This expansion is short-lived and, similar to the effects of isotonic crystalloids, the majority of fluid will ultimately diffuse into the interstitium. Hypertonic saline was initially developed for use in the battlefield because it allowed medics to carry small volumes of fluids and still provide resuscitative triage. The rapid and significant expansion of the intravascular volume using small-volume resuscitation allowed field stabilization of the patients before transport to a hospital unit. Because of the variation in reflection coefficients for sodium, HSS principally pulls volume from the intracellular space, not the interstitial space. This is particularly beneficial in the shock state, where endothelial volume rises with loss of membrane pump function. The decrease in endothelial cell volume increases capillary diameter and improves perfusion. In addition, HSS appears to blunt neutrophil activation and may alter the balance between inflammatory and anti-inflammatory cytokine responses to hemorrhage and ischemia.[13] The recommended dose of HSS is 2 to 4 mL/kg or 1 to 2 L for a 500-kg horse. Hypertonic saline is invaluable in equine surgical emergencies when rapid increases in blood volume and perfusion are needed to stabilize a patient before general anesthesia. The use of these fluids enables the clinician to quickly improve CO and perfusion to allow immediate surgical intervention. Additional blood volume expansion will be needed and can be provided during and after surgery to further restore homeostasis.

COLLOIDS

Colloids are solutions containing large molecules that, because of their size and charge, are principally retained within the vascular space. Because colloid concentrations are higher in the intravascular space, they exert an oncotic pressure that opposes the hydrostatic pressure and helps retain water in or draw it into the intravascular space. Normal equine plasma has a colloid oncotic pressure (COP) of about 20 mm Hg. Colloids with a high COP can actually draw additional fluid into the intravascular space. Consequently, infusion of certain synthetic colloids such as hetastarch (HES) (COP ~30 mm Hg) will increase intravascular volume by an amount that is greater than the infused volume. Although this effect is similar to HSS, the benefits of colloids are prolonged. Colloid therapy is recommended in patients that are hypo-oncotic, patients with capillary leak syndrome, patients with cardiac disease where fluid overload may be detrimental, and patients with fluid excess (edema) in which fluid therapy needs to be carefully titrated to prevent further overload.

Both synthetic and natural colloids are available. Natural colloids include plasma, whole blood, and bovine albumin. The advantage of natural colloids is that they provide protein, such as albumin; antibodies; critical clotting factors; and other plasma constituents. Because fresh frozen plasma must be thawed before infusion, it is often not useful in an emergency situation where immediate fluid therapy may be indicated. In addition, hypersensitivity reactions occur in up to 10% of horses receiving plasma.[14] The most common synthetic colloids are HES and dextrans, with HES being the most commonly used product in equine practice. HES contains amylopectin molecules of sizes ranging from 30 to 2300 kDa (average 480 kDa) and exerts a COP of 30 mm Hg. The elimination of HES occurs via two major mechanisms: renal excretion and extravasation. Larger molecules are degraded over time by α-amylase. The presence of molecular substitutes on the amylopectin chains slows this process of degradation to smaller colloid particles, and consequently the effect of HES is prolonged. A dose of 10 mL/kg will significantly increase oncotic pressure for longer than 120 hours.[15] Though evidence of spontaneous bleeding in healthy horses has not been documented, an increase in the cutaneous bleeding time was seen with larger doses (20 mL/kg) and has been associated with a decrease in von Willebrand factor antigen (vWf:Ag). Consequently, the use of large volumes of HES should be considered in light of bleeding tendencies of patients.[15] Measurement of COP must be used to assess the response to HES, because its infusion is not reflected in the total solids or total protein measurements, making these inaccurate estimates of the COP after HES infusion. HES infusions will actually decrease total protein because of the dilutional effect of the volume expansion.

HYPERTONIC SALINE SOLUTION AND DEXTRAN

The combination of hypertonic crystalloids and synthetic colloids offers the advantage of both rapid and persistent volume support and also provides some of the anti-inflammatory benefits of hypertonic saline. HSS with dextran (HSS-D) has been shown to expand the plasma volume,[16,17] restore hemodynamics,[18,19] and improve microcirculatory perfusion[20,21] in animal models of hemorrhagic shock. In addition, HSS-D has been shown to decrease neutrophil adhesion and blunt the hemorrhage-induced inflammatory response. The majority of human clinical trials have yet to show that it has a benefit over other fluid therapies.

WHOLE BLOOD

Whole blood is the ideal replacement fluid in shock due to blood loss. The use of blood or plasma provides clotting factors and prevents dilutional coagulopathy. By providing red blood cells (RBCs) and protein it helps retain fluid within the intravascular space and improves oxygen content of the blood. However, there are several disadvantages to whole blood. It is unusual for

most equine referral hospitals to store whole blood; consequently, it must be collected each time it is needed. In addition, because of its viscosity, it is difficult to rapidly infuse large volumes in an emergency situation. However, despite these drawbacks, the use of blood or blood components can be a valuable adjunct in preventing some of the potential side effects of large-volume resuscitation, namely dilutional coagulopathy, dilutional hypoproteinemia, and anemia. Ironically, data in human medicine suggests that blood products should be replaced in a ratio of plasma, RBCs, and platelets that approximates whole blood.[22] Because the most commonly available blood product in equine clinics is whole blood, the determination of an ideal ratio is a moot point! The use of whole blood is generally unnecessary in the patient with mild to moderate hypovolemia because restoration of perfusion often results in adequate oxygen delivery despite dilutional anemia. In more severe cases of hypovolemia or in cases with ongoing bleeding, whole blood may be indicated to provide oxygen-carrying capacity, colloid oncotic support, platelets, and coagulation factors.

CURRENT RECOMMENDATIONS

The debate regarding the use of crystalloids versus colloids is extensive. Despite this intense focus, clear benefits of colloids or hypertonic solutions over isotonic crystalloids have not yet been demonstrated. Rather than always using one or the other, the choice should depend on the situation. In a case of severe blood loss, hypovolemia, and impending circulatory collapse, the rapid expansion of blood volume using hypertonic and isotonic crystalloids may be imperative. The addition of colloids, whether synthetic or natural, and whole blood should depend on the severity of shock and the underlying disease process as well as the response to initial treatment.

When presented with an adult horse in hypovolemic shock it is critical to use a large 10- or 12-gauge catheter and large bore extension set to maximize flow rate in the initial resuscitation phase. Because crystalloids have the lowest viscosity, they can be infused more rapidly than colloids or blood. If necessary, a fluid pump can be used to increase the rate of infusion. The general recommendation is to calculate a shock dose of fluids using the following formula: percent blood volume (L/kg body weight $\times$ 100) $\times$ body weight. In an adult horse the percent blood volume is estimated to be 7% to 9% of the total body weight or 35 to 45 L for a 500-kg horse. Given the pros and cons of large-volume resuscitation fluid, goals should be estimates and not absolutes. Frequent reassessment of the patient's cardiovascular status and blood gases is important for adequate resuscitation without causing secondary problems. Signs of improved intravascular volume include a decreased heart rate and improved capillary refill time, skin temperature, and mentation. If possible, the measurement of urine output is extremely useful in assessing perfusion, although urine specific gravity is less accurate because it will be affected by the infusion of large quantities of crystalloids and will no longer accurately reflect hydration status. In humans, the assessment of blood pressure can be useful in monitoring trends (i.e., an improvement of pressure toward normal). In situations where bleeding is uncontrolled, normalization of blood pressure should not be the goal because this may promote continued bleeding.

VASOPRESSORS

Vasopressors are rarely used in the standing adult horse in hypovolemic shock. Restoration of volume is the primary treatment goal. However, if the administration of appropriate fluid volumes and types is insufficient to stabilize the patient, vasopressors may be indicated, particularly as shock progresses and vasomotor tone and cardiac ischemia cause a further fall in perfusion. The most commonly used drug in the awake, adult horse is dobutamine. Dobutamine is a strong β_1-adrenoreceptor agonist with relatively weaker β_2- and α-adrenoreceptor affinity. Its primary use is to improve oxygen delivery to the tissues via its positive inotrophic action. Dobutamine has been shown to have benefit in improving splanchnic perfusion in multiple species, although clinical data are currently lacking in the horse. Recommended dosages are 1 to 5 µg/kg/min. Higher doses have been reported to cause hypertension in the adult horse.[23]

Norepinephrine has been reported to be useful in neonatal foals to restore adequate organ perfusion in vasodilatory shock. Norepinephrine has strong β_1- and α-adrenergic affinity, resulting in vasoconstriction and increased cardiac contractility. Norepinephrine has been successfully used in combination with dobutamine in persistently hypotensive foals with improved arterial pressure and urine output reported.[23] The use of norepinephrine in the awake adult horse has not yet been evaluated.

At this time, there is little published information on the use of vasopressors to treat hypovolemic shock in the awake adult horse. Consequently, it is difficult to make recommendations for their use at this time. Close monitoring of urine output and blood pressure is recommended when using vasopressor therapy. Readers are directed to Chapter 2 for additional treatment recommendations for septic shock.

Monitoring

The body's compensatory responses are designed to restore many of the parameters used to assess hypovolemia or perfusion deficits. Consequently, in the early stages of shock, there is no perfect measure to assess progression. Despite this, there are several physical and laboratory parameters that can be useful in monitoring the patient's progression and response to treatment.

Repetitive physical exams focusing on assessment of CO and perfusion may be the most sensitive method to assess a patient, especially during early compensated shock when subtle changes may indicate impending decompensation. Heart rate, CRT, jugular venous fill, extremity temperature, pulse pressure, and mentation are all useful when repeatedly evaluated. Steady improvement and stabilization of these parameters in response to treatment would suggest a positive response. Continued tachycardia and poor pulse pressure, CRT, jugular fill, and deteriorating mentation despite treatment suggest that additional blood loss or decompensation is occurring.

Capillary Refill Time

Capillary refill time (CRT) is usually prolonged in hypovolemic shock. However, CRT can also be affected by changes in vascular permeability such as seen with endotoxemia or sepsis. In these situations, CRT may actually decrease because of vascular congestion and pooling of blood in the periphery. Though CRT at any one time point can be misleading, if assessed over time, it is useful in evaluating the progression of shock. Jugular fill is a relatively crude assessment of venous return or central venous pressure.

Central Venous Pressure

Central venous pressure (CVP) assesses cardiac function, blood volume, and vascular resistance or tone. Holding off the jugular vein should result in visible filling within 5 seconds in a normally hydrated horse that is standing with an elevated head. If filling is delayed, venous return or CVP is decreased. A more accurate estimate of CVP can be obtained with a water manometer, attached to a large-bore jugular catheter and placed at the level of the heart base or point of the shoulder. Normal CVP in standing horses ranges from 7 to 12 mm Hg, with pressure measured by inserting a catheter into the right atrium.[24-26] Measurement of pressure in the jugular vein using a standard IV catheter will result in falsely elevated CVP; however, this measurement can still be a useful estimation. During an experimental blood loss model, CVP fell to zero or below with a loss of 15% to 26% of circulating volume. Because CVP is a measure of venous return it can be used to assess the adequacy of fluid resuscitation and prevent fluid overload, especially in patients at risk for edema. If clinical signs are deteriorating despite a normal CVP, hypovolemia alone is not the cause. Low CVP can occur with hypovolemia or a fall in effective circulating volume, as occurs with distributive shock. Cardiogenic shock (or fluid overload) can result in an elevated CVP, because forward failure of the cardiac pump results in backup of blood within the venous side of the system. In this case, jugular veins may appear distended even with the head held high. Cardiogenic shock is a relatively uncommon cause of shock in adult horses.

Urine Output

Urine output is a sensitive indicator of hypovolemia with normal urine production being approximately 1 mL/kg/hr or more, depending on how much water an individual is drinking. Urine production of less than 0.5 mL/kg/hr suggests significant volume depletion, and fluid therapy is indicated to prevent renal ischemia. Urine output is rarely measured in adult horses, though it is relatively simple to perform and commonly done in neonatal medicine. Urine production can be useful for monitoring resuscitative strategies and determining endpoints in such therapies. Urine production coupled with improvement in physical exam parameters suggests a positive response to treatment. Though urine specific gravity can be used to assess renal concentrating efforts and consequently the water balance of the animal, it will be affected by intravenous fluid therapy and is not an accurate reflection of dehydration or volume status once bolus intravenous fluids have been begun.

Arterial Blood Pressure

Arterial blood pressure is a reflection of CO and total vascular resistance. Consequently, the measurement of a normal blood pressure does not directly correlate with adequate perfusion. Because of the compensatory increase in peripheral resistance, blood pressure does not consistently fall below normal until blood volume is profoundly decreased (30% or more). Though a normal blood pressure does not rule out hypovolemic shock, a low blood pressure is often an indicator of significant blood loss. Treatment goals should be to maintain mean arterial pressure above 65 mm Hg to ensure adequate perfusion of the brain. Blood pressure can be measured directly via arterial catheterization of the transverse facial artery in the adult horse or the transverse facial, metatarsal, radial, and auricular arteries in a neonate. Indirect measurement of the blood pressure can be achieved using the coccygeal artery in adult horses and the metatarsal artery in foals.[27] In healthy individuals there is good agreement between both direct and indirect measurements.[27-29] Direct, invasive blood pressure monitoring is more accurate during states of low flow and significant vasoconstriction.[28,29] Normal systolic blood pressure using indirect measurement at the coccygeal artery is 80 to 144 mm Hg. Because blood pressure will increase with increased vascular resistance, it is not an accurate reflection of oxygen delivery.

Lactate

Lactate is the end product of the anaerobic metabolism of glucose. Aerobic metabolism of glucose results in the production of 36 moles of adenosine triphosphate (ATP) per molecule of glucose. In the absence of adequate oxygen to meet energy demands, anaerobic metabolism of glucose to lactate results in production of only 2 moles of ATP. The shift to anaerobic metabolism of glucose with inadequate oxygen delivery to tissue increases blood lactate concentrations. Less commonly, hyperlactatemia can result from hepatic dysfunction (impaired clearance), pyruvate dehydrogenase inhibition, catecholamine surges, and sepsis or SIRS, although the increase in lactate level is generally less than what is seen with hypovolemia. Because lactate level generally correlates with oxygen delivery and uptake by the tissues, it is a useful marker for determining perfusion deficits and response to treatment. Delayed lactate clearance has been shown to be associated with a poorer prognosis in many human and veterinary studies.[30-34] A decrease in lactate following therapy indicates improved oxygen delivery and use, suggesting improved perfusion. Conversely, an increased or persistently elevated lactate level indicates continued tissue oxygen deficits. The anion gap will mimic lactate changes and has been used to assess oxygen debt; however, it can be affected by changes in other anions, such as plasma proteins, and is therefore not as accurate as blood lactate concentration.

Oxygen Extraction

The normal response to a decrease in perfusion or CO is to increase the oxygen extraction ratio (O_2ER) of the blood as it moves through the capillaries. By increasing the oxygen extraction, the body is able to maintain oxygen delivery to the tissue despite a fall in blood flow. Oxygen extraction is determined by the difference between the oxygen saturation of arterial blood (SaO_2) and oxygen saturation of venous blood (SvO_2):

$$O_2ER = SaO_2 - SvO_2$$

and can be determined by measuring central venous saturation and arterial oxygen saturation. Alternatively, it can be estimated by measuring jugular venous saturation and by using a pulse oximeter to assess arterial oxygen saturation. In the normovolemic, healthy individual, oxygen delivery (DO_2) far exceeds oxygen need or uptake (VO_2), and the O_2ER ranges from 20% to 30%. The O_2ER can increase with decreased perfusion to a maximum of 50% to 60%, at which point oxygen delivery becomes supply or flow dependent and a further drop in perfusion will result in a decrease in oxygen delivery. Because of this relationship, the O_2ER can be used to estimate the severity of

global perfusion deficits and is also a useful measurement in evaluating the response to resuscitative strategies.

Mixed Venous Partial Pressure of Oxygen

Mixed venous partial pressure of oxygen (PvO_2) is a useful measure to assess oxygen delivery for the same reasons that O_2ER is. In low-perfusion states, more oxygen is extracted per volume of blood and, consequently, PvO_2 will fall. Mixed venous blood is ideally measured by catheterizing the pulmonary artery, because a sample from the jugular vein or cranial vena cava only assesses venous blood returning from the head. Jugular venous PvO_2 is usually greater than mixed venous blood in the shock state, but it still has utility in estimating global tissue hypoxia.[35,37] Normal jugular vein PvO_2 ranges from 40 to 50 mm Hg and SvO_2 from 65% to 75%.[35,36] Increased PvO_2 in the presence of significant perfusion or supply deficits (DO_2) can signify impaired oxygen consumption caused by mitochondrial or cellular dysfunction, termed dysoxia. This syndrome has been recognized in septic shock or after cardiopulmonary resuscitation.

Cardiac Output

Cardiac output monitoring evaluates both volume return to the heart and cardiac function.[38] With prolonged or specific types of shock (septic), cardiac function may deteriorate and increasing fluid resuscitation will not resolve clinical signs of end organ perfusion deficits. The gold standard for CO monitoring is the pulmonary thermodilution method, which requires catheterization of the pulmonary artery. This technique is rarely performed in the equine clinical setting. An alternative technique, lithium dilution, is relatively easy to use once experienced and has been validated in the equine clinical setting. Injection of lithium dye into the venous system results in generation of a lithium concentration–time curve, which is used to calculate CO. Lithium dilution has been used successfully to monitor CO in adult horses and critically ill foals,[39-42] although repetitive sampling can result in toxic accumulation of lithium.[43] Alternatively, ultrasound measurement of CO has been validated using both transesophageal and transthoracic Doppler measurements.[40,44] Because Doppler measurement requires the beam to be parallel with flow there is large variability in the accuracy of this technique. Transesophageal measurements improve this accuracy but can be difficult to obtain in the standing horse.[40,45] A recent paper described an ultrasound velocity dilution method in foals.[46] This technique uses a bolus injection of saline and an arteriovenous loop connected to ultrasound velocity sensors.

CO measurement has its greatest benefit in cases with cardiac disease and is of great help in monitoring the response to vasopressor treatment. Because CO does not assess local tissue perfusion, its accuracy in evaluating tissue oxygenation is poor. Many of the standard monitoring techniques are limited because they principally assess global function (CO) and global oxygen debt (mixed venous lactate), not regional tissue deficiencies. These global measures, while helpful, do not assess the perfusion to high-risk organs such as the gastrointestinal tract, and they may provide a false sense of security when used to monitor treatment response. With the exception of urine output, none of the measurements just described evaluate perfusion to regional vascular beds. Because of the large variation in perfusion to specific tissues, such as the gastrointestinal tract and the brain, these global measures have poor sensitivity in determining oxygen delivery and uptake to "less important tissues."

Regional Perfusion

Several techniques have been developed in an effort to more specifically assess these differences in regional perfusion. Noninvasive measures of regional tissue perfusion include sublingual capnometry, near-infrared spectroscopy to monitor muscle tissue oxygen saturation, transcutaneous tissue oxygenation, and capnometry.[47-49] Slightly more invasive techniques include gastric tonometry, which evaluates CO_2 production in the stomach wall; infrared spectroscopic assessment of splanchnic perfusion; and measurement of bladder mucosal pH.[50,51] These alternative techniques are based on the idea that the body preferentially shunts blood away from the skin and gastrointestinal tract to spare more vital organs. As such, these techniques will detect abnormalities in perfusion before many of the more established techniques. Although these techniques have yet to be evaluated in the veterinary field, they have been shown to be sensitive markers of regional perfusion deficits manifest in early shock in humans.

Hypotensive Resuscitation and Delayed Resuscitation

As previously discussed, aggressive large-volume fluid therapy to restore blood pressure to normal values has potentially negative consequences. In situations of uncontrolled bleeding, this treatment will result in increased blood loss. Dilution of blood components (platelets and clotting factors) may additionally worsen bleeding. Increasing systolic blood pressure to normal values may dislodge or "blow out" a tenuous clot, leading to further bleeding. Hypotensive resuscitation has been advocated to prevent or minimize further blood loss until surgical control or formation of a stable clot has occurred. In these situations resuscitation to a lesser end point is recommended. The ideal end point or goal in hypotensive resuscitation is unclear. Strategies include achieving a mean blood pressure (MBP) of 40 to 60 mm Hg, using a predetermined, lower fluid infusion rate, or in some situations, completely delaying resuscitation until bleeding is surgically controlled.[22] In multiple animal models, controlled resuscitation (goal of MBP 40 to 60 mm Hg, or systolic blood pressure of 80 to 90 mm Hg) resulted in decreased blood loss; better splanchnic perfusion and tissue oxygenation; less acidemia, hemodilution, thrombocytopenia, and coagulopathy; decreased apoptotic cell death and tissue injury; and increased survival.[3,52-59] In cases of severe or ongoing bleeding, resuscitation with blood components is recommended to minimize the risk of coagulopathy, although data with respect to outcome compared to resuscitation with crystalloids is currently lacking. This strategy of hypotensive resuscitation (with whole blood as part of the fluid plan) is indicated in situations such as a bleeding of the uterine artery in a pregnant mare, where ligation of the vessel is unlikely and of great risk to the mare and fetus. There are currently no specific recommendations for end points of treatment in large animal species. If using blood pressure as the end point, direct measurement is currently recommended to ensure accuracy.

PREDICTING OUTCOME

In a critical review, high-risk surgical patients were used as a model for shock because time relationships were precisely documented.[60] In this study, nonsurvivors had reduced CO and DO_2 in the intraoperative and immediate postoperative period. Survivors had lower O_2ER; higher hematocrits, VO_2, and blood volume; and normal blood gases. In human trials, time is a strong predictor of survival, with survivors showing improvement or normalization in indices of CO, perfusion, oxygen uptake, and clinical parameters.[61] To this end, rapid control of hemorrhage, restoration of perfusion, normalization of blood gas values, and prevention of dilutional coagulopathy are predictors of survival. In patients with ongoing blood loss, controlled hypotension has been shown to decrease in-hospital complications and possibly increase survival rates. Lactate values, particularly lactate clearance, have been shown to be strongly associated with survival in both clinical and experimental studies of shock.[30-33] Though the data are not as robust, single lactate measurements and delayed lactate clearance have been shown to be associated with higher mortality rates in both adult horses and foals.[62-64] A poor or absent response to resuscitative attempts with continued evidence of perfusion deficits or the development of clinical evidence of organ dysfunction, or both, are associated with a poorer outcome.

ON THE HORIZON

Treatment

Although a perfect fluid protocol for treatment of hypovolemic shock remains elusive, liposome encapsulated hemoglobin may offer more benefits than other fluids because of its oxygen-carrying capacity. The presence of hemoglobin reduces the need for blood products, thereby lowering the associated risks to the patient.[65,66] In contrast to other synthetic oxygen carriers, liposome encapsulated hemoglobin vesicles do not appear to cause peripheral vasoconstriction and in a rat model of hemorrhage appear to be as effective in restoring hemodynamic and blood gas parameters.[67]

Monitoring

The ideal method to assess shock and treatment response would enable measurement of oxygen delivery at the tissue level as well as oxygen uptake and use. The ability to measure end organ perfusion, particularly in "less important" organs like the epidermis and gastrointestinal tract, in our veterinary patients has potential implications in assessing the severity of the shock state, developing treatment goals, and predicting outcomes. The implementation and evaluation of these techniques in equine critical care medicine is warranted.

Prognostic Indicators

Recent epidemiologic and experimental data have shown a sex-specific difference in the response to trauma and shock. Estrogen administration to castrated male mice improved immune responses after trauma and hemorrhage compared to castrated untreated mice.[68] Treatment of intact male mice with estradiol improved the survival rate and immune response to trauma, hemorrhage, and sepsis.[69-70] In a prospective study that evaluated more than 4000 trauma patients, hormonally active women tolerated trauma and shock better than men.[71] Dehydroepiandrosterone (DHEA) has estrogenic effects and has been shown to decrease morbidity in mice after trauma or hemorrhage. Because DHEA is used clinically to enhance the immune response, it may have use in the trauma or hemorrhage patient. Conversely, it may be the lower levels of male hormones in women that confer protection. Male mice depleted of testosterone either through castration or by treatment with the drug flutamide had improved cardiovascular and immune function compared to intact mice after hemorrhage and resuscitation. In the future, the use of hormone therapy may help improve outcome in hemorrhagic shock.

In addition to gender, genetic markers have been found to segregate with response to hemorrhage and trauma.[72,73] Currently, genomic markers are being evaluated as prognostic factors; however, there may come a time when genetic markers are used to direct therapy.[74]

Physiologic Response to Trauma

The metabolic response to trauma or injury has classically been divided into two phases—the ebb phase, which occurs during the first several hours after injury, and the flow phase, which occurs in the ensuing days to weeks. The ebb phase is characterized by hypovolemia and low flow or perfusion to the injured site. Once perfusion is restored, the flow phase begins. The flow phase is divided into a catabolic period and an anabolic period. The catabolic period is triggered by many of the same mediators discussed in the earlier section on the pathophysiology of shock, and many of the clinical signs will mimic those seen in shock. The anabolic period is characterized by the return to homeostasis. Cortisol levels fall during this final period and normalization of physiology occurs. The physiologic response to trauma is complex, and the duration and progression will vary depending on the injury site, severity, and underlying condition of the patient. For more specific information regarding trauma of specific organs or body cavities, the reader is referred to chapters dealing with those specific systems. This section is designed to provide an overview of the complex pathophysiology of trauma.

Mediators of the Stress Response: Ebb Phase

The stress response to trauma is initiated by pain, tissue injury, hypovolemia, acidosis, shock, hypothermia, and psychological responses. Direct tissue injury, ischemia, and inflammation activate afferent nerve endings, which exert local and systemic effects via the central nervous system. Hypovolemia, acidosis, and shock exert their effects via baroreceptors and chemoreceptors located in the heart and great vessels. Fear and pain have conscious effects in the cortex, and they stimulate cortisol secretion via the hypothalamic-pituitary-adrenal axis (HPA), which increases sympathetic output. Because of this effect, modulation of pain has been shown to be important in controlling the stress response to trauma, and pain control should be strongly considered in the trauma patient.

The sympathoadrenal axis is stimulated through direct input from injured nerves and by hypovolemia, acidosis, shock, and psychological responses (fear, pain, anxiety). Catecholamines have widespread effects on cardiovascular function (see "Pathophysiology of Shock," earlier in this chapter) and metabolism (see "Metabolic Response to Injury" in Chapter 6), and they

stimulate release of other mediators, including cortisol and opioids. The catecholamine response is beneficial; however, prolonged sympathoadrenal stimulation can be detrimental because of its effects on general body condition. Catecholamines increase peripheral vascular resistance, so ongoing stimulation leads to long periods of tissue ischemia.

Other triggers of cortisol secretion in trauma and shock include vasopressin, angiotensin II, norepinephrine, and endotoxin. The degree of hypercortisolemia correlates with the severity of injury and persists until the anabolic phase of healing begins. Cortisol secretion results in sodium and water retention (edema), insulin resistance, gluconeogenesis, lipolysis, and protein catabolism. Cortisol also affects leukocytes and inflammatory mediator production and, although cortisol is critical for recovery from acute injury, prolonged cortisol secretion can result in pathologic suppression of the immune response.

Vasopressin and the renin-angiotensin system are important mediators of the stress response. The reader is referred to the section on pathophysiology of shock for a review of these mediators.

Endogenous opioids released from the pituitary gland as well as from the adrenal glands in response to sympathetic stimulation are important mediators in the modulation of pain, catecholamine release, and insulin secretion. Endogenous opioids modulate lymphocyte and neutrophil function and may act to counter cortisol's effect on immune function.

Local mediators released in response to injury trigger a multitude of cascades. Tissue factor exposure activates the coagulation and complement cascades and ultimately stimulates the inflammatory response. Cell membrane injury results in release and activation of the arachidonic acid cascade and production of various cytokines, including prostaglandins, prostacyclines, thromboxanes, and leukotrienes. These mediators have a multitude of functions, including further activating coagulation and platelets, altering blood flow via vasoconstriction and vasodilation, and increasing chemotactic activity mediating the influx and activation of inflammatory cells, with subsequent release of lysosomal enzymes and reactive oxygen species. Microvascular thrombosis at the site of endothelial damage causes further pathologic changes in perfusion. If perfusion is restored, further damage may ensue because elevated local concentrations of reactive oxygen species coupled with influx of desperately needed oxygen can induce further oxidative stress with production of highly toxic reactive oxygen species and further tissue injury. Amplification of this response coupled with reperfusion can lead to the development of SIRS and multiorgan dysfunction (see Chapter 2).

Response to Trauma: Catabolic Period

Psychological response to trauma and shock is manifest in changes in behavior, withdrawal, immobilization or reluctance to move, fear, anxiety, aggression, and malaise. These psychological responses can persist for long periods depending on the severity of the injury and pain. In people, the psychological effect may persist long after the injury has resolved. Whether the same happens in horses has yet to be determined.

Many of the changes in vital signs will mimic those seen with hypovolemic shock. Cardiovascular changes including tachycardia, tachypnea, and other clinical signs of the hyperdynamic response may be seen. Fever during the early period after injury is typically a response to injury and inflammation itself, particularly in patients with head trauma. Infectious causes of fever should be suspected if fever persists or is recurrent days after the injury. Other clinical signs will depend on the severity of blood loss and the organ injured. Cardiovascular changes including hypotension, decreased perfusion, decreased urine output, and reduced cardiac contractility are likely to occur with significant blood loss or thoracic contusion. Endotoxemia and bacteremia are likely with gastrointestinal injury, such as strangulating injury to the intestine.

Edema at the site of injury is caused by vascular injury from both the trauma and the inflammatory response, which results in loss of capillary integrity and extravasation of protein and fluid. In severe injury, edema may become generalized. This generalized edema results from systemic inflammatory, hormonal, and autonomic responses that increase capillary pressure and salt and water retention. The presence of hypoproteinemia can exacerbate clinical edema as colloid oncotic pressure is decreased.

The metabolic response to trauma is complex and results in changes in the metabolic rate as well as the mobilization and utilization of energy stores. Decreased appetite and malaise are also seen in response to pain, cytokines, and hormones. The reader is referred to Chapter 6 for a more detailed description of the metabolic changes occurring with injury.

Coagulation is activated by endothelial injury and the expression of tissue factor. Tissue factor also activates complement and inflammation. These changes combined with release of arachidonic acid from damaged cell membranes stimulate production of multiple inflammatory mediators, platelet activation and adhesion, and fibrinolysis. Blood loss coupled with crystalloid replacement can further dilute platelets and coagulation factors, which, in combination with factor consumption to control bleeding at the site of injury, can result in development of a hypocoagulable state. Coagulation dysfunction is recognized in many types of injury including large colon volvulus, severe traumatic injury, SIRS, and septic shock.

Circulating leukocytes increase in the initial response to injury with subsequent accumulation in injured microvascular beds. This accumulation may be exacerbated by vasoconstriction in response to hypovolemia and catecholamine surges and may play a role in reperfusion injury, because activated neutrophils are a major source of reactive oxygen metabolites. In addition to changes in circulating leukocytes, the immune response can be altered significantly with severe trauma. Decreases in antibody production, neutrophil chemotaxis, and serum opsonic activity; increases in serum immunosuppressive factors; and activation of T-cell suppressors mediated by neurohormonal stress response are just some of the changes that may occur.

Response to Trauma: Anabolic Period

The final stage in recovery is the anabolic phase of flow. During this period many of the responses return to normal. Appetite returns, body protein is synthesized, and weight is restored, resulting in improved organ function and energy stores. Metabolic demands diminish, water balance is restored, and as hormonal levels decrease, a generalized feeling of well-being develops. The length of this period will depend on the severity of the injury, the number and type of complications, the patient's condition before injury, and the length of the catabolic period of recovery. Healthy individuals that do not develop complications will likely recover more rapidly than debilitated

patients that suffer complications, such as infection, and have a prolonged catabolic phase of recovery.

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CHAPTER 2

The Systemic Inflammatory Response

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The systemic inflammatory response and failure of multiple organ systems are syndromes that result from an inappropriate and generalized inflammatory response to stimuli, which may or may not result from an infectious process. Although it appears that the phagocytic activation of the monocyte/macrophage cell lineage is directly responsible for the development of clinical signs and symptoms, identifying the bacteria and neutralizing their toxins has not drastically changed the outcomes of patients affected by these syndromes. As a result, current management strategies and research efforts have been directed at addressing infectious and noninfectious causes and identifying effective ways of modulating the associated immune-mediated responses. The pathophysiology of these inflammatory-based syndromes has not been clarified in people or lab animal models, and very little original work has been produced in the horse. A generally accepted summary of these conditions is that bacteria or their endotoxins, or both, induce and sustain a marked inflammatory response by the host, which eventually overwhelms sensitive organs and often results in a fatal outcome. This chapter reviews the pathophysiology of systemic inflammatory response and multiple organ failure with the viewpoint that inflammation, not bacterial overgrowth, may directly generate these syndromes in the horse.

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME

With microbial invasion or any process that results in tissue damage, the ultimate goal of the immune system is to contain infection, alarm the host to defend, and to promote tissue repair. Whether these goals are achieved or defeated, the host relies on a defense and repair response that is appropriate for the insult. If the host overzealously responds, the same innate components that are meant for protection and repair may ironically turn out to be just as detrimental or even more harmful to the host than the initial insult. When the response to infection and injury results in an incongruous and exaggerated systemic inflammatory reaction, the clinical state is referred to as the *systemic inflammatory response syndrome,* or *SIRS,*[1] which can be initiated by infection, endotoxemia, or noninfectious insults, such as severe trauma, ischemia, immune-mediated disease, surgery, hypothermia, hyperthermia, or intense hypoxemia (i.e., hemorrhagic shock). To counteract the proinflammatory response and deter the state of SIRS, the host relies on anti-inflammatory opposition that includes production of cytokines, soluble cytokine receptors, receptor antagonists, prostaglandin E_2, and corticosteroids.[2] If there is over-recruitment of the anti-inflammatory processes, a state of anergy, increased

susceptibility to infection, and inability to repair damaged tissues ensues. This scenario is referred to as *compensatory anti-inflammatory response syndrome,* or *CARS*.[1] In some circumstances, a *mixed anti-inflammatory response syndrome,* or *MARS*, arises in which surges of both SIRS and CARS coexist.[1] In the circle of equilibrium, if SIRS and CARS are ultimately appropriately balanced, then homeostasis resumes. Predominance of SIRS may culminate in adverse pathophysiologic events, such as disseminated intravascular coagulopathy (DIC), shock, organ failure, and death. In this later scenario, dissonance has occurred and the patient is defined as having *multiple organ dysfunction syndrome (MODS)* or the presence of organ dysfunction associated with acute illness in which homeostasis cannot be restored without intervention (see "Multiple Organ Dysfunction Syndrome," later).[1]

Pathophysiology of SIRS

The key event in the initiation and propagation of SIRS is the release of endogenous molecular substances by host cells, each with a diverse array of biological activities. The enormity of the molecular response to injury, redundancy in action and location in various tissues, the dynamic discovery of new molecules, and rediscovery of new roles for previously identified molecules complicates their discussion and classification. There are literally thousands of molecules involved in the inflammatory cascade of injury. This discussion will focus on the main molecular categories of cytokines, lipid-derived autocoids, acute phase proteins, reactive oxygen species, and vasoactive and neutrophil-associated substances, as they relate to the horse.

Cytokines

Cytokines are protein substances that are the "early responders" to infectious agents or tissue damage. The cytokines can be further classified by whether their biological activities are primarily proinflammatory or anti-inflammatory and by their cell of origin. Examples of pro-inflammatory cytokines include tumor necrosis factor (TNF); interleukin 1, 6, and 8 (IL-1, IL-6, IL-8); and interferon-γ (INF-γ).[2,3] Monocytes and macrophages are universal sources for the pro-inflammatory cytokines, though other cell types contribute as well, including neutrophils (TNF), endothelial cells (IL-1, IL-8), fibroblasts, keratinocytes, lymphocytes (IL-1, IL-6) and natural killer cells (TNF, INF-γ). Some of the main functions of TNF, IL-1, and IL-6 are to initiate coagulation, fibrinolysis, complement activation, the acute phase response, and neutrophil chemotaxis. TNF and IL-1 also induce pyrogenic activities and augment further cytokine production. The importance of TNF and IL-1 is clearly exemplified by the fact that administration of these substances to otherwise healthy laboratory animal species mimics many of the events of septic shock. In horses, experimental infusion of endotoxin results in increased circulating levels of TNF and IL-6 (see "Endotoxemia" later). Less specific information is known about the anti-inflammatory cytokines (IL-4, IL-10, IL-11, IL-13, transforming growth factor-β [TGF-β]) in the horse, though in septic foals that did not survive, IL10 gene expression was significantly greater than in surviving ones.[4] The anti-inflammatory cytokines are released from monocytes, macrophages, and T-helper cells and serve to restrain the inflammatory campaign by inhibiting macrophage activation, proinflammatory cytokine release, antigen-presenting cells, and chemotaxis.

Lipid-Derived Mediators

Arachidonic acid is a 20-carbon fatty acid that is a major constituent of the phospholipids of all cell membranes.[2] It also serves as the parent molecule for eicosanoid synthesis, but it must first be released from the cell membrane. Endotoxin, TNF, and IL-1 all upregulate the activity of phospholipase A_2, the enzyme responsible for cleavage of arachidonic acid. Once released, arachidonic acid is further metabolized by either lipoxygenase, to form the family of leukotrienes, or cyclooxygenase, to form the prostanoids: thromboxane A_2 (TxA_2) and the prostaglandins (PGs). The prostanoids are vasoactive substances: TxA_2 and $PGF_{2\alpha}$ are potent vasoconstrictors, whereas PGI_2 and PGE_2 are vasodilators. The prostanoids also play important roles in primary hemostasis: TxA_2 promotes platelet aggregation, but PGI_2 inhibits aggregation. Finally, PGE_2 is a pyrogen. The prostanoids have been extensively studied in endotoxemic horses (see "Endotoxemia" later). Less specific attention has been given to the investigation of the leukotrienes in horses, although they serve as chemoattractants and increase vascular permeability.

Platelet-Activating Factor

Like the eicosanoids, platelet-activating factor (PAF) is released from cell membrane (mononuclear phagocytes, endothelial cells, and platelets) phospholipids by phospholipase A_2. The released alkyl-lyso-glycerophosphocholine is then acetylated to form PAF. The biologic effects of PAF include vasodilation, increased vascular permeability, platelet aggregation, and recruitment and activation of phagocytes. It also is a negative inotrope. Use of a PAF receptor antagonist in horses experimentally challenged with endotoxin significantly delayed the onset of fever, tachycardia, neutropenia, and lactic acidosis.[5]

Acute Phase Proteins

An acute phase protein is any protein whose blood concentration significantly increases (or decreases) during an inflammatory response.[6] Collectively, the hundreds of acute phase proteins are responsible for many of the well-recognized reactions to microbial invasion, such as fever; anorexia; depression; alterations in metabolism, hemodynamics, and coagulation; and leukocyte activation. The liver is a key site of synthesis. Cytokines, principally TNF, IL-1, and IL-6; glucocorticoids; and growth factors stimulate and modulate gene expression and the transcription of the acute phase proteins. The serum concentrations of the major acute phase proteins, serum amyloid A (SAA) and C-reactive protein (CRP) can each increase as much as 100-fold during the acute phase response. Interestingly, despite their intense synthesis during the acute phase reaction, the roles of each of these major proteins are still not entirely clear. SAA may be involved in cholesterol regulation, chemotaxis, and mediation of anti-inflammatory events, such as downregulation of fever, phagocytosis, and prostanoid synthesis. CRP can activate complement, induce phagocytosis, and stimulate cytokine and tissue factor expression. In horses, SAA and CRP concentrations have been determined by several methodologies. Using the latex agglutination immunoturbidimetric assay, the expected SAA concentration in healthy neonatal foals and adult horses is less than 27 mg/L.[7] SAA nonspecifically increases with either infectious or noninfectious (but inflammatory) conditions, with values greater than 100 mg/L, suggestive of an infectious process in foals. In horses with acute gastrointestinal diseases, higher

SAA levels are correlated with risk of death. Using radial immunodiffusion, CRP concentrations have been established in healthy foals and adult horses (5 to 14 mg/mL).[8] Although CRP increased 3 to 6 times in experimentally induced inflammation in adult horses, its utility in determining an inflammatory or infectious response in naturally occurring diseases in the horse has not been established.

The remaining acute phase proteins have widely diverse pathophysiologic effects. The complement system is represented by the acute phase synthesis of C3a, C4a, C5a, C4b, C3b, C5b-C9, factor B, and C1 inhibitor.[9] Collectively, these compounds induce bacteriolysis, increase vascular permeability, are chemotactic for neutrophils, and enhance opsonization of both microbes and damaged host cells. Balanced activation of the coagulation and fibrinolytic systems by the acute phase response of factor VIII, fibrinogen, plasminogen, tissue plasminogen activator, plasminogen activator inhibitor, fibronectin, von Willebrand factor, and tissue factor leads to formation of intravascular and extravascular "clots" that capture and contain infectious organisms and inflammatory debris and provide a scaffold for tissue repair. Of these coagulation factors, hyperfibrinogenemia is a well-recognized clinicopathologic finding in horses with inflammation. The release of the acute phase transport and scavenger proteins, such as ceruloplasmin, haptoglobin, lipopolyscharride-binding protein, soluble cluster of differentiation antigen 14 (CD14), and lactoferrin, bind bacterial nutrient components, such as copper and iron, and neutralize or transport toxic bacterial components.

Reactive Oxygen Species

The reactive oxygen species encompass all oxygen-derived toxic mediators that most commonly originate from mononuclear phagocytes or neutrophils.[10] Oxygen free radicals are oxygen-containing molecules that contain an unpaired electron (superoxide anion O_2^-; hydroxyl radical, $OH^\bullet$). Free radicals can react with essentially any molecular component in their quest to "repair" the unpaired electron. In doing so, more radicals are generated and molecular damage ensues with loss of protein function, cross-linking of DNA, lipid peroxidation, vasoconstriction, and pain. Oxygen free radicals also induce cytokine production and endothelial adhesion molecules. Other reactive oxygen species that do not contain unpaired electrons include hydrogen peroxide (H_2O_2) and nitric oxide (NO). NO is generated enzymatically in phagocytes by inducible NO synthetase, which is activated by endotoxin and cytokines.

Vasoactive Mediators

In addition to the prostaglandins and NO, bradykinin, a by-product of activation of the contact coagulation system, and histamine are vasodilators. Angiotensin, endothelin, TxA_2, and leukotrienes (LTC_4, D_4, and E_4) have vasoconstrictive activities. Numerous molecular substances promote vascular leakage, including PAF, leukotrienes, complement components (C3a, C5a), NO, and bradykinin.[2]

Diagnosis of SIRS

In 1992, Bone and colleagues[1] proposed the following specific diagnostic criteria for SIRS in human patients. More than one of the following clinical manifestations had to be present: (1) a body temperature greater than 38° C (100.4° F) or less than 36° C (96.8° F); (2) a heart rate greater than 90 beats per minute; (3) tachypnea, manifested by a respiratory rate greater than 20 breaths per minute, or hyperventilation, as indicated by a $PaCO_2$ of less than 32 mm Hg; or (4) an alteration in the white blood cell count, such as a count greater than 12,000/mL, a count less than 4000/mL, or the presence of more than 10% immature neutrophils ("bands"). There has not been a similar consensus on diagnostic criteria for SIRS in horses; however, with some adjustments that would be appropriate relative to normal findings in the horse, these criteria could be applied to the horse (Table 2-1). Since the average adult horse's body temperature is higher than the average human's, a rectal temperature greater than 38.6° C (101.5° F) or less than 36.6° C (98° F) would seem more appropriate for horses. The heart rate criterion is based on an approximately 25% increase over the high end of the normal average adult human heart rate. Thus for adult horses, a heart rate greater than 60 beats per minute would represent a similar rate increase. Because the upper end of the normal total white blood cell count for horses is 12,000/mL, a white cell count greater than 14,000/mL might be a more appropriate upper cutoff for SIRS in the horse.

Criteria for SIRS in foals would have to be adjusted by age-relative criteria. Since the most common trigger of SIRS in foals is sepsis, the sepsis score system developed in the 1980s for foals[11] might be an equally effective SIRS score (Table 2-2). Note that in the sepsis score, the human SIRS criteria for rectal temperature and white blood cell count are included.

TABLE 2-1. Diagnostic Criteria for SIRS in Adult Horses*

Parameter	Criteria
Rectal temperature	>38.6° C (101.5° F) or <36.6° C (98° F)
Heart rate	>60 beats/min
Respiratory	Respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg
White blood cell count	>14,000/μL or <4000/μL or >10% bands

*The diagnosis of SIRS can be made when at least two parameters' criteria are present.

Treatment of SIRS and Prognosis

The treatment of SIRS is largely directed at controlling the primary disease process that triggered the response. Considering the underlying theme of overzealous inflammation in SIRS, anti-inflammatory agents are indicated. In light of the complexity of the pathophysiology of SIRS and the diverse array of endogenous mediators, there is unlikely to be a single therapeutic panacea. In people with SIRS, scoring systems have been developed that offer prognostic information.[1] Because SIRS is not defined by consensus in horses, similar comparisons are not directly possible. However, there is evidence that foals meeting proposed criteria for SIRS had a higher mortality rate than those without SIRS.[12]

TABLE 2-2. Sepsis Score System for Neonatal Foals: Each of the Following Parameters is Evaluated and a Score is Assigned[11]

	Score					
Parameter	**4**	**3**	**2**	**1**	**0**	**Score for This Case**
HISTORY						
1. Placentitis, vulvar discharge, dystocia, long transportation of mare, mare sick, induced, prolonged gestation (>365 days)		Yes			No	
2. Premature (days of gestation)		<300	300-310	311-330	>330	
CLINICAL SIGNS						
1. Petechiae, scleral injection		Severe	Moderate	Mild	None	
2. Fever			>102° F	<100° F		
3. Hypotonia, coma, depression, seizure			Marked	Mild	Normal	
4. Anterior uveitis, diarrhea, respiratory distress, swollen joints, open wounds		Yes			None	
LABORATORY DATA						
1. Neutrophil count (per µl)		<2000	2000-4000 or >12,000	8,000-12,000	Normal	
2. Bands (per µl)		>200	50-200		None	
3. Any toxic change in neutrophils	Marked	Moderate	Mild		None	
4. Fibrinogen (mg/dL)			>600	400-600	<400	
5. Blood glucose (mg/dL)			<50	50-80	>80	
6. IgG (mg/dL)	<200	200-400	400-800		>800	
7. Arterial oxygen (mm Hg)*		<40	40-50	50-69	>70	
8. Metabolic acidosis*				Yes	No	
				Total points for this case: _____		

A score of ≥11 predicts sepsis.
*If these two parameters are included, the positive cutoff value is 12.
IgG, Immunoglobulin G.

MULTIPLE ORGAN DYSFUNCTION SYNDROME

Multiple organ dysfunction syndrome (MODS) refers to the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.[1] Any disease process inducing a well-defined injury that affects the function of organs at the initial site of insult (primary MODS) or organs remotely positioned from the primary injury (secondary MODS), not as a direct response to the insult, but as a consequence of the host's response to the injury, can culminate in MODS. Multiple organ dysfunction syndrome can be relative or absolute and is best characterized as a dynamic process that is a continuum of pathophysiologic change over time.[1]

The same key inflammatory catalysts that are responsible for the development of SIRS (see earlier) can subsequently progress to MODS.[13] In essence, MODS is simply an extension and potential consequence of SIRS. In people, disease processes commonly associated with induction of MODS include sepsis, endotoxemia, DIC, ischemia, surgery, anesthesia, massive trauma, thermal injury, drug reaction, and anaphylaxis. Immune-mediated inflammatory injury, altered hemodynamics, and reduced tissue perfusion are the pivotal pathophysiologic events that drive MODS. Because appropriate function of one organ often depends on adequate function of another, the systemic effects of single organ failure can contribute to a self-perpetuating cycle of multiple organ involvement and ultimately, multiple organ dysfunction and failure.

Pathophysiology of MODS

The gastrointestinal, hemostatic, cardiovascular, hepatic, musculoskeletal, renal, respiratory, adrenal, and nervous systems are commonly affected by MODS in people.[13] Each of these organ systems will be briefly discussed here with particular reference to what is currently recognized in the horse.

Because the *gastrointestinal tract* is a frequent site of injury in adult horses and is a major portal of entry of bacterial pathogens in the neonatal foal, gastrointestinal dysfunction is a common manifestation of primary MODS in horses. In people with MODS, gastrointestinal dysfunction is defined as the presence of ileus, inability to tolerate enteral nutrition, and the presence of mucosal ulceration.[14] Intestinal injury and ileus are major complications of acute gastrointestinal disease of the horse and are addressed elsewhere in detail (see Chapters 35 and 40). Additional signs and complications of primary inflammatory insults (enteritis and colitis) and strangulation obstruction of intestine (see "Ischemia," later) are gastrointestinal reflux, alteration in enteric resident flora, diarrhea, and protein-losing enteropathy. Protein-losing enteropathy will compromise oncotic pressure, and electrolyte derangements can further affect distant organs, especially cardiac, muscular, and neural tissue function. Mucosal damage enhances the translocation of luminal bacteria and endotoxin with subsequent septicemia and endotoxemia, further fueling the drive of SIRS and MODS.

In its most simplified description, the major elements that define dysfunction of the *coagulation system* associated with the development of MODS are (1) excessive procoagulation, (2) loss of controlled fibrinolysis, and (3) loss of natural anticoagulant activities.[15,16] These events promote clot formation, especially in the microvascular space, which contributes to reduction of blood flow to vital tissues.[16] With prolonged or excessive thrombi formation, platelets, coagulation, anticoagulation, and fibrinolytic factors are consumed, balance is lost, and hemorrhage may ensue. This state of clinical coagulopathy is referred to as DIC.[16] Endotoxemia and sepsis are commonly reported primary ailments that initiate DIC,[16] rendering the coagulation system probably one of the most frequently described and studied systems affected by SIRS and MODS in horses.[15,17-21] Approximately 30% of horses with acute colitis,[22] 70% of horses with a colon torsion,[23] and 25% of septic foals[24] fit diagnostic criteria for DIC at admission. Evidence in both humans and horses suggests that the primary mechanism responsible for the initial hypercoagulative state of DIC is activation of the extrinsic coagulation cascade (see Chapter 4) via enhanced expression of membrane tissue factors (i.e., thromboplastin).[25,26] Constitutively expressed membrane tissue factor on endothelial cells and mononuclear phagocytes in the circulation and tissues is upregulated, either *directly* by pathogen-associated molecular pattern antigens such as lipopolysaccharide (endotoxin), or *indirectly* by cytokines that are generated in the process of self defense or response to injury.

In addition to events that activate coagulation, failure of appropriate fibrinolysis and consumption of anticoagulation factors further promote thrombosis. In horses, endotoxin appears to favor activation of plasminogen activator inhibitor (PAI) over tissue plasminogen activator (tPA). In a large study of 153 horses with acute colic, mean plasma PAI activity increased approximately fivefold, whereas tPA activity dropped twofold.[18] Both PAI and α_2-antiplasmin were significantly greater in septicemic foals, compared to healthy age-matched foals.[19] These types of responses would be expected to enhance clot formation and be negatively correlated with prognosis for survival in equine colic patients.[18] The natural anticoagulants antithrombin (AT) and protein C are rapidly consumed in sepsis or inactivated by neutrophil enzymes released during the inflammatory response. AT and protein C also have several anti-inflammatory effects, including induction of prostacyclin synthesis, diminution of endotoxin-induced cytokine and tissue factor synthesis, and reduction of chemotaxis and neutrophil adhesion.[26] Clinical studies on horses with acute gastrointestinal disease and septic foals consistently report that these populations at risk for endotoxemia have significantly reduced AT and protein C activity. Correlation of these activities with the development of complications such as jugular thrombi, peritoneal adhesions, and laminitis is significant and is associated with a reduced chance of survival.[19,20,27] The ultimate consequence of insufficient AT and protein C activity is both increased clot formation and heightened inflammatory response, the latter effect serving to "fuel the fire" of driving thrombus formation. Ultimately, platelets, coagulation, anticoagulant, and fibrinolytic factors are consumed and the normally regulated balance between clot formation and dissolution is lost, culminating in unregulated hemorrhage, shock, multiple organ hypoxia, and thus organ dysfunction and failure.

The exact mechanism for *cardiac dysfunction* and failure in MODS is not entirely understood, though both systemic changes in autonomic nervous system balance and direct effects of pathogen-associated molecular patterns and SIRS-induced cytokines on the myocardium play key roles. Specifically, upregulation of sympathetic drive relative to vagal tone and direct endotoxin impairment of the signal transduction pathways and (or) ion channels mediating the autonomic nervous signals in cardiac pacemaker cells contribute to reduced myocardial contractility, increased heart rate, and reduced heart rate variability.[28] Furthermore, electrolyte disturbances that may be present as a result of the primary insult or subsequent to renal or gastrointestinal dysfunction can also negatively affect myocardial function. Reduced cardiac output, arrhythmias, and hypotension ensue, further compromising hemodynamic homeostasis. There is little information on myocardial function in horses with SIRS or MODS; however, serum cardiac troponin concentration, a sensitive biochemical marker of acute myocardial injury, is increased in foals with septicemia[29] and horses with small intestinal strangulating lesions that underwent surgical correction.[30] In the latter, there was a significant association between serum lactate and troponin concentrations, indicating a correlation between hypoperfusion and ischemic myocardial degeneration.

Overt *hepatic failure* is not commonly recognized in horses with MODS; however, alterations in hepatic perfusion, via local or systemic effects, and delivery of endotoxin to the liver from both intestinal and systemic sources, can contribute to hepatic injury. The presence of gastrointestinal reflux, ileus, and obstruction of bile flow may lead to ascending infection of the biliary system or pressure necrosis around the bile cannaliculi, or both. In a review of horses with acute small intestinal disease, horses with proximal enteritis were 12 times more likely to have increased γ-glutamyltransferase activity, as serum biochemical evidence of acute hepatic injury, compared to horses with strangulating small intestinal disease.[31] The liver is a remarkable organ with a diverse range of functions, including protein, carbohydrate, and lipid nutrient regulation, metabolism, and storage; synthesis of albumin, coagulation factors, and components of the acute phase response; digestion; detoxification; vitamin storage; and extramedullary hematopoiesis. Loss of any of these functions is detrimental and can lead to the development of nutrient crisis, encephalopathy, and coagulopathy.

The laminae of the equine digit are highly susceptible to direct concussive damage and altered weight distribution, as well as injury evoked by the systemic response to endotoxemia, acute gastrointestinal disease, sepsis, metabolic disease, dietary changes, or acute renal failure. Thus *laminitis* represents a unique manifestation of MODS in the horse. In a recent retrospective case-controlled study in hospitalized horses, endotoxemia was the only risk factor in multivariate analysis that significantly correlated with development of laminitis.[32] The pathophysiology of laminitis is complex and incompletely understood, though alterations in systemic and local hemodynamics; local endothelial dysfunction; and inflammatory, enzymatic, endocrine, and metabolic components all appear to contribute to the development of laminitis.[33] Laminitis is discussed in detail in Chapter 90.

Acute *renal failure* is defined as the presence of azotemia or oliguria, or both, in a normovolemic patient that does not have signs of postrenal obstruction.[14] In people, the primary mechanism leading to the development of acute renal failure is acute tubular necrosis.[34] The mechanism for renal tubular necrosis follows the same general theme of systemic or local reduction in blood flow, leading to cellular hypoxemia and cell death. Swelling of epithelial cells and infiltration with inflammatory

cells contributes to renal tubular obstruction. Ischemic glomerular damage further contributes to reduction in glomerular filtration. The combined effects are loss of autoregulation of renal blood flow, vasoconstriction, fluid retention, protein-losing nephropathy, and electrolyte derangement. These effects are often confounded by the adverse renal side effects of drugs that are used to combat the primary initiating cause of MODS, such as aminoglycosides for treatment of sepsis, or drugs that are used to control the systemic inflammatory response of MODS (nonsteroidal anti-inflammatory drugs [NSAIDs]). Although the exact incidence of acute renal failure in horses with MODS is not known, in a recent retrospective study on horses with acute gastrointestinal disease, azotemia was a frequent finding at admission.[35] Furthermore, those horses in which azotemia resolved within 72 hours had a better survival rate than those in which azotemia persisted.

Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) have been defined as the clinical conditions of acute onset of *respiratory failure* characterized by hypoxemia and diffuse bilateral pulmonary infiltrates on thoracic radiographs, in the absence of left atrial hypertension.[36] ALI and ARDS develop when injury to the alveoli and pulmonary endothelium causes thromboembolism and protein-rich pulmonary edema. Subsequently, type II pneumocytes and fibroblasts are recruited to replace damaged areas in the alveoli. Collectively, these injuries severely impair gas exchange. Likely triggers for ALI and ARDS in horses include sepsis, aspiration of gastric contents, smoke inhalation, severe trauma, and transfusion reaction.[37] ALI and ARDS have been described in foals in which the primary insult was pulmonary in origin and apparently triggered by bacterial or viral infection.[38] Fulminant pulmonary edema without left-sided heart failure has also been reported in adult horses with transient airway obstruction and air embolism as anesthetic complications.[39,40]

The term *critical illness–related cortisosteroid insufficiency (CIRCI)* is used to describe the clinical scenario in which the cortisol response is insufficient for the degree of stress induced by illness.[41] Mechanisms that have been proposed for CIRCI are cytokine-induced inhibition of any component of the hypothalamic-pituitary-adrenal axis, downregulation and sensitivity of tissue cortisol receptors, and direct inflammatory, hemorrhagic, or hypoxic damage to the adrenal gland (Waterhouse-Friderichsen syndrome). CIRCI has been recently described in septicemic foals.[42,43] Furthermore, septic foals that met diagnostic criteria for CIRCI were more likely to have clinical signs of shock and MODS and were less likely to survive.[43]

Hypoxia, electrolyte derangements, alterations in glucose homeostasis, hepatic encephalopathy, and microthrombosis all affect neural function. *Encephalopathy* of MODS could be easily overlooked when it simply manifests as depression or altered behavior. In severe cases, stupor and coma may develop.

Diagnostic Criteria for MODS

Multiple organ dysfunction is a syndrome that is definition dependent, and unfortunately a consensus definition has not yet been proposed for horses. When the definition of MODS was first suggested for human patients, criteria for organ dysfunction had not yet been defined. Currently the syndrome is defined by identifying criteria for individual organ dysfunction and dysfunction that are correlated to prognosis. Unfortunately, with the exception of ALI/ARDS, consensus definitions for individual organ systems have not been established for the horse. Therefore, diagnostic criteria supporting dysfunction or failure are proposed in the following sections. They are based on current practical information pertaining to the horse, as guided by criteria used in human patients.

Respiratory Criteria

Clinical signs of ALI and ADRS in horses would include tachypnea, labored breathing, nasal flare, cough, bilateral foamy nasal discharge, and pulmonary crackles. Acute lung injury and ARDS can be suspected in animal species in which the following criteria are met: (1) the patient has acute onset (less than 72 hours) of respiratory distress at rest; (2) the patient has a known risk factor for ALI or ARDS; (3) there is pulmonary capillary leakage without increased pulmonary capillary pressure as evidenced by the presence of any one of the following: bilateral/diffuse infiltrates on thoracic radiographs, proteinaceous fluid within the conducting airways, or increased extravascular lung water; and (4) there is inefficient gas exchange evidenced by hypoxemia (defined as one of the following: partial pressure of oxygen in arterial blood/fraction of inspired oxygen ratio (PaO_2/FIO_2) of less than 300 mm Hg for ALI and less than 200 mm Hg for ARDS, increased alveolar-arterial oxygen gradient, increased venous admixture, or increased dead space ventilation.[37]

Hemostasis Criteria

Clinical signs of DIC include thrombi, petechiae, and ecchymoses; any sign of excessive hemorrhage at sites of trauma or at mucocutaneous orifices; or compartmental hemorrhage. Disseminated intravascular coagulopathy can be suspected in the presence of at least three of the following: thrombocytopenia; prolonged prothrombin; activated partial thromboplastin time; decreased fibrinogen concentration or prolonged thrombin time; increased fibrin degradation products or D-dimer concentrations; or decreased antithrombin activity.

Gastrointestinal Criteria

Diagnosis of gastrointestinal dysfunction is mostly subjective and dependent on the clinical signs of ileus. In the horse this would include colic, decreased borborygmi, abdominal distension, diarrhea, and excessive gastrointestinal reflux or colic-like signs that necessitate withholding of enteral nutrition. The presence of gastric ulceration, protein-losing enteropathy, and intra-abdominal hypertension would also fit criteria for gastrointestinal dysfunction in people.

Renal Criteria

Clinical signs of renal dysfunction include decreased urine production and subcutaneous or pulmonary edema. In people, acute renal failure is defined diagnostically by the presence of (1) an increased serum creatinine concentration or (2) oliguria with normovolemia (acceptable central venous pressure and systolic pressure), without signs of postrenal obstruction with either criterion.[13] Exact quantification of urine output is not easy or practical in adult horses, though it is possible in catheterized neonatal foals. In the latter, mean urine production is 6 mL/kg/hr.[44]

Hepatic Criteria

In people, liver dysfunction is defined by progressive increases in plasma transaminase activities and bilirubin concentration. Since icterus is a common feature of anorexia in the horse, progressive hyperbilirubinemia would not be an appropriate criterion. Liver-specific indices in the horse would include persistently increased serum sorbitol dehydrogenase or γ-glutamyltransferase activities, and increased serum bile acid concentration.

Cardiovascular Criteria

There is no consensus on criteria for cardiovascular failure in people. However, several parameters have been suggested, including lactatemia; the presence of hypotension (either decreased systolic or mean arterial pressure); the presence of pathologic arrhythmias, particularly ventricular in origin; evidence of myocardial ischemia (such as increased cardiac troponin concentrations); loss of heart rate variability; decreased cardiac output and contractility; and the need for vasopressor therapy. In people, pressure-adjusted heart rate (PAR) is frequently used to quantify cardiovascular dysfunction. It is the product of the heart rate (HR) and the ratio of central venous pressure (CVP) to mean arterial pressure (MAP) (HR × [CVP/MAP]). Increasing values reflect worsening cardiovascular dysfunction. Reference values for lactate and cardiac troponin concentration are available for horses. They can be run "stall side" and thus are practical biochemical markers of cardiovascular health in horses.[45] Accurate measurement of blood pressure in adult horses would require arterial catheterization. Most clinically significant pathologic arrhythmias would be detected via auscultation and confirmed with electrocardiography. Fractional shortening is frequently used as an echocardiographic parameter of cardiac contractility in horses. Normal fractional shortening for most light-breed adult horses is 28% to 45%.[46]

Musculoskeletal Criteria

Clinical signs suggestive of musculoskeletal dysfunction include weakness, recumbency, and reluctance to move. Laminitis is a unique and common manifestation of MODS in the horse; therefore, signs specific to digital pain would be present, such as Obel grades of lameness (see Chapter 90), increased digital pulse quality, hoof wall heat, and depression at the coronary band. A positive response to hoof testers or mitigation of signs following digital nerve block would be sufficient evidence for diagnosis of early acute laminitis. Persistently increased serum concentrations of creatinine phosphokinase activity would be evidence of skeletal muscle damage.[47]

Neurologic Criteria

Diagnosis of neurologic dysfunction is mostly dependent on the presence of clinical signs of alteration in behavior, depression, weakness, ataxia, and stupor.

Treatment of MODS and Prognosis

Identification and treatment of the primary initiating cause should be undertaken, with particular attempts to control the proinflammatory response and to maintain adequate hydration, tissue perfusion, oxygenation, and nutrition. Specific therapy for individual organ failure is indicated. As one might logically assume, with an increasing number of organ systems that fail, the likelihood of death increases. Mortality is typically close to 100% in human patients with four or more failing organ systems.[48]

COMMON CAUSES OF SIRS AND MODS IN HORSES

Sepsis

Colonization, or the presence of proliferating microbes without a host response, is an expected, and in many tissues, beneficial event that prevents invasion by unwanted microbes. However, if microbial exposure or colonization leads to invasion of normally sterile tissue or evokes a response from the host, infection has been initiated. Sepsis is the systemic inflammatory response to infection. In association with infection, manifestations of sepsis are the same as those previously defined for SIRS.[1]

Infection is initially prevented by innate immunity, the branch of the host defense system that does not require previous exposure to a microbe to immediately counteract with a protective response. The innate immune system is composed of phagocytic cells (principally monocytes, macrophages, and neutrophils) and an extensive array of molecular substances that are present in the circulation, on cell surfaces, and inside cells. Whether microbial tissue invasion is contained locally or continues to spread depends heavily on an appropriate and competent response from the innate immune system. Common portals of entry for microbes include the respiratory, gastrointestinal, and urogenital tracts and the skin. Microbial invasion into the bloodstream, with a concurrent systemic host response, is termed *septicemia*. Septicemia is often cited as one of the most common causes of illness and death in neonatal foals and primarily involves gram-negative bacterial invasion.[49] However, septicemia appears to be far less common in mature horses.

A main feature of the innate immune system that enables immediate discrimination is *pattern-recognition receptors* (PRRs) that are capable of detecting a variety of microbial ligands, referred to as *pathogen-associated molecular patterns* or PAMPs.[9] These ligands are evolutionally conserved molecules that are unique to microbes, are often shared by a broad range of organisms, and are usually essential for microbial survival or virulence. Examples of PAMPs include bacterial cell wall extracts, such as endotoxin, peptidoglycan, and lipoteichoic acid, and prokaryotic DNA. Ultimately, the interaction of a PRR with its PAMP can directly neutralize the PAMP or microbe, or it may activate other components of the host immune system to deploy further defense mechanisms, initiate an inflammatory response, or commence tissue repair. There is a complex and overlapping arsenal of mammalian PRRs; however, they can generally be divided into three main types: (1) secreted PRRs, such as defensins; (2) cell membrane PRRs that are involved in phagocytosis; and (3) cell membrane PRRs that are involved in signal transduction. A well-characterized cell signaling PRR-ligand relationship is CD14–Toll-like receptor (TLR) and its well-characterized PAMP, endotoxin (see later section, "Endotoxemia").

In summary, infection results from a combination of exposure to microbes and colonization of host tissue coupled with failure of the innate immune system to prevent microbial proliferation and invasion into deeper sterile tissue. By the definition of Bone and coworkers,[1] sepsis is the systemic response

evoked from the host to serve as an alarm of invasion and thus to activate defense and repair mechanisms. The outcome of infection is determined by the appropriateness of the host's defense response. If the response is appropriate, the host is altered, defense is activated, and infection is contained. When the innate immune system is overwhelmed by infection, the infection advances and cannot be overcome without intervention. Severe sepsis is defined when organ dysfunction and shock ensues (see shock in Chapter 1 and MODS in this chapter).[1]

Diagnosis of Sepsis

Sepsis can be documented cytologically by the presence of microbes in normally sterile tissue with evidence of an inflammatory response, such as the presence of neutrophils, particularly degenerative neutrophils or neutrophils with intracellular organisms. Isolation of microbes by culture provides convincing proof of infection, especially when consistent with morphologic findings on cytology. Because microbial isolation and identification can take several days, the sepsis score system was developed to identify foals at risk for septicemia. The score includes historical, physical, and laboratory findings that were statistically associated with the diagnosis of sepsis, as based on culture or necropsy.[11] This score system was developed before Bone and colleagues proposed their criteria for SIRS, and thus it is particularly interesting to note that several criteria used for SIRS in people, such as fever and leukogram changes, are included in the sepsis score for fools (see Table 2-2).

Treatment of Sepsis

Successful treatment of sepsis-induced SIRS depends on identification of the invading organism coupled with appropriate antimicrobial therapy.

Endotoxemia

German scientist Richard Pfeiffer first described endotoxin in 1892 as a toxin that was an integral part of gram-negative bacteria that was distinctly different from actively secreted heat-labile exotoxins.[50] These observations led to adaptation of the term *endotoxin*. Indeed, endotoxin is a heat-stable toxin consisting of lipopolysaccharide comprising approximately 75% of the outer cell membrane of gram-negative bacteria. It serves as a structural permeability barrier that is an essential component for survival of gram-negative bacteria. Pfeiffer's original observations were correct: bacteria do not actively secrete endotoxin. Rather, when gram-negative bacteria multiply or lyse upon bacterial cell death, endotoxin is released from the outer cell membrane. Thus the enormous resident population of gram-negative bacteria in the equine intestinal tract serves as a tremendous reservoir of endotoxin. This source of endotoxin is normally confined to the lumen of the healthy intestine by protective mucosal barriers. However, if the intestinal wall is damaged, as occurs in acute gastrointestinal diseases that cause mural inflammation or ischemia, the otherwise contained endotoxin gains access to the circulation. Although endotoxemia is often associated with acute gastrointestinal disease in horses, the release of endotoxin during log-phase bacterial growth puts any horse with gram-negative bacterial infection at risk of becoming endotoxemic. For example, as many as half of neonatal foals with gram-negative septicemia are endotoxemic at the time of admission to referral hospitals.[19] Septic metritis, pleuropneumonia, and septic peritonitis are other common primary diseases associated with endotoxemia in mature horses. Whether released from endogenous sources from the intestinal lumen or from overwhelming gram-negative sepsis, the net effects of endotoxin are similar once it accesses the circulation.

Pathophysiology of Endotoxemia

Endotoxin consists of three structural domains: a highly variable outer polysaccharide "O-antigenic" region, a core region consisting mostly of monosaccharides, and the highly conserved toxic moiety, lipid A. Variation in the number, length, saturation, and position of fatty acids on the glucosamine disaccharide backbone of lipid A confers its degree of toxicity. Endotoxin is not directly toxic to mucous membranes or skin; rather, it must enter the circulation to fully manifest its pathologic effects. Once in the blood, endotoxin's amphipathic properties cause it to form aggregates resembling micelles that otherwise spontaneously disperse into monomers at a very slow rate.

Lipopolysaccharide binding protein (LBP) is a 65-kDa plasma constituent that is primarily synthesized by the liver and belongs to the family of lipid transfer proteins.[51] It efficiently extracts molecules of endotoxin from aggregated micelles in the blood and transports them to various locations. LBP is not consumed by this transfer, which appears to be catalytic. As an acute phase protein, the blood concentration of LBP can increase several hundredfold within 24 hours following an inflammatory stimulus. In a 2005 clinical study, serum concentrations of LBP at admission were up to 30 times greater in horses with acute gastrointestinal disease, as compared to healthy horses, though LBP concentrations did not correlate with the cause of colic or the outcome.[7] Through its interaction with lipid A, LBP effectively imprisons endotoxin, with its potential for toxicity determined by the complex's final destination. LBP can rapidly deliver monomers of endotoxin to the cell surface of host inflammatory cells to evoke an inflammatory response or it can be transferred to other neutralizing lipoproteins, such as high-density lipoprotein, for eventual removal from the blood. Thus LBP may both enhance and hinder the biologic activities of endotoxin. Although LBP greatly facilitates "disaggregation" and transport of endotoxin to the surface of inflammatory cells, its presence is not mandatory for the interaction of endotoxin at the cell surface.

Once at the cell surface, endotoxin is transferred to CD14, a well-conserved receptor attached by a glycosylphosphatidylinositol anchor.[52] Mononuclear phagocytes (monocytes and macrophages) express abundant CD14, though other inflammatory cells also express minute amounts. CD14 is a 53-kDa glycoprotein that exists as both a cell membrane receptor (mCD14) and a soluble form (sCD14) in the circulation, the latter being essentially identical to membrane bound CD14 minus the glycosylphosphatidylinositol anchor. Similar to LBP's roles, sCD14 is normally present in the circulation and has dual functions. It may bind and neutralize circulating endotoxin, thereby competing with membrane CD14. However, it may also enhance endotoxin's toxic effects by transferring it to membrane CD14 or to cells that do not express it. CD14 does not structurally cross the cell membrane. Thus it must associate with a secondary protein, TLR that contains a transmembrane portion that is capable of communication with the intracellular domain.

Both CD14 and TLR are classified as bacterial recognition receptors, indicating that they are evolutionarily conserved receptors that are a part of the innate immune system. These receptors accept a wide variety of bacterial, fungal, and viral ligands that express a similar "pattern of recognition." Previous exposure to the pathogen is not required for recognition by these receptors, which therein provide an immediate response to infection. CD14 binds to isolated monomers of endotoxin, intact bacteria, and several components of gram-positive bacteria, including peptidoglycan and lipoteichoic acid. The name "Toll-like" receptor was adapted because of homology with a receptor found in *Drosophila*, called *Toll*, that is responsible for dorsal-ventral polarity and innate immunity in fruit flies. Approximately a dozen TLRs have been identified, but TLR type 4 (TLR4) appears to be the most important isotype in the recognition of endotoxin, whereas TLR type 2 primarily confers recognition of gram-positive bacteria.[53] The presence of TLR4 is mandatory for responsiveness to endotoxin, and transfection experiments have clearly demonstrated that the type and degree of response is conferred by the species of origin of the TLR4.[54] Furthermore, polymorphism in TLR4 is associated with differences in the degree of response to endotoxin in people and offers an explanation for the well-recognized clinical phenomenon that some individuals react violently to endotoxin, whereas others have little to no response.[55] The development of tolerance, or the tachyphylactic response to prolonged endotoxin exposure, has been linked to downregulation of TLR4 and CD14 mRNA. CD14, TLR4, and TLR2 have been cloned and sequenced in the horse.

Once the CD14-TLR4-endotoxin complex is compiled at the cell surface, TLR4 requires the help of a 160–amino acid helper molecule, MD2, to transmit a signal to the cytosol.[53] Numerous intracellular signaling pathways have been reported to link endotoxin-occupied cell surface receptor to a response from the cell, including protein tyrosine kinases, mitogen-activated tyrosine kinases (ERK, JNK, p38), protein kinases A and C, G proteins, various phospholipases, and translocation of nuclear factor κB (NFκB). The latter pathway is well characterized in mammalian endotoxin-induced cell signaling, including horses.[53,56] In the NFκB pathway, the cytosolic domain of TLR4 associates with myeloid differentiation factor 88 (MyD88) that recruits interleukin-1 receptor-associated kinase (IRAK), resulting in its phosphorylation. Phosphorylation of IRAK promotes its association with tumor necrosis factor receptor–associated factor (TRAF6), NFκB inducing kinase, and inhibitor (IκB) kinase (IKK). IKK phosphorylates IκB, which cleaves IκB from the IκB-NFκB complex. NFκB is a heterodimer of two proteins, p50 and p65. Once freed from IκB, the NFκB dimers translocate to the nucleus to activate the promoter sites for genes encoding inflammatory mediators.

Although there is more to learn about the regulation of the intracellular signaling systems involved in endotoxin-induced cell activation, there is solid agreement that most of the deleterious effects of endotoxin are the result of overzealous endogenous synthesis of proinflammatory mediators and initiation of SIRS. The most widely studied of these mediators are the metabolites of arachidonic acid (the prostaglandins, thromboxane, and the leukotrienes), PAF, cytokines (TNF and interleukins 1, 6, and 8), vasoactive and chemotactic peptides (histamine, serotonin, bradykinin, complement components), tissue factor, proteolytic enzymes, and reactive oxygen species. Several of these endogenous mediators have been quantified in horses with both experimentally induced and naturally occurring endotoxemia.[4,5,19,57-60] Correlation of peritoneal and serum concentrations of these mediators with adverse sequelae and mortality in horses solidifies the importance of their roles in endotoxemia.[59,60] The culmination of events in endotoxemia is uncontrolled inflammation, immunosuppression, hemodynamic changes, and coagulopathy. Decreased peripheral vascular resistance, myocardial depression, hypovolemia, and microvascular thrombosis all contribute to reduction of blood flow to vital organs. If flow is sufficiently reduced, it may become irreversible (see "Common Causes of SIRS and MODS in Horses").

Clinical Findings and Diagnostic Approach

The clinical signs of endotoxemia are often overshadowed by signs of the primary disease process that initiated the response. However, through experimental infusion studies with purified lipopolysaccharide, the response to endotoxin is well characterized in horses.[25,57,61-64] Within the first 30 to 60 minutes of sublethal intravenous challenge with endotoxin, horses yawn frequently; develop mucous membrane pallor; become depressed, anorectic, tachypneic, tachycardic, and restless; develop fasciculations and mild to moderate signs of colic; and pass loose feces. This period is the *early hyperdynamic phase of endotoxemia* that is characterized by pulmonary hypertension (increased pulmonary arterial and wedge pressures and increased pulmonary vascular resistance) and ileus associated with increased levels of thromboxane A_2, though other vasoconstrictors likely contribute. By 1 to 2 hours after challenge, depression and anorexia continue and are affiliated with the onset of fever and hypotension. This *hypodynamic phase of endotoxemia* is caused by decreased systemic vascular resistance from the release of prostaglandins. Mucous membranes are often hyperemic, and capillary refill time is prolonged. With reduced tissue perfusion, the classic "toxic line" develops as a red to blue-purple line (a few millimeters in width) at the periphery of the gums. This is particularly notable at the upper incisors. If hypotension advances, mucous membranes become diffusely congested, progressing to cyanosis and then a grayish-purple pallor.

As per its definition, endotoxemia technically means the presence of endotoxin within the blood. The gold standard for measurement of endotoxin, the limulus amebocyte lysate (LAL) assay, has been used to detect endotoxin in the plasma, portal circulation, or peritoneal fluid of horses and foals with naturally occurring endotoxemia.[59,60,65,66] The lower limit of detection of the assay is picograms per milliliter, though nanograms of endotoxin per milliliter of plasma or peritoneal fluid have been identified in horses with acute gastrointestinal diseases and foals with septicemia.[60,66] As might be expected in horses with colic, peritoneal fluid and plasma endotoxin concentrations are greatest in horses with inflammatory or ischemic gastrointestinal injuries. Although the LAL assay is a highly specific test for the detection of endotoxin, its tedious nature makes it impractical as a routine diagnostic test. Likewise, quantification of specific endotoxin-induced inflammatory mediators can provide a presumptive diagnosis as well as prognostic information, though again, these types of assays are rarely available in clinical settings. Therefore the diagnosis of endotoxemia relies heavily on identification of clinical signs and diagnostic markers in diseases known to be associated with the release of endotoxin.

One cardinal diagnostic marker of endotoxemia and acute overwhelming bacterial infection is profound neutropenia with toxic neutrophil morphology (basophilic cytoplasm, vacuolization, Döhle bodies) with a left shift. Neutropenia will occur within an hour of onset of endotoxemia and is proportionate to the degree of endotoxemia. Other diagnostic markers of endotoxemia typically reflect nonspecific secondary changes from stress (hyperglycemia), hypovolemia, (relative polycythemia, hyperproteinemia, azotemia, metabolic acidosis, increased anion gap, and lactic acidosis), and specific organ damage from decreased perfusion (azotemia; increased creatine phosphokinase, liver enzyme, or cardiac troponin activities).

Clinical Management

The rapid response to endotoxin coupled with the diverse biologic actions of the endogenous mediators present a difficult therapeutic challenge. Although neutralization of endotoxin or blockade of a single class of mediators may provide some benefit, no single treatment is a panacea. Rather, treatment of endotoxemia should include a combination of therapeutic targets. Many different treatments for endotoxemia have been evaluated in the horse and other species, both experimentally and clinically. The following discussion will be limited to therapies that have been specifically evaluated in the horse or are available for clinical use.

When treating endotoxemia, the first goal should be diminishing the source. When endotoxemia is the result of acute intestinal inflammation or ischemia, translocation of luminal endotoxin might be abated by administering smectite orally (4 ounces orally, twice daily), which absorbs bacteria and bacterial toxins, and by specifically treating the underlying disease, such as surgically removing the ischemic intestine. If endotoxemia is the result of gram-negative sepsis, tissue débridement and lavage should be undertaken when possible. Caution should be exercised when antimicrobials are used, because the bactericidal actions of certain drug classes, particularly the β-lactam family, may increase the release of endotoxin, and in such scenarios, concurrent treatment with endotoxin-neutralizing drugs (see later) is indicated.[67,68]

Once endotoxin enters the circulation, the most effective therapeutic strategy is to bind and neutralize endotoxin before it interacts with inflammatory cell receptors. Although this may seem to present a narrow therapeutic opportunity, until the source of endotoxin is controlled, use of endotoxin-neutralizing drugs is beneficial. Furthermore, there are several clinical situations in which development of endotoxemia can be anticipated; specific examples include the release of endotoxin following use of bactericidal antimicrobials in the face of gram-negative infection, following grain engorgement, and immediately after correction of a strangulating lesion.

Currently, there are two commercially available products for horses that directly bind to endotoxin: anti-endotoxin antibodies and polymyxin B. It must be noted that both of these products directly bind endotoxin, and thus their effectiveness is a factor of both the dose administered and the amount of free, unbound circulating endotoxin. Anti-core antibodies have been used in several clinical and experimental trials, often with conflicting results.[69-71] Endoserum (Immvac Inc., Columbus, MO), hyperimmune serum from horses vaccinated with the *Salmonella typhimurium*, Re mutant, has the disadvantage of requiring refrigeration. Dilution of this product with sterile isotonic saline or lactated Ringer solution (1:10 to 1:20) and administration of the diluted product intravenously over 1 to 2 hours may reduce the risk of another complication: hypersensitivity reactions. Hyperimmune anti-core plasma to *Escherichia coli* J5 (Equiplas J, Plasvacc USA Inc., Templeton, CA) can be used in foals for the concurrent treatment of endotoxemia, septicemia, and failure of transfer or passive immunity, given at the dose recommended for treatment of failure of transfer of passive immunity (20 to 40 mL per kg body weight) or in adult horses. The disadvantages in using plasma products are that they are expensive, require freezer storage, and must be thawed before administering, which delays treatment.

Polymyxin B is a cationic antibiotic that, in addition to its bactericidal properties, also binds to and neutralizes endotoxin through direct molecular interactions with the lipid A region. Because lipid A is structurally conserved among gram-negative bacteria, polymyxin B hypothetically should have broader endotoxin-binding capabilities than anti–core-endotoxin antibodies. In one study in foals, polymyxin B provided more protection against endotoxemia than did anti-core antiserum.[72] Polymyxin B has the additional advantage of being a lyophilized product that may be stored at room temperature. Clinical use of polymyxin B has been hindered by its inherent nephrotoxic and neurotoxic side effects when used intravenously at bactericidal doses. Recent studies in human patients, foals, and horses have shown that doses considered suboptimal for bactericidal treatment are remarkably effective in neutralizing endotoxin without causing toxic side effects. Currently, in horses and foals, the recommended dose of polymyxin B is 1000 to 6000 IU per kg body weight, administered intravenously every 8 to 12 hours.[72-75] As with anti-core antibodies, the effectiveness is dose related. If large circulating concentrations of endotoxin are anticipated, the higher dose range for polymyxin B is warranted. Although nephrotoxicity has not been reported in experimental trials with polymyxin B at doses less than 6000 IU per kg body weight, polymyxin B should be used judiciously in azotemic patients.

In many clinical scenarios, endotoxemia has not been anticipated and thus its recognition is delayed until clinical signs of endotoxemia are well established. Unfortunately, at this point, halting the multitude of biologic actions of the numerous endogenous mediators presents a difficult therapeutic challenge. To this end, seemingly countless antidotes for specific mediators or regimens for general shock therapy have been studied as treatments for endotoxemia; however, none have been shown to dramatically improve survival in the clinical setting. Thus, the mainstays of treatment that are most commonly employed are general supportive fluid therapy and the use of anti-inflammatory drugs. Therapy directed specifically for shock (see Chapter 1) or organ failure should be addressed if these complications develop in advanced endotoxemia. The following discussion will be limited to drugs that have been specifically tested in endotoxemic horses and are available for use in the clinical setting.

NSAIDs were one of the first classes of drugs described for the treatment of endotoxemia in horses. In this regard, flunixin meglumine, a nonspecific cyclooxygenase inhibitor, is highly effective in preventing endotoxin-induced prostanoid synthesis and associated clinical signs of endotoxemia. Administration of 0.25 mg/kg body weight intravenously every 8 hours is commonplace. Advantages of this low-dose flunixin meglumine regimen are reduced risk of potential toxic side effects and

effective inhibition of prostanoid synthesis without completely masking physical manifestations of endotoxemia that are necessary for accurate clinical assessment of the patient's progress.[57] However, if the underlying cause of endotoxemia has clearly been identified, and a more effective level of cyclooxygenase inhibition is desirable for more potent analgesic and anti-inflammatory effects, higher doses (1.1 mg/kg) may be used. Other NSAIDs, such as ketoprofen, phenylbutazone, and firocoxib, have either not been fully evaluated in endotoxemic horses or do not appear to offer any advantage over flunixin meglumine when used for endotoxemia.

Inhibition of other mediators associated with endotoxemia is more difficult. Although monoclonal antibodies to TNF and PAF receptor antagonists have been shown to be advantageous in experimentally induced endotoxemia in horses, these drugs are prohibitively expensive to use in horses or they are not commercially available. There is a general lack of conclusive evidence that blockade of lipid peroxidation is beneficial in experimentally induced endotoxemia in horses. Nonetheless, dimethyl sulfoxide (DMSO) frequently is used intravenously at doses ranging from 0.1 to 1 g/kg body weight, diluted to at least 10% in isotonic fluid. In an experimental model of endotoxemia in horses, DMSO had minimal effects on clinical signs with the only effect being reduction of endotoxin-induced fever.[76] Other anti-inflammatory agents that have been used in endotoxemic horses are pentoxifylline and lidocaine[77,78]; however, the clinical efficacy of these drugs has not been critically evaluated.

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CHAPTER
3

Fluids, Electrolytes, and Acid-Base Therapy

Joanne Hardy

Fluid administration for maintenance or replacement purposes is one of the mainstays of equine critical care, and the technology should be readily accessible in any equine hospital. The availability of commercial materials and fluids for use in large animals makes fluid administration easy and cost effective in most situations. This chapter reviews fluid and electrolyte balance, materials needed, and principles to follow when planning fluid administration.

NORMAL FLUID AND ELECTROLYTE BALANCE

Distribution of Fluids

Fluids in the body are distributed in two compartments: the *intracellular fluid (ICF)* volume and the *extracellular fluid (ECF)* volume. The ECF is composed of interstitial fluid, plasma, lymph, and transcellular fluids such as synovial, pleural, abdominal, and cerebrospinal fluids. The transcellular fluids do not normally contribute to signficant fluid losses, but in disease states such as pleuropneumonia or peritonitis, they can contribute significantly to volume deficits. For example, it is not unusual to drain 10 to 20 L of fluid from the pleural cavity of horses with severe pleuropneumonia. Additionally, the volume of gastrointestinal secretions in horses plays an important role in fluid distribution. The normal volume of gastrointestinal secretion in horses is approximately equivalent to the extracellular fluid volume, representing about 100 L every 24 hours in a 500-kg horse.[1] Therefore significant fluid sequestration and loss can occur with intestinal obstruction or colitis. The volume of total body water (TBW) represents 60% of body weight in adults and up to 80% in neonates. The ECF volume represents ⅓ of TBW or 20% of body weight (in adults) to ½ of TBW or 40% of body

weight (in neonates) of water, and the ICF volume represents approximately ⅔ of TBW or 40% of body weight. Recent estimates of fluid distribution in horses report values of 0.67 L/kg (67%) for TBW, 0.21 L/kg (21%) for ECF, and 0.46 L/kg (46%) for ICF.[2,3] In neonates, the ECF is approximately 40% of the TBW, and it decreases to approximately 30% by 24 weeks of age.[4] For calculation purposes on substances distributed across the ECF, a factor of 0.3 is used for adults and 0.4 for young animals. Blood volume in sedentary horses represents approximately 8% of body weight.[5] In fit horses, this value can reach 14% of body weight.[6] In neonates, blood volume represents 15% of body weight and decreases to adult values by 12 weeks of age.[4]

Body solutes are not distributed equally through TBW. In plasma, sodium is the main cation, and bicarbonate and chloride are the main anions. Proteins contribute to the negative charges, and they also provide oncotic pressure. Albumin or molecules of similar size are the main contributors to oncotic pressure. The *interstitial fluid* accounts for about 75% of the ECF, and it is composed mainly of sodium, bicarbonate, and chloride, but the concentration of protein there is lower than in plasma. The slightly increased concentration of anions and decreased concentration of cations in interstitial fluids occurs because of the greater concentration of protein in plasma (according to the Gibbs-Donnan equilibrium). In clinical practice, this difference is small, so that the measured concentration of solutes in plasma is thought to reflect the concentration of solutes throughout the ECF. Table 3-1 lists normal plasma concentrations of electrolytes in adult horses. The composition of the intracellular fluid compartment is different: the important cations are potassium and magnesium, and the important anions are phosphates and proteins (Figure 3-1).

Transfer of fluid between compartments is an important consideration when planning fluid administration. Some important concepts govern these mechanisms. *Osmolality* is defined as the concentration of osmotically active particles in solution per kilogram of solvent (mOsm/kg), whereas *osmolarity* is the number of particles of solute per liter of solvent (mOsm/L). In biologic fluids, the difference between the two concentrations is negligible, and the two terms are often used interchangeably. Normal plasma osmolality in adult horses ranges from 275 to 312 mOsm/kg, and it varies slightly between breeds. Lower values are reported for normal foals.[7,8] The effective osmolality, or tonicity, is the osmotic pressure generated by the difference in osmolality between two compartments. Colloid oncotic pressure is the osmotic pressure generated by proteins, mainly albumin, and is measured using a colloid osmometer (Wescor, Logan, UT). Normal values of 15.0 to 22.6 mm Hg for foals and 19.2 to 31.3 mm Hg for adult horses have been reported.[9,10] Water and ionic solute exchange between the vascular and

TABLE 3-1. Normal Hematologic Values in Adult Horses

Plasma Parameter (Units)	Normal Concentration Range
CATIONS	
Sodium (mmol/L or mEq/L)	132-146
Potassium (mmol/L or mEq/L)	2.4-4.7
Calcium (mmol/L)	2.8-3.4
Ionized calcium (mmol/L)	1.0-1.3
Magnesium (mmol/L)	0.9-1.15
Ionized magnesium (mmol/L)	0.4-0.55
ANIONS	
Chloride (mmol/L or mEq/L)	99-109
Total CO_2 (mmol/L or mEq/L)	24-32
VENOUS BLOOD GAS	
pH	7.32-7.44
PCO_2 (mm Hg)	38-46
PO_2 (mm Hg)	37-56 (arterial, 80-100)
HCO_3^- (mmol/L or mEq/L)	20-28
Base excess (mmol/L or mEq/L)	−2 to +2
OTHER	
Creatinine (mg/dL)	0.9-1.9
Plasma protein (g/dL)	5.8-8.7
Albumin (mg/dL)	2.9-3.8
Plasma lactate (mmol/L)	1.11-1.78

Data from Kaneko JJ, Bruss ML: Clinical Biochemistry of Domestic Animals. 5th Ed. Academic Press, San Diego, 1997.

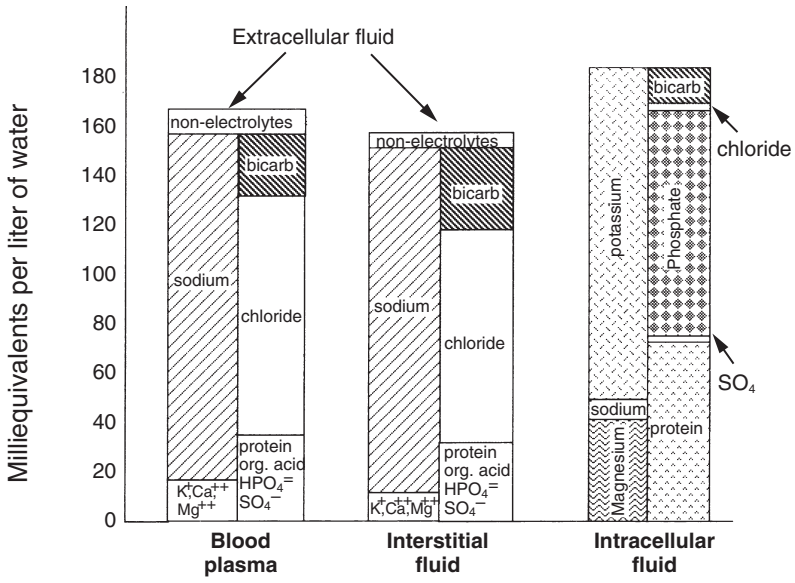

Figure 3-1. The compositions of plasma, interstitial fluid, and intracellular fluid. (Adapted from Guyton AC: Textbook of Medical Physiology, 7th Ed. Saunders, Philadelphia, 1986, p. 386; originally modified and reprinted by permission of the publisher from Gamble JL: Chemical Anatomy, Physiology and Pathology of Extracellular Fluid: A Lecture Syllabus. Cambridge, Mass, Harvard University Press, 1954; copyright 1942 by JL Gamble.)

interstitial compartments occurs at the capillary level and is rapid; equilibrium is reached within 30 to 60 minutes. The rate of exchange or net filtration that occurs between these compartments is controlled by a balance between the forces that favor filtration (capillary hydrostatic pressure and tissue oncotic pressure) and the forces that tend to retain fluid within the vascular space (plasma oncotic pressure and tissue hydrostatic pressure). These relationships are described by *Starling's law:*

$$\text{Net filtration} = K_f\,[(P_{cap} - P_{int}) - \sigma(\pi_p - \pi_{int})]$$

where K_f is the filtration coefficient, which varies depending on the surface available for filtration and the permeability of the capillary wall; P_{cap} and P_{int} represent the hydrostatic pressures in the capillary or in the interstitium; π_p and π_{int} are the oncotic pressures in the plasma or interstitial fluid; and σ is the reflection coefficient of proteins across the capillary wall.

Exchanges between the interstitial and the intracellular compartment are governed by the number of osmotically active particles within each space. Sodium is the most abundant cation in the ECF. Consequently, sodium accounts for most of the osmotically active particles in the ECF. Other osmotically active compounds that make a significant contribution to ECF osmolarity are glucose and urea. The most commonly used formula for estimation of serum osmolarity is as follows[11]:

$$\text{ECF osmolality} = 2[Na^+] + \frac{\text{glucose}}{18} + \frac{\text{urea}}{2.8}$$

Cell membranes are permeable to urea and K^+. Therefore the effective osmolarity is calculated as follows:

$$\text{ECF osmolality} = 2[Na^+] + \frac{\text{glucose}}{18}$$

The osmolar gap is the difference between measured osmolarity and calculated osmolarity; an increased osmolar gap can exist when unmeasured solutes, such as mannitol, are present.[12]

Exchanges between the extracellular and intracellular compartments are comparatively slow, taking up to 24 hours to reach equilibrium.

Acid-Base Balance

The concentration of hydrogen ions, and therefore the pH, is closely regulated in the body to vary between 7.35 and 7.45. This narrow range is maintained by the presence of buffers within different body compartments; a buffer is a compound that can accept or donate protons to maintain the pH within a narrow range. In the body, bicarbonate is the primary buffer system of the extracellular fluid, whereas protein and inorganic and organic phosphates represent the principal intracellular buffers.

The importance of bicarbonate as a buffer in the ECF stems from the fact that it is an open system. The dissociation of carbonic acid is expressed by the law of mass action:

$$H^+ + HCO \rightleftharpoons H_2CO_3 \rightleftharpoons CO_2 + H_2O$$

In the body, this system is open, and carbonic acid, in the presence of carbonic anhydrase, forms CO_2, which is eliminated entirely from the system by alveolar ventilation.

The relationship between pH, bicarbonate, and carbonic acid is expressed in the Henderson-Hasselbalch equation:

$$pH = 6.1 + \log\left[\frac{HCO_3^-}{0.03PCO_2}\right]$$

where PCO_2 is the partial pressure of carbon dioxide. This is the clinically relevant form of the equation, which shows that in body fluids, pH is a function of the ratio of HCO_3^- to PCO_2; this ratio is normally approximately 20:1.

The responses to acid or base alterations in the body all combine to normalize pH. For example, an acute increase in hydrogen ions from a fixed acid load is immediately buffered by bicarbonate and intracellular buffers. This is the acute physiochemical response. Alveolar ventilation is subsequently modified, and this is complete within hours to further minimize changes in pH by normalizing the ratio of HCO_3^- to PCO_2. Finally, renal responses result in regeneration of HCO_3^-, resulting in a long-term response. The renal response begins within hours and is complete within 2 to 5 days. An acute increase in volatile CO_2, in contrast, cannot be buffered by HCO_3^-; therefore, the hydrogen ions generated from the dissociation of carbonic acid must be buffered by intracellular buffers. Renal adaptation, characterized by increased HCO_3^- reabsorption and net acid secretion, takes 2 to 5 days to achieve maximal effectiveness.

ACID-BASE DISORDERS

Terminology

Acidosis and alkalosis refer to the *processes* that cause net accumulation of acid or alkali in the body, respectively. Acidemia and alkalemia refer to the pH of the ECF: in acidemia, the pH of the ECF is lower than normal, and in alkalemia the pH of the ECF is higher than normal. The distinction between these terms is important; for example, a horse with chronic reactive airways disease may have a normal blood pH because of effective renal compensation, but in this setting the patient will have increased bicarbonate. This patient has alkalosis but does not have alkalemia.

Primary Acid-Base Disorders

There are four primary acid-base disorders: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. The metabolic disorders refer to the net excess or deficit of nonvolatile or fixed acid, whereas the respiratory disturbances refer to a net deficit or excess of volatile acid (dissolved CO_2).

Metabolic acidosis is present when there is a decrease in HCO_3^- caused by either loss or buffering of nonvolatile acids. Common causes of metabolic acidosis in horses include accumulation of lactic acid as a result of poor perfusion, and HCO_3^- losses in the gastrointestinal tract resulting from diarrhea. *Metabolic alkalosis* is present when there is an increased concentration of HCO_3^-. Metabolic alkalosis is commonly associated with a disproportionate loss of chloride ions. *Respiratory acidosis* is present when the PCO_2 is increased in response to alveolar hypoventilation. *Respiratory alkalosis* is present when the PCO_2 is decreased. Table 3-2 lists examples of primary acid-base disorders in horses.

For each primary acid-base disturbance, there is a secondary or adaptive response that involves the component opposite the primary disturbance in an attempt to return the pH toward

normal. The secondary response never restores the pH completely to normal. For metabolic disorders, the secondary or adaptive respiratory response begins immediately and is complete within hours. In respiratory disorders, the adaptive response begins with an acute, immediate titration by nonbicarbonate buffers that results in an initial change in plasma HCO_3^- concentration. This is followed by a chronic response mediated by the kidney that involves net acid secretion and bicarbonate resorption. This response begins within hours and takes 2 to 5 days to be complete. Table 3-3 lists expected adaptive responses to acid-base disorders. These expected responses vary slightly across species.[13]

Mixed Acid-Base Disorders

When a primary disorder occurs with the expected secondary response, it is considered a simple acid-base disorder. A mixed disorder means that two separate primary disorders are present in the same patient. A mixed disorder is suspected when the adaptive response is lower or higher than the expected response from the primary disorder.

MEASUREMENT AND INTERPRETATION OF BLOOD GASES

Measurement

For accurate blood gas analysis, appropriate sampling methods should be followed. Blood (arterial or venous) is collected anaerobically from the puncture site using a syringe that contains the appropriate anticoagulant for the analyzer, usually lithium heparin, taking care to not dilute the sample with excess heparin. Introduction of room air into the sample will falsely increase the PO_2 and decrease the PCO_2. If a delay in analysis is anticipated, the blood should be placed on ice to decrease cell metabolism. Most blood gas analyzers perform their analysis at 37° C. At extremes of body temperature, a patient's actual value may differ from expected values according to the gas law: with increases in temperature, gas is less soluble and therefore its partial pressure in the solution increases; this will increase the PO_2 and PCO_2 of a solution. Similarly, with extreme hypothermia, gas is more soluble, resulting in decreases in PO_2 and PCO_2. Although available in many blood gas analyzers, temperature correction is usually not performed for several reasons: (1) the small changes in body temperature usually do not affect blood gas analyses significantly; (2) the patient's temperature, if extreme, will usually be corrected shortly; and (3) there are no established normal values for extremes in body temperature.

Routine blood gas analyzers provide three measured values, pH, PCO_2, and PO_2, and three calculated values, total CO_2 (TCO_2), HCO_3^-, and base excess (BE). Measured values that are outside of physiologic ranges should be considered a malfunction of the analyzer.

- pH is the negative base 10 logarithm of hydrogen concentration and is a measured value.
- PCO_2 (mm Hg) is the measured partial pressure of dissolved carbon dioxide in the sample. A venous sample will have a

TABLE 3-2. Traditional Approach* to Simple Acid-Base Disorders in Adult Horses

Parameter	Metabolic Acidosis	Metabolic Alkalosis	Respiratory Acidosis	Respiratory Alkalosis (Arterial Sample)
pH	7.390	7.49	7.30	7.50
PCO_2 (mm Hg)	28.4	49	60	32
PO_2 (mm Hg)	42	43	38	55
HCO_3^- (mEq/L)	16.5	34	28	22
Base excess (BE) (mEq/L)	−5.8	+9	−1	−2
Comments	There is a secondary decrease in PCO_2 to compensate for the primary disorder.	There is a secondary increase in PCO_2 in an attempt at compensation.	This is an acute disorder with increased bicarbonate of 1-2 mEq/L per 10 mm Hg increase in PCO_2. Note that the BE is normal, indicating no metabolic disturbance.	Increased ventilation in response to hypoxemia is the cause of this disorder.

*This traditional approach to the diagnosis of simple acid-base disorders depends on interpretation of the clinical parameters in the left-hand column, without examining the contribution of electrolytes, unmeasured anions, or protein concentrations.

TABLE 3-3. Secondary (Adaptive) Responses to Primary Acid-Base Abnormalities

Disorder	Primary Change	Secondary Response
Metabolic acidosis	↓ HCO_3^-	PCO_2 decreases by 1.2 mm Hg for every 1 mEq/L decrease in bicarbonate
Metabolic alkalosis	↑ HCO_3^-	PCO_2 increases by 0.6-1 mm Hg for every 1 mEq/L increase in bicarbonate
Acute respiratory acidosis	↑ PCO_2	[HCO_3^-] increases by 1 mEq/L for every 10 mm Hg increase in PCO_2
Chronic respiratory acidosis	↑ PCO_2	[HCO_3^-] increases by 3-4 mEq/L for every 10 mm Hg increase in PCO_2
Acute respiratory alkalosis	↓ PCO_2	[HCO_3^-] decreases by 1-3 mEq/L for every 10 mm Hg decrease in PCO_2
Chronic respiratory alkalosis	↓ PCO_2	[HCO_3^-] decreases by 5 mEq/L for every 10 mm Hg decrease in PCO_2

From Brobst D: J Am Vet Med Assoc 183:773, 1983.

slightly higher (5 mm Hg) value than an arterial sample. An increase in PCO_2 is termed *hypercapnia* or *hypercarbia*, and it usually reflects alveolar hypoventilation.

- PO_2 (mm Hg) is the measured partial pressure of dissolved oxygen in blood. This is different from oxygen content, which is the total concentration of oxygen carried by blood and includes the portion carried by hemoglobin.
- TCO_2 (mEq/L) is the concentration of total CO_2 in the sample, obtained by adding a strong acid to the sample and measuring the amount of CO_2 produced; it includes both dissolved CO_2 and HCO_3^-. Because HCO_3^- represents 95% of total CO_2, this measurement is indirectly a measurement of HCO_3^-, and it is 1 to 2 mEq/L higher than the concentration of HCO_3^-.
- HCO_3^- (mEq/L) is reported as actual bicarbonate, which is the calculated concentration of bicarbonate in the sample, and standard bicarbonate, which is the calculated concentration of HCO_3^- after the sample has been equilibrated to a PCO_2 of 40 mm Hg.
- BE (mEq/L) is the amount of strong acid or base required to titrate 1 L of blood to a pH of 7.40 at 37° C with the PCO_2 held constant at 40 mm Hg. Because the base excess is changed only by nonvolatile fixed acids, it is considered to reflect metabolic acid-base disturbances.

Normal values for the horse are presented in Table 3-1.

Interpretation

To interpret blood gases, a practiced method should be followed. First the pH is examined, and if it is outside the normal range, an acid-base disorder is present. The clinician examines next the HCO_3^- level and PCO_2 and determines if an abnormality is present that could explain the abnormal pH. An *acidemia* is caused by an increase in PCO_2 or a decrease in HCO_3^-, whereas an *alkalemia* is caused by a decrease in PCO_2 or an increase in HCO_3^-. Once the primary disorder has been characterized, the clinician determines whether a secondary response is present. The absence of a secondary response, or a change in the direction opposite the expected response, is an indication of a mixed disorder. The clinician then determines whether the acid-base disturbance is consistent with the patient's history and clinical findings. Table 3-2 lists examples of simple acid-base disorders in horses.

Another important component of blood gas interpretation is the PO_2. The normal PO_2 of arterial blood (PaO_2) is approximately 5 times the fraction of inspired oxygen (FiO_2), or 80 to 100 mm Hg in room air at sea level (FiO_2, 21%). *Hypoxemia* refers to a decreased PaO_2; common causes include a decreased FiO_2 (an example is a decreased barometric pressure associated with high altitude), hypoventilation, ventilation/perfusion mismatch, shunt, or diffusion impairment. The normal PO_2 of venous blood (PvO_2) is 40 mm Hg. A low mixed PvO_2 (mixed refers to a sample collected centrally, ideally from the pulmonary artery) in the presence of normal PaO_2 should alert the clinician that tissue perfusion is poor.

Anion Gap

The anion gap (AG) is the difference between the sum of the commonly measured cations and the sum of the commonly measured anions in serum, calculated as follows:

$$AG = ([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-]).$$

The sum of cations always exceeds the sum of anions, and the difference is an attempt to estimate the concentration of unmeasured anions—for example, lactate. A normal AG of 10.4 ± 1.2 mEq/L has been reported in adult horses.[14] Neonates have a slightly higher AG because of their increased levels of phosphates and globulins.[14] In exercising horses, the AG is useful to estimate plasma lactate concentrations in the presence of relatively normal plasma protein concentrations.[15] In horses with abdominal pain, the correlation between lactate concentration and the AG is excellent, but the presence of other strong ions results in a higher AG than would be expected from lactate measurement.[16] The AG is considered a good prognostic indicator of survival in horses with abdominal disorders: a value greater than 25 mEq/L is associated with a significantly lower survival rate.[17,18]

Lactate

Measurement of lactate concentration is now a routine part of the assessment of perfusion in equine patients, and it is available in most chemistry and point-of-care analyzers.[19] Samples should be analyzed immediately to prevent *in vitro* lactate production by erythrocytes; alternatively, collection in fluoride-containing tubes, storage on ice, and separation of plasma can help minimize this problem. Lactate is the end product of anaerobic glycolysis, and its concentration is another indicator of tissue perfusion and oxygen delivery; an increased blood lactate concentration is most often a result of tissue hypoxia. Although inadequate oxygen delivery to tissues as a result of hypovolemia, decreased oxygen content, or impaired myocardial function (absolute hypoxia) is the most common cause of hyperlactatemia, hypermetabolic states or impaired oxygen use as a result of mitochondrial dysfunction (relative hypoxia) can also increase blood lactate concentration. Less commonly, increased lactate may result from impaired clearance because of hepatic dysfunction, thiamine deficiency, or increased catecholamine production.[20] Normal blood lactate concentrations in resting adult horses are less than 2 mmol/L; concentrations higher than this in the adult are an indication of inadequate oxygen delivery. Neonates have higher blood lactate concentrations that decrease to adult values by 24 hours of age.[21] Serial measurement of lactate is a useful tool to monitor the adequacy of fluid therapy (see Chapter 1).

Nontraditional Approach to Acid-Base Evaluation

In the traditional approach, the relationship between PCO_2, HCO_3^-, and pH is explained by the Henderson-Hasselbalch equation and appears to stand alone as an explanation for acid-base derangements. This is still the approach most commonly used by clinicians, and it serves to initiate and target therapeutic intervention. However, what this approach fails to do is provide explanations for the influence of other electrolytes, weak acids, and plasma protein on acid-base balance.

The nontraditional approach (or Stewart's approach) to acid-base balance is based on three physical laws: maintenance of electroneutrality, satisfaction of dissociation equilibrium for solutes that are incompletely dissociated, and conservation of mass. In this approach, independent variables are those that can be altered externally; dependent variables change only when

independent variables change. Independent variables include the strong ion difference (SID), PCO_2, and the total concentration of weak acids, or A_{tot}. The SID is the difference between the concentration of strong cations and the concentration of strong anions. The most important cation is sodium; chloride and other unmeasured anions make up the strong anions. Because many strong anions are not routinely measured, the normal SID accounts for the presence of these anions. An increase in SID indirectly indicates an accumulation of unmeasured anions. The concentration of weak acids in plasma mostly derives from protein and phosphates. Bicarbonate is a dependent variable that changes in response to a change in independent variables. Hypoproteinemia (a decrease in weak acid) results in alkalosis (an increase in HCO_3^-); conversely, an increase in phosphates, such as that occurring with acute renal failure, causes an acidosis.

In Stewart's approach, the primary disturbance is therefore defined as a change in one or more of the independent variables: SID, PCO_2, or A_{tot}. To calculate the contribution of these variables to an acid-base disturbance, determination of the total concentration of nonvolatile weak acids and the effective dissociation constant for weak acids is required, which is impractical in most clinical situations. A simplified version of Stewart's approach, proposed and validated for equine plasma, allows the determination of A_{tot} and the effective dissociation constant for plasma weak acids (K_a) and accurately predicts pH.[15]

Another approach to Stewart's concepts involves the characterization of four components of base excess: changes in free water reflected by changes in sodium, changes in chloride, changes in serum albumin concentration, and changes in unmeasured anions. This method has also been used successfully in horses to better characterize acid-base disorders.[22] The example in Table 3-4 illustrates the contributions of protein, chloride, and unmeasured anions to acid-base balance, and it shows how the traditional approach to acid-base balance can sometimes fail to recognize abnormalities when complex disorders are present.

The preceding discussion emphasizes the complexity of interactions between solutes in body fluids and the importance of recognizing the limits of the traditional approach to acid-base interpretation. Although the traditional approach provides a working method for identification of problems, it falls short in complex mixed acid-base disorders and does not provide a satisfactory explanation when electrolyte, colloidal, and unmeasured anion disorders coexist.

TABLE 3-4. Example of Acid-Base Measurements in a Horse with Intestinal Strangulating Obstruction

Parameter	Value
pH	7.49
PCO_2 (mm Hg)	37
Base excess (BE) (mEq/L)	4.6
Na^+ (mEq/L)	137
Cl^- (mEq/L)	93
HCO_3^- (mEq/L)	32.7
K^+ (mEq/L)	2.7
Total protein (g/dL)	4.4
Packed cell volume (%)	58
Anion gap (mEq/L)	25
BE_{fw} (mEq/L)	0.9
BE_{Cl} (mEq/L)	9
BE_{tp} (mEq/L)	7.5
BE_{ua} (mEq/L)	−12.8

From Whitehair KJ, Haskins SC, Whitehair JG, et al: J Vet Intern Med 9:1, 1995. Using the traditional approach to diagnosing acid-base disorders, the interpretation of blood gas analyses on this horse would indicate a metabolic alkalosis with no secondary or adaptive response (PCO_2 is normal). Further examination reveals hypochloremia and hypoproteinemia, which are responsible for the alkalosis (as indicated by the calculation of their respective base excesses [BE_{Cl} and BE_{tp}]). However, examination of the anion gap and calculation of the BE contributed by unmeasured anions (BE_{ua}) reveal an underlying acidosis that was masked by the hypoproteinemia and hypochloremia. Measurement of lactate would be indicated in this case to further characterize the disorder. Most likely, lactic acidosis is present as a result of poor perfusion (indicated by the marked increase in packed cell volume). BE_{Cl}, BE contributed by chloride; BE_{fw}, BE contributed by free water; BE_{tp}, BE contributed by total protein; BE_{ua}, BE contributed by unmeasured anions.

DESIGNING A FLUID THERAPY REGIMEN

Volumes of Fluid to Administer

Fluids can be administered for the purpose of maintenance or replacement. Maintenance regimens are often provided via the oral route in equine patients, and oral electrolyte formulations are available for this purpose (see later in this chapter). Intravenous maintenance fluids are lower in sodium and higher in calcium, potassium, and magnesium than replacement fluids. An appropriate maintenance fluid is 0.45% saline to which potassium, magnesium, and calcium were added.

More commonly, a replacement fluid therapy regimen is given to equine patients to replace fluids lost through dehydration and ongoing losses. When designing a replacement fluid therapy regimen, three questions must be answered:

1. What volume of fluid must be given?
2. What type of fluid will be given?
3. What will be the rate of administration?

Furthermore, the volume of fluids given must equal the maintenance requirements plus the volume needed to correct hypovolemia plus that needed to compensate for ongoing losses.

Maintenance

In adult horses, maintenance fluid requirements have been estimated at 60 mL/kg/day. This figure probably overestimates the actual needs of a resting, fasted animal in a normothermic environment, but it appears to be safe in most situations. In horses with renal failure, when elimination of excess fluids is difficult, monitoring of body weight and central venous pressures is indicated to help prevent fluid overload. If weight gain, edema, or increased central venous pressures are noted, the fluid rate should be decreased.

Dehydration

Dehydration is the general term used to indicate loss of total body water; hypovolemia is a form of dehydration resulting from loss of effective circulating volume. This distinction is important; for example, when the lack of water intake is prolonged, heart rate and parameters of perfusion remain within normal limits, as fluid shifts from the intracellular space to maintain normal circulating volume. In this case, intracellular

fluid volume should be replenished relatively slowly, to allow time for slow fluid shifts to occur. In contrast, acute intestinal obstruction results in loss of circulating blood volume manifested by altered cardiovascular parameters, such as increased heart rate, poor perfusion, and decreased pulse quality. Rapid reinstitution of effective circulating blood volume is important in this situation. Parameters that may be used for estimation of dehydration include serial body weights, heart rate, mucous membrane color, capillary refill time, skin elasticity (skin tenting), palpation of extremities, and urine output. Useful laboratory parameters include packed cell volume (PCV), total protein, creatinine and lactate concentrations, and urine specific gravity. Table 3-5 lists parameters useful for estimating hypovolemia (loss of effective circulating volume) in the horse.

Once an estimate of hypovolemia has been obtained, the amount of fluids to administer is calculated as follows:

$$\text{Correction of hypovolemia} = \text{Estimate of loss (\%)} \times \text{body weight (kg)}$$

Ongoing Losses

Ongoing losses can sometimes be measured and recorded—for example, when nasogastric reflux is present—but usually they must be estimated. Therefore patient monitoring is used to determine if the calculated fluid volume is meeting the ongoing losses. Monitoring, which may include serial measurements of cardiovascular parameters, PCV and total protein, lactate concentration, and blood gas analyses, is done at least twice a day when patients are on intravenous fluids, but it should be done more frequently (every 2, 4, or 6 hours) depending on the severity of cardiovascular compromise. Creatinine concentration should also be monitored at least once daily when initially elevated, to ensure adequate return to normal. Additional means of monitoring adequate fluid delivery include measurement of central venous pressure, arterial blood pressure, and urine output (see Chapter 1).

Type of Fluid

The type of fluid chosen depends on the evaluation of the chemistry profile and on the disease state. The first step is to choose a baseline fluid (saline or balanced electrolyte solution), and the second is to decide which additives to include in the baseline fluid. The choice of additives depends on the specific deficits or excesses, such as hypoglycemia or acid-base disorders, or when concentrations of sodium, potassium, calcium, or magnesium ions are too high or too low.

The two categories of crystalloids commonly used for fluid replacement are 0.9% saline and balanced electrolyte solutions (BESs). Table 3-6 lists the compositions of various commercially available fluids. In general, BESs are chosen when serum electrolytes are close to normal. The BES provides a bicarbonate precursor, which is either lactate, or acetate plus gluconate. Lactate requires hepatic metabolism for conversion to bicarbonate, whereas acetate and gluconate are metabolized by other tissues. All BESs contain some potassium. As noted in Table 3-6, calcium or magnesium is present in different types of BES. Saline (0.9%) is higher in sodium and much higher in chloride than serum concentrations and is used when $[Na^+]$ is lower than 125 mEq/L. Saline is also used in disease processes associated with high $[K^+]$, such as hyperkalemic periodic paralysis or renal failure, where a potassium-free solution is preferred. In cases of long-term maintenance fluid therapy (greater than 4 to 5 days), if the oral route is not available, half-strength basic fluids, to which potassium, calcium, and magnesium are added, should be considered. Long-term fluid therapy solely with a BES will result in hypernatremia, hypokalemia, hypomagnesemia, and hypocalcemia.

TABLE 3-5. Parameters to Estimate Degree of Hypovolemia in Adult Horses*

% Loss of Effective Circulating Volume	Heart Rate (beats/min)	Capillary Refill Time (sec)	PCV/TP (%, g/L)	Creatinine (mg/dL)
6%	40-60	2	40/7	1.5-2
8%	61-80	3	45/7.5	2-3
10%	81-100	4	50/8	3-4
12%	>100	>4	>50/>8	>4

*These parameters are useful to estimate the degree of hypovolemia in adult horses, assuming a normal packed cell volume (PCV) of 35% and total protein (TP) of 6.5 g/dL.

TABLE 3-6. Composition of Commonly Used Intravenous Solutions

Fluid	Na^+ (mEq/L)	K^+ (mEq/L)	Ca^{2+} (mEq/L)	Mg^{2+} (mEq/L)	Cl^- (mEq/L)	Buffer Source (mEq/L)	Osmolality (mOsm/L)
Plasma	132-146	2.8-5.1	9.0-13	1.8-3	99-110	(TCO_2) 20-36	285 ± 10
Lactated Ringer	130	4	3	0	109	(lactate) 28	274
Normosol-R	140	5	0	3	98	(acetate, gluconate) 50	295
0.9% NaCl	154	0	0	0	154		308
5% Dextrose	0	0	0	0	0		253
2.5% Dextrose in 0.45% NaCl	77	0	0	0	77		280
1.25% $NaHCO_3$	149	0	0	0	0	149	298

Adapted from Morris DD: Vet Med 34:164, 1987.

In horses, routine fluid replacement also includes calcium, potassium, and magnesium supplementation, particularly when there is no oral intake because of gastrointestinal disease. Low concentrations of serum ionized calcium (iCa) and magnesium (iMg) are more prevalent in horses with surgical gastrointestinal disease, particularly in those with small intestinal or large and small colon nonstrangulating infarction or strangulation and in horses with postoperative ileus.[23-25] Horses with enterocolitis also have low iCa and iMg and a decreased fractional clearance of calcium.[26] Total magnesium and calcium concentrations are less reliable for identification of calcium and magnesium status—it is preferable to determine ionized concentrations.[23-25] Measurement of total calcium can be misleading if total protein is low (ionized calcium may still be normal) or if the horse is alkalotic (total calcium may be normal, with a low ionized fraction). Fractional excretion of magnesium has been suggested as a diagnostic tool for assessment of magnesium status in horses.[27] Based on this information, supplemental calcium and magnesium appears beneficial for fluid therapy in horses.

Administration of 50 to 100 mL of 23% *calcium* gluconate in every 5 L of fluid is usually sufficient to maintain normocalcemia. In the presence of severe hypocalcemia (iCa less than 4.0 mg/dL), administration of 500 mL of calcium gluconate in 5 L of BES is indicated. Hypocalcemia that is refractory to calcium therapy may indicate hypomagnesemia, and concurrent magnesium replacement is required. The maintenance requirement of *magnesium* in horses is estimated at 13 mg/kg/day of elemental Mg, which is provided by 31 mg/kg/day of MgO, 64 mg/kg/day of $MgCO_3$, or 93 mg/kg/day of $MgSO_4$.[28] In critically ill patients, the requirement may be increased, as indicated by the high prevalence of hypomagnesemia in hospitalized patients.[25] When considering magnesium supplementation, the concentration of elemental magnesium in the compound should be considered. Some crystalloid fluids such as Plasma-Lyte A and Normosol-R contain 3 mEq/L of elemental Mg. This amount may be insufficient to account for the increased losses in sick horses. Administration of 150 mg/kg/day of $MgSO_4$ (0.3 mL/kg of a 50% solution), equivalent to 14.5 mg/kg/day or 1.22 mEq/kg/day of elemental magnesium, administered in saline, dextrose, or polyionic fluids, would provide the daily requirement for the horse.[28]

Hypokalemia may develop because of lack of intake, diuresis, and gastrointestinal loss through diarrhea. Horses with a metabolic acidosis can become hyperkalemic, and potassium excretion can occur after correction of the acidemia. Measurement of serum potassium as an estimate of total body potassium can be misleading, because most of the potassium ions are intracellular. Routine *potassium* supplementation is indicated if lack of intake and fluid therapy are continued for more than 24 hours. To prevent complications, it is recommended that animals not receive more potassium than 0.5 mEq/kg/hr. Most horses will benefit from the addition of 20 mEq of potassium in the form of potassium chloride per liter of fluids.

Bicarbonate supplementation may also be required in horses with metabolic acidosis. Because the most common cause of nonrespiratory acidosis is lactic acidemia resulting from poor perfusion, providing fluid replacement should be the first and principal means of correcting this problem. The following are rules of thumb for bicarbonate supplementation in acute metabolic acidosis:

- The horse should have normal respiratory function. If it is unable to exhale the generated CO_2 because of a respiratory problem, worsening of the acidosis will result.
- The blood pH should be less than 7.2. In acute acidosis associated with dehydration, fluid replacement will result in restoration of urine output, and renal compensation will follow and usually be complete if the pH is greater than 7.2.
- Half of the calculated amount should be administered rapidly, followed by the rest over 12 to 24 hours.
- IV bicarbonate should not be given with calcium-containing solutions.

The amount of bicarbonate required can be calculated using the following formula:

$$\text{mEq of bicarbonate} = \text{Base excess (mEq/L)} \times \text{BW (kg)} \times 0.3$$

Alternatively, total CO_2 (TCO_2) can be used, remembering tCO_2 represents bicarbonate content:

$$(\text{Normal } tCO_2 - \text{actual } tCO_2) \times \text{BW (kg)} \times 0.3$$

In chronic metabolic acidosis, particularly when there are ongoing losses of bicarbonate (e.g., with diarrhea), the full calculated amount is usually required, partially because the bicarbonate loss is distributed over all fluid compartments, not just the extracellular fluid. Oral supplementation is a good means of dealing with ongoing losses in horses with diarrhea. Bicarbonate is available as an injectable solution in two concentrations: a 5% solution, which contains 0.59 mEq/L of bicarbonate, and an 8.4% solution, which contains 1 mEq/L. To make an isotonic solution for intravenous administration, 1 part of 5% bicarbonate can be diluted in 3 parts of sterile water. Alternatively, 150 mL of 8.4% bicarbonate can be added to 850 mL of sterile water. Bicarbonate can be given orally as a powder (baking soda), where 1 g $NaHCO_3$ = 12 mEq HCO_3^-).

Administration of *dextrose* is indicated for the treatment of hypertonic dehydration, for animals that are susceptible to or that have hyperlipemia (miniature horses and donkeys, adult horses with azotemia), and for pregnant mares as a source of energy for the fetoplacental unit.[29,30] Because glucose is metabolized rapidly, administration of dextrose in water results in the administration of free water, which is useful for the correction of intracellular dehydration. As a source of energy, 5% dextrose can be administered at a rate of 1 to 2 mg/kg/min.

Administration of *colloids* is indicated when the total protein concentration is less than 4 g/dL, the albumin concentration is less than 2.0 g/dL, or the colloid oncotic pressure is less than 12 mm Hg. Plasma and hetastarch are commonly used colloids in horses (see Chapter 1). Plasma administration is indicated when administration of other plasma products such as coagulation factors or antithrombin is desired in addition to administration of colloids. The amount of plasma to be administered can be calculated as follows:

$$\text{Plasma to be administered (L)} = \frac{(TP_{des} - TP_{pt}) \times 0.05\text{BW (kg)}}{TP_{don}}$$

where TP_{des} is the desired protein concentration, TP_{pt} is the total protein concentration of the patient, TP_{don} is the total protein

concentration of the donor plasma and 0.05BW (kg) is an estimate of the plasma volume. If the goal of colloid therapy is to restore oncotic pressure, then synthetic colloids can be used. Before the advent of hetastarch, dextran was commonly used, but its administration was associated with more anaphylactic reactions, and because of its lower molecular weight average, it was effective for a shorter duration.[5] Hetastarch is preferred and is used at a dosage of 10 mL/kg. Higher dosages (20 mL/kg) were associated with increased coagulation times caused by a decrease in von Willebrand factor antigen (vWf:Ag) activity and factor VIII coagulant (FVIII:C), and should probably not be used in sick animals with increased susceptibility to coagulopathies.[9] Hetastarch registers at a lower value than protein on a refractometer and can therefore decrease the value of the total protein concentration that is measured. To accurately monitor hetastarch therapy, use of a colloid osmometer is indicated.

Administration of blood or blood substitutes (see Chapter 4) is indicated when loss of oxygen-carrying capacity has occurred through red blood cell loss. Ideally, fresh whole blood collected in a plastic container (to preserve platelet function) from a donor that is negative for the red blood cell antigens A and Q should be given. The blood should also contain an appropriate anticoagulant. Commercially available kits (Dynavet, Plasvacc USA Inc., Templeton, CA) consist of 2-L collection bags, collection and administration sets, and sodium citrate as an anticoagulant. This anticoagulant is not suitable for blood storage for longer than 24 hours. For longer storage, acid citrate dextrose (ACD) can be used. However, blood stored in ACD for greater than 10 days has a decreased concentration of 2,3-diphosphoglycerate, resulting in decreased oxygen release into tissues, increased red blood cell fragility, and increased potassium concentration. For prolonged storage of equine blood, the use of citrate phosphate dextrose with supplemental adenine is recommended.[31] In cases of chronic blood loss, the amount of blood required can be calculated as follows:

$$\text{Amount required (L)} = \frac{(\text{PCV}_{\text{des}} - \text{PCV}_{\text{pt}}) \times 0.08\text{BW (kg)}}{\text{PCV}_{\text{don}}}$$

where PCV_{des} is the target packed cell volume, PCV_{pt} is the patient's packed cell volume, PCV_{don} is the PCV of the donor, and BW is body weight. When the blood loss is acute, the packed cell volume does not reflect the amount of blood lost for up to 24 hours. If blood loss is considered severe, 10 to 20 mL/kg of whole blood can be administered. When a large volume of anticoagulated whole blood is administered, the patient should be monitored for anaphylactic reaction and hypocalcemia.

Hemoglobin glutamer-200 of bovine origin (Oxyglobin, Biopure Corp, Cambridge, MA) is a glutaraldehyde-polymerized bovine hemoglobin solution that has been administered safely to horses for restoration of oxygen-carrying capacity.[32-34] After administration, volume expansion also occurs because of the colloidal nature of the solution. In one study performed in ponies with experimentally induced normovolemic anemia, administration of 15 mL/kg given at the rate of 10 mL/kg/hr improved hemodynamics and oxygen transport parameters without adverse renal or coagulation effects; however, one pony suffered an anaphylactoid reaction during infusion.[32] The half-life of Oxyglobin is relatively short; therefore, the patient should be monitored if the need for another transfusion may arise.[31] Expense may limit its use in adult horses.

Rate of Administration

In severe shock, a shock dose of fluids (60 to 90 mL/kg or 30-45 L per 500 kg horse) should be administered in the first hour. This can be done only with pressurized bags or a pump. In other situations, the rate of administration is calculated on the basis of 24-hour requirements and estimated as a volume per hour. It is important to keep a tally of the fluids given to ensure that the correct amount is reached.

Oral Fluids

Although the oral route of fluid administration has been neglected with the advent of commercially available intravenous fluids for horses, interest is being revived, particularly in the treatment of impaction colic. Oral fluids should be considered when the gastrointestinal tract is functional and maintenance requirements are needed, for example, in a dysphagic horse. Oral fluids also may be the principal treatment of impaction colic. Enteral fluid therapy may complement and even supplement intravenous fluids. Advantages of enteral fluid therapy include administration of fluid directly into the gastrointestinal tract, stimulation of colonic motility through the gastrocolic reflex, decreased expense, and decreased need for precise adjustment of fluid composition.[35] Enteral fluids may be administered by intermittent nasogastric intubation or by placement of an indwelling feeding tube (18-French equine enteral feeding tube), allowing continuous fluid administration.

An isotonic electrolyte solution can be made by mixing 5.27 g of NaCl, 0.37 g of KCl, and 3.78 g of $NaHCO_3$ per liter of tap water.[36] This solution results in the following electrolyte concentrations: 135 mEq/L of Na^+, 95 mEq/L of Cl^-, 5 mEq/L of K^+, and 45 mEq/L of HCO_3^-, with a measured osmolarity of approximately 255 mOsm/L, representing a balanced, slightly hypotonic electrolyte solution compared with plasma. Plasma electrolyte concentrations remain within normal range with this solution compared with the marked hypernatremia and hyperchloremia observed when 0.9% saline is administered enterally.[35]

Although normal horses can tolerate up to 10 L hourly, it is usually not possible to administer more than 5 L every 2 hours to horses with impactions, because they start to reflux when more fluid is given.[37] As a consequence, intermittent intubation allows administration of approximately 60 L of fluids per day. When continuous enteral fluids are given, a greater rate of administration is tolerated, and horses can be given between 4 and 10 L/hr. At the higher rate of 10 L/hr, mild signs of abdominal pain were observed in normal horses, and in horses with large colon impaction, a rate of 5 L/hr is better tolerated.[36] In one study, right dorsal colon ingesta hydration was significantly increased after enteral fluid therapy compared with intravenous fluid therapy combined with enteral administration of magnesium sulfate.[38]

FLUIDS USED FOR RESUSCITATION

Isotonic Crystalloids

Isotonic crystalloid fluids are administered intravenously and immediately reconstitute the circulating volume. However, because they are crystalloids, they are distributed to the entire extracellular compartment within a matter of minutes. Because the ECF compartment is approximately 3 times the volume of

blood, three times as much isotonic crystalloid must be administered to gain the desired amount of circulating volume expansion. As an example, if blood loss is estimated at 30% of blood volume, representing 12 L for a 500-kg horse, then 36 L of a crystalloid fluid is required. An estimated shock dose for crystalloids is therefore 60 to 90 mL/kg/hr.

Hypertonic Crystalloids (7.2% NaCl)

Hypertonic crystalloid fluids (7.2% NaCl) have approximately 8 times the tonicity of plasma and ECF (composition: Na^+, 1200 mOsm/L; Cl^-, 1200 mOsm/L). Their immediate effect is to expand the vascular volume by redistribution of fluid from the interstitial and intracellular spaces. Each liter of hypertonic saline will expand blood volume by approximately 4.5 L. However, this effect is short-lived. As the electrolytes redistribute across the ECF, fluids shift back and the patient once again becomes hypovolemic. Because the principal effect of hypertonic saline is fluid redistribution, there still exists a total body deficit, which must be replaced. The duration of effect of hypertonic solutions is directly proportional to the distribution constant, which is the indexed cardiac output. In horses, the duration of effect is estimated at approximately 45 minutes. The recommended dosage is 4 mL/kg or 2 L per 500 kg horse, administered as rapidly as possible. Because of its short duration of effect, hypertonic saline administration must be followed with isotonic volume replacement at shock doses (see earlier).

Colloids

Colloids are fluids that contain a molecule that can exert oncotic pressure. These molecules do redistribute to the ECF, but at a much slower rate than crystalloids, so the effect is prolonged compared with crystalloids. Hetastarch, because of its long duration of effect, is the most commonly used fluid for volume expansion in horses. Each liter of administered colloid will further expand the circulating blood volume by approximately 1 L, resulting in a total fluid expansion of 2 L. If hetastarch is used at a dosage of 10 mL/kg or 5 L per 500 kg horse, the resulting increased colloid pressure will be significant for up to 120 hours in horses.[9] For shock therapy, the combination of hypertonic saline at 4 mL/kg and hetastarch at 4 mL/kg will prolong the resuscitation efforts and be more beneficial than either fluid alone.[39,40]

MATERIALS FOR FLUID ADMINISTRATION

Intravenous Catheters

Intravenous catheters are available in varying materials, constructs, lengths, and diameters (Tables 3-7 and 3-8). In choosing a catheter, the desired fluid rate, the fluid viscosity, the length of time the catheter will remain in the vein, the severity of the systemic illness, and the size of the animal should be considered. The rate of fluid flow is proportional to the diameter of the catheter and inversely proportional to the length of the catheter and the viscosity of the fluid. Standard adult horse catheter sizes are usually 14 gauge in diameter and 13 cm (5.25 inches) in length. For more rapid administration rates (shock), a 12- or 10-gauge catheter should be used. Plasma and blood products flow more slowly because of their increased viscosity, so if volume replacement is also needed, administration of these fluids can be combined with a BES. Teflon catheters should be changed every 3 days, whereas polyurethane catheters may remain in the vein for up to 2 weeks. Horses that are very ill (bacteremic, septicemic, endotoxic) are more likely to encounter catheter problems and benefit from polyurethane or silicone catheters.

The catheter construction needs also to be considered (see Table 3-8). Through-the-needle catheters are most common for standard size adult horses. An over-the-wire catheter is best for foals and miniature horses or when the lateral thoracic vein is

TABLE 3-7. Commercially Available Catheter Materials

Material	Comment
Polypropylene, polyethylene tubing	Highly thrombogenic
Teflon	Less thrombogenic
Polyurethane	Much less thrombogenic
Silastic	Least thrombogenic

TABLE 3-8. Catheter Constructs Commercially Available

Type	Description	Advantage	Disadvantage
Butterfly	Needle is attached to tubing	Ease of use	Laceration of vessel Vessel puncture Extravascular administration
Over-the-needle	Stylet is inside catheter for venipuncture	Available in large diameter	Limited length of catheter Insertion more difficult Break at junction of catheter and hub
Through-the-needle	Short needle is inserted, catheter is threaded through needle	All lengths available	Trocar must be removed or protected
Over-the-wire	Needle serves as guide to insert wire, which is the guide for catheter	Trocar is removed after catheter insertion Long catheters available Ensures proper catheter placement	More technical expertise required Expensive

catheterized. Short and long extension sets are available, as well as small- and large-bore diameters. It is best to use an extension that screws into the hub of the catheter, to prevent dislodgement. In horses with low central venous pressures, disconnection of the line may result in significant aspiration of air and cardiovascular collapse. Double extensions are also available when other medications need to be administered with the fluids.

Sites for Intravenous Catheterization in Horses

Common sites for insertion of intravenous catheters in horses include the jugular, lateral thoracic, cephalic, and saphenous veins. The lateral thoracic vein makes an acute angle as it enters the chest at the fifth intercostal space. Therefore a short (7.5-cm [3-inch]) or an over-the-wire catheter is best used when catheterizing this vein. When catheters are placed in any location other than the jugular vein, more frequent flushings (every 4 hours) are required, because these catheters tend to clot more easily. Limb catheters are usually bandaged, because they are more prone to dislodgment than jugular catheters.

Catheter Maintenance

In adult horses, catheters usually are not covered with a bandage but rather are sutured in place, so that any problem is quickly identified. Bandages may need to be applied in foals if they are tampering with the catheter. A triple antibiotic ointment or antiseptic skin sealant, an iodine/alcohol disinfectant solution (DuraPrep, 3M US, St. Paul, MN) (see Chapter 10), may be applied at the insertion site on the skin to decrease the risk of infection. Catheters should be flushed with heparinized saline (10 IU/mL) four times a day if they are not used for fluid administration. When administering a medication, the injection cap should be wiped with alcohol before insertion of the needle. The injection cap should be changed daily. All infected catheters should be cultured for identification of the causative organism and for possible nosocomial infection. In addition, culturing noninfected catheters at removal is a good practice in preventing hospital-wide nosocomial events.

Coil Sets and Administration Sets

Coil sets are used for in-stall fluid administration. They are essential as they allow the horse to move around, lie down, and eat without restraint. An overhead pulley system with a rotating hook prevents fluid lines from getting tangled.

Administration sets are used for short-term fluid or drug administration and are available at 10 drops/mL and 60 drops/mL. When using a calibrated fluid pump, care should be taken to use the appropriate set calibrated for the brand of pump. Long coiled extension sets may then be used to introduce fluids into the horse. Foal coil sets (18-French equine enteral feeding tube) are also available that deliver 15 drops/mL.

Pump Delivery

Calibrated pumps are available that allow delivery at various rates. These pumps have alarms that signal when air is in the line, fluid bags are empty, or there are problems with the catheter. The maximum fluid rate these pumps can deliver is 999 mL/hour, which is usually not rapid enough to provide fluid replacement in adult horses, but they are useful for foals or for constant-rate infusions. For large-volume fluid delivery, peristaltic pumps are available that can deliver up to 40 L/hr. These must be constantly supervised, because the pumps will continue to run even if fluids run out. Large-bore catheters should be used to prevent trauma from the jet effect on the endothelium of the vein.

Oral Feeding Tubes

Oral fluid administration offers a good alternative to intravenous fluid therapy in animals that require maintenance fluids because of an inability to swallow, or in horses with impaction colic. Enteral nutrition (see Chapter 6) can also be administered for complete or partial nutrition in foals and adults. Commercially available feeding tubes for foals, weanlings, and adults enable fluid or liquid diet supplementation while the horse continues to nurse or eat.

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CHAPTER
4

Hemostasis, Surgical Bleeding, and Transfusion

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PHYSIOLOGY OF HEMOSTASIS

Physiologic hemostasis is required for the control of bleeding related to surgery and trauma. A delicate balance of procoagulant, anticoagulant, fibrinolytic, and antifibrinolytic activities is required for effective control of bleeding without pathologic thrombosis. Over the last two decades, our understanding of physiologic hemostasis has evolved to include the pivotal role of the cells, rather than just the coagulation factors. It is still useful to understand the more simplistic cascade model of coagulation, because this is the basis for many coagulation tests. The surgeon should be familiar with predisposing factors for bleeding and coagulopathy as well as management of the bleeding patient, including blood transfusion and topical hemostatic agents.

Blood Vessels and the Role of the Vascular Endothelium

The vascular endothelium is critical in preventing inapproriate clot formation. Healthy, intact endothelium has antiplatelet, anticoagulant, and fibrinolytic properties. Anticoagulation and fibrinolysis are discussed in further detail later in this chapter, but an initial understanding of the role of the endothelium is needed to understand how coagulation events are set in motion after vessel trauma.

The synthesis of prostacyclin (PGI_2) and nitric oxide (NO) is largely responsible for the antiplatelet properties of the endothelium. Both of these substances inhibit platelet aggregation, and NO also inhibits platelet adhesion.[1] The vasodilation induced by NO also helps to prevent clot formation by promoting low-turbulence blood flow. Platelet aggregation and adhesion are also prevented by enzymes on the endothelial surface that degrade adenosine diphosphate (ADP).

The electronegative charges on endothelium and platelets physically prevent adhesion. Additionally, endogenous heparin-like substances are present on the endothelial surface, contributing substantially to anticoagulation. Glycosaminoglycans act as cofactors for antithrombin, which inactivates thrombin and coagulation factors VIIa, IXa, Xa, and XIa. Endothelial cells also express thrombomodulin, tissue plasminogen activator, and tissue factor pathway inhibitor, contributing further to anticoagulation and fibrinolysis.

The immediate response of the blood vessel to injury is vasoconstriction. This is mediated through local signaling from damaged endothelial cells, perhaps through interruption of the release of endothelial-derived relaxation factors. Prompt vasoconstriction prevents unnecessary blood loss and promotes rapid fibrin formation. Alternatively, inappropriate or excessive activation of these procoagulant properties may play a role in the hemodynamic dysfunction and end-organ failure often

observed in severe endotoxemia or sepsis.[2] The endothelium is metabolically active and able to respond to changes in environment, including hypoxia, shear stress, pH, and trauma. When vessel injury occurs, endothelial cells can express tissue factor (TF) and downregulate expression of thrombomodulin, becoming procoagulant. Activated endothelial cells release von Willebrand factor (vWF) from the Weibel-Palade bodies, promoting platelet adhesion. Local vasoconstriction is a crucial component of primary hemostasis, along with platelet activation, adhesion, and aggregation, all leading to formation of a temporary platelet plug.

Platelets and Primary Hemostasis

The interaction of activated platelets with the exposed subendothelium of blood vessels is the basis of primary hemostasis. Platelets also play a key role in secondary hemostasis: once activated, they undergo conformational changes, exposing binding sites for specific coagulation factors.

Platelets are derived from the cytoplasm of bone marrow megakaryocytes. They contain dense granules, α-granules and lysosomes, which store the majority of platelet proteins needed for the initiation of coagulation. The α-granules are the largest and most prevalent storage granules, comprising the majority of the storage capacity of platelets. They contain a number of proteins involved in platelet aggregation and cohesion, including fibrinogen, factor V (FV), factor VIII (FVIII), fibronectin, vWF, platelet-derived growth factor (PDGF), and platelet factor 4. Dense granules store calcium, a common cofactor in platelet–phospholipid interactions, as well as ADP, adenosine triphosphate (ATP), and serotonin. Thrombin is the strongest stimulant for the release of the contents of the dense granules, but other agonists for release have also been reported. Platelet lysosomes contain predominantly acid hydrolases, responsible for degradation of unwanted cellular debris after complete activation of fibrin formation.[3]

The platelet is the initial responder to vascular damage and subsequent endothelial exposure. Platelet adhesion is mediated by expression of P-selectin on the activated endothelium and by the platelet receptor GPIbα, which attaches to vWF. Once attached to the endothelium, platelets rapidly change shape and provide an effective monolayer in what is known as the adhesion phase. This results in a primary platelet plug (primary hemostasis) that is responsible for preventing leakage of blood from the minute vessel defects that occur daily. If blood flow in this area remains nonturbulent, further platelet aggregation does not occur, and the monolayer generally suffices to plug the small defects or the area of vascular attenuation.[3]

With large vessel disruption, blood flow becomes quite turbulent, resulting in large platelet aggregates coating the exposed endothelium. Activation of platelets results in degranualation of platelet contents, releasing agonists. Thrombin, collagen, ADP, and thromboxane A_2 promote platelet activation. After the platelet plug bridges the gap between endothelial cells, prostacyclin, produced by neighboring healthy endothelial cells, prevents unwanted expansion of platelet aggregates by decreasing further ADP release. The activated platelet serves as a congregation site for the coagulation factors via the integrin $\alpha_{IIb}\beta_3$ receptor (see "Secondary Hemostasis and Models of Coagulation").

Secondary Hemostasis and Models of Coagulation

Secondary hemostasis involves the activation of soluble coagulation factors, ultimately resulting in formation of a stable fibrin clot. The traditional cascade model divides coagulation into intrinsic, extrinsic, and common pathways. These pathways are useful when interpreting *in vitro* plasma-based coagulation tests. The more recently described cell-based model of coagulation demonstrates that these traditional pathways are quite interconnected and are dependent on cell signals and receptors.

Coagulation Cascade

The coagulation cascade is the traditional model that describes the process of coagulation. This model is centered around the coagulation factors and is an excellent model for *in vitro,* plasma-based coagulation. The *intrinsic pathway,* or "contact activation" pathway, is initiated by the activation of factor XII (FXII) and subsequently factor XI through the exposure of blood to a negatively charged surface (Figure 4-1). Contact proteins such as high-molecular-weight kininogen (HMWK) and prekallikrein interact with FXII to acclerate its activation. Factor XIa (activated factor XI) in turn activates factor IX in the

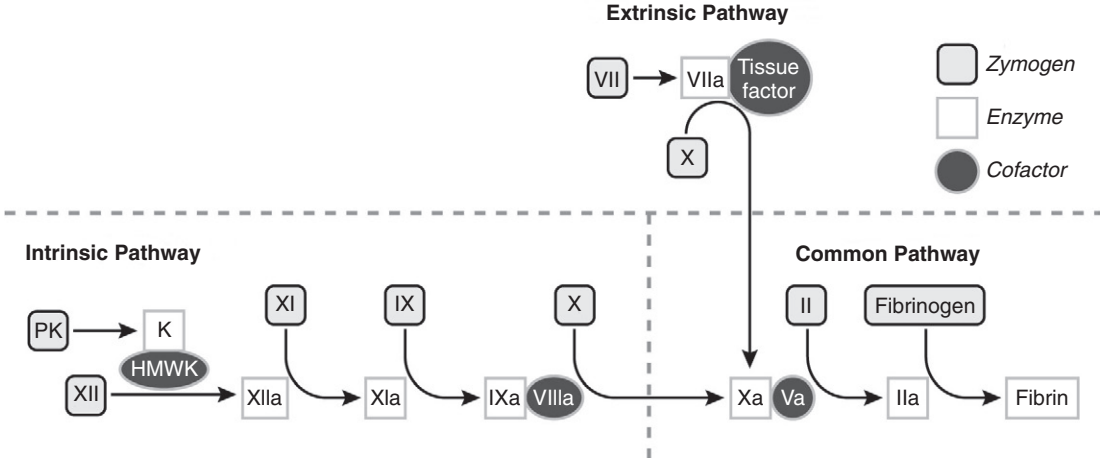

Figure 4-1. The traditional coagulation cascade: intrinsic, extrinsic, and common pathways. Roman numerals indicate factors. *HMWK,* High-molecular-weight kininogen; *PK,* prekallikrein.

presence of calcium. Factor IXa then binds to procoagulant VIIIa in the presence of calcium. It is this complex that activates the common coagulation pathway, marked by the activation of factor X.

The *extrinsic pathway* is initiated by the activation of factor VII by TF present in fibroblasts or other tissue factor–bearing cells. The TF-FVIIa complex activates factor X, leading into the common pathway. The common pathway is initiated by the activation of factor X, which, in the presence of activated factor V (Va), calcium, and a platelet phospholipid, converts prothrombin (factor II) to thrombin (IIa). In the final step of clot formation, factor IIa converts fibrinogen to fibrin. Factor XIIIa stabilizes the fibrin clot by cross-linking strands of fibrin monomer in the presence of calcium.

Cell-Based Model

Physiologic hemostasis occurs in three overlapping phases: initiation, amplification, and propagation.[4,5] The intrinsic and extrinsic coagulation pathways are still incorporated in this model, but the pathways are shown to be highly interconnected (Figure 4-2).

INITIATION

When there is disruption of the endothelium, tissue factor–bearing cells such as fibroblasts are exposed to blood, and coagulation is initiated. TF is the primary initiator of coagulation, and the first steps of coagulation are limited to the cell membrane. Under pathologic (inflammatory) conditions, TF can be upregulated on endothelium, monocytes, and other cells and cell particles. Factor VII circulates in plasma and is available to bind to TF, leading to activated FVII. The TF-FVIIa complex then activates factor X and factor IX. Although FXa in plasma is readily inactivated, the membrane-bound FXa can combine with FVa to produce small amounts of thrombin.

AMPLIFICATION

Once a small amount of thrombin is formed during initiation, the coagulation process can move to the platelet surface. The adherence, activation, and aggregation of platelets, along with the accumulation of activated cofactors, constitute the amplification of coagulation. Some platelets have already adhered to the site of injury, but thrombin fully activates platelets via protease-activated receptors. Factor V is present in the α-granules of the platelet, and during platelet activation, FV moves to the surface of the platelet. FV is then fully activated by thrombin and FXa. Thrombin cleaves vWF/FVIII, allowing vWF to stimulate platelet adhesion. FVIII is bound to the platelet surface and is available to continue the propagation phase of coagulation. FXI is also activated by thrombin on the platelet surface.

PROPAGATION

Coagulation complexes assemble on the activated platelet surface and the resulting generation of large amounts of thrombin leads to the propagation of the coagulation process. FIXa is able to reach the platelet surface via diffusion, since it is not inactivated by antithrombin (AT) and other plasma protease inhibitors. FIX is also activated on the platelet surface by FXIa. FIXa and FVIIIa combine as the tenase complex on the platelet surface, and subsequently activate FX on the platelet surface. FXa and FV combine to form the prothrombinase complex, which produces a thrombin burst.

Fibrinolysis

Simultaneous activation of the fibrinolytic system occurs with activation of coagulation. This is the primary mechanism of clot dissolution and is responsible for prevention of excessive fibrin deposition and restoration of nutrient blood flow to affected tissues. Fibrinolysis, in conjunction with prostacyclin released by surrounding healthy endothelial cells, inhibits unwanted expansion of the fibrin clot.

Plasminogen, an inactive zymogen produced primarily in the kidney and liver, is the principal component of the fibrinolytic system. Plasminogen activators such as tissue plasminogen activator (tPA) and urokinase plasminogen activator (uPA) convert plasminogen to plasmin. Plasmin degrades fibrinogen and fibrin into soluble fibrin(ogen) degradation products (FDPs).

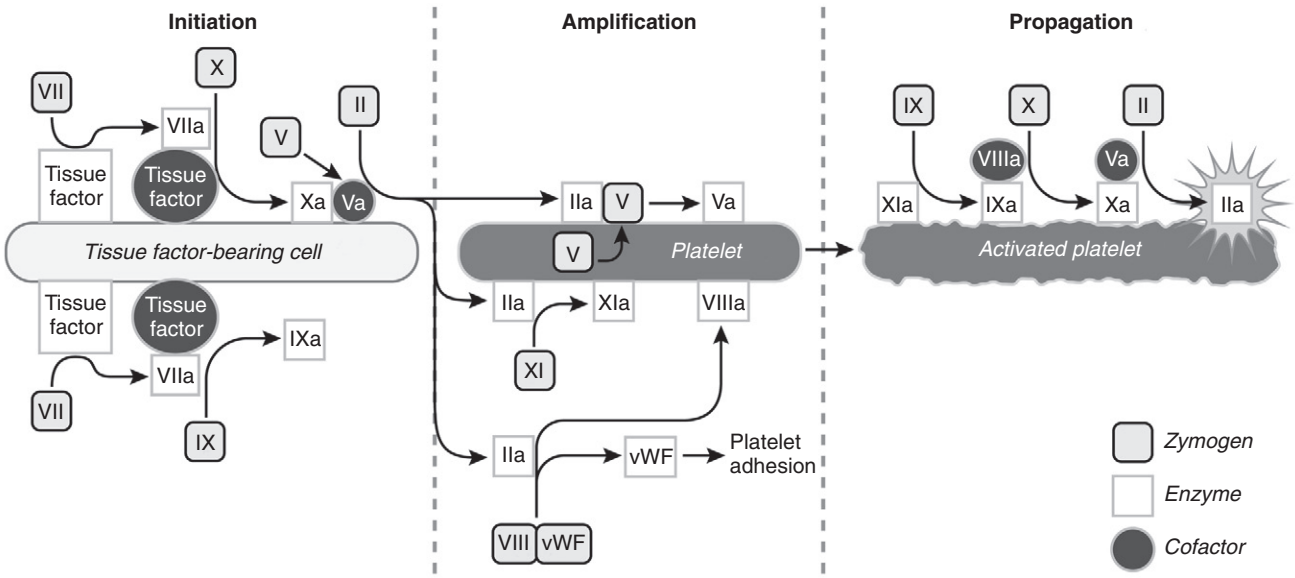

Figure 4-2. The cell-based model of coagulation: initiation, amplification, and propagation. Roman numerals indicate factors. *vWF,* von Willibrand factor.

The activation of the intrinsic pathway also activates plasminogen conversion to plasmin, through the action of kallikrein. Plasmin also inactivates other members of the coagulation cascade, such as factors Va and VIIIa, and actively degrades prekallikrein and HMWK. Through these mechanisms, plasmin not only degrades fibrin(ogen) but also downregulates coagulation.

The products of fibrinogen or fibrin degradation are the FDPs designated fragment X, fragment Y, and fragments D and E.[6] Plasmin degradation of cross-linked fibrin results in the D-dimer fibrin degradation product. These fragments are removed by the mononuclear phagocytic system of the liver, and accumulation of these fragments indicates increased fibrin production (and degradation) or liver dysfunction. During the maintenance of physiologic hemostasis, a critical balance between fibrin formation and degradation exists. Proper functioning of the fibrinolytic system controls unwanted clot expansion, prevents premature fibrin lysis, and provides appropriately timed restoration of nutrient blood flow to tissues. Increased levels of FDPs, D-dimers, or soluble fibrin monomer in the circulation lead to increased fibrinolysis. This can be interpreted either as being the result of a thrombogenic disease process, or as the patient being in a hypercoagulable state.

Inhibitors of Coagulation and Fibrinolysis

Inibitors of Coagulation

Inhibitors of coagulation are composed of a family of proteins that enzymatically bind with coagulation factors to form inactive complexes. In some instances, coagulation cofactors or surface receptors are destroyed to downregulate clot formation. The principal inhibitors of coagulation are antithrombin, heparin, protein C, protein S, and tissue factor pathway inhibitor (TFPI) (Table 4-1).

AT is responsible for 70% to 80% of thrombin inhibition in the coagulation system. It is the key player in a family of serine protease inhibitors responsible for modulation of clot formation. Antithrombin is a glycoprotein produced in the liver and in endothelial cells that binds aggressively to thrombin. A stable thrombin-antithrombin (TAT) complex is the result of this reaction, and this complex is removed by the reticuloendothelial system. The cofactor heparin alters the arginine site of AT and dramatically increases its ability to interact with thrombin. AT is also capable of neutralizing factors XIIa, XIa, Xa, and IXa. The AT–heparin complex also slowly inactivates factor VIIa.[7] The horse appears to have higher concentrations of AT than some other species, such as dogs and humans.[8]

Heparin is a highly sulfated glycosaminoglycan, ranging in molecular weight from 3 to 30 kDa. It is produced primarily in mast cells located in the lung, liver, kidney, heart, and gastrointestinal tract. Heparin causes a conformational change in AT, which increases the activity of AT 1000-fold.[7] Its presence in an area of coagulation activation decreases thrombin-generated fibrin formation significantly. Heparin also releases TFPI from endothelial cells, thereby liberating one of the most effective inhibitors of the factor VIIa-TF complex.

The thrombomodulin–protein C–protein S pathway has received a lot of attention in recent years. Protein C is a vitamin K–dependent zymogen with primary inhibitory action on factors Va and VIIIa. Protein C is activated by thrombomodulin-thrombin complexes. This reaction is potentiated by the endothelial protein C receptor, which is located mainly in large vessels. When activated protein C is released into circulation, it associates with protein S and is able to inactivate factors Va and VIIa. Activated protein C is also profibrinolytic, since it inhibits plasminogen activator inhibitor-1 (PAI-1) and indirectly inhibits thrombin-activatable fibrinolysis inhibitor (TAFI) as a result of thrombin inhibition.

TFPI is a group of lipoprotein-bound proteins produced primarily by platelets and endothelial cells. Heparin enhances the release of TFPI into the circulation. In the presence of calcium, TFPI inhibits factor VIIa-TF activation of factor X, thereby dramatically decreasing the primary cellular initiator of coagulation.

TABLE 4-1. Anticoagulation Factors and Their Inflammatory Effects

Factor Name	Action	Changes Associated with Inflammation
Antithrombin	Anticoagulant Inhibits factors VIIa, IXa, Xa, XIa, XIIa	Decreases
Protein C	Anticoagulant Inhibits factors Va, VIIa Decreases fibrinolysis	Decreases
TFPI	Anticoagulant Inhibit factors Xa and TF-VIIa complex	Variable
PAI-1	Antifibrinolytic Inhibits plasminogen	Increases
TAFI	Antifibrinolytic Reduces conversion of plasminogen to plasmin	Increases

PAI-1, Plasminogen activator inhibitor-1; *TAFI*, thrombin-activatable fibrinolysis inhibitor; *TFPI*, tissue factor pathway inhibitor.

Inhibitors of Fibrinolysis

PAI is the principal regulator of plasminogen through inhibitory effects on tPA (see Table 4-1). PAI is present in endothelial cells and is stored in α-granules of platelets.[9] The main physiologic inhibitor of plasmin is α-2-antiplasmin. An alternative inhibitor of plasmin, α-2-macroglobulin, may inhibit plasmin in a limited fashion, particularly if α-2-antiplasmin is overwhelmed. Prevention of premature fibrinolysis and clot dissolution is mediated principally through these inhibitors of plasminogen and plasmin. Another inhibitor of fibrinolysis, TAFI, is activated by thrombin, the thrombin-thrombomodulin complex, and plasmin. As a negative-feedback mechanism, plasmin can also activate TAFI.

Coagulation Testing

Screening tests consist of assays of primary and secondary hemostasis. Coagulation inhibitors and fibinolytic pathway inhibitors can also be assayed, including AT, FDPs, and D-dimer. Many point-of-care tests are available, and automated blood coagulation analyzers can perform a variety of coagulation tests, including activated partial thromboplastin time (APTT), prothrombin time (PT), fibrinogen, and AT testing (see later).

Tests of Primary Hemostasis

Defects in primary hemostasis are suspected with clinical signs of mucosal bleeding, petechiation, ecchymoses, and epistaxis. The platelet count is the first step in the evaluation of primary hemostasis. Horses tend to have lower platelet counts than other species, typically in the 150,000 to 250,000/μL range. A platelet count of less than 100,000/μL is considered abnormal, although clinical bleeding may not be seen until the platelet count is below 30,000/μL.

Platelet function tests should be performed when there are clinical signs of thrombocytopenia with a normal to increased platelet count. Template bleeding time (TBT) can be performed on the buccal mucosa or on the caudolateral aspect of the forelimb. TBT will be prolonged with thrombocytopenia, thrombocytopathia, and lack of vWF, and it may also be prolonged in cases of vasculitis. Unfortunately, TBT has been shown to have poor reproducibility and a very wide reference range in horses.[10] Additional platelet function tests include platelet aggregation studies and platelet function analysis (PFA-100, Siemens, Deerfield, IL). The PFA-100 has been validated in the horse.[11]

Prothrombin Time

PT measures the function of the extrinsic and common coagulation pathways. Platelet-poor plasma is mixed with thromboplastin and calcium, and time to clot formation is measured. Deficiencies in FV, FVII, FX, prothrombin, and fibrinogen can result in prolonged PT. Typically, an increase in time by 20% indicates an abnormal test result. In human patients, PT becomes prolonged when fibrinogen is less than 100 mg/dL, prothrombin is less than 30% of its normal plasma concentration, or factors VII, V, and X are decreased to 50% of their normal concentrations.[12]

Activated Partial Thromboplastin Time

APTT measures the function of the intrinsic and common coagulation pathways. The test is performed by adding an activating agent to platelet-poor plasma in a glass tube containing phospholipid emulsion and calcium. Deficiencies of FXII, FXI, FX, FIX, FVIII, FV, prothrombin, and fibrinogen can result in prolonged APTT. FXII, HMWK, or prekallikrein deficiencies can prolong APTT but are not associated with bleeding tendencies in humans.[13] As with PT, an increase in time by 20% is usually considered abnormal.

Both PT and APTT serve as variables to evaluate the coagulation cascade portion of the hemostatic system. Although PT and APTT are certainly useful indications of significant problems with the coagulation cascade, they may not be sensitive enough to adequately identify early stages of hypercoagulability or DIC. Prolonged PT or APTT may be associated with body cavity bleeding, significant hematuria, or hematochezia. Normal reference ranges for PT and APTT should be established for individual laboratories, and separate reference ranges for neonatal foals should be determined.

Activated Clotting Time

Activated clotting time (ACT) measures the time required for whole blood to clot after contact with diatomaceous earth, simulating the intrinsic and common coagulation pathways. Blood is collected directly into a tube containing the diatomaceous earth and is incubated at 37° C. The ACT will be prolonged with deficiencies of FVIII, FIX, prothrombin, and fibrinogen. ACT has the advantage of being a rapid, patient-side test; however, it is less sensitive than APTT for coagulation factor deficiencies.

Anticoagulant Testing

AT is the most commonly measured anticoagulant. It is measured by chromogenic assay in an automated analyzer, and results are reported as a percentage of activity. A decrease in AT levels may occur through consumption via increased thrombin formation; through protein loss, such as nephropathies or enteropathies; or via failure of adequate production. Protein C can also be measured with a chromogenic assay. Decreased AT and protein C levels are associated with hypercoagulability. AT is an acute phase reactant, so AT levels may be increased with some acute inflammatory conditions.

Thrombin-antithrombin (TAT) is an irreversible inactive complex between thrombin and antithrombin. TAT levels can be measured using a sandwich enzyme-linked immunosorbent assay (Enzygnost), which has been evaluated and validated for use in the horse.[14] Activation of coagulation and the procoagulant state result in elevated plasma levels of TAT. In human patients, TAT is elevated in states of disseminated intravascular coagulation (DIC) and sepsis.[15]

Fibrin(ogen) Degradation Products

FDPs are produced by the proteolytic degradation of fibrin(ogen) by plasmin. They are routinely cleared by the mononuclear phagocytic system (MPS), and an accumulation of FDPs indicates a failure of the MPS to adequately remove them from the circulation. This can be the result of local or systemic hyperfibrinolysis, and it may be indicative of a dramatic increase in clot formation. FDP evaluation is usually performed as a semiquantitative test, resulting in the following possible ranges for FDPs: 0 to 10 μg/mL, 10 to 20 μg/mL, 20 to 40 μg/mL, or greater than 40 μg/mL, with FDPs greater than 10 μg/mL considered abnormal. An evaluation of FDP assays in horses with severe colic demonstrated that FDP assays had a very low sensitivity and were not useful for the diagnosis of DIC in this patient population.[16]

Fibrinogen

The measurement of fibrinogen as part of a standard coagulation profile is an attempt to document hypofibrinogenemia, which is a somewhat consistent feature of overt DIC in humans. Fibrinogen can be measured by the heat precipitation method, von Clauss technique, or automated photometric detection. It is not unusual for human patients with significant hemostatic dysfunction to develop a fibrinogen level of less than 100 mg/dL. This does not seem to be a consistent feature of DIC in the horse, however, since fibrinogen increases with inflammatory conditions.[17,18] Horses with DIC do not consistently demonstrate true hypofibrinogenemia, but they do have lower fibrinogen concentration than would be expected for horses with inflammatory conditions.[19]

D-Dimer

D-dimer is an epitope resulting from the plasmin degradation of fibrin. It is a cross-linked dimer of the two smallest fibrin degradation products, fragment D-D. The D-dimer assay is specific for plasmin degradation of fibrin, as opposed to FDPs, which indicate degradation of either fibrin or fibrinogen. D-dimer can be measured semiquantitatively by latex agglutination or by latex-enhanced turbidimetric immunoassay performed on a standard coagulation analyzer.[16] Increased D-dimer levels indicate increased fibrinolysis or inability to clear the products from the circulation. In critically ill human patients, D-dimer has been used to better characterize acute pulmonary thromboembolism and to diagnose deep vein thrombosis. D-dimer can be increased in horses as a physiologic response to the primary disease or surgical procedure or as a pathologic coagulopathy.

Viscoelastic Monitoring

Viscoelastic analyzers may hold some promise for evaluation of coagulation in the veterinary surgical patient. Thromboelastography (TEG), rotational thromboelastometry (ROTEM), and the Sonoclot analyzer are three currently available analyzers that use viscosity, elasticity, or both to evaluate clot formation in whole or citrated blood samples. These analyzers evaluate all phases of clot formation and retraction from a single small volume (i.e., 330 µL) of blood. A tracing or signature is provided from which values can be derived to assess platelet or coagulation function. Software is provided with each analyzer, resulting in a user-friendly interface and easy storage of data.

In human surgical patients, viscoelastic analyzers are most commonly used as point-of-care testing to monitor coagulation inhibition during cardiopulmonary bypass procedures and liver transplantations and to evaluate perioperative hemorrhage. TEG has been used to identify hypercoagulable states in dogs with parvoviral enteritis, neoplasia, and immune-mediated hemolytic anemia.[20-22] Normal values have been reported for adult horse TEG and neonatal foal Sonoclot, and viscoelastic testing has been used in populations of septic foals and adult horses with gastrointestinal disorders.[23-28] There appears to be significant individual variation in TEG values in horses, and this variability may limit the use of TEG as a first-line point-of-care coagulation test.

HEMOSTATIC DYSFUNCTION

Hypocoagulability with subsequent surgical bleeding may be related to an inherited condition in the patient or to an acquired coagulopathy or thrombocytopathy. Hemostatic dysfunction may also consist of hypercoagulability, thrombotic tendencies, and DIC, especially associated with acute inflammatory diseases.

Inherited Conditions

Inherited conditions that result in coagulopathy or thrombocytopathy are relatively uncommon in the horse and much more common in the dog, cat, and human. These conditions include von Willebrand disease, thrombasthenia, hemophilias, and specific coagulation factor deficits. In horses, deficits of prekallikrein and of factors VIII, IX, and XI have been reported.[29,30] These may be difficult to detect preoperatively; a thorough history obtained from the client or observation of clinical signs may indicate a need for specific coagulation testing. If a deficit is identified, adequate preparation for surgery is critical, possibly consisting of pretreatment with plasma or component therapy.

Acquired Conditions

Acquired conditions resulting in hemostatic dysfunction may manifest clinically as DIC or as a specific coagulopathy or thrombocytopathy. Hemostatic dysfunction can be the result of inappropriate use of heparin (particularly unfractionated), aspirin, or other anticoagulants. The administration of certain drugs such as sulfonamides, penicillin, phenylbutazone, ibuprofen, estrogens, antihistamines, and cardiovascular drugs has been associated with thrombocytopenia in humans and animals. Other diseases associated with hemostatic dysfunction in the horse are severe liver disease, equine infectious anemia, *Anaplasma phagocytophilum*, and equine viral arteritis. In general, if any acquired condition that could result in a coagulopathy is noted in the history or detected in the clinical progression of a surgical candidate, appropriate and complete evaluation of the hemostatic system must be performed. If the surgeon is presented with an emergency situation, arrangements should be made for the availability of a blood donor or possible component therapy to attenuate the situation.

Inflammation and Coagulation

Hemostatic dysfuction has long been recognized in horses with severe inflammatory diseases such as gastrointestinal disease and sepsis, and there is a growing body of evidence that demonstrates the intricate interplay between inflammation and coagulation. Severe inflammation can cause increases in coagulation, decreases in anticoagulation, and inhibition of fibrinolysis, resulting in a *procoagulant state*. Cytokines and endotoxin can induce increased expression of tissue factor on monocytes, macrophages, and microparticles.[7,31] Endotoxin and proinflammatory cytokines can also activate platelets and induce the release of vWF from endothelium. Levels of AT are decreased as a result of impaired synthesis, increased consumption (because of increased thrombin generation), and negative acute-phase response. Protein C also decreases as a result of increased consumption, decreased production by the liver, and decreased activation by thrombomodulin. Fibrinolysis is impaired because tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β) stimulate an increase in PAI-1. Coagulation derangements can acually contribute to further inflammation, since AT and protein C have anti-inflammatory effects. Activation of protease-activated receptors during coagulation also enhances inflammation through increased production of TNF-α, IL-6, and IL-8.[31]

This procoagulant state induced by inflammatory conditions can lead to DIC. The initial hypercoagulable state does not commonly lead to clinically evident thrombotic events in horses, except for catheter-related jugular thrombophlebitis.[32] There are only single reports of a few cases of thrombosis (distal limb and pulmonary) related to gram-negative bacteremia or endotoxemia.[33,34] In the early stages of DIC (subclinical), there will be clinicopathologic evidence of platelet consumption, coagulation factor consumption, and hyperfibrinolysis. With severe activation of coagulation, DIC can lead to massive fibrin deposition in tissues in the lungs, liver, and kidneys, potentially

leading to multiorgan failure.[35] The syndrome of DIC places patients at risk of bleeding if intravascular coagulation is severe enough to result in coagulation factor depletion and thrombocytopenia, although the bleeding form of DIC is rare in the horse. Primary diseases that could result in DIC and that may be encountered by a surgeon include neoplasia, sepsis, trauma, severe acute hemorrhage, clostridial myositis, and severe endotoxemia associated with acute gastrointestinal disease.

The diagnosis of DIC requires the horse to have a primary disease that places it at risk, as well as clinicopathologic evidence of coagulopathy. The testing recommended for diagnosis of DIC includes platelet count (thrombocytopenia), clotting times (prolonged PT and APTT), fibrinogen concentration (decreased), and D-dimer concentration or FDPs (increased).[36] Scoring systems have been developed to aid in the diagnosis of DIC in human and canine patients, but there is not a comparable consensus scoring system for the equine patient.[37,38]

Reports of DIC in the equine veterinary literature most commonly describe the process as occurring secondary to a gastrointestinal disorder. Earlier reports describe clinical DIC, in which horses had overt clinical signs such as epistaxis, surgical bleeding, and venipuncture bleeding, whereas more recent reports have documented larger numbers of horses with subclinical DIC (an abnormal coagulation profile but lacking signs of a thrombohemorrhagic crisis).[17,19,39,41] Ischemic and inflammatory conditions of the large colon are most commonly associated with clinicopathologic coagulopathy, but simple obstructions such as large colon impaction are rarely associated with coagulopathy. Approximately one third of horses presenting to a referral facility with acute colitis had evidence of subclinical DIC, defined as abnormal findings in at least three of six coagulation tests.[19] The coagulation testing included platelet count, fibrinogen, PT, APTT, AT, and FDPs. Horses with subclinical DIC were 8 times more likely to die than those without evidence of DIC. Although fibrinogen was not below the reference range in coagulopathic horses, it was lower in horses with DIC compared to horses with no evidence of DIC, and fibrinogen decreased over the first 48 hours of hospitalization in nonsurvivors. Despite the frequent diagnosis of subclinical DIC, none of these horses with acute colitis demonstrated a clinical bleeding condition.

Horses with large colon volvulus commonly demonstrate subclinical DIC, with 70% reported to have at least three of six coagulation tests abnormal.[41] In this group of horses, development of prolonged PT, increased TAT, and thrombocytopenia were associated with a poor prognosis. Horses with four of six abnormal coagulation tests were also more likely to be euthanized. Other investigators have demonstrated that increased TAT, PT, APTT, PAI-1 and FDPs and decreased AT, protein C, and platelet count are associated with nonsurvival in horses with colic.[14,18,42-46]

Neonatal foals with sepsis have been shown to have a high incidence of clinicopathologic coagulopathy. Compared to healthy foals, septic neonates have prolonged PT and APTT; increased levels of fibrinogen, FDPs, α-2-antiplasmin, and PAI-1; and decreased levels of AT and protein C.[47] Foals with septic shock were reported to have coagulopathy (at least three abnormal coagulation tests) in 25% of cases, with 67% demonstrating clinical bleeding disorders, including petechiation and epistaxis.[48] Septic foals (not in shock) had clinical signs of bleeding in 39% of cases. Because horses with inflammatory conditions such as gastrointestinal disease and sepsis are at risk of coagulation abnormalities, hemostasis testing should be strongly considered in these patient populations, and treatment should be initiated if indicated.

Treatment of DIC

Since DIC is not a primary disease, there is no specific treatment that will effectively reverse the process of coagulopathy. Identification of horses at risk and aggressive treatment of the primary underlying disease are the best strategies for preventing DIC. Prevention and treatment of endotoxemia, including treatment with hyperimmune plasma, polymyxin B, and nonsteroidal anti-inflammatory drugs, are reasonable strategies for prevention of DIC (see Chapter 2). In both human and veterinary patients, plasma and platelet transfusions are recommended in cases with active bleeding or with a high risk of bleeding (e.g., surgical procedure).[49,50] Transfusions with specific coagulation factors such as prothrombin complex concentrates are not recommended as prophylactic treatment in nonbleeding human patients, because the addition of activated factors may worsen intravascular coagulation.[50] Antithrombin concentrate is not available for horses, but treatment with fresh frozen plasma will provide AT, an anticoagulant factor, which is frequently decreased in critically ill and septic horses.

Anticoagulant treatment early in the course of DIC may limit the activation of coagulation. Heparin is the anticoagulant most commonly used for this purpose in human and veterinary medicine. Heparin increases the activity of AT, thereby inhibiting thrombin and factor Xa. Low-molecular-weight heparin (LMWH) has greater inhibition of FXa, dose-dependent clearance, and a longer half-life than unfractionated heparin (UFH).[51] In horses, administration of UFH has been associated with prolonged APTT and decreased packed cell volume (PCV), whereas these side effects are not seen with administration of LMWH.[52] The following regimen is recommended: heparin calcium, 150 IU/kg SQ initially, then 125 IU/kg SQ q12h for 3 days, followed by 100 IU/kg SQ q12h. When using sodium heparin, a dose of 40-80 units/kg q12h is recommended. The following regimen for LMWH is recommended: Dalteparin 50 to 100 anti-Xa units/kg SQ q24h; enoxaparin 40 to 80 anti-Xa units/kg (0.35 mg/kg) SQ q24h. Although the use of heparin has been reported for treatment of DIC in horses, there are no controlled studies to evaluate treatment of DIC in horses.[17] There is some evidence to support heparin anticoagulant treatment for DIC in human patients; however, studies indicate that the use of exogenous heparin may negate some of the beneficial anti-inflammatory effects of AT, which are mediated via endogenous heparans on the endothelium.[53,54] Treatment with recombinant human activated protein C was shown to be beneficial in human patients with severe sepsis and DIC.[55] A recombinant equine protein C is not available, and therefore this drug has not been evaluated in horses.

Risk of Surgical Bleeding

Certain surgical procedures in equine surgery are associated with a significant risk for intraoperative and postoperative hemorrhage. Surgery involving the sinuses or ethmoid area, the cranial reproductive tract, the spleen, or certain neoplasias may result in significant intraoperative hemorrhagic challenges. Because some of these surgeries can be performed electively, careful preoperative planning may alleviate many of the complications of

perioperative hemorrhage. Options include planned autologous transfusion and normovolemic or isovolemic hemodilution, preoperative crossmatch and subsequent whole blood transfusion (see later), or availability of stored blood products and components. Autologous transfusion and normovolemic hemodilution involve collection of the patient's blood in the weeks before surgery (banking) or in the immediate preoperative period, followed by administration of crystalloids before induction. Throughout any surgical procedure, it is critical to employ proper hemostatic techniques (see Chapter 12).

BLOOD TRANSFUSION

Indications

Whole Blood

Whole blood (WB) transfusions are most often indicated for horses that have suffered acute blood loss from trauma, surgery, or other conditions such as splenic rupture or uterine artery hemorrhage. In cases of blood loss, the transfusion serves to restore blood volume as well as oxygen-carrying capacity. Although there are no set variables that serve as "transfusion triggers," a combination of physical examination and clinicopathologic parameters can be used to guide the decision to transfuse. It is important to remember that the PCV may remain normal for up to 12 hours following acute hemorrhage because of the time required for fluid redistribution and the effects of splenic contraction. Serial monitoring of PCV and total solids (TS) as the horse is rehydrated with intravenous fluids will give an indication of the extent of blood loss. Suspicion of large-volume blood loss, combined with tachycardia, tachypnea, pale mucous membranes, lethargy, and decreasing TS may lead to the decision to transfuse.[56] A blood transfusion is likely needed during an acute bleeding episode when the PCV drops below 20%, although in acute severe cases, transfusion may be needed before there is a significant drop in PCV.

Estimation of blood loss at surgery can be used to guide the decision to transfuse, with loss of greater than 30% of blood volume generally requiring transfusion.[57] Anesthetized horses may have very stable heart rate and PCV despite massive blood loss; pale mucous membranes with prolonged capillary refill time (CRT), decreasing TS, hypotension, and hypoxemia are better indicators of blood loss.[58]

Oxygenation status can help to determine the need for blood transfusion in cases of both acute hemorrhage and chronic anemia. A rise in blood lactate concentration despite volume replacement with crystalloid or colloid fluids may indicate continued tissue hypoxia and a need for blood transfusion.[59,60] Oxygen extraction ratios are also useful measures; a ratio greater than 40% to 50% in the context of blood loss may indicate a need for blood transfusion (Box 4-1).[61]

Transfused red blood cells (RBCs) have been reported to have a very short half-life; however, a recent study indicates that autologous transfused red blood cells have longer survival than originally reported, and allogeneic (donor) transfused RBCs may also have a longer half life than was reported in the original chromium label studies.[62-64] Red blood cells from allogeneic transfusions do have a much shorter half-life than autologous red cells, so transfusion should still be considered a temporary measure to restore oxygen-carrying capacity, relying on the horse's erythropoeitic response or resolution of underlying disease to provide long-term resolution.

Box 4-1. Formulas

OXYGEN EXTRACTION RATIO

O_2 ER = ~(SaO_2 – SvO_2)/SaO_2

O_2 ER = Oxygen extraction ratio

SaO_2 = Arterial oxygen saturation

SvO_2 = Mixed venous oxygen saturation

BLOOD TRANSFUSION VOLUME (L)

Body weight (kg) × 0.08 × [(Desired PCV – Actual PCV)/Donor PCV]

PLASMA TRANSFUSION VOLUME (mL)

Body weight (kg) × 45 mL/kg × [(Desired TP – Actual TP)/Donor TP]

ER, Extraction ratio; *PCV,* packed cell volume; *TP,* total protein.

Fresh whole blood can also provide platelets, though generally not in concentrations high enough to treat severe thrombocytopenia. For patients with primary thrombocytopenia or thrombocytopathia, platelet concentrates can be given. Platelet concentrates can be obtained by plateletpheresis or by centrifugation using a slow-spin technique.

Packed Red Blood Cells

Packed red blood cells (pRBCs) are indicated for normovolemic anemia, such as neonatal isoerythrolysis, erythropoietic failure, and chronic blood loss. In cases of chronic or hemolytic anemia, markers of tissue oxygenation, such as lactate and oxygen extraction are still useful. PCV is a better "transfusion trigger" for chronic anemia compared to acute hemorrhage, with transfusions suggested for horses with evidence of tissue hypoxia and a PCV less than 10% to 12%. Transfusions may be given at a higher PCV for horses with concurrent conditions (e.g., respiratory disease, anesthesia, sepsis) or risk of further blood loss. When pRBCs are not available, WB may be used for the same indications, although attention should be paid to the total volume given so that volume overload is avoided.

Plasma

Plasma transfusion is indicated for the treatment of clotting factor deficiency, hypoalbuminemia, and neonatal failure of transfer of passive immunity. Fresh and fresh frozen plasma (FFP) contain immunoglobulins, coagulation factors (fibrinogen and factors II, VII, IX, X, XI, and XII), and cofactors (factors V and VIII), and the anticoagulant proteins antithrombin, protein C, and protein S. Plasma has also been used for treatment of DIC in horses.[17]

Colloid support is generally recommended in patients with a total protein less than 4.0 g/dL or serum albumin concentration less than 2.0 g/dL. Other indications for colloid support are colloid oncotic pressure less than 14 mm Hg, clinical signs such as ventral edema, and conditions that increase microvascular permeability, such as sepsis. When plasma is not necessary for clotting factor replacement, a synthetic colloid such as hydroxyethyl starch (hetastarch) is preferred for volume expansion and more effective oncotic support. For more information on this subject please review Chapter 1.

Preoperative evaluation of neonatal foals should include testing IgG concentration. Failure of transfer of passive

immunity (FPT) in neonatal foals greater than 12 hours of age is best treated by plasma transfusion, because colostrum absorption is greatly diminished after 12 hours.[65] An IgG concentration less than 200 mg/dL is considered complete FPT, and IgG between 400 and 800 mg/dL is considered partial FPT. Although plasma transfusion is not always needed for foals with partial FPT, it is recommended for foals that have preexisting infection or exposure to pathogens. Commercially available fresh frozen hyperimmune plasma is most commonly used for treatment of neonatal foals. Equine FFP is licensed by the U.S. Department of Agriculture, and most products have a minimum guarantee for IgG concentration and a 2- to 3-year shelf life when frozen. Although commercially available hyperimmune plasma has very high IgG concentrations (1500 to 2500 mg/dL), plasma from local donor horses may provide better protection against specific local pathogens.

There are multiple hyperimmune plasma products with bacterial- or viral-specific antibodies. There is some evidence for the efficacy of *Escherichia coli* (J5) and *Salmonella typhimurium* hyperimmune plasma for the treatment of equine endotoxemia; however, there are also reports that dispute the efficacy of such products.[66,67] The use of *Rhodococcus equi* hyperimmune plasma for the prevention of *R. equi* infection has also been controversial.[68,69] Other plasma products available for specific disease treatment include botulism antitoxin, West Nile virus antibody, and *Streptococcus equi* antibody.

Oxyglobin

Oxyglobin is a hemoglobin-based oxygen-carrying solution that is indicated for treatment of anemia. Oxyglobin has been used experimentally in ponies with normovolemic anemia.[70] In this study, Oxyglobin improved hemodynamic and oxygen transport parameters; however, one pony had an anaphylactic reaction. The use of Oxyglobin was also reported for treatment of a pony mare with chronic hemorrhage and a history of acute transfusion reactions.[71] Although Oxyglobin is currently commercially available, the cost and volume (125 mL) per bag limit its utility for equine treatment.

Donor Selection and Management

There are 8 recognized equine blood groups, and 30 different factors identified within 7 of these groups.[72] Because of the large number of blood groups and factors, there are no true universal donors for horses. The ideal equine blood donor is a healthy, young gelding weighing at least 500 kg. Donor horses should be up-to-date on vaccinations, including rhinopneumonitis, tetanus, eastern and western equine encephalomyelitis, rabies, and West Nile virus. Donors should be tested annually for equine infectious anemia. Because RBC antigens Aa and Qa are the most immunogenic, the ideal donor should lack the Aa and Qa alloantigens. There are breed-specific blood factor frequencies, so a donor of the same breed as the recipient may be preferable, especially when blood typing is not available. Horses that have received blood or plasma transfusions and mares that have had foals are not suitable as donors because they have a higher risk of carrying RBC alloantibodies. Donkeys have an RBC antigen known as "donkey factor," which is not present in horses; therefore, donkeys or mules should not be used as donors for horses, because the horses receiving transfusion can develop anti–donkey factor antibodies.[73] In the referral practice setting, it may be practical to establish a group of blood donor horses. These donor horses should be blood typed and should also be tested for alloantibodies.

When a surgical procedure is planned in advance and there is a high risk of substantial blood loss, preoperative autologous donation should be considered, because the horse would be its own ideal blood donor.[74] The life span of transfused autologous RBCs after 28 days of storage is approximately 30 days, compared to a 14-day half-life for fresh, crossmatched allogeneic blood.[64,75] Intraoperative or posthemorrhage cell salvage is also an option for autotransfusion, and its use has been reported in a horse with postcastration hemorrhage.[76] RBC recovery can be performed with specialized cell salvage equipment, which washes and filters collected blood, but cell salvage can also be performed with simple anticoagulation and filtration.[77] The technique of cell salvage is limited to cases in which the salvaged blood is not in an area of infection or malignancy, unless specialized washing and filtering equipment is used.

Blood Typing and Crossmatching

In an emergency situation, an immediate blood transfusion may be given without a crossmatch for the first time, with a very minor risk of serious transfusion reaction. Horses can develop alloantibodies within 1 week of transfusion, so blood typing and crossmatching are recommended before a second transfusion is performed.[78] However, a second blood transfusion may be performed safely within 2 to 3 days of the first transfusion without a blood crossmatch.

Blood typing and alloantibody screening can be used to help find the most appropriate donor horse for the patient requiring transfusion. Unfortunately, since blood typing is time-consuming and laboratories performing blood typing are very limited, this is not often a practical method of donor selection. Blood typing and antibody screening before initial transfusion are more important for horses that may require subsequent blood transfusions and for broodmares that may produce foals with neonatal isoerythrolysis (NI) if sensitized to other blood group factors.[78] A rapid agglutination method for detection of equine RBC antigens Ca and Aa has been developed that may be a more practical method of pretransfusion testing.[79]

A blood crossmatch is recommended before a transfusion, especially for any horse that may have previously been exposed to RBC antigens. Hemagglutination crossmatching is widely available and rapidly performed; however, it will not predict all transfusion reactions, namely the hemolytic reactions. Rabbit complement can be added to the reaction mixture to detect hemolytic reactions.[80] The major crossmatch involves mixing the donor's washed red blood cells with the recipient's serum, whereas the minor crossmatch involves mixing the recipient's red cells with the donor's serum. If the minor crossmatch is incompatible, but the major crossmatch is compatible, the transfusion can still be performed after washing the donor red blood cells.

Blood Collection and Administration

Collection Technique

Blood is collected from the jugular vein of the donor horse, either via direct needle cannulation or catheterization. When a large volume of blood is needed, a 10- or 12-gauge catheter is recommended, although a 14-gauge catheter is also sufficient.

Blood flow may be improved by placing the catheter opposite the venous blood flow (catheter directed toward the head). A healthy horse can donate approximately 20% of its total blood volume every 30 days.[81] When 15% or greater blood volume is collected, volume replacement with intravenous crystalloid fluids is recommended. The donor horse's heart rate, respiratory rate, and attitude should be monitored during the blood collection. Vital parameters should normalize within 1 hour of collection.

Plastic bags and vacuum-collection glass bottles are available for blood collection in sizes ranging from 450 mL to 2 L. The glass bottles are preferred by many because of the speed of collection; however, the glass inactivates platelets and causes some damage to RBCs.[82,83] When blood is collected for immediate transfusion, anticoagulation with 3.2 % sodium citrate (1:9 anticoagulant to blood ratio) is adequate. However, when blood is stored for later transfusion, optimal pH and support of RBC metabolism are necessary to sustain RBC viability. Biochemical and hematologic parameters suggest that WB may be stored in citrate-phosphate-dextrose-adenine (CPDA)-1 bags for at least 3 weeks.[82] A posttransfusion viability study on equine blood stored for 28 days demonstrated a 24-hour labeled RBC survival of 73% and a half-life of 29 days.[64] RBC concentrates stored in saline-adenine-glucose-mannitol solution may be suitable for transfusion for up to 35 days after collection.[84] Blood should be stored in a dedicated blood bank refrigerator at 4° C.

Equine blood can be processed to provide plasma and pRBC components. Because of the rapid sedimentation of equine RBCs, the RBC component can be administered without specialized processing; however, the pRBCs will still contain plasma components unless centrifugation and repeated washing are performed. Washing of RBCs is the preferred technique when a transfusion is given to an NI foal using the mare as a donor. When RBC washing or other processing is planned, blood should be collected into bags rather than bottles because of ease of centrifugation and sterile transfer.

Plasma processing can be performed by gravity sedimentation, centrifugation using a double-bag system, or plasmapheresis. Plasmapheresis is the preferred technique because it is more rapid than WB collection and processing and yields plasma with minimal RBCs and leukocytes.[85] Plasmapheresis of 4 to 11 L can be performed every 30 days on donor horses.[86] Immunoglobulins are well-maintained for at least 1 year in FFP; however, coagulation factor activity may decrease after 2 to 4 months of storage.[87]

Administration and Adverse Reactions

The volume of blood to be transfused depends on estimated blood loss, estimated total blood volume, and donor PCV. In cases of acute blood loss, PCV is often not useful for estimates of volume to be transfused since it does not accurately reflect blood loss. Instead, estimates of blood loss and evaluation of clinical parameters are used to determine the volume of blood needed. From 25% to 50% of the total blood lost should be replaced by transfusion since much of the circulating volume will be replaced by fluid shifts. It is important to remember that up to 75% of RBCs lost into a body cavity (e.g., hemoperitoneum) are autotransfused back into circulation within 24 to 72 hours.[88] Therefore lower percentages of blood volume replacement may be needed in cases of intracavitary hemorrhage. The volume of blood required to treat horses with normovolemic or chronic anemia can be estimated based on the target PCV (see Box 4-1). Blood and plasma products should be delivered with an in-line filter to remove small clots and fibrin.

Volumes of plasma for treatment of hypoproteinemia can be estimated by total protein or albumin concentrations (see Box 4-1), although the use of plasma to normalize severe hypoproteinemia can be prohibitively expensive in the adult horse. Volume of plasma given for treatment of hypoproteinemia or coagulopathy is often determined by clinical and clinicopathologic response. A starting point for treatment of coagulapathy is approximately 4 to 5 mL/kg plasma. Follow-up monitoring with hemostatic testing is recommended to help determine the end point of treatment.

To facilitate monitoring for transfusion reactions, blood should be delivered at a rate of approximately 0.3 mL/kg over the first 10 to 20 minutes, while monitoring heart rate, body temperature, and respiratory rate. Horses should also be monitored for signs of muscle fasciculation, piloerection, and urticaria. Adverse reactions reported in horses receiving blood transfusions include urticaria, hemolysis, and acute anaphylactic reactions. The rate of adverse reaction to WB transfusion has been reported as 16%, with 1 of 44 horses (2%) having a fatal anaphylactic reaction.[56] If no signs of reaction are seen, the rate of administration can be increased to 5 mL/kg/hr for normovolemic horses and up to 20 to 40 mL/kg/hr for hypovolemic horses. If signs of anaphylaxis are present, epinephrine (0.01 to 0.02 mL/kg IV of 1:1000 solution) should be administered immediately. More mild transfusion reactions, such as urticaria, fever, and tachypnea, may be treated with an NSAID (e.g., flunixin meglumine 1.1 mg/kg IV) or an antihistamine (e.g., tripelennamine 1.1 mg/kg IM).

Similar to the risk in other veterinary species, bacterial contamination of blood, transmission of blood-borne disease from donor to recipient, and hypocalcemia associated with citrate toxicity are all potential concerns related to transfusion in the equine patient. An additional concern in horses is the possible sensitization of a broodmare to blood group antigens, leading to the risk of NI in subsequent foals.[78] Although plasma transfusions are not commonly associated with serious adverse reactions, serum hepatitis has been reported in association with transfusions of commercial plasma.[89]

TOPICAL HEMOSTATIC AGENTS

Topical hemostatic agents are needed for control of diffuse capillary bleeding from bone or parenchymal organs, such as liver or spleen. These agents can also be useful for control of bleeding during dental and nasal surgery. Surgical hemostasis techniques, including mechanical, thermal, chemical, and physical hemostasis are discussed in Chapter 12. This section will focus only on topical products available for augmentation of hemostasis. The most common veterinary use of topical hemostatic agents is in canine spinal surgery, and there are no specific equine studies available to guide the use of these hemostatic agents.

Mechanical Hemostatic Agents

These topical agents exert their main hemostatic effect by applying pressure on the area of diffuse bleeding. Some of these products also act as a scaffold for platelets and coagulation factors. The mechanical hemostatic agents are generally

appropriate for control of smaller areas of discrete bleeding rather than more severe bleeding. Although there are numerous topical hemostatic products on the market, the major long-standing products are described later.

Purified Gelatin Sponge

The gelatin sponge is made from purified animal gelatin. It binds well to tissue and exerts a hemostatic effect by swelling as it is soaked with blood. Gelatin sponges can be soaked in thrombin to help promote coagulation directly.[90] Gelatin sponges can potentiate infection, and their use should be avoided in contaminated wounds. This product is absorbed over a period of 4 to 6 weeks.

Oxidized Regenerated Cellulose

Oxidized regenerated cellulose is a chemically altered form of cellulose, which is particularly useful to control diffuse bleeding from broad surfaces. Surgicel has mechanical hemostatic effects as a result of swelling from blood absorption, and it activates coagulation on the collagen surface. Surgicel also acts as a caustic hemostatic agent because of its low pH. The low pH additionally confers antibacterial properties and therefore is preferred over gelatin foam for use in contaminated areas.[91] Surgicel should not be soaked in thrombin, because the biologic agents will be inactivated in the low-pH environment. The low pH may also lead to tissue inflammation and delayed wound healing, so any excess product should be removed from the surgical site. This product is absorbed in 7 to 14 days, although residue from the material may persist for several months to years.[92]

Microfibrillar Collagen Hemostatic Agents

Microfibrillar collagen agents (Avitene, Instat) are derived from bovine dermal collagen, and are available in fibrous (flour), sheet, and sponge forms. These products are absorbed in 8 to 10 weeks. Microfibrillar collagen agents do not swell, and they do not rely as much on their mechanical effect as does Gelfoam. The product does bind tightly to the bleeding surface, so there is likely some mechanical blockage of injured vessels. Platelets adhere to the collagen and are activated, and the resultant platelet degranulation and aggregation lead to hemostasis. These products are less effective in patients with thrombocytopenia.[93] Microfibrillar collagen products have been associated with allergic reactions in human patients, likely related to the bovine origin of the materials. Microfibrillar collagen can interfere with bacterial clearance and wound healing, and it is therefore recommended that it be removed from the surgical site before closure of the wound.[90]

Polysaccharide Hemostatic Agents

Microporous polysaccharide hemispheres (TraumaDex) have a porous surface that allows absorption of blood, thereby concentrating platelets and coagulation factors and reducing the time required for coagulation. This product is absorbable and does not appear to inhibit wound healing.[94] It does not appear to be as effective for severe arterial or venous bleeding compared to other topical hemostatic agents. Another type of polysaccharide, chitosin, is present in hemostatic dressings designed to control bleeding from traumatized extremities (HemCon Bandage).

Bone Wax

Bone wax is composed of beeswax and petroleum jelly and, as its name suggests, is used to control bleeding from bone surfaces. It mechanically stops blood flow from vessels in bone, and it does not have any biologic hemostatic effect. Bone wax inhibits bone healing, so it should not be used when fracture union is desired. It has also been shown to inhibit bacterial clearance from cancellous bone, and therefore it should not be used in areas of bacterial contamination or infection.[95] Bone wax has been reported to cause additional adverse effects such as allergic reaction, granulomatous reaction, and embolization.[90]

Adhesives and Sealants

Thrombin Products

Thrombin is available as a stand-alone product and is also a component of other biologic hemostatic agents.[92] Bovine-derived thrombin actively promotes coagulation by converting fibrinogen to fibrin and activating platelets. The stand-alone thrombin products (Thrombin-JMI) are packaged as a powder that is reconstituted for use. The liquid solution can be difficult to apply accurately during surgery. Thrombin is also available in a variety of combination preparations. A combination of human thrombin and collagen-derived gelatin matrix (FloSeal) is available as a "flowable" product, applied to the bleeding surface. Bovine-derived thrombin has been shown to induce antibody formation in human patients, especially to factor V. Recombinant human thrombin products are available, but similar veterinary recombinant products do not exist.

Fibrin-Based Sealants

These products are applied directly to the tissue and promote hemostasis by adhesion and formation of a fibrin clot, reducing the size of the open bleeding defect. Fibrin glues (TISSEEL) contain thrombin and fibrinogen, which are combined at the time of application through a dual-chamber syringe. Fibrin sealants replicate the last stage of coagulation and do not require that the patient have normal platelets or coagulation factors. Fibrin sealants are biodegradable and have not been associated with tissue inflammation or foreign body reaction.

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CHAPTER 5

Wound Healing

Patricia J. Provost

CLASSIFICATION OF WOUNDS

Wound healing is inherent to all species and is the biologic process by which the body repairs itself after injury, whether it be traumatic or surgical. Understanding the basics of wound healing can improve patient outcome, reducing morbidity and often expense. Wounding may be restricted to the skin but often will involve underlying and adjacent tissues. Wounds have been traditionally classified as open or closed, and further as clean or contaminated.[1] These traditional classification schemes are useful because they provide a basis for general therapeutic guidelines. Closed wounds include crushing or contusion injuries, which at the time of impact do not have skin loss. However, substantial disruption to the underlying blood supply can occur, which may lead to future skin loss and often a prolonged recovery period. Open wounds can be classified by the type of trauma, such as abrasions, avulsions, incisions, and lacerations (Table 5-1); partial or full-thickness; or alternatively, they can be classified based on their potential for bacterial presence.[1]

Surgical wounds created under aseptic conditions are *clean* wounds. *Clean-contaminated* wounds are surgical wounds in which the respiratory, alimentary, or urogenital tracts are entered under controlled conditions without unusual contamination, whereas *contaminated* wounds are open, acute, accidental, or surgical wounds in which there has been a major break in sterile technique. *Dirty* or *infected* wounds are those that are old, have devitalized tissue, or have gross contamination with foreign debris. Clean, clean-contaminated, and contaminated wounds by definition contain less than 1×10^5 bacteria per gram of tissue, whereas those with greater than 1×10^5 are infected.[2] When in doubt, all nonincision open wounds should be handled as if they are infected, as should any incision from which there is purulent drainage. In the past, open wounds were often classified on duration since the time of injury and the degree of contamination: Class 1 (less than 6 hours duration with minimal contamination), Class 2 (6 to less than 12 hours duration with significant contamination), and Class 3 (longer than 12 hours duration with gross contamination).[3] This type of classification is less useful in equine veterinary medicine because all wounds regardless of the duration have the opportunity for marked contamination considering the environment in which horses live.

TABLE 5-1. Wound Classification

Classification	Description
Crush	Injury occurring when the body part is subjected to a high degree of force between two heavy objects.
Contusion	A blow to the skin in which blood vessels are damaged or ruptured.
Abrasion	Damage to the skin epidermis and portions of the dermis by blunt trauma or shearing forces.
Avulsion	Loss of skin or tissue characterized by tearing of the tissue from its attachments.
Incision	A wound created by a sharp object that has minimal adjacent tissue damage.
Laceration	An irregular wound created by tearing of tissue. Skin and underlying tissue damage can be variable.
Puncture	A penetrating injury to the skin resulting in minimal skin damage and variable underlying tissue damage. Contamination with dirt, bacteria, and hair is common.

Choice of wound closure primarily depends on the type of wound (i.e., puncture versus laceration) and the degree of contamination. Closure of open, full-thickness wounds may be by *primary*, *delayed primary*, or *secondary closure* techniques, or they may be left to heal by *second intention* (Table 5-2).[1] The decision to proceed with one method versus another is guided by the wound's location, its initial classification, and often the surgeon's past experience with similar injuries. The biology of wound healing is similar regardless of the choice of wound closure, but outcome results can be directly influenced, especially in horses, by knowledge of the processes involved.

PHASES OF WOUND HEALING

Wound healing is a dynamic process, similar in all adult mammalian species, that is initiated whenever there is a break in tissue integrity. The repair process involves complex interactions between cellular and biochemical events that coordinate healing (Tables 5-3 through 5-5), which are similar whether injury is confined to the skin or extends to deeper structures. Our understanding of what is occurring is continually evolving. This is especially true in the horse. For the sake of simplicity, the healing process has been divided into three phases: (1) the *inflammatory* or *lag* phase, which involves hemostasis and acute inflammation; (2) the *proliferative* phase, during which tissue formation occurs; and (3) the *remodeling* phase, during which the healing tissue regains strength.[4] These three phases overlap

TABLE 5-2. Wound Closure

Classification	Wound Type	Recommendations
Primary closure	Clean or clean-contaminated wound converted to clean wound	Immediate suture closure without tension
Delayed primary closure	Clean-contaminated or contaminated wound with questionable tissue viability, edema, skin tension	Performed 2-5 days after injury; tissue débridement and wound lavage before closure
Secondary closure	Contaminated or infected wound	Performed at least 5 days after injury; granulation tissue and epithelialized skin edges excised at the time of closure
Second intention healing	Wound tissue unsuitable for closure; large skin defect and/or extensive tissue devitalization	Healing by granulation tissue, wound contracture, and epithelialization

TABLE 5-3. Inflammatory Cells in Tissue Repair

Cell Type	Function	Mediators
PMN	Phagocytosis of microbes	Reactive oxygen species, cationic peptides, eicosanoids, proteases
	Macrophage activation	
	Amplify inflammatory response	TNFα, IL-1β, IL-6
	Stimulate repair process	VEGF, IL-8
Macrophage	Phagocytosis of PMN, damaged tissue, and microbes	TNFα, IL-1β, IL-6
	Amplify repair process	PDGF, VEGF, bFGF, TGF-α, and TGF-β
	Stimulate angiogenesis and fibroplasia	
	Fibrolysis	tPA, uPA (tissue and urokinase-type plasminogen activator)
Mast cell	Control vascular permeability	Histamine
	Control influx of PMN	Chymase, tryptase
	Regulate tissue remodeling	

bFGF, Basic fibroblast growth factor; *IL,* interleukin; *PDGF,* platelet-derived growth factor; *PMN,* polymorphonuclear; *TGF,* transforming growth factor; *TNF,* tumor necrosis factor; *VEGF,* vascular endothelial growth factor.

TABLE 5-4. Cytokines Involved in Wound Repair

Name	Abbreviation	Source	Major Function
Colony-stimulating factor	CSF	Macrophage, lymphocyte, fibroblast, endothelial cell	Differentiation and maturation of hematopoietic stem cells
Interferon	IFN	Monocyte, macrophage, lymphocyte, mesenchymal cell	Proinflammatory; release of other cytokines; inhibit fibrosis
Interleukin	IL	All nucleated cells, in particular macrophage and lymphocyte	Proinflammatory; enhances epithelialization, angiogenesis, and remodeling
Tumor necrosis factor	TNF	Macrophage, lymphocyte, mast cell	Proinflammatory; enhances angiogenesis, epithelialization, and remodeling
Connective tissue growth factor	CTGF	Fibroblast	Mediator of TGF-β activity (cell proliferation and ECM accumulation)
Epidermal growth factor	EGF	Platelet, saliva	Epithelialization; chemotactic and mitogenic to fibroblast; protein and MMP synthesis (remodeling); angiogenesis (TGF-α)
Transforming growth factor-α	TGF-α	Macrophage, epithelial cell	
Fibroblast growth factor	FGF	Inflammatory cell, fibroblast, endothelial cell	Chemotactic and mitogenic to fibroblast and epithelial cell; protein synthesis; angiogenesis
Insulin-like growth factor	IGF	Liver, platelet	Chemotactic and mitogenic to epithelial cell; migration of epithelial cell; fibroblast proliferation, protein and GAG synthesis
Keratinocyte growth factor	KGF	Fibroblast	Chemotactic and mitogenic to epithelial cell
Platelet-derived growth factor	PDGF	Platelet	Chemotactic to inflammatory cell and fibroblast; mitogenic to mesenchymal cell; protein synthesis, contraction?
Transforming growth factor-β	TGF-β	Platelet, lymphocyte, mast cell, monocyte and macrophage, endothelial cell, epithelial cell, fibroblast	Chemotactic to inflammatory and mesenchymal cell; fibroblast proliferation; protein synthesis; ECM deposition (inhibition of MMP; induction of TIMP); wound contraction
Vascular endothelial growth factor	VEGF	Macrophage, fibroblast, endothelial cell, epithelial cell	Angiogenesis

ECM, Extracellular matrix; *GAG,* glycosaminoglycan; *MMP,* matrix metalloproteinase; *TIMP,* tissue inhibitor of metalloproteinase.
From Theoret CL: Wound Repair. p.54. In Auer JA, Stick JA (eds): Equine Surgery, 3rd Ed. Saunders Elsevier, St. Louis, 2006.

in time, with numerous interactions occurring at all levels (Figure 5-1). When wounds proceed through these steps in a timely manner and achieve functional and anatomic integrity, they are considered acute wounds. Alternatively they become chronic, which is not an uncommon outcome in horses.[5]

Inflammatory Phase

Known also as the *lag* phase of wound healing, this early response, which involves hemostasis and inflammation, is a very metabolically active period lasting for several days, during which wound healing is jump started. The response is directed at stopping blood loss, protecting against infection, and providing the substrate and cellular signals that will facilitate the subsequent steps in the process of healing.[4] Hemostasis is initiated immediately through the contributions of vasoconstriction, platelet aggregation, and fibrin deposition.

Reflex vasoconstriction occurs by smooth muscle contraction mediated by release of endothelin and thromboxane A_2 from the injured vessels and platelet-derived serotonin. The response is transient, lasting only 5 to 10 minutes, after which vasodilators such as prostacyclin, histamine, and nitric oxide predominate, facilitating diapedesis of cells, fluid, and protein into the

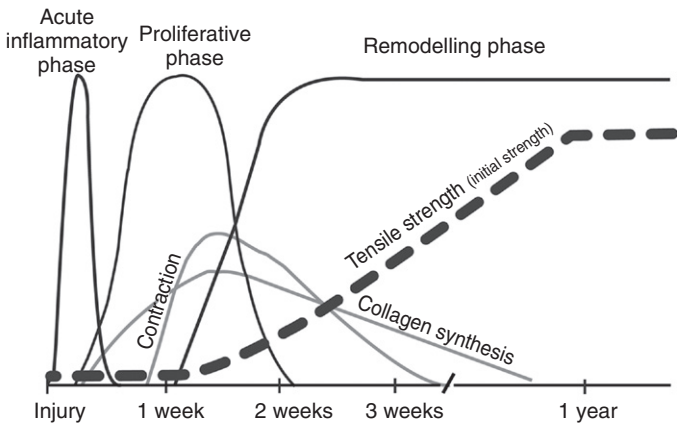

Figure 5-1. Temporal profile of various processes and gain in tensile strength occurring during normal cutaneous wound repair. (From Theoret CL: Wound Repair. p.45. In Auer JA, Stick JA (eds): Equine Surgery, 3rd Ed. Saunders Elsevier, St. Louis, 2006.)

TABLE 5-5. Matrix Metalloproteinases Involved in Wound Repair

MMP Name	MMP Number	Substrates	Source
COLLAGENASES			
Interstitial collagenase	MMP-1	Collagen (I, II, III, VII, IX)	Epithelial cell, fibroblast
Neutrophil collagenase	MMP-8	Collagen (I, II, III)	PMNs
Collagenase 3	MMP-13	Collagen (I, II, III)	–
STROMELYSINS			
Stromelysin 1	MMP-3	PGs, laminin, fibronectin	Epithelial cell
Stromelysin 2	MMP-10	Collagen (III, IV, IX, X)	Epithelial cell, fibroblast
Stromelysin 3	MMP-11	Collagen IV, fibronectin, gelatin, laminin	–
GELATINASES			
Gelatinase A (72 kDa)	MMP-2	Gelatin, collagen (I, IV), elastin	Most cells
Gelatinase B (92 kDa)	MMP-9	Gelatin, collagen (IV, V), elastin	Inflammatory cell, epithelial cell, fibroblast
Matrilysin	MMP-7	PGs, elastin, fibronectin, laminin, gelatin, collagen IV	Epithelial cell
MEMBRANE-TYPE (MT) MMPS			
MT1-MMP	MMP-14	Collagen (I, III), fibronectin	Membrane bound
MT2-MMP	MMP-15	Vitronectin, pro-MMPs	–
MT3-MMP	MMP-16	–	–
MT4-MMP	MMP-17	–	–
MT5-MMP	MMP-20	–	–

MMP, Matrix metalloproteinase; *PG,* proteoglycan; *PMN,* polymophonuclear granulocyte.
From Theoret CL: Wound Repair. p.52. In Auer JA, Stick JA (eds): Equine Surgery,3rd Ed. Saunders Elsevier, St. Louis, 2006.

wound and extracellular space.[6-9] Hemostasis is ultimately achieved through compression of vessels by soft tissue swelling and formation of a fibrin-platelet plug within the wound defect. Thrombin, the principal factor in clot formation, is instrumental in this process.[10,11]

Released by activation of both the intrinsic and extrinsic coagulation pathways, thrombin cleaves fibrinogen into fibrin monomers, which upon polymerization into fibrin fibers interact with plasma fibronectin to stabilize the hemostatic plug that fills the wound site.[12-14] This early wound clot is known as provisional wound matrix. If left unbandaged, the surface of the clot dessicates to form a scab, beneath which the provisional matrix will be replaced by granulation tissue during the proliferative phase of healing. Although the clot provides tenuous protection and stability to the wounded area and adjacent skin edges, there is no meaningful return of tissue integrity or breaking strength, hence the descriptive term *lag*.[15] Despite this, blood and fluid loss is halted, and microbial invasion through the open wound is minimized.

The activated platelets within this fibrin plug complex direct and amplify the early inflammatory phase of healing through the release of wound repair mediators, most importantly platelet-derived growth factor (PDGF) and transforming growth factor beta (TGF-β), from their storage granules.[12,14] As early wound healing progresses, polymorphonuclear cells (PMNs), macrophages, and fibroblasts can bind selectively to the provisional wound matrix through expression of cell surface integrin receptors as they migrate into the wound to initiate immune and synthetic functions.[16,17]

Leukocyte migration into the wound is activated by exposed collagen, elastin breakdown products, complement factors, and cytokines. PMNs are the first cell type to enter the wound in large numbers.[4] They appear soon after injury, with numbers peaking on about day 2, and decline as debris is cleared from the injured site. The neutrophils have two primary roles: to remove damaged tissue and bacteria, and to release chemoattractants to further augment the early cellular inflammatory response. The principal degradative proteinases released by PMNs to remove damaged tissue include cathepsin G, neutrophil-specific interstitial collagenase, and neutrophil elastase.[18] By 24 hours, circulating monocytes begin to enter the wound and differentiate into macrophages.[4] Macrophages are regarded as the major inflammatory cells responsible for regulating most of the important molecular signals for wound repair mechanisms through generation and release of oxygen free radicals, inflammatory cytokines, and tissue growth factors.[19] Macrophages proliferate in the wound and, similar to neutrophils, remove necrotic tissue as well as bacteria. The proteinases released by macrophages—elastase, collagenase, and plasminogen activator—aid in the débridement. Macrophages may be present for a period lasting from a few days to weeks, depending on wound characteristics. Their synthesis and release of tissue growth factors initiates the proliferative phase of the repair process, including angiogenesis, fibroplasia, and epithelialization. Neutrophil and macrophage apoptosis occurs as the inflammatory phase subsides.

Despite the fact that animal models of wound healing have demonstrated that neither neutrophils nor macrophages are essential to wound healing in sterile conditions, in the presence of bacteria, healing is delayed compared to that in animals with available PMNs.[2,18-20] In wound healing studies in horses and ponies, their presence has always been noted.

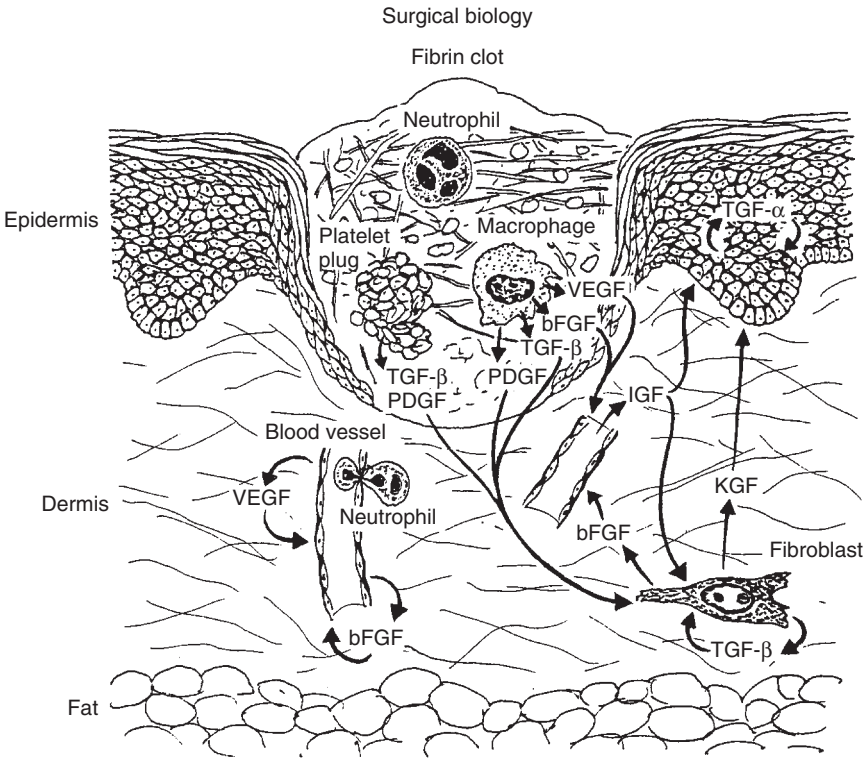

Figure 5-2. Cutaneous wound 3 days after injury. *bFGF,* Basic fibroblast growth factor; *IGF,* insulin-like growth factor; *KGF,* keratinocyte growth factor; *PDGF,* platelet-derived growth factor; *TGF,* transforming growth factor; *VEGF,* vascular endothelial growth factor. (Modified from Singer AJ, Clark RAF: N Engl J Med 341:738-746, 1999.)

Tissue Formation Phase

The *proliferative* phase of acute tissue repair is active by the third day following injury. It is characterized by angiogenesis, fibrous and granulation tissue formation, collagen deposition, epithelialization, and wound contraction (Figure 5-2).[21,22] As in the previous phase of wound healing, steps in the proliferative phase do not occur in series but rather overlap in time.

Angiogenesis

The wound healing process requires a continuous oxygen and nutrient supply. Decreased oxygen tension, high lactate levels, and low pH within the wound initiate the process of angiogenesis.[21] The endothelial cells at the tips of capillaries adjacent to the wounded area are attracted to the area by fibronectin, found within the provisional matrix, and grow in response to cytokines released by platelets and macrophages at a rate of 0.4 to 1.0 mm per day.[23] The development of vascular outgrowths requires endothelial cell proliferation that organizes into vessel architecture. Growth factors such as vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF) play central regulatory roles in neovascularization and subsequent tissue repair.[24] The tissue in which angiogenesis has occurred is dense in capillary loops, resulting in the characteristic granular red appearance of granulation tissue.[23] When macrophages and other growth factor–producing cells are no longer in a hypoxic, lactic acid–filled environment, their production of angiogenic factors stops.[25] Thus when tissue is adequately perfused, migration and proliferation of endothelial cells is reduced through the action of matrix metalloproteinases (MMPs). Eventually blood vessels that are no longer needed undergo endothelial cell apoptosis.[18]

Fibroplasia and Granulation Tissue Formation

Fibroblasts begin to arrive by the second day after injury, and by the fourth day they are the major cell type in the wound bed.[4,26-28] Recruitment from adjacent tissue, local proliferation, and transformation of undifferentiated local and systemic mesenchymal stem cells into fibroblasts all contribute to the peak in fibroblast numbers at 7 to 14 days after injury.[29] Fibroblast migration into the wound and their subsequent proliferation is largely regulated by PDGF, TGF-β and bFGF.[11] In the first several days after injury, fibroblasts proliferate and migrate, whereas later they synthesize and reorganize the components, which will eventually replace provisional matrix within the wound site. Fibroblasts synthesize and release collagen; glycosaminoglycans, including hyaluronan (which facilitates cell migration); glycoproteins (fibronectin and laminin); and proteoglycans.[30] Simultaneously they also secrete proteases, including MMPs, which digest the fibrin clot so that replacement with the new components can occur.[17]

Collagen production begins slowly on the second or third day after wounding and reaches peak production within 1 to 3 weeks.[4,17] Although wound fibroblasts produce type I collagen, which predominates in unwounded dermis, almost 30% to 40% of the collagen found in the acute wound will be type III. This is reflective of the dense population of blood vessels containing type III collagen, which then comprises granulation tissue. As the wound heals and vascularity is reduced, there is a shift in the balance of the collagen content toward type I.[4] In addition to collagen production, fibroblasts within the wound organize the collagen molecules into fibers and then into bundles, which are aligned parallel to the wound surface, usually along lines of maximum tension. The presence of collagen and its arrangement contribute to tissue strength. When

the wound defect is filled and homeostasis of collagen production and collagen degradation is achieved, macrophage and fibroblast numbers are reduced by apoptosis, and tissue maturation and remodeling begin.[31,32]

Epithelialization

The slow process of reepithelialization, to restore the barrier function of skin, starts immediately after wounding.[33] Suprabasal keratinocytes residing above the basement membrane of the epidermis and lining hair follicles and the sweat and sebaceous glands facilitate the repair.[34] Reepithelialization initially begins with the migration of these existing cells, but within a few days keratinocyte proliferation at the wound margins contributes to the number of available cells.[4,35] The location, and therefore the number of the keratinocytes available, depends on the type of injury. There is rapid reepithelialization in superficial injuries, such as an abrasion, as the basement membrane and epidermal appendage populations of keratinocytes remain available across the entire wounded area to participate in the repair. In contrast, in full-thickness wounds there is no residual epithelium, or epidermal appendages, from which keratinocytes can be recruited. In wounds of similar surface area, it is this last type of injury that requires the longest duration to heal, because reepithelialization can only occur through centripetal movement of the keratinocytes from the wound margins.[7,36]

Participating keratinocytes undergo phenotypic changes in response to a loss of contact inhibition and exposure to cellular products, including nitric oxide, which enable them to migrate and to phagocytize debris in their way.[37] The interaction between keratinocytes and fibroblasts is quite important. Keratinocytes stimulate fibroblasts to synthesize and release growth factors and cytokines, which in turn stimulate keratinocyte proliferation.[38] Upon detaching from neighboring cells they develop pseudopods that contain actin filaments.[36,39] During migration, integrins on the pseudopods attach to the extracellular matrix (ECM), and the actin filaments enable the pseudopod to pull the cell along.[39] Keratinocyte migration, however, requires healthy tissue over which to migrate.[36] Migration is impaired by fibrin, by inflammatory products, and by the presence of exuberant granulation tissue.[40,41] In surgical incisions, the tissue is healthy and the wound surface area following suture apposition is small, which enables epithelialization to occur within days.[7] In open traumatic injuries, however, there is a delay in epithelialization, because the necrotic tissue must first be eliminated and then a bed of healthy granulation tissue must be developed. Keratinocytes synthesize and release collagenases, proteases (MMPs), and plasminogen activator to clear a path across the wound surface.[33,36,42,43] Thus the time of onset of migration is variable, and new epidermis is often not apparent at the wound edges until 4 to 5 days following wounding. In most instances, because they must dissolve any scab that forms, keratinocyte migration is best enhanced by a moist environment, because the drier the environment, the thicker the eschar.[35,44,45]

Keratinocytes continue centripetal migration across the wound bed until cells from either side meet in the middle, at which point contact inhibition causes them to stop migrating, assume their normal phenotype, and begin the process of reestablishing the strata found in normal skin.[18,46,47] The new epidermis differs from that found in uninjured skin; it lacks rete pegs, which anchor it into the underlying connective tissue matrix; and in full-thickness wounds it lacks a dermal layer, without which there is a loss in tissue strength and elasticity.[4,48] There is no regeneration of lost epidermal appendages such as sweat glands and hair follicles. The fragile nature of the resultant epithelium makes the process of healing by epithelialization alone without the contribution of wound contraction less than ideal.[4] Time until complete reepithelialization occurs depends on the wound surface area and, in horses, on the location of the wound.

Contraction

Contraction usually begins in full-thickness wounds in the second week following injury, once the wound is heavily populated by fibroblasts, and can continue for several weeks.[4] The process is beneficial because it reduces the surface area of the original wound by 40% to 80%.[49] The centripetal movement of the adjacent uninjured dermis and epidermis over a full-thickness wound minimizes the area that requires epithelialization. In areas with loose skin, rates of contraction can be as high as 0.75 mm per day.[50]

The differentiation of fibroblasts into myofibroblasts is considered by most investigators to be necessary for contraction to occur.[31,51] The primary inducer of fibroblast-to-myofibroblast differentiation appears to be TGF-β1 released from macrophages and keratinocytes.[52,53] Fibroblast density and mechanical tension on fibroblasts within the ECM can also impart transition.[14,54-56] The acquisition of an alpha smooth muscle actin microfilament system signifies the change from the fibroblast to myofibroblast phenotype.[57] Although not completely understood, myofibroblasts form specialized connections between themselves and molecules, including collagen and fibronectin, within the ECM at the wound's edges.[47] When the actin filaments within the myofibroblast contract, force is transmitted through these connections to the edges, causing wound contraction.[35,58,59] Fibroblasts lay down collagen to reinforce the contracted wound.[60] Contraction usually does not occur symmetrically, rather, most wounds have an "axis of contraction," which allows greater organization and alignment of cells with collagen.[61] The process slows and ceases when either the wound edges meet, tension within the surrounding skin becomes equal to or greater than that generated by the contracting myofibroblasts, or when the number of myofibroblasts within the wound bed become low. At the conclusion of contraction, myofibroblasts either disappear by apoptosis or revert back to a fibroblastic phenotype.[61]

Remodeling and Maturation Phase

Remodeling and maturation of the extracellular matrix found in granulation tissue represents the final phase of wound healing. It is a phase that begins during the second week of repair and ends in the formation of scar tissue 1 to 2 years later, which remains 15% to 20% weaker than the original tissue (Figure 5-3).[18] The processes occurring during this phase begin with the replacement of the hyaluronan content within the provisional matrix by proteoglycans in the extracellular matrix. This gradually stops fibroblast proliferation and migration.[62] The cellular content within the ECM slowly decreases as cytokine and growth factor signals decline and the collagen content increases. Angiogenesis decreases and wound metabolic activity slows. The collagen deposited during the period of fibroplasia is oriented randomly, providing minimal tissue

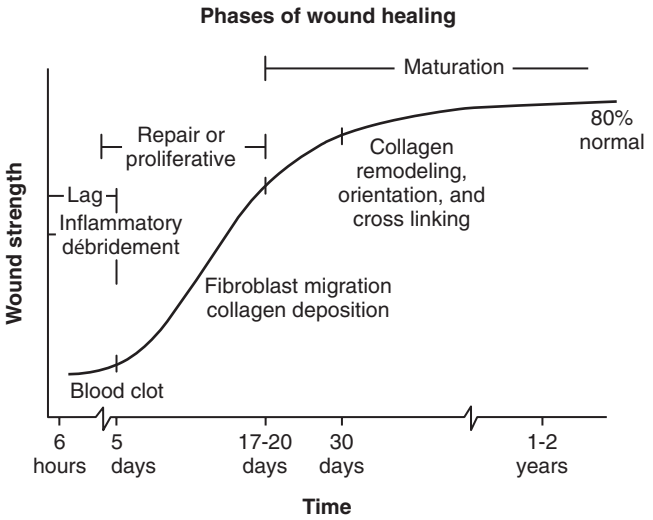

Figure 5-3. Changes in wound strength during the phases of wound repair. Note that the time axis is not to scale. (From Bassert JM: McCurnin's Clinical Textbook for Veterinary Technicians. 7th Ed. Saunders, Philadelphia, 2010.)

strength. During remodeling, collagen synthesis continues, but because of simultaneous lysis there is no net gain in content. MMPs (collagenase, stromelysins, and gelatinases), which are derived from macrophages, epithelial cells, endothelial cells, and fibroblasts within the ECM, are responsible for the degradation of collagen within the wound. Collagen fibers, which were once haphazardly arranged, are reestablished in bundles, cross-linked, and aligned along lines of tension by fibroblasts to progressively increase the tensile strength. There is a gradual gain in tissue strength from 20% of that of normal tissue at 3 weeks, to 50% within 3 months, and 70% to 80% of the strength of original tissue at the conclusion of maturation.[63]

These phases of acute wound healing normally progress with efficiency to stop blood loss, reestablish an immune barrier, and replace lost tissue. Yet of the six possible reported outcomes for acute wounds in humans, five are undesirable: dehiscence, herniation, wound infection, delayed healing, and keloid formation. Although the latter is rare in horses, it can easily be replaced with the problem of excessive or exuberant formation of granulation tissue.[64] In a retrospective study of traumatic wounds involving both ponies and horses, of the 217 wounds in horses and 41 wounds in ponies closed by primary intention, 74% of those in horses and 59% of those in ponies dehisced.[5] Uncomplicated healing in a timely manner is not always a given. Several factors are known to complicate the process.

WOUND HEALING DIFFERENCES IN THE HORSE

Wound healing in horses can be distinguished from that in other animals by several unique characteristics, including marked *differences within the equine species, variations in the rate of healing based on body location,* and a great propensity for the *development of exuberant granulation tissue* during the healing process.

Ponies Heal Faster

The ability of ponies to heal more rapidly than horses was first reported in 1985 and confirmed later in a large retrospective study and a series of experiments.[5,65-69] These found both primary and second intention wound healing in ponies to proceed more rapidly than that of horses. In the experimental studies, 2×3.5 cm full-thickness wounds created on the metatarsus and buttocks of horses and ponies and allowed to heal by second intention yielded a quicker and more intense inflammatory response in ponies than in horses. Leukocytes produced higher levels of reactive oxygen species, interleukin-1, tumor necrosis factor, chemoattractants, and TGF-β1, likely explaining why ponies' wounds are more resistant to infection and why wound contraction is greater than in horses. In ponies, unlike horses, within 2 weeks after wounding, myofibroblasts were found organized and oriented parallel to the wound surface for optimal wound contraction.[68] Metatarsal bone involvement resulted in a greater periosteal reaction and new bone formation in horses than in ponies, leading to prolonged enlargement of their limbs.[66] In all five experimental ponies, body and limb wounds healed within 7 to 9 weeks, whereas only two body wounds in the five horses had healed by the conclusion of the 12-week study.[66]

Not surprisingly, outcome in clinical cases involving traumatic wounds undergoing primary closure was also found to be better in ponies than in horses. Wounds dehisced less frequently in ponies, and ponies developed fewer bone sequestra despite receiving, in many instances, less optimal treatment than their larger counterparts.[5] Based on the results of the experimental studies, the less intense but more chronic inflammatory response, which occurs in horses likely increases their risk for wound infection and for the development of exuberant granulation tissue, both of which can explain the clinical findings and, in general, their tendency for delayed wound healing. Although there is no definitive explanation for why these differences exist between horses and ponies, it is speculated that during domestication of the horse, humans took on the role of wound care provider, which decreased natural selection for efficient healing.[41] Pony breeds were spared because they were less popular and therefore subjected to less intensive breed selection. Lastly, horses incurring wounds precluding them from performing are often retired and kept as breeding stock, which would also contribute over time to the genetic selection for poor wound healing. Regardless of the reason, in patients with similar injuries, a better prognostic outcome should be associated with ponies over horses.[41]

Distal Limb Wounds

In horses, delayed healing of wounds on limbs compared to those involving the upper body has been recognized for many years.[40,70] Experimental, full-thickness, excisional wounds of the metacarpus or metatarsus allowed to heal by second intention have repeatedly been shown to heal more slowly than those of equal size created on the upper body.[40,66] Current knowledge indicates that this occurs because of differences in the rate of epithelialization and the rate of contraction, both of which are adversely influenced by excessive motion, infection, and the development of exuberant granulation tissue.[66] The latter is a result of an inefficient inflammatory response (in horses), an imbalance in collagen homeostasis, a shift towards a profibrotic

environment, microvascular occlusion, and inappropriate cell apoptosis.[71] For the process of epithelialization to proceed in a timely manner, keratinocytes require healthy granulation tissue on which to migrate. This is impaired by chronic inflammation, as is the process of wound contraction.[41]

Wound Expansion

Acute wounds in horses, regardless of their location, expand in size in the first 1 to 2 weeks because of the tensional forces of the adjacent tissues. Expansion can be significant. This contributes to the duration of healing.[65,72] In 2.5 × 2.5 cm full-thickness limb wounds, wound areas expanded 1.4 to 1.8 times the original size during the first 2 weeks.[73] This is then followed by progressive contraction of the granulation tissue bed, once it is formed, and a visible decrease in the wounded area, provided the process is undisturbed. In second intention healing, contraction is desirable; coverage of the wound site with full-thickness skin containing epidermal appendages is more cosmetic and durable than coverage by epithelium alone. Contraction rates of 58% to 76% for 2.5-cm^2 full-thickness lesions created on the metacarpal and metatarsal areas were reported.[74,75] With published rates of reepithelialization as slow as 0.09 mm/day for small experimental distal leg wounds, it is not surprising that traumatic clinical wounds require a prolonged period for healing.[76]

Effect of Motion

The shape of the wound does not influence the rate of contraction, but location does.[77] Wounds on the body contract more efficiently (0.8 to 1 mm/day) than those located on the legs (0.2 mm/day).[76] In addition, wounds in ponies contract more rapidly than those in horses.[66] Unlike wounds of the upper body, leg wounds commonly involve areas of high motion and high tension, or tissues that are poorly vascularized.[72] Wounds located over or adjacent to a joint, over tendons, or in opposition to the lines of skin tension contract more slowly or cease contraction before complete epithelialization, delaying wound healing.[40,65] Full-thickness 4 × 3 cm wounds created over the dorsum of the fetlock took significantly more time to heal compared to wounds of identical size over the metatarsus.[65]

Exposed Bone

The process is further delayed if bone is exposed, whether it is extensive, as with degloving injuries, or it involves a much smaller area. Exposed bone, devoid of periosteum, develops granulation tissue slowly because of the poor vascularity present.[78] Ironically however, development of granulation tissue occurs more rapidly in horses than in ponies.[66] In the interim, dessication of the bone's surface may lead to formation of a sequestrum, further delaying granulation tissue development and ultimately contraction and epithelialization.[78]

Infection

Infection also contributes to delays in wound healing and is the primary reason for wound dehiscence.[20] In contaminated traumatic wounds, those located on the limb are at a greater risk of infection than those of the upper body, because soil and fecal contamination are more likely in distal wounds. Soil components have been shown to reduce white blood cell effectiveness, decrease humoral defenses, and neutralize antibodies, thereby significantly reducing the number of bacteria needed to overburden the host's immune system. It has been reported that contamination with as few as 100 microorganisms in the presence of soil can result in infection.[79] As mentioned earlier, horses are unable to mount a rapid, intense inflammatory response after wounding, which facilitates the establishment of bacteria.[68] Regional differences in the number of tissue macrophages have been documented, less in the leg than in the neck, which may also affect the adequacy of the immune response and difference in healing rates.[68] Considering these findings and that feces may harbor up to 10^{11} bacteria per gram, it is not surprising that infection is often more problematic in the limb than body.[80] Use of systemic, regional, or topical antimicrobial therapy, or a combination of these three, is often warranted.

Development of Exuberant Granulation Tissue

Prolonged Inflammatory Phase

The development of exuberant granulation tissue can be considered both a cause and a result of delayed healing in traumatic wounds that are allowed to heal by second intention. Characterized by an abundance of capillaries surrounded by collagen, exuberant granulation tissue, or *proud flesh*, is a common development in wounds involving the limbs of horses managed by second-intention healing. The production of excess granulation tissue can be traced back to the horse's inefficient protracted inflammatory phase, which leads to an excessive proliferative phase in which fibroblasts retain their synthetic role rather than differentiate into myofibroblasts or disappear.[81] Although the influx of PMNs in horses was much slower than that seen in ponies, PMN numbers remained higher in horses than in ponies for a longer period of time, resulting in chronic inflammation.[68] It is hypothesized that the imbalance of the mediators released by PMNs, including TNF-α (tumor necrosis factor alpha), interleukin 1 and 6 (IL-1, IL-6), PDGF, TGF-β, and bFGF, contributes to a profibrotic state leading to the formation of exuberant granulation tissue.[41] TGF-β1 enhances migration and proliferation of fibroblasts and subsequent collagen production. It also delays fibroblast apoptosis.[82,83] In experimental limb wounds, its presence persists beyond the initial inflammatory phase, which is significantly different than in thoracic wounds.[84-86] Simultaneously, there is a downregulation of the MMPs required for collagen turnover and, in leg wounds compared to those of the thorax, an increase in tissue inhibitor of metalloproteinase (TIMP).[86] TIMP inhibits the activity of MMP-1. Granulation tissue becomes excessive, which contributes to wound expansion, delays contraction, and inhibits epithelialization (Figure 5-4).[60,66]

Microvascular Occlusion

Other mechanisms leading to exuberant granulation tissue also appear to be important. *Microvascular occlusion* of the small capillaries within granulation tissue has been documented (and found to be three times more likely to occur in limb wounds than in thoracic wounds).[81] The resultant local hypoxia signals upregulation of angiogenic and profibroblastic signals. Hypoxia stimulates the synthesis of TGF-β1, which in addition to its

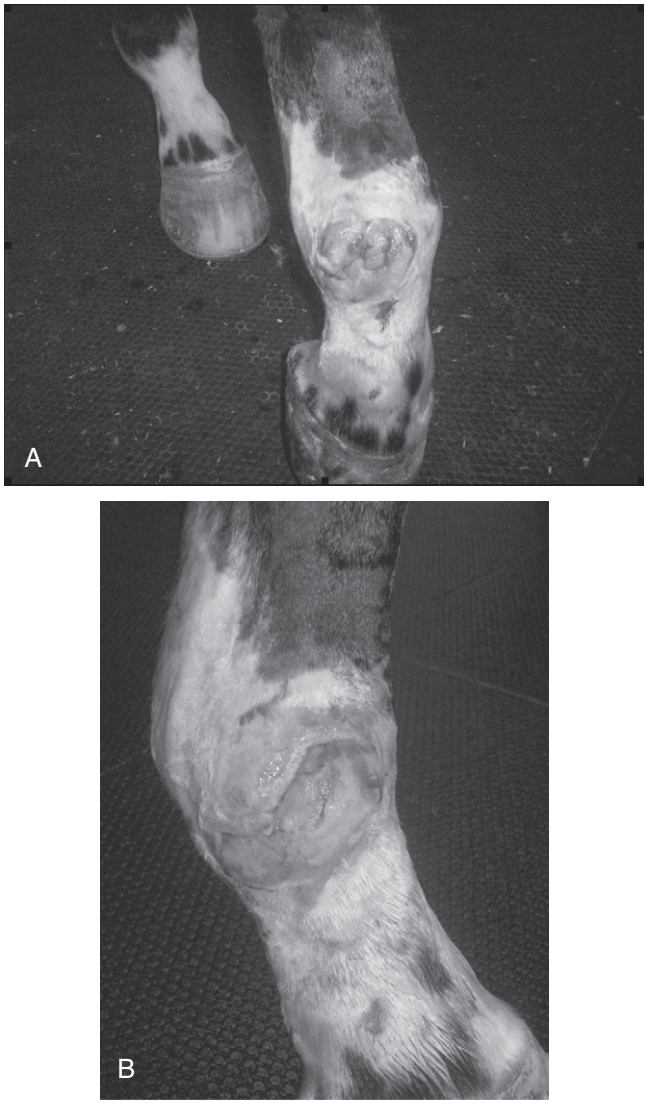

Figure 5-4. A, Traumatic wound over the dorsomedial aspect of the hind fetlock of several months duration. Chronic inflammation and movement has led to development of exuberant granulation tissue and fissures within the granulation bed. Wound contraction and epithelialization is delayed. **B,** Excessive granulation tissue has been excised to below the level of the adjacent skin edges to allow contraction and epithelialization to proceed. Removal of the excess granulation tissue also removed the fissures, which decreases the accumulation of exudates and bacteria that can lead to chronic inflammation and the development of exuberant granulation tissue.

antiapoptotic effect on fibroblasts, is an inhibitor of keratinocytes.[81] Keratinocyte migration is further delayed when the height of the granulation tissue exceeds that of the adjacent skin edges. In the absence of migrating keratinocytes, signaling for apoptosis of fibroblasts is delayed, thus perpetuating the development of granulation tissue.[25] Hence exuberant granulation tissue can be both the cause and the result of delayed wound healing.

Bandaging

Interestingly, bandaging of limb wounds in horses and ponies has long been associated with development of excessive granulation tissue and has been reported to be "detrimental to the goal of healing."[75] This has led to recommendations to eliminate its use when possible.[75,87] Bandaging contributes to local hypoxia, which stimulates angiogenesis, and to the accumulation of exudates on the dressing against the wound surface, which provide a constant source of inflammatory mediators. However, bandaging in clinical cases is often unavoidable and may be beneficial if used during an appropriate time frame. Bandaging can reduce environmental contamination, protect vital structures, provide mechanical stabilization, and reduce edema. Several studies have examined the effects of bandaging and dressing types.[73,75,88,89]

Although a moist wound environment is desirable in most species for optimum healing, this has not been found to be uniformly true in horses.[74,75] Wound dressing development in human health care is a multibillion dollar industry resulting in an abundant number of dressings that equine veterinarians can use. General guidelines are to use occlusive dressings in clean, acute wounds until a healthy bed of granulation tissue develops, then switch to a semiocclusive dressing. In dirty or infected wounds, adherent, hydrophilic, or antimicrobial dressings should be used until healthy granulation tissue develops. The use of a semiocclusive dressing should then follow (for more information on the management of wounds see Chapters 26 and 27).[90]

Management of Granulation Tissue

Control of exuberant granulation tissue should be aimed at minimizing inflammation once healthy granulation tissue fills the wound site. Excessive granulation tissue can be managed by excising it when it protrudes above the wound margins.[87] When this method is employed as needed, no delay in healing occurs regardless of bandaging.[75,91] For wounds that need to be bandaged beyond the initial development of the granulation bed, but in which excision of granulation tissue is undesirable, use of either topical corticosteroids or a nonadherent silicone dressing (CicaCare, Smith-Nephew Canada Inc, St-Laurent, QC, Canada) have been shown to be successful at eliminating development of exuberant granulation tissue.[92,93] Equine amnion applied as a dressing is another option. It has been shown in some but not all studies to decrease development of granulation tissue and to accelerate epithelialization.[74,88] Methods for collection and storage of amnion have been reported.[88] Proponents recommend applying amnion after a healthy granulation bed has developed.[90] Skin grafting and delayed closure techniques are strongly recommended in all large granulating wounds to reduce their area and associated inflammation to eliminate the problem of exuberant granulation tissue (see Chapter 25).[94]

GENERAL FACTORS THAT INFLUENCE WOUND HEALING

To further optimize wound healing in the horse, it is important to acknowledge not only the differences unique to the species but also to appreciate other general factors and management techniques that are known to influence wound healing. Many of the factors cannot be manipulated to the benefit of healing, such as the type of injury incurred or the nutritional status of the patient at the time of injury, but they should remain thought-provoking when determining a treatment plan for a given patient.

Age

Although advancing patient age is known to influence the rate of healing in humans and in many experimental animal models, this has not been investigated in horses.[95,96] In humans as well as companion animals, with increasing age many comorbid conditions are encountered, including diabetes, chronic renal insufficiency, cardiac insufficiency, and acute or chronic liver disease, that are known to affect healing. These, however, with the exception of Cushing's disease, are generally not age-related problems common to horses.[97] In horses with pars intermedia dysfunction, high endogenous cortisol levels may delay wound healing through suppression of the inflammatory phase and increase the risk of wound infection because of immunosuppression.[97]

Nutritional Status

Tissue repair is an anabolic process, and data suggest that healing may be improved with diets containing adequate protein.[98,99] Malnutrition preceding surgery or at the time of trauma can greatly influence outcome. In animal studies, protein deficiency directly delayed the rate of wound healing through the suppression of fibroblast proliferation, angiogenesis, collagen synthesis, and remodeling.[100] In a large study involving war veterans, low preoperative serum albumin level was identified as the most significant variable for predicting surgical complications, including wound infection and acute wound failure.[101] Although comparable studies do not exist for the horse, it seems reasonable to expect similar results. Vitamins and micronutrients are also known to affect healing when either deficient or in excess.[4,102] Vitamin A is essential for normal cell differentiation, and deficiencies can result in impaired collagen synthesis and cross-linking and in delays in epithelialization.[103] Vitamin C and the B vitamins (thiamine, pyridoxine, and riboflavin) are important cofactors in collagen cross-linking reactions, whereas vitamin E stabilizes cell membranes. Iron not only is necessary for red blood cell production but also is required as a cofactor in collagen synthesis. Zinc is a cofactor in many enzymatic reactions including DNA and protein synthesis. All of these mechanisms are necessary steps in the healing process.

Type of Injury

Injuries can be classified into one of seven types based on cause (see Table 5-1). The greater the force of impact, the greater the soft tissue damage will be, and the greater the risk of subsequent wound infection.[104,105] Of the seven types, those with the least risk of developing infection are caused by sharp objects (e.g., an incision, a laceration caused by a nail). Contusion and crush injuries, which often include vessel thrombosis, are those most prone to infection. Puncture wounds, although seemingly innocuous, often develop infection because the puncture tract heals at the surface before the deeper soft tissues, thereby creating an ideal environment for bacterial growth. Horses with these latter types of injuries are also most prone to developing tetanus. In general, infection prolongs wound healing, decreases wound tensile strength, and is the most common reason for wound dehiscence.[20,106]

Tissue Perfusion

Wound healing depends on adequate arterial circulation to supply tissue with oxygen. The surgical practice of débriding wounds until bleeding tissue is reached is supported by clinical and experimental findings that "healing progresses more quickly in optimally perfused tissues."[4] In human patients, transcutaneous oxygen tension ($TcPO_2$) and tissue oxygen levels are good indicators of ischemia and can be used to predict healing.[107,108] Repair processes, including fibroblast replication, collagen production, and epithelialization, are impaired when $TcPO_2$ is less than 40 mm Hg; with tensions less than 10 mm Hg, tissues die.[21] Anemia has less of an impact on wound healing, provided blood flow to the wound is maintained and the patient is able to increase cardiac output. Even profound hemodilution does not appear to interfere with wound healing.[109] However, shock and hypotension, even if brief, can negatively impact wound healing.[4] Tissue oxygen tensions can be improved provided arterial circulation is intact by increasing the fraction of inspired oxygen and by increasing the pressure at which oxygen is delivered, as with hyperbaric oxygen therapy (HBOT). However, if arterial circulation to the wound is interrupted, the two management actions proposed earlier will not improve oxygen tension within the wound.[110] Use of HBOT has shown benefits in human surgery and in many skin graft animal models, but no advantage over nontreated horses was found in experimental acute skin grafting studies.[111,112] Horses receiving HBOT had diminished neovascularization, which affected graft take. Angiogenesis and the delivery of oxygen remain necessary steps in the process of wound healing.

Hemostasis and Hematoma Formation

Seromas and hematomas impede wound healing by mechanically distracting the wound edges, by reducing capillary perfusion secondary to exertion of pressure, and by increasing the risk of infection.[4] The incidence of acute hematoma formation can be influenced by surgical technique (Halsted's principles—see Chapter 12). A surgical plan that minimizes undermining of tissue edges and includes techniques that minimize dead space should be pursued. Drains should be placed in areas that are at risk of fluid accumulation and removed when nonproductive.[113] Electrocautery should be used judiciously because excessive use can delay wound healing.[114] Within the last 10 years, vacuum-assisted wound closure (see Figure 17-8) has become commonplace in human medicine. The technique applies negative pressure to the wound and removes accumulated fluid. It has been shown to promote wound healing in part by decreasing the duration of wound drainage and by reducing hematoma development.[115] Its use for treatment of deep cervical wounds in a horse has been reported.[116] The procedure was tolerated well and resulted in the horse returning to light work within 4 weeks. Other benefits attributed to vacuum-assisted wound closure include improved wound perfusion and decreases in wound infection rates.[115] In select cases, incorporation of vacuum-assisted wound closure may be advantageous.

Débridement

Early wound débridement affects wound healing positively. The goal is to reduce bacterial numbers, foreign debris, and the necrotic tissue that would otherwise need to be removed during the cellular inflammatory phase. Repeated débridement benefits chronic and indolent wounds. Fibroblasts within these wounds become senescent. Surgical removal can initiate the healing

process by resulting in platelet accumulation, thereby re-initiating the wound-healing process.[4]

Débridement can be performed surgically using a scalpel, CO_2 laser, or hydrosurgical unit or nonsurgically with dressings, topical compounds, or maggots.[104] Surgical débridement has the advantage of being quick but can be imprecise and painful. Serial or staged sharp débridement over a period of several days can reduce the uncertainty by allowing time for wounded tissues to clearly demarcate themselves as either healthy or not.

Nonsurgical débridement can be divided into mechanical, chemical (enzymatic and nonenzymatic), and autolytic methods, all of which are slower than sharp dissection but in general are tissue sparing and less painful. Wet-to-dry dressings mechanically débride the surface of the wound when removed without re-wetting. This method is efficient at removing fibrin but can also remove newly formed epithelial cells if use is continued too long. Mechanical débridement can also be achieved using wound irrigation. For maximum benefit, fluid should be delivered at an oblique angle to the tissue surface and at a pressure of 7 to 15 pounds per square inch.[104,117,118] A 35-mL syringe combined with a 19-gauge needle is a simple tool that meets these guidelines, although other methods may also be employed.[104] There are also battery-operated handheld pulsed irrigation units with a variety of irrigation tips (e.g., Interpulse, Stryker Corporation, Kalamazoo, MI) that are convenient to use. Autolytic débridement is achieved by placing an occlusive dressing over the wound, trapping the body's own proteases within the wound to liquefy necrotic tissue. Granulex spray, meat tenderizers containing papain and bromelain, and papain/urea-based proteinase are examples of chemical débridement agents. Granulex, which contains trypsin, peruvian balsam, and castor oil, is the product more commonly used in veterinary medicine. It is reported to hydrolyze a variety of proteins, increase perfusion, and possibly promote epithelialization.[119] Collagenase-containing products digest collagen and elastin but do not degrade fibrin.[4] The papain/urea combination degrades fibrin and denatures collagen and skin.[4] Their use therefore is not appropriate for all wounds. Traditional gauze dressings hydrated in saline were found to be 47% more effective in removing fibrin in blood clots from horses than enzymatic formulations.[120]

A unique method of débridement is to use sterile maggots from the common green bottle fly *Lucilia sericata*. Maggots produce potent proteolytic enzymes and can consume up to 75 mg of necrotic tissue per day.[121-123] In addition, they are capable of destroying bacteria.[123] Maggots can be applied to the wound in either a direct (free range) or indirect (contained) manner. Successful outcomes have been associated with their use in penetrating hoof wounds of the horse (see Chapters 26 and 27 for more information on wound dressings).[124]

Wound Closure Technique

The appropriate size and type of suture for a given wound site should be selected. The goal should be to select a suture that is similar in strength to the tissue in which it is to be used.[125] Appropriate selection limits the foreign body effect that each suture possesses, and therefore the risk of infection.[126,127] Suture placement should be directed at minimizing excessive tension at skin edges. Blood flow to the skin edge is inversely proportional to the wound closure tension.[128] Suture tension, which increases the interstitial pressure within the center of the incision above capillary pressure (30 to 40 mm Hg), can lead to tissue necrosis. Study results examining the effects of suture tension on incision strength over time favored loosely apposed skin edges.[129] In most tissue locations simple interrupted sutures are preferred if excessive tension is present and there is a potential of impaired wound healing.[130]

Topical Therapy

A plethora of topical products available to horse owners and veterinarians claim to improve wound healing. Unfortunately some are beneficial and some are not. Treatment choice can affect outcome. Selection should be based on sound information regarding the effects of the product selected and the phase of wound healing. Use of commercially available soaps, such as Ivory or Dove, should be avoided in favor of wound cleansers with neutral pH.[131] Low pH, such as that occurring with products containing benzethonium chloride, is associated with cell toxicity. Tap water can be safely used initially during cleaning to reduce bacterial load, but it should be replaced with an isotonic fluid once a granulation tissue bed has developed to avoid cellular swelling and destruction.[119,132-135] Fluids should be warmed to approximately 30° C to prevent vasoconstriction, which may cause further tissue ischemia.[136] Antiseptics, such as chlorhexidine diacetate and povidone-iodine (10%), should be diluted appropriately when added to lavage solutions. Chlorhexidine solutions (2%) diluted to 0.05% (25 mL/975 mL solution) or less is recommended.[137] Concentrations higher than this are cytotoxic to both tissue and bacteria.[138] If povidone-iodine is used, it should be diluted to a concentration of 0.1% to 0.2% (10 to 20 mL/L).[139-141] Concentrations greater than this have been shown to be toxic to canine fibroblasts, lymphocytes, and monocytes and to inhibit neutrophil migration. Concentration of the antiseptic ointments and gels should also be kept in mind when used topically. Povidone-iodine ointment (10%) had deleterious effects on wound healing in human patients, but in a study in horses, no delay was encountered.[75,142] Lastly, hydrogen peroxide is cytotoxic to fibroblasts and its routine use cannot be recommended.[143]

When selecting a topical antibiotic for use, knowledge of its antimicrobial spectrum and the potential complications should be considered before choosing. Triple antibiotic ointment (bacitracin, polymixin B, and neomycin) and silver sulfadiazine (SSD) have broad spectrums of activity, but silver sulfadiazine, unlike triple antibiotic, is effective against *Pseudomonas* spp. and fungi. Both have been reported to increase epithelialization but both may decrease wound contraction.[119] When used in combination with a bandage, investigators found SSD cream increased development of exuberant granulation tissue.[75] Gentamicin sulfate has a narrow spectrum of activity, primarily against gram-negative organisms. The 0.1% oil-in-water cream is reported to slow wound contraction and epithelialization.[141,144] The use of nitrofurazone ointment, despite its broad spectrum of antimicrobial activity, has several drawbacks.[145] It has been shown to decrease epithelialization and to delay wound contraction. It also possesses carcinogenic properties.[119]

Topical application of individual growth factors has had generally disappointing results during attempts to accelerate wound healing in horses. Recombinant TGF-β1 was selected to stimulate granulation tissue development and enhance wound

contraction in a second-intention wound healing model in horses. No benefit was found over untreated wounds.[146] Platelet-rich plasma (PRP) on the other hand has shown promise. Platelets are rich in TGF-β, PDGF, epidermal growth factor (EGF), transforming growth factor-α (TGF-α), VEGF, serotonin, and histamine. They also secrete fibrin, fibronectin, and vitronectin, which act as provisional matrix and provide a surface for epithelial migration. This characteristic of platelets may explain the positive advantage of PRP over that of topical use of individual cytokines.[147,148] In PRP, platelet numbers are increased over that of whole blood, increasing TGF-β1 concentration nearly threefold.[149] In rabbits, the application of PRP to the full-thickness skin wounds improved overall healing in full-thickness wounds by reducing contraction, stimulating angiogenesis, and producing a trend toward more rapid epithelialization.[148] PRP has been used for the treatment of a variety of equine musculoskeletal pathologies and was reported to induce accelerated epithelial differentiation and well-organized collagen bundles in healing skin wounds.[147] In a larger study, no improvement was found in the quality or speed of wound healing in the treatment of experimental acute 6.25 cm^2 wounds in horse limbs.[149] The authors of this latter study speculate that PRP use may be more appropriate for larger or more chronic wounds. Harvesting autologous PRP is quick and relatively inexpensive and its use may be warranted in many cases.[94]

Various other wound products are also available. Many have little but anecdotal support for their use. Application of products containing lye, gentian violet, or pine tar can lead to further damage of wounded tissues and are not recommended.[119] Other products can be beneficial when used during the appropriate wound phase. Ketanserin-containing products (Vulketan gel, Jannsen Animal Health, Toronto, Canada) block serotonin-induced macrophage suppression and vasoconstriction and can be used during the inflammatory phase to promote a strong inflammatory response.[150] Acemannan, the active ingredient of aloe vera, stimulates macrophages to release fibrogenic and angiogenic cytokines. Its use can be beneficial during the inflammatory phase and early period of fibroplasia and will accelerate the development of granulation tissue over exposed bone.[143,151] Once a granulation bed has developed, its use should be discontinued. In the later phases of wound healing, the use of topical corticosteroids may be warranted to limit fibroblast and endothelial cell proliferation.[93,152] Lanolin cream may be useful to increase the rate of epithelialization.[153] Identifying the phase of wound healing and understanding the product being used is important to facilitate rather than impede the process of wound healing.

Pharmaceuticals

Many drugs are known to impair wound healing. Chemotherapeutic drugs, which target rapidly dividing cells, comprise the largest group. Based on information from human medicine, risks for wound complications are greatest when drugs are given preoperatively, although drug, dose, and frequency also matter.[154] Data in horses receiving biweekly local treatment of cisplatin (1 mg/cm^3) during the perioperative period did not reveal any adverse affect on wound healing. Rate of epithelialization was similar to that reported in other wound-healing studies, although some primarily sutured wounds developed partial dehiscence.[155]

Local Anesthetics

Local anesthetic agents are commonly used to facilitate wound cleansing, débridement, and suture repair in standing equine patients. The use of 2% mepivacaine or lidocaine is most common. Studies in animal wound healing models report conflicting results on the impact that surgical wound infiltration of local anesthetics have on healing. In a rat model, use of 2% lidocaine was found to reduce wound breaking strength and to impair healing of acute wounds.[156,157] In another study, 1% lidocaine had no effect on wound breaking strength at 8 days after wounding.[158] In a recent study, wounds treated with local infiltration of lidocaine (0.5% or 1%) or bupivacaine (0.25% or 0.5%) healed at similar rates to control wounds when wound areas and extent of reepithelialization were compared. Neutrophil numbers increased in a dose-dependent manner.[159] However, a trend was seen by the third day for reduced collagen levels and an increase in MMP-2 (collagenase).[159] Based on the available literature, it seems reasonable whenever possible to avoid local infiltration of anesthetic in areas where wound breaking strength is important, even when diluted. Because of its vasoconstrictive effects, adding epinephrine to local anesthetics should also be avoided.

Anti-Inflammatory Drugs

Anti-inflammatory drugs, in general, inhibit the normal inflammatory response to wounding. Systemic and local use of glucocorticoids have global effects: decreased fibroblast proliferation, protein synthesis, and wound contraction; inhibition of keratinocyte growth factor (KGF) production; and reduced angiogenesis.[160-162] Single-dose administration of a therapeutic dose at the time of surgery likely has no untoward effect, but frequent administration or high concentrations can lead to impairment. Chronic behavioral stress has also been shown to suppress inflammatory gene expression during early wound healing, resulting in delayed healing.[163]

Administration of nonsteroidal anti-inflammatory drugs (NSAIDs), through repression of cyclooxygenase (COX) activity, has been implicated in several studies to adversely affect migration and degranulation of neutrophils, angiogensis, infection rate, and healing.[162-168] In ponies, flunixin meglumine administration delayed linea alba repair.[169] The decision to use an NSAID during wound healing should be made on a case-by-case basis and tailored according to the phase of wound healing. If possible, NSAIDs should be avoided during the inflammatory phase because the influx of inflammatory cells and mediators are important for efficient healing. This, however, must be balanced with the need to control pain and minimize tissue swelling, which may further contribute to tissue ischemia.

Malignancy

Neoplastic transformation should be ruled out in all chronic nonhealing wounds. Squamous cell carcinoma and equine sarcoid can be similar in appearance to granulation tissue. Both are known to occur at previous wound sites.[170]

SUMMARY

Wound healing is a dynamic process involving complex interactions between cellular and biochemical events that coordinate healing. In the horse it is important to support an initial strong

inflammatory response and to prevent chronic inflammation for optimum results. Hippocrates stated, *"Healing is a matter of time, but it is sometimes also a matter of opportunity."*[171] Although wound healing is a physiologic process, our actions can directly influence it, positively or adversely. Understanding the basics of wound healing can lead to improved patient outcome.

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CHAPTER

6 Metabolism and Nutritional Support of the Surgical Patient

Elizabeth A. Carr

Tremendous advances in the care and treatment of the critically ill equine patient have occurred during the last two decades. Survival rates from colic surgery have increased, and large animal intensive care units are found in most, if not all, major university hospitals and referral practices across the United States and most of Europe. Critical to this success is presurgical evaluation and patient triage. The physical examination data as well as laboratory and diagnostic information are carefully collected and analyzed to determine the severity of the disease process. Any underlying abnormalities and any metabolic derangements that may affect the outcome negatively are carefully determined. Patients may receive fluids, anti-inflammatories, colloids, oxygen insufflation, and other medications before anesthesia to ensure that a hemodynamically and metabolically stable patient is taken to the induction stall. The surgical technique is designed to minimize trauma, resolve the underlying problem, and keep postoperative complications at a minimum. After recovery, the patient usually continues a regimen of intravenous fluids to maintain hydration and other therapeutics that minimize or prevent postoperative complications, including ileus, pain, and infection, and maximize the chance of recovery. Despite this proactive approach to treatment and support of adult equine patients, rarely is their nutritional status considered in the initial therapeutic plan. Preoperative and postoperative nutritional status and support are clearly linked to morbidity and mortality in humans.[1,2] Malnutrition has been shown to reduce survival, immune function, wound healing, and gastrointestinal function, and it probably negatively affects numerous other processes.[2-6]

This chapter will discuss the metabolic consequences of food deprivation, the pathologic metabolic responses to illness, nutritional requirements in health and disease, and the indications for and types of nutritional supplementation.

INDICATIONS FOR NUTRITIONAL SUPPORT

The need for interventional nutritional support depends on a number of factors. The healthy adult horse that is undergoing elective surgery and has a body condition score of 4 or 5 (out of 9) rarely requires nutritional supplementation (Box 6-1). These individuals can easily tolerate food deprivation for 48 hours. The majority of healthy adult horses undergoing elective surgery have food withheld for 6 to 12 hours preoperatively, and it is reintroduced after recovery when the animal is deemed capable of eating and swallowing effectively. During this period of starvation, energy demands are met by glycogen reserves, with little effect on overall metabolism.

Regardless of the type and complexity of the surgical procedure, nutritional support should be considered in patients with an increased metabolic rate (e.g., young growing animals), individuals presenting with a prior history of malnutrition or hypophagia, patients with underlying metabolic abnormalities that could worsen with food deprivation, and individuals with disorders such as severe trauma, sepsis, or strangulating bowel obstruction that result in an increased energy demand. Underweight horses require nutritional support earlier. Obese or overconditioned individuals, particularly pony breeds,

Box 6-1. Body Condition Score

1. Poor: Animal extremely emaciated. Spinous processes, ribs, tailhead, hooks, and pins projecting prominently. Bone structure of withers, shoulders, and neck easily noticeable. No fatty tissue can be felt.
2. Very thin: Animal emaciated. Slight fat covering over base of spinous processes, transverse processes of lumbar vertebrae feel rounded. Spinous processes, ribs, tailhead, hooks, and pins prominent. Withers, shoulders, and neck structures faintly discernible.
3. Thin: Fat buildup about halfway on spinous processes, transverse processes cannot be felt. Slight fat cover over ribs. Spinous processes and ribs easily discernible. Tailhead prominent, but individual vertebrae cannot be visually identified. Hook bones appear rounded but easily discernible. Pin bones not distinguishable. Withers, shoulder, and neck accentuated.
4. Moderately thin: Negative crease along back. Faint outline of ribs discernible. Tailhead prominence depends on conformation; fat can be felt around it. Hook bones not discernible. Withers, shoulders, and neck not obviously thin.
5. Moderate: Back level. Ribs cannot be visually distinguished but can be easily felt. Fat around tailhead beginning to feel spongy. Withers appear rounded over spinous processes. Shoulders and neck blend smoothly into body.
6. Moderate to fleshy: May have slight crease down back. Fat over ribs feels spongy. Fat around tailhead feels soft. Fat beginning to be deposited along the sides of withers, behind the shoulders, and along the sides of the neck.
7. Fleshy: May have crease down back. Individual ribs can be felt, noticeable filling of fat between ribs. Fat around tailhead is soft. Fat deposited along withers, behind shoulders, and along the neck.
8. Fat: Crease down back. Difficult to feel ribs. Fat around tailhead very soft. Area along withers filled with fat. Area behind shoulder filled in flush. Noticeable thickening of neck. Fat deposited along inner buttocks.
9. Extremely fat: Obvious crease down back. Patch fat appearing over ribs. Bulging fat around tailhead along withers, behind shoulders, and along neck. Fat along inner buttocks may rub together. Flank filled in flush.

Scoring is based on visual appraisal and handling (particularly in scoring horses with long hair).

miniature horses, and donkeys, as well as lactating mares are at risk for developing hyperlipemia and should receive nutritional support if their serum triglycerides are higher than normal values. Older horses, or individuals diagnosed with equine Cushing's syndrome or equine metabolic syndrome, are insulin resistant and at greater risk for developing hyperlipemia and fatty infiltration of the liver. If food deprivation is prolonged or there is a concern regarding the individual's desire or ability to eat, early intervention is indicated to prevent more severe malnutrition.

PURE PROTEIN/CALORIE MALNUTRITION

The average, healthy adult horse can easily tolerate food deprivation (pure protein/calorie malnutrition [PPCM] or simple starvation) for 24 to 72 hours with little systemic effect. A decline in blood glucose concentration occurs with food deprivation, insulin levels fall, and energy demands are initially met via glycogenolysis, which increases the breakdown of liver glycogen stores. As starvation progresses, glycogen is mobilized within other tissues, including muscle. Lipid mobilization is triggered by alterations in insulin or glucagon levels and the activity of hormone-sensitive lipase. As glucose becomes limited, many body tissues begin to rely on fatty acid oxidation and the production of ketone bodies as energy sources. Glycerol produced from lipid degradation, lactate from the Krebs cycle, and amino acids from muscle tissue breakdown continue to be used for gluconeogenesis to provide energy to glucose-dependent tissues (central nervous system and red blood cells). This response to starvation correlates with an increase in circulating levels of growth hormone, glucagon, epinephrine, leptin, and cortisol and a decrease in insulin and thyroid hormones. These hormone fluxes are an afferent stimulus for the hypothalamic response to starvation, which increases the drive to eat and decreases energy expenditure. Metabolism slows in an effort to conserve body fuels, and the body survives primarily on fat stores, sparing lean tissue.

Individuals with preexisting PPCM are at a disadvantage when intake is restricted because of surgery or illness. In the malnourished or cachectic human patient, presurgical nutritional supplementation has been shown to positively influence both survival and morbidity. Early nutritional supplementation should be strongly considered in animals presenting with preexisting PPCM.

METABOLIC RESPONSE TO INJURY

The metabolic response to injury (e.g., surgical manipulation, critical illness, sepsis, trauma), unlike the response to PPCM, is characterized by an increased metabolism and the onset of a catabolic process leading to excessive breakdown of tissue proteins. This metabolic state results from a complex interaction of inflammatory cytokines (interleukin [IL]-1, IL-2, IL-6, tumor necrosis factor [TNF]-α, and γ-interferon; see Chapters 1 and 2) released at the site of injury or inflammation, circulating hormones released in response to stress and injury (hypothalamic-pituitary-adrenal axis), and neurotransmitters (sympathoadrenal axis).[7] Infusion of cytokines including IL-6 and TNF-α results in stimulation of corticotrophin, cortisol, epinephrine, and glucagon, leading to an increase in the resting metabolic rate and lipolysis.[8,9] TNF-α activation of nuclear factor kappa B (NFκB) results in stimulation of proteolytic pathways.[10] In response to injury, there is an increased metabolic activity of the brain. Afferent nerve activity and brain stimulation may cause autonomic nerve stimulation with direct effects on hormone secretion; for example, splanchnic nerve stimulation caused by injury increases glucagon secretion and hyperglycemia.[11] Afferent nerve activity from the injured site also results in hypothalamic-pituitary activation, increasing activity of cortisol, catecholamines, growth hormone, aldosterone, and antidiuretic hormone.[7] In fact, in humans, prolonged infusions of glucagon, cortisol, and epinephrine increase protein breakdown and elevate resting metabolic rate.[12] Prolonged elevation of cortisol is associated with onset of insulin resistance. In addition, peripheral nerve endings have been shown to exist on adipocytes, and the stimulation of adipocytes increases lipolysis.

During illness or after trauma, food intake frequently falls. However, despite this decline, the adaptive responses to starvation do not occur. Hepatic gluconeogenesis continues and rapid protein catabolism develops. There is an increased mobilization of stored fuels and metabolic cycling, resulting in heat production and energy loss. Insulin resistance develops and hyperglycemia may occur despite the absence of food intake. In severe metabolic stress, the body appears to preferentially use skeletal muscle as a metabolic fuel (as opposed to the situation in PPCM, when fat metabolism is the principal source of energy). The adaptive switch to fat use is limited, in part because of increased levels of circulating insulin. The result is an increase in lean tissue breakdown, visceral organ dysfunction, impaired wound healing, and immunosuppression.[7,13] Nitrogen losses during this catabolic response may be as high as 20 to 30 g/day versus 4 to 5 g/day in an adult human experiencing PPCM. Excess protein breakdown and muscle disuse because of inactivity cause muscle weakness and increased morbidity. Because sodium and water retention are a component of this response, weight loss frequently goes unnoticed. Cytokine production results in behavioral changes, including anorexia and decreased activity. Food deprivation during this hypermetabolic and catabolic state causes a much greater loss of lean muscle mass and visceral protein than would be expected during simple starvation. A healthy human allowed access to water can survive approximately 3 months with food deprivation or PPCM. In contrast, the same individual with a critical illness would survive approximately 1 month, and those with preexisting malnutrition, less than 2 weeks.

Although nutritional supplementation will reverse the catabolic processes occurring during simple starvation, it will not completely reverse those occurring during metabolic stress. As long as tissue injury persists, catabolic processes are maintained. In the critically ill patient, protein catabolism continues despite protein supplementation in the diet. However, nutritional supplementation does have benefits in minimizing the severity of protein loss, providing both essential and conditionally essential amino acids, vitamins, and minerals, and in decreasing morbidity associated with illness.

Although the metabolic response to surgical injury is not likely to be as severe as that expected with sepsis, severe trauma, or other critical illnesses, an increase in metabolic rate is seen postoperatively in humans undergoing simple elective surgery. The combination of an increased energy demand and the metabolic processes already discussed can cause significant loss of lean body mass. These changes may not affect survival, but they can inhibit the return to performance of a competitive

athlete. In equine patients with severe surgical trauma, prolonged recoveries, or complications such as infection and laminitis, food deprivation almost certainly affects overall recovery.

METABOLIC REQUIREMENTS

The total energy of a feedstuff is divided into the digestible energy (DE) and the nondisgestible energy. Digestible energy is further divided into metabolic energy (which is used to provide energy) and lost or nonmetabolizable energy, such as gases produced and urea excreted in the urine. By convention, energy requirements are calculated in terms of digestible energy.

Adults

The amount of DE needed to meet the maintenance energy requirements (DE_m) of the normally active, nonworking horse can be estimated using the following formulas:

- For horses weighing less than 600 kg,

$$DE_m\ (Mcal/day) = 1.4 + (BW \times 0.03)$$

- For horses weighing greater than 600 kg,

$$DE_m\ (Mcal/day) = 1.82 + (BW \times 0.0383) - (BW \times 0.000015)$$

where BW is body weight in kilograms, and 1 Mcal equals 1000 kcal. Alternatively, these requirements can be estimated to be approximately 33 kcal/kg/day.

The resting energy requirement (DE_r) is the amount of energy required for maintenance (neither weight gain nor weight loss) of the completely inactive animal and is determined using a metabolism stall in a thermoneutral environment. The result is approximately 70% of the maintenance energy, and it can be calculated using the following formula:

$$DE_r\ (Mcal/day) = (BW \times 0.021) + 0.975$$

The maintenance energy requirements of a horse can be affected by several factors, including its age, size, and physical condition; the amount and type of activity; and environmental factors. Even when all these factors are controlled, individual variation occurs.

Increased Energy Demand

Energy requirements in the pregnant mare do not significantly increase until late gestation and are estimated to be 1.1, 1.13, and 1.2 times the DE_m, respectively, in the last 3 months of gestation. During lactation, energy demands peak over the first 3 months and then decline toward weaning. They can be calculated using the following equations:

- In the first 3 months of lactation
  - For 300- to 900-kg mares,

$$DE\ (Mcal/day) = DE_m + (0.03 \times BW \times 0.792)$$

  - For 200- to 299-kg mares,

$$DE\ (Mcal/day) = DE_m + (0.04 \times BW \times 0.792)$$

- After 3 months of lactation
  - For 300- to 900-kg mares,

$$DE\ (Mcal/kg) = DE_m + (0.02 \times BW \times 0.792)$$

  - For 200- to 299-kg mares,

$$DE\ (Mcal/day) = DE_m + (0.03 \times BW \times 0.792)$$

The energy and protein requirements for the hospitalized surgical patient are not known and probably vary depending on disease state, environment, and level of fitness of the individual. However, they are likely to be close to the resting or maintenance energy requirements. In humans, multipliers have been used to estimate the energy requirements in certain conditions, including severe sepsis, trauma, and burn injuries. However, the increased metabolic demands of illness or surgical trauma and recovery are likely to be balanced by the inactivity of the patient during hospitalization. Consequently, these multipliers may overestimate the caloric requirement of certain illnesses. The exceptions to this are individuals with extreme trauma, burns, or severe sepsis; surgical conditions that require intestinal resection; and patients with large areas of devitalized tissue (e.g., patients with clostridial myositis undergoing multiple fasciotomies). When estimating the energy requirements of the majority of surgical patients, resting energy requirements are an acceptable target. If the patient tolerates nutritional supplementation at this rate, the amount can be gradually increased to meet maintenance needs.

Foals and Weanlings

Foals and young horses that are growing have the highest energy demands. Mare's milk has been reported to provide between 500 and 600 kcal of energy per liter. A healthy 1-week-old neonatal foal drinks between 20% and 30% of its body weight in milk a day, which means that a 45-kg foal drinking 9 to 13.5 L of mare's milk consumes between 4500 kcal (4.5 Mcal) and 7800 kcal per day. This equates to a metabolic rate of between 100 and 173 kcal/kg/day. The resting metabolic rate in the healthy sedated foal has been calculated to be between 45 and 50 kcal/kg/day. As previously discussed, it is unclear whether a sick individual truly has a higher metabolic rate than a healthy individual. A recumbent sick foal is expending significantly less energy than its healthy counterpart in terms of activity level, but disease and its effect on metabolic rate and catabolism must be considered. As with adults, it is probably best to start nutritional supplementation at approximately the DE_r, particularly if starting with the enteral route. If supplementation is tolerated, it is recommended that this be gradually increased toward growth requirements over a shorter time than might be used to increase the adult animal's caloric intake. If using mare's milk or a milk replacement of similar caloric content, DE_r would equate to feeding the equivalent of 10% of the foal's body weight per day. Clinical experience suggests that this would be sufficient in the initial 12 to 24 hours, but additional nutritional support would be required to ensure adequate intake for healing and growth.

The largest growth rate occurs during the first month of life.[14] The following formula can be used to estimate and adjust the

energy requirement, in Mcal DE per day, for growth of weanlings and young growing horses:

$$DE_m + \{[4.81 + (1.17 \times M) - (0.023 \times M^2)] \times ADG\}$$

where M is months of age, ADG is average daily weight gain in kilograms, and BW is body weight.

More comprehensive reviews related to the metabolic needs of active and young growing horses are available for readers who need them.[15-17]

PROTEIN REQUIREMENTS

Protein intake must be adequate not only for energy requirements but also to ensure that protein catabolism is minimized. Maintenance requirements for crude protein (CP) in the adult horse can be estimated using the following equation:

$$CP\ (\text{in grams}) = 40 \times DE_m\ (\text{in Mcal/day})$$

For example, a 500-kg horse with a DE_m of 16.5 Mcal/day would require 660 g of protein per day. Alternatively, protein requirements can be estimated as 0.5 to 1.5 g protein per kilogram of the horse's body weight per day, or 250 to 750 g/day for a 500-kg horse. The middle to higher end of this estimate should be used when calculating protein needs in a sick patient.

VITAMIN REQUIREMENTS

Vitamins are organic compounds that are important in many enzymatic functions and metabolic pathways. Fat-soluble vitamins include vitamins A, K, D, and E. Water-soluble vitamins include the B vitamins and vitamin C. Vitamin K and all the B vitamins with the exception of niacin are synthesized by the microbial population in the horse's large colon and cecum. Vitamin D, vitamin C, and niacin are produced by the horse, whereas the precursors to vitamin A, β-carotene, and vitamin E must be ingested. The need for supplemental vitamins and minerals depends on the type and duration of supplementation. Fat-soluble vitamins are stored in body tissues and generally do not require supplementation for short periods of anorexia. Complete pelleted diets have vitamins and minerals added to meet the requirements set by the National Research Council. When feeding a component diet or a parenteral diet, vitamin and mineral supplementation is necessary to ensure adequate intake.

ASSESSMENT OF NUTRITIONAL SUPPORT

Body weight should be measured daily to determine if nutritional support is adequate to maintain body weight. The most accurate method is to use a walk-on floor scale. A weight tape is a useful alternative when a scale is not available. Weight tapes are used to measure the girth just behind the elbow; the circumference correlates with pounds or kilograms. Weight tapes are relatively accurate in predicting the weight of small horses (less than 350 kg) and large ponies (350 to 450 kg). Weight tapes have been shown to be less accurate in estimating weight in heavy stock breeds and Thoroughbred horses.[18] However, in the hospital setting, their value lies in determining the overall trend of body weight, not the actual number.

Body condition scores are used to subjectively determine the animal's body fat stores and are useful to evaluate the long-term nutritional status of the animal (see Box 6-1). Body condition scores are less useful than a scale for determining smaller weight gains and losses in a hospital situation, but they are more accurate for predicting fat stores.

Diet and hydration status can alter body weight by as much as 5% to 10%. For example, a 500-kg horse that presents with colic may be 7% dehydrated at admission. Rehydration of this animal would result in a weight increase of 35 kg. At the time of exploratory celiotomy, the large colon may be emptied to facilitate correction of a surgical lesion. The large colon and cecum can hold between 75 and 90 L of ingesta; removal of a portion of the contents could result in a weight loss of 50 kg or more. Consequently, weight changes need to be considered in light of hydration status, feed intake, and any procedures that have occurred. In an ideal situation an animal should be weighed postoperatively after rehydration to try to remove variables that can affect weight assessment.

ENTERAL NUTRITION

In the critically ill patient with poor perfusion and decreased oxygen delivery to the tissues, the gastrointestinal tract is frequently the most vulnerable organ. Decreased oxygen delivery has been shown to increase mucosal permeability, resulting in increased translocation of bacteria and absorption of bacterial toxins.[19,20] Inflammatory mediators, produced in the gut as a result of ischemia, are absorbed across the damaged mucosa and enter the portal and systemic circulations; this absorption has been implicated in the onset of septic shock or multiorgan failure.[21] Enteral nutrition increases total hepatosplanchnic blood flow in healthy patients, resulting in greater oxygen delivery to the mucosa. In a rat model of *Escherichia coli* sepsis, enteral feeding of glucose improved intestinal perfusion rates.[22] Enteral nutrition maintains functional and structural integrity of the gut; the absence of enteral nutrition causes mucosal atrophy, increased gut permeability, and enzymatic dysfunction in critically ill human patients.[23]

Enteral nutrition is a trophic stimulus for the gastrointestinal tract both directly via the presence of nutrients and indirectly via stimulation of trophic hormones such as enteroglucagon. Early enteral nutrition (EEN) refers to the initiation of enteral feeding within 48 hours after surgery. In a large clinical study of surgical and trauma patients, EEN significantly decreased morbidity and length of stay when compared with delayed enteral nutrition and parenteral nutrition.[21] Enteral nutrition has a protective effect against bacterial translocation across the ischemic intestinal wall. In addition, EEN has been shown to blunt the hypermetabolic and catabolic responses to injury in several human and animal models.[2] During the hypermetabolic, catabolic state seen with injury or illness, many amino acids, such as glutamine, become conditionally essential. Glutamine is an important fuel for lymphocytes, hepatocytes, and mucosal cells of the gut. During catabolism, glutamine levels may become insufficient to meet these energy demands. The addition of glutamine to both enteral and parenteral diets may improve gastrointestinal function and mucosal cell healing.[24]

Although the decision to supply supplemental nutrition may be clear, the route of supplementation must be considered in light of the original insult, surgical manipulations, and postoperative status of the patient. The enteral route is always preferred

when the gastrointestinal tract can be used. Patients with extensive bowel ischemia, intestinal resection, and anastomosis or postoperative ileus may not be the best candidates for EEN. However, concerns about the strength and diameter of anastomotic sites after surgical resection and about the risk of leakage if enteral feeding is introduced prematurely are not valid. Enterally fed dogs had higher bursting pressures at colonic anastomotic sites and better wound collagen synthesis than unfed controls.[25] Because horses are commonly fed high-fiber diets, the risk of obstruction at the anastomotic site is a valid concern; consequently, when enteral feeding is to be instituted, the type of diet should be carefully considered. Patients with a high risk of postoperative ileus or with a narrow anastomotic site may be better off if they are initially fed parenterally and gradually reintroduced to enteral nutrition. Alternatively, a liquid enteral diet may be instituted until healing is sufficient to allow introduction of roughage.

Types of enteral nutrition can vary from normal feedstuffs (i.e., grains, hay, and complete pelleted diets), to slurry diets composed primarily of normal feedstuffs (Table 6-1), and liquid diets containing component requirements (Table 6-2). In horses with decreased appetite or complete anorexia, the choices are limited to those diets that can be administered through a nasogastric tube. Complete pelleted diets offer several advantages: they are relatively inexpensive, they are well balanced for the maintenance requirements of the adult horse, and they contain fiber. Fiber is beneficial in increasing colonic blood flow, enzymatic activity, colonic mucosal cell growth, and absorption.[26] The major disadvantage of pelleted diets is the difficulty of administering them via nasogastric intubation. Both human and equine liquid formulations are available and have been used as enteral nutrition support in horses.[27-30]

Alternatively, diets prepared using specific components have been described.[31] Corn oil may be added to the diet to increase the caloric content. The use of human products for the full-size horse can be very expensive, and these products have been associated with diarrhea. Liquid diets may be given via continuous flow through a small nasogastric tube, or larger meals may be given by periodic intubation. When using pelleted diets, approximately 1 kg of a pelleted complete feed is soaked in approximately 4 L of water. Once the feed is dissolved, an additional 2 L of water is added and the slurry is administered via a large-bore nasogastric tube. Slurry diets made from complete pelleted feeds will not pass through a nasogastric tube using gravity alone and must be pumped in with a marine supply bilge pump. If a bilge pump is not available or a large-bore tube cannot be passed, pulverizing the pellets before adding water may improve flow. The horse should be checked for the presence of gastric reflux before administration, and the slurry should be pumped slowly with attention paid to the horse's attitude and reaction.

The stomach volume of an adult, 450-kg horse is approximately 9 to 12 L, and a feeding should not exceed 6 to 8 L. This volume should be adjusted for smaller horses. Long-term placement of nasogastric tubes is not without the risk of complications.[32] Small-bore, softer (polyurethane) tubes are recommended if intubation is prolonged, but these generally preclude the use of slurry diets. Alternatively, intermittent placement of a nasogastric tube is effective in decreasing complications, but this can be difficult and at times traumatic for the patient. When instituting enteral feeding, particularly in a patient with prolonged anorexia, it is best to start gradually, increasing the amount fed over several days. The recommended regimen is to feed a maximum of 50% of the calculated requirements during the first 24 hours. If the patient tolerates the supplementation, it can be increased over the next few days until full supplementation is achieved. Rapid changes in intake, particularly with component feeding or high-fat diets, may be associated with colic or diarrhea.

TABLE 6-1. Nutritional Contents of Selected Horse Feeds

Component	Equine Senior	Strategy	Purina Horse Chow
Crude Protein	14%	14%	10%
Fat	4%	6%	2%
Fiber	16%	8%	30%
Kcal/kg feed	2695	3300	454

TABLE 6-2. Nutritional Content of Selected Liquid Diets

Component	Vital HN	Osmolite	Critical Care Meals/Packet
Cal/L	1000	1008	1066
Protein	41.7 g/dL	40 g/dL	12%
Fat	10.8 g/dL	34 g/dL	1%
Carbohydrate	185 g/L	135.6 g/L	73%

PARENTERAL NUTRITION

Parenteral nutrition (PN) is used to supply nutrition when the enteral route is unavailable. Parenteral nutrition can provide partial nutritional support (PPN) or total nutritional support (TPN). In the adult horse, it is most commonly used to supply partial nutrition when oral intake is insufficient or inappropriate. Horses with proximal enteritis, colitis, postoperative ileus, esophageal lacerations, or obstructions can receive nutritional support until resolution of the underlying problem allows reinstitution of enteral feeding. Recumbent or dysphagic animals at risk for aspiration pneumonia, individuals with preexisting protein calorie malnutrition or increased energy demands (late gestation, early lactation, and young, growing animals), and those with decreased feed consumption should also be considered as candidates for PPN or TPN.

Depending on the desired goals and duration of supplementation, solutions containing various amounts of carbohydrates, amino acids, lipids, vitamins, electrolytes, and minerals may be formulated. Carbohydrates are commonly provided with 50% dextrose solutions (2525 mOsm/L) that contain 1.7 Kcal/mL. Isotonic lipid emulsions contain principally safflower and soybean oils, egg yolk phospholipids, and glycerin and come in 10% and 20% solutions. Amino acid preparations are available in several concentrations; 8.5% and 10% solutions are most commonly used in veterinary medicine. Solutions containing both essential and conditionally essential amino acids are preferable.

Although not always ideal, providing calories with dextrose infusions alone is a simple and inexpensive method to supply limited nutritional support to a postsurgical patient for a brief

period (2 days) before transitioning to oral nutrition or more complex parenteral nutrition. Intravenous dextrose has been shown to help reverse serum hypertryglyceridemia and more severe hyperlipemia, and therefore it may be useful in preventing these metabolic derangements in postsurgical patients.[33,34] Dextrose is less calorie dense than lipids and provides no amino acids for protein production; therefore it cannot be recommended as a long-term solution for nutritional support. Hyperglycemia can occur, particularly when attempting to provide a large percentage of energy requirements (as an example, 50% of resting energy levels) to a patient. Accordingly, blood glucose should be monitored during dextrose therapy.

Components that may be added to parenteral nutrition include electrolyte solutions and vitamin and mineral supplements. Multivitamin supplements for humans are available and may be added directly to PN solutions. Some vitamins can be given orally (vitamins C and E) or added to crystalloid solutions (B vitamins). Fat-soluble vitamins are stored in body tissues and rarely need to be supplemented unless prolonged periods (weeks) of anorexia occur. Macrominerals, if required, are best supplemented in separate crystalloid solutions, because divalent cations may destabilize lipid emulsions. Although sick animals require trace minerals, such supplementation is rarely given except to patients receiving parenteral nutrition as their sole nutritional source for prolonged periods (greater than 7 days).

Resting energy requirements should be used when calculating PN volumes for adult animals, but protein requirements should be based on maintenance needs (see Box 6-1) or estimated using the following formula (as described under "Protein Requirements," earlier):

0.5 to 1.5 g protein per kilogram BW per day

The higher end of this formula is recommended for sick, compromised patients. The ratio of nonprotein calories to nitrogen should be at least 100 : 1 in the final solution. We often use lipids to provide approximately 30% to 40% of the nonprotein calories if possible, although many clinicians prefer to use solutions containing amino acids and dextrose but not lipids in the formulation. The addition of lipids to PN is beneficial in patients with persistent hyperglycemia or hypercapnia, because this reduces the dependency on glucose as the principal energy source. The amount of fat use will depend on the amount of carbohydrate provided, with fat storage occurring in the presence of excess carbohydrate calories.

PN formulas should be prepared in a laminar flow hood using aseptic techniques. Lipids should be added last to prevent destabilization of the emulsion in acidic dextrose solutions. Parenteral solutions are an excellent medium for growth of bacteria and should be used within 24 hours of preparation. Before use, they should be kept in a dark, cool area to minimize degradation and loss of vitamins. Because these solutions are hyperosmolar, delivery through a central venous catheter is recommended. Ideally, a separate catheter or portal is designated for PN only. Catheter placement and line maintenance should be performed using strict aseptic technique, and all lines should be changed daily. I generally place a 14-gauge double-lumen catheter (Arrow catheter) and designate one port for PN. Gradual introduction of PN is recommended to decrease risk of complications. Initial infusion rates should provide approximately 25% to 50% of the calculated requirement during the first 24 hours. If tolerated, the rate of infusion can gradually be increased over the next few days to provide 100% of the calculated requirement.

Complications of PN include hyperglycemia, hyperammonemia, hyperlipemia, elevation of serum urea nitrogen, thrombophlebitis, and sepsis.[13,35-38] Lipid infusions have been associated with allergic reactions, hyperlipemia, alterations in liver function, and fat embolism. Insulin resistance seen with systemic inflammatory response syndrome can result in hyperglycemia and rebound hypoglycemia when rates are altered too rapidly. Although solutions containing lipids are very useful in providing additional calories, their use should be determined on a case-by-case basis. Lipids should be avoided in patients with a predisposition to or a preexisting hyperlipemia or an underlying liver dysfunction. Thrombocytopenia, fat embolization, coagulopathies, and alterations in cellular immunity are reported with lipid infusions. Triglyceride levels and platelet counts should be monitored regularly when lipids are added to PN solutions.

Monitoring should include daily assessment of serum electrolytes, blood urea nitrogen, triglycerides, and ammonia and liver function during the acclimation period. Blood glucose should be monitored every 4 to 6 hours and the rate adjusted to maintain blood glucose within the established normal range. If costs are a concern, blood values may be monitored less frequently once a steady state has been achieved.

Box 6-2. Sample Calculations for Feed Supplementation

- Daily nutritional requirements of a 450-kg horse:
  DE_r = (450 kg × 0.021 Mcal/kg) + 0.975 = 10.4 Mcal
  DE_m = (450 kg × 0.03 Mcal/kg) + 1.4 = 14.9 Mcal
  Crude protein requirement = 40 g/Mcal × 14.9 Mcal = 596 g protein
  Alternatively, 0.5 to 1.5 g protein/kg × 450 kg = 225 to 675 g protein

ENTERAL FORMULATION

Equine Senior (horse feed) = 2695 kcal/kg
Corn oil = 1.6 Mcal/cup

- To meet daily DE_r requirements:
  10.4 Mcal/2.7 Mcal/kg = 3.8 kg Equine Senior
- Daily protein requirements (maintenance):
  12% protein = 120 g/kg feed
  3.8 kg × 120 g = 456 g crude protein
- To meet daily DE_m requirements:
  4.9 kg Equine Senior (2.7 Mcal/kg × 5.0 kg) = 13.3 Mcal
  plus 1 cup corn oil = 1.6 Mcal = 14.9 Mcal
  4.9 kg × 120 g protein/kg = 588 g protein

PARENTERAL FORMULATION

1 L of 50% dextrose = 1.7 Mcal
1.5 L of 10% amino acids = 0. 57 Mcal and 150 g of protein
0.5 L of 20% lipids = 1.0 Mcal
Total = 3.27 Mcal/3 L or 1.09 Mcal/L
DE_r = 10.4 Mcal/day
10.4 Mcal/day ÷ 1.09 Mcal/L = 10 L/24 hours = 416 mL/hour
500 g protein/day
Ratio of nonprotein calories to nitrogen = 117 : 1

DE_m, Maintenance energy requirement for active horse; *DE_r*, resting energy requirement.

The same approach should be used when discontinuing PN. The infusion rate should be gradually decreased over at least 24 hours. Frequent monitoring of blood glucose during withdrawal is warranted because of the risk of transient hypoglycemia. For examples on how to calculate PN requirements, see Box 6-2.

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CHAPTER
7 Surgical Site Infection and the Use of Antimicrobials

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Probably the single most important advance in surgery throughout history was the understanding and application of aseptic technique. Before the 1840s, Semmelweis in Austria and Holmes in the United States independently demonstrated that the simple act of hand washing before seeing each patient could dramatically reduce morbidity and mortality in obstetrical wards.[1] When Pasteur more fully developed the germ theory of disease, these practices were validated, but application in surgery was slow to develop. Surgery was a last resort, for good reason, because the consequences of hospitalization and surgery were

virtually always worse than the disease itself. The first champion of primitive aseptic technique was Sir Joseph Lister in the late 1860s.[2] He used carbolic acid (phenol) as an antiseptic and made history when he made a surgical incision to repair a fractured patella and was able to achieve healing of the wound without infection. These early surgical discoveries are common pillars of modern surgical technique. Despite the earlier and more modern continued advances in preventing and managing surgical site infections (SSIs) in the equine patient, it remains a significant problem. The importance of SSIs cannot be overestimated; in humans 77% of deaths among patients with SSIs were directly attributable to the SSI.[3] Similarly, equine orthopedic patients with a SSI were 7.25 times less likely to survive to discharge from hospital than patients without an SSI.[4]

SURGICAL SITE INFECTION CLASSIFICATION

The identification of SSIs involves the combined interpretation of clinical and laboratory findings. To ensure uniformity in the reporting of SSI, it is essential that standardized classification systems be used. Variation in the definition of what constitutes an SSI results in marked variation in reported rates and results in confusing and potentially misleading information.[5,6] The Centers for Disease Control and Prevention (CDC) has developed standardized surveillance criteria for defining SSIs that were redefined in 1999.[3] There are three different types of SSI defined by the CDC: superficial incisional, deep incisional, and organ/space (Table 7-1). An alternative system that has been more commonly used in veterinary medicine is based on the National Research Council's wound classification and is based on the extent of operative contamination (Table 7-2). This system has four classification levels—clean, clean-contaminated, contaminated, and dirty—as the degree of contamination increases.

INFECTION AND SOURCES OF MICROORGANISMS

The goal of aseptic technique is the elimination of all infection-causing organisms from the surgical environment. Although this is not actually possible, it is important that surgeons proactively pursue the objective of preventing SSIs. To achieve this goal, knowledge of the common sources and types of bacteria

TABLE 7-1. Classification of Surgical Site Infections

Surgical Site Infection	Qualifications	Includes at Least One of the Following
Superficial incisional	Within 30 days after operation Involves only skin or subcutaneous tissue of the incision	Purulent drainage from the superficial incision Organisms isolated from aseptically obtained culture of fluid or tissue from the superficial incision At least one of the following signs or symptoms of infection: pain or tenderness, localized swelling, redness, or heat *and* superficial incision is deliberately opened by surgeon, *unless* incision is culture negative Diagnosis of superficial incisional infection by surgeon or attending clinician
Deep incisional	Within 30 days after operation if no implant Within 1 year if implant is in place and infection appears to be related to the operation *and* involves deep soft tissues (fascial and muscle layers)	Purulent drainage from the deep incision but not from the organ/space of the surgical site Deep incision spontaneously dehisces or is deliberately opened by surgeon when patient has one of the following symptoms: fever, localized pain, or tenderness, unless site is culture negative An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathologic or radiologic examination Diagnosis of a deep incisional SSI by a surgeon or attending clinician
Organ/space	Within 30 days after operation if no implant Within 1 yr if implant is in place and infection appears to be related to the operation *and* involves any part of the anatomy (organs and spaces) other than the incision, which was opened or manipulated during an operation	Purulent drainage from a drain that is placed through a stab wound into the organ/space Organisms isolated from aseptically obtained culture of fluid or tissue in the organ/space An abscess or other evidence of infection involving the organ/space that is found on direct examination, during reoperation, or by histopathologic or radiologic examination Diagnosis of an organ/space SSI by a surgeon or attending clinician

TABLE 7-2. Classification of Surgical Wounds

Classification	Criteria
Clean	Elective, primarily closed, and undrained Nontraumatic, uninfected No break in technique No inflammation encountered Respiratory, alimentary, genitourinary tracts not entered
Clean-contaminated	Gastrointestinal or respiratory tracts entered without significant spillage Oropharynx entered Vagina entered Genitourinary tract entered in absence of infected urine Minor break in technique
Contaminated	Major break in technique Gross spillage from gastrointestinal tract Traumatic wound, fresh (<4 hr after trauma) Entrance of genitourinary tract or biliary tract in presence of infected urine or bile
Dirty	Acute bacterial inflammation encountered Transection of "clean" tissues for the purpose of surgical access to a collection of pus Traumatic wound with retained devitalized tissues, foreign bodies, fecal contamination, and/or delayed treatment (>4 hr) after trauma

for any given surgery is essential. There are four basic sources of bacteria in any surgical procedure: the air, the patient, the surgeon, and the surgical instrumentation used. The relative importance of each of these sources obviously varies greatly according to the surgical environment. For example, a surgery performed in a horse's stall will have a greater likelihood of infection from the air compared to that in a hospital operating suite. Thus, attention must be focused as is appropriate for the surgical situation.

Airborne bacteria and debris should be controlled by locating the operating room in a low-traffic location of the hospital and by minimizing the number and activity of personnel. Published guidelines in 2003 from the CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC) may not be readily achievable in an equine hospital but can serve as guides. It recommends maintaining positive air pressure in the operating room, filtering greater than 90% of the air, exchanging air 15 times per hour, and ensuring that air is introduced from the ceiling and exhausted at the floor. It does not recommend the use of ultraviolet light to reduce SSIs.[7]

Preparation of the horse when possible should include basic grooming to minimize overall contaminants. Hair is not directly associated with an increased risk of SSIs, but it is harder to clean and may make aseptic preparation of the surgical site more difficult.[8,9] Hair removal did not affect the number of colony-forming units (CFUs) postscrub over the midcarpal or distal interphalangeal region.[10] If the hair is removed, shaving is associated with an increased risk of SSI compared to clipping, especially if performed earlier than immediately before surgery.[9] This increased risk of SSI has been attributed to microscopic cuts in the skin that later serve as foci for bacterial multiplication.[3] A No. 40 blade is commonly used to clip horses. Preparation of the patient's skin can be performed using a variety of agents, the most common of which are iodophors, alcohol-containing products, and chlorhexidine gluconate (see Chapter 9).[11] Final draping by the surgeon (see Chapter 10) completes the patient preparation before surgery and produces a surgical field that is isolated from contamination. Draping has not been conclusively shown in humans to reduce SSIs, but because of the nature of the equine patient it is highly likely to reduce wound contamination.[12] A newly developed microbial sealing approach has had good success in reducing human SSIs and may be applicable to equine surgery in the future (see Chapter 10).[13,14]

Surgical aseptic preparation and attire are discussed in Chapter 10. Studies evaluating the efficacy of gloves in maintaining a sterile field have demonstrated that surgical contamination is common and that culture results will be positive on gloves after only 15 minutes of surgical time.[15,16] Techniques such as double gloving and discarding the outer layer after draping should reduce glove contamination and resultantly reduce SSIs.[16-18]

Surgical instrumentation is the only component of the surgical procedure that can be sterilized. A variety of sterilization methods are available, with some types of instruments being more suited to different techniques (see Chapter 9). Sterilization indicators should be checked before instrument use to ensure appropriate sterilization conditions have been met. If possible, instruments should not be opened before completion of patient preparation and draping, because airborne bacteria counts are significantly higher at this time.[19]

Microbial contamination of the surgical site is virtually unavoidable. However, development of an SSI depends on many factors. Operating room design, patient, surgeon, and instrument preparation are designed to reduce the number of bacteria at the surgical site. Quantitatively, it has been shown that if a surgical site is contaminated with more than 10^5 microorganisms per gram of tissue the risk of SSI is markedly increased.[20-22] However, the dose required to result in an SSI is a complex interplay between the quantity of bacteria, the virulence of the inoculum, and the immune resistance of the patient.[23] The presence of foreign material in the surgical site, such as suture material or orthopedic implants will dramatically reduce the dose required to produce a SSI.[24-26] Doses of 10^2 *Staphylococcus pyogenes* per gram of tissue produced infections in the presence of foreign material, such as suture material, in humans.[23,27] Microorganisms can adhere to foreign material and evade the host immune response.[28-31]

Infection of a surgical wound occurs most commonly as a result of direct inoculation of the patient's *endogenous* flora from the skin, mucous membranes, or hollow viscera. The most common musculoskeletal pathogen in humans and animals is *Staphylococcus aureus* and has been reported to cause between 19% and 21% of equine orthopedic infections; furthermore,

Staphylococcus spp. are reported to cause up to 60% of equine cases of cellulitis.[4,32,33] This is not surprising because staphylococci are a common part of the resident flora of the skin and nasopharynx. *Enterobacter* spp. were the most common isolate (25%) in a large retrospective study of equine long bone fractures and arthrodeses, similar to other orthopedic (23%) and musculoskeletal infections (28%) reported.[4,32,34] *Enterobacter* spp. are endogenous bacteria that are common resident flora of the genitourinary and gastrointestinal tracts. These opportunistic bacteria cause infection if the host's defense mechanisms are impaired. Bacteria isolated from equine distal limb skin were *Staphylococcus, Bacillus,* and *Micrococcus*.[10] Table 7-3 summarizes reported common bacterial isolates from horses.[4,34-41] *Exogenous* sources of bacteria include surgical personnel, the operating room environment, instrumentation and materials bought to the sterile field during the surgical procedure. These bacteria are primarily aerobes, especially gram-positive organisms (e.g., staphylococci and streptococci).[3] Fungi from endogenous or exogenous sources rarely cause SSIs and their pathogenesis is not well understood.

TABLE 7-3. Common Bacterial Isolates in Horses

Disease Process	Bacterial Isolates
Orthopedic surgery	Enterobacteriaceae, *Staphylococcus, Streptococcus, Pseudomonas*
Cellulitis	*Staphylococcus, Streptococcus*
Chronic wounds	*Pseudomonas, Staphylococcus, Serratia, Enterococcus, Providencia*
Enterocolitis	*Salmonella, Clostridium*
Iatrogenic septic arthritis	*Staphylococcus aureus*
Wounds	*Streptococcus, Staphylococcus,* Enterobacteriaceae, *Pseudomonas,* and anaerobes
Peritonitis after abdominal surgery	*Streptococcus,* Enterobacteriaceae, *Actinobacillus,* anaerobes
Penetrating wounds to synovial structures	Enterobacteriaceae, anaerobes
Septic physitis/arthritis (foals)	*Escherichia coli, Rhodococcus equi*
Paranasal sinus and guttural pouch	*Streptococcus equi* subsp. *equi, Streptococcus zooepidemicus, Aspergillus, Cryptococcus*

RISK FACTORS FOR SURGICAL SITE INFECTION

The risk of SSI is a result of the complex interplay of microbe, host, and surgery-related factors (Table 7-4). This can be conceptualized according to the following relationship, in which surgery-related factors can affect the dose and the innate resistance of the host[3]:

$$\frac{\text{Dose of bacterial contamination} \times \text{virulence}}{\text{Resistance of host}} = \text{Risk of SSI}$$

TABLE 7-4. Risk Factors for SSIs in the Horse

Risk Factors	Examples
Host-related factors	Extremities of age Gender (female) Immunocompromise (failure of passive transfer, corticosteroid administration) Weight (>300-325 kg) Distant sites of infection Hypoxia—systemic and local Foreign material—e.g., clay, dirt
Surgery-related factors	Emergency procedures Patient and surgeon preparation—shaving, scrubbing technique Duration of surgery Surgical skill Foreign material—suture and prostheses Bandage—Incise drape reduces SSI, stent >3 days increases SSI, postcolic abdominal bandage reduces SSI

Microbe-Related Factors

Many types of microorganisms may be present at a surgical site, however they do not always cause an infection. A microorganism's virulence, or its ability to adhere to eukaryotic cell surfaces, multiply, and evade the host immune response, is variable. Resultantly, a low number of virulent *S. aureus* will cause an SSI, whereas a large number of less virulent microorganisms may not. Bacterial adhesion molecules are one type of virulence factor and referred to as microbial surface components recognizing adhesive matrix molecules (MSCRAMMs).[42,43] The exact role of each bacterial adhesion molecule has been difficult to elucidate in animal models.[44] However, bacterial adherence is thought to play a key role in the pathogenesis of *S. aureus* infections.[45] Fibronectin-binding genes, *fnbA* and *fnbB*, were detected in 98% and 99% of *S. aureus* infections recovered from human orthopedic infections, respectively.[46] Bacteria, including *S. aureus,* can also produce a variety of exotoxins such as hemolysin and leukotoxins that are produced to avoid the host immune response.[46] Virulence or antibiotic resistance can be conferred by secreted proteins, including flagellar proteins, type III secretion factors, pili, and enzymes such as proteases and β-lactamases.[47,48] A promising new approach to combat emerging antibiotic resistance is by targeting bacterial virulence, rather than bacterial viability.[49] For example, *Pseudomonas aeruginosa* expresses a periplasmic protein, DsbA, that is essential for the folding and function of almost all exported virulence factors.[47] The development of drugs targeting these important virulence determinants may allow the development of more effective drugs with a lower propensity for inducing bacterial resistance.[50]

Production of a biofilm is another virulence factor. Microorganisms such as gram-positive staphylococci that can adhere to foreign materials and produce a biofilm, or extracellular glycocalyx, are resultantly resistant to host defenses and antimicrobial agents. These microorganisms are problematic in horses.[28,51] SSIs involving implants with biofilm formation can be so effective that removal of the implant may be required to resolve the infection.[4] Novel implant coatings are being developed that are

resistant to this biofilm formation and may offer an attractive option to reduce SSIs in horses.[52]

Host-Related Factors

Systemic Risk Factors

Intrinsic, patient-related, and preoperative risk factors are important considerations for SSIs. Extremes of age are an important risk factor in humans, although the relationship between age and SSIs may be secondary to comorbidities (the appearance of multiple illnesses) or immune senescence (aging).[3,53] In horses, increasing age has been identified as a risk factor for infection during arthroscopic surgery but not for orthopedic repair of long bone fractures and arthrodeses.[4,36] This is likely because there was a broader age range in patients undergoing arthroscopic surgery, and as a result the effect of age became more evident. Concomitant infections such as pneumonia or separate sites of infection (e.g., foals with umbilical infections) should be evaluated and appropriately managed before surgery, when possible, to reduce the incidence of SSI secondary to bacteremia.[3,54] In humans, remote infections result in a twofold to threefold increase in SSIs.[55] Complication rates are lower in young (younger than 1 year but older than 1 month, ~15%) horses compared to adults (older than 1 year, ~43%) with ventral midline incisions.[56] However, in cases of long bone fracture repair in horses younger than 1 year, there was an increased rate of SSI in one study, but interestingly this was not associated with a reduced rate of overall survival.[4] One possible explanation for the increase in SSIs in younger horses is that attempts to treat complicated fractures, which carry a higher risk of SSIs, were more likely to be made in the younger rather than the older patients.

Female horses have an increased risk of SSIs in arthroscopic and orthopedic surgery compared to males and geldings.[4,36] This gender relationship with SSI has not been evident in other types of equine surgery and may potentially be because of the increased economic and breeding value of female horses and the acceptance of greater risk before surgery.

Obesity (20% over ideal body weight) is strongly related to increased SSI in humans.[3,57] In horses, the relationship between weight and SSI is not as clear. Horses weighing greater than 300 kg and undergoing a ventral midline celiotomy were twice as likely to have incisional complications than lighter horses.[56] This may actually result from anesthesia-related hypotension and reduced tissue perfusion rather than absolute weight.

Host resistance against infection is a function of the immune system and is an important factor in determining if a surgical site becomes infected. In adult horses, immunocompromise is infrequently a concern. However, it certainly is a concern in neonates, and preoperative failure of passive transfer (IgG less than 800 mg/dL) should be evaluated and corrected before surgery.[39] In general, the immune status can be suppressed by local or systemic administration of corticosteroids, which may increase rates of infection.[53,58] The direct causative effect of corticosteroids has not been conclusively agreed upon in human medicine.[3,53,57] The role of endocrine diseases, such as pituitary pars intermedia dysfunction, on SSI has not been clearly delineated in the horse. Nutritional status has been found to be important in severely malnourished human patients and has been managed with preoperative and postoperative nutritional support.[3] However, the effect of nutritional status in horses undergoing surgery has not been determined. As a basic principle, where possible, patients should be maintained in a good nutritional status. Cardiovascular disease and severe metabolic derangements are important risk factors for SSIs in humans.[55] These systemic states have a detrimental effect on the host's ability to resist infection. Hypothermia (temperature lower than 36° C/96.8° F) triples the rate of SSI in human surgery, and it may be a concern in foals and miniature horses, particularly if the skin and hair coat becomes wet.[59,60] Maintenance of normothermia positively affects a patient's immune system and therefore improves its ability to resist SSIs.

Other factors that are important in humans, such as nicotine use, diabetes, and the perioperative transfusion of certain blood products, are not or are unlikely to be important in the horse.

Local Risk Factors

The perioperative supplemental supply of oxygen to the surgical site has been linked to a reduction in SSIs in human surgery.[61-64] The use of hyperbaric oxygen therapy as an adjunctive treatment to improve healing for skin grafts in horses was not effective in one recent study.[65] The effect of supplemental oxygen on reduction of SSI in horses has not been examined. In humans, wound infection rates decrease as tissue oxygen tension increases to 100 mm Hg.[66] Surgical site perfusion and resultantly oxygenation should be maintained as a surgical priority. The distal limbs of horses have little muscle and soft tissue coverage and as a result are more likely to suffer from regional hypoxia and as such heal more slowly compared to the head.[67] This may lead to an inhibition of the host's local resistance to infection and increase the incidence of SSIs.

Disruption of the physical barrier (skin) at the surgical site because of wounds, dermatitis, or inappropriate surgical preparation (e.g., shaving) can increase rates of SSI.[53] Long bone fractures that are open at presentation were 4.2 times more likely to become infected after surgical repair compared to closed fractures.[4] This results from disruption of the host's normal barrier to infection (an intact epidermis) in addition to likely direct inoculation of the fracture. More extensive long bone fractures are 5.1 times more likely to develop an SSI than fractures only involving the articular surface.[36]

Any foreign material, such as sutures, prostheses, or organic materials, alter the local immune response and may result in SSI, even with relatively low levels of contamination. These materials allow biofilm formation and serve as a nidus for infection.[51] Careful débridement of contaminated surgical sites to remove foreign material is a basic principle of surgery. Foreign materials have differing abilities to potentiate an SSI. Silk suture material is 3.4 times more likely than polyglactin 910 (Vicryl) to be correlated with an infection.[24] Furthermore, a single strand of silk suture reduces the number of *S. aureus* required to cause an infection by a factor of 10^5.[68] Similarly some soils, most notably montmorillonite clay, contain highly charged particles and are very potent potentiators of infection.[69] Surgical techniques to remove and minimize the presence of foreign material at the surgical site will reduce the incidence of SSIs by allowing efficient function of the host immune response.

Careful attention to surgical technique will reduce the presence of blood clots, ischemic tissue, dead space, and pockets of fluid that will prolong the inflammatory phase of wound healing and potentiate SSIs.

Surgery-Related Risk Factors

Factors related to the surgical procedure are readily manipulated by the surgeon and resultantly should be an important consideration. A summary of the reported rates of SSI for common surgical procedures are shown in Table 7-5. With attention to detail and a systematic approach, many of the following surgery-related risk factors can be controlled and therefore significantly reduce SSIs.

Surgical Procedure

Patients undergoing gastrointestinal surgery are at an increased risk of wound complications when treated during an emergency rather than an elective procedure.[70,71] Horses undergoing surgery for acute abdominal discomfort had a 39% incidence of incisional complications compared with 9% for elective celiotomies.[56] In horses with abdominal discomfort requiring immediate surgical intervention, this cannot be altered; however, in patients with concomitant infections, surgery should be delayed if possible.[39] Stabilization of the patient will improve the physical status of the patient (lower the American Society of Anesthesiology [ASA] score—see Chapter 18) and likely be associated with a reduced risk of SSI in horses as it is in humans.[72,73]

Patient and Surgeon Preparation (See Chapters 9 and 10)

Basic grooming of the equine surgical candidate before induction will reduce bacterial contamination. Preparations include picking the feet, cleaning the coat of debris and loose hair, and possibly covering the feet and tail.[39] Surgery of the foot should involve trimming and soaking the hoof overnight to reduce bacterial contamination before surgery.[22] Preoperative hair removal is acceptable, because it may help reduce anesthesia duration but should not be done by shaving. In human medicine, when hair was removed using a razor, the rate of SSIs was 5.6% compared with 0.6% when hair was removed by depilatory agents or when hair was not removed.[9,73,74] This increased rate of SSI is attributed to microscopic cuts in the skin that later serve as foci for bacterial infection. Hair removal with clippers immediately before surgery reduced SSIs (1.8%) compared with removal the night before (4.0%).[3] The initial patient preparation ideally should be performed in a designated area separate from the operating room to reduce contamination of the surgical field.

Antiseptics available for skin preparation include iodophors (e.g., povidone-iodine [PI]), alcohol-containing products, and chlorhexidine gluconate. Iodophores or chlorhexidine have broad antimicrobial activity, are either aqueous- or alcohol-based, and are common choices for skin preparation.[3,11,75,76] Recently, preoperative cleansing of the patient's skin with chlorhexidine-alcohol (CA) was shown to be superior to PI for preventing SSIs associated with superficial skin incisions (4.2% with CA; 8.6% with PI) and deep incisions (1% with CA; 3% with PI), but not organ space infections.[76] Additionally, chlorhexidine-alcohol was found to have greater residual antimicrobial activity compared to 4% chlorhexidine gluconate and 7.5% PI.[77] In horses undergoing ventral midline incisions, evaluation of iodophor-alcohol and a film-forming iodophor complex as the means of skin preparation revealed no difference between the techniques.[78] Other factors that influence the effectiveness of the surgical scrub are appropriate technique and duration of scrub.[3,77] Scrub duration of at least 2 minutes has been shown to be as effective as 10-minute scrubs in reducing bacterial colony counts.[3] Another study found that a 1-minute scrub with povidone-iodine followed by an alcohol foam was superior to a traditional 5-minute scrub.[79] Overall, the best length of time to scrub is unclear and depends on the antiseptic used.[80] Newer alcohol-based rubs (e.g., Avagard [chlorhexidine gluconate 1% solution and ethyl alcohol 61% wt/wt]) have been shown to be an effective alternative to traditional aqueous scrubbing.[57,80] In Europe, a similar product consisting of 2-propanol (45%), 1-propanol (30%), and mecetronium ethyl sulfate (0.2%) (Sterillium) has shown significantly better results than chlorhexidine gluconate and povidone-iodine.[81] Before the

TABLE 7-5. Rate of SSIs for Common Surgical Procedures

Procedure	Rate of SSI	Risk Factors	Protective Factors
CELIOTOMY			
Emergency	7.4%-39%	Reoperation, inexperienced surgeon, near-far-far-near suture pattern, staples, polyglactin 910	Lavage of linea alba, topical antibiotics to surgical site at closure, incise drape for recovery, minimize surgical duration
Elective	9%		
CASTRATION			
Routine	2%-3.2%	Lack of drainage, lack of antibiotic prophylaxis, standing nonsutured technique	Laparoscopic technique, recumbent sutured technique
Laparoscopic cryptorchid	0%		
Laryngoplasty	0%-4%	Laryngotomy, draft breed	
Arthroscopy	0.5%-1.5%	Draft breed, tibiotarsal joint	
ORTHOPEDIC PROCEDURES			
Clean	8.1%	Procedure classification, long bone affected, surgical duration >90 min, female patients	
Clean-contaminated	52.6%		
Long bone fractures	28%-32%	Open fracture configuration, surgical duration >180 min	Minimally invasive reduction

See references 4, 36, 78, 102, 103, 114, 115, 150, 172-184.

first scrub of the day using these products, the nail bed should be cleaned and briefly washed to remove soil and debris.[82]

Drapes, Gloves, and Gowns

Scrub suits, surgical masks, caps and hoods, and shoe covers are all parts of traditional surgical attire. Though there is limited evidence demonstrating a direct effect on SSI by surgical attire, it seems prudent to limit the exposure of patients to potential contamination from members of the surgical team. Surgical gloves fail during about 20% of operations.[57] Wearing two pairs of gloves has been shown to reduce the incidence of failure.[3] Careful attention should be paid to inspection of glove integrity during procedures in an attempt to identify and correct breaks in barrier integrity promptly. In one study of gloving procedure using either open or closed technique there was a 100% incidence of contamination, and scrub staff–assisted technique was associated with no contamination.[15] A barrier type of gown should be worn and disposable impervious drapes used when possible, although the effectiveness of this has been disputed.[3,83] However, distal limbs of dogs wrapped with impervious drapes resulted in reduced bacterial contamination compared to more traditional techniques.[84] Any option that may reduce contamination in cases where infection is devastating, such as equine orthopedic repair using metallic implants, should be actively pursued by surgical teams.

Duration of Surgery

Increased surgical duration has been strongly associated with increased SSI in horses.[4,36,56,67,85] For general equine orthopedic procedures, a surgical duration longer than 90 minutes increased the risk factor for SSI by 3.6 times.[36] For long bone fractures and arthrodeses, as surgical duration increased so did the risk of SSI.[4] Postoperative incisional complications in horses are twice as likely after abdominal surgery longer than 2 hours.[85,86] The exact effect of increasing surgical duration is likely a combined result of many factors, including more complicated procedures, tissue drying, reduced tissue perfusion, and increased tissue trauma. Obviously, surgery should not be rushed to prevent SSIs. However, careful surgical planning and surgeon training are essential components that will help to minimize surgical duration and possibly reduce SSIs.[4]

Surgical Technique (See Chapter 12)

Surgical skill and careful attention to and adherence to Halsted's principles play an important role in SSIs.[3] Techniques focusing on careful tissue handling, débridement of devitalized tissue, eradication of dead space, appropriate use of drains and suture materials, and effective hemostasis while maintaining perfusion are essential to reduce the incidence of SSIs.[3,39,54,67,87] Careful attention to appropriate surgical and aseptic technique have a direct effect on the amount of contamination at the surgical site. Regardless of the host immune status or specific bacterial virulence, poor technique will result in increased SSIs. Excellent surgical training, anatomic knowledge specific to the procedure being performed, and attention to detail are vital factors in prevention of SSIs.

The presence of foreign material in a surgical site should be reduced and minimized by the surgeon to reduce the incidence of SSIs. Greater than 10^5 microorganisms per gram of tissue will increase the risk of SSIs, and the presence of foreign material will dramatically reduce this number.[20-23,27] Surgical techniques to reduce the presence of these materials in surgical sites, such as débridement and lavage, coupled with appropriate use of suture materials, drains, and implants will result in reduced SSIs.[39,54,67] Selecting appropriate suture and minimizing excessive tension on the skin edges will likely reduce SSIs in horses undergoing celiotomies.[85]

Incision

Skin incisions can be made using either a conventional scalpel, laser tools, or electrosurgical devices.[88-91] A benefit of laser and electrosurgical devices is improved hemostasis compared to the traditional scalpel.[88,90] However, these devices also cause collateral tissue damage, resulting in eschar formation.[92,93] Numerous electrosurgical devices are available for surgical use, such as monopolar and bipolar cautery, harmonic scalpel, and LigaSure units. The LigaSure unit and harmonic scalpel are associated with less lateral thermal damage compared to cautery devices and thus are less likely to produce necrotic tissue that may serve as a focus for infection.[39,91] Conventional scalpels cause the least collateral damage compared to CO_2 laser and electrosurgical tools, and skin incisions made with steel scalpels heal faster.[88,90,93,94] As a result a conventional scalpel should be used unless surgical circumstances dictate otherwise.

Minimally Invasive Techniques

The advent and application of minimally invasive techniques in humans and subsequently in horses has been a major advance in surgical practice.[4,87,95-101] In humans, SSIs are reduced in laparoscopic procedures compared to conventional surgical approaches.[57,95] In horses there is little information regarding SSI rates for these procedures. However, minimally invasive plate fixation and laparoscopic procedures will likely be associated with reduced infection rates, but more cases are required before definitive conclusions can be drawn.[4,99-101] The reduced rate of SSI has been attributed to preservation of immune function and reduction in the inflammatory response compared to open surgery.[57,100]

Suture Materials and Surgical Implants

Any foreign material in a surgical site will increase the likelihood of developing an SSI. All appropriate methods to minimize the amount of material introduced to a surgical site will reduce bacterial colonization and resultantly SSIs. The strength and elasticity of the tissue should be matched to the selected suture material to minimize excessive retention in the surgical site. Suture patterns can affect SSI; for example the near-far-far-near suture pattern is associated with an increased rate of SSI compared to a simple interrupted patttern.[102] The use of polyglactin 910 has been associated with increased SSI when used to close the linea alba in horses.[103] In contaminated surgical sites, multifilament, nonabsorbable suture materials should be avoided (e.g., silk), because these materials are prone to contamination by drug-resistant bacteria and cause SSIs.[24,104] The use of tissue glue (cyanoacrylate) to close surgical incisions has been associated with reduced rates of SSIs in humans and may warrant evaluation in the horse.[105]

An alternative method to reduce SSIs that has recently been developed is the incorporation of antibacterial materials in the suture material or to the surface of implants.[52,86,106-109] Coating suture material with triclosan has been shown experimentally to prevent in vitro and in vivo bacterial colonization.[110,111] However, use of triclosan-coated polyglactin 910 to close ventral midline celiotomies in 100 horses did not reduce the rate of incisional infection.[86] In fact, the use of this material was associated with a slight increase in incisional edema in these horses.[86] The principle of coating implants with various materials to prevent or reduce the likelihood of bacterial adherence has been extended to include virtually any type of implant material.[52,109,112,113] These emerging technologies are very exciting for equine surgeons because the consequences of an implant related infection is often devastating to our patients.[4]

Bandages and Drains

Despite the best efforts of equine surgeons, horses are returned to relatively dirty housing environments immediately after surgery. It has been shown that application of an abdominal bandage postoperatively may reduce the rate of SSIs following celiotomies.[114] Also, the application of an Incise drape (Steri-Drape) to ventral midline incisions for recovery has been associated with a reduced rate of SSI.[102] Application of bandages for longer than 24 to 48 hours is likely not warranted, and the beneficial effect on SSIs is unclear.[3] In horses, application of a stent bandage for 3 days following celiotomy procedures increased rates of incisional infections.[115] Drains should exit distant to and not from the primary surgical incision.[3] Where possible, use of a closed suction drain is preferable to an open one, and drain removal as early as appropriate will help to reduce the likelihood of infection.[3]

NOSOCOMIAL INFECTIONS

Nosocomial, or hospital-acquired, infections are caused by exposure in a hospital to pathogens that were not present or incubating in a patient before admission. They commonly occur after at least 48 hours of hospitalization. In human medicine, the estimated cost of nosocomial infections may be as high as $4 billion annually.[116] In equine hospitals the financial cost is much lower but still considerable. For example, in one large equine hospital the cost of a nosocomial outbreak of *Salmonella* was $4.12 million.[117]

Perhaps the most devastating disease agent associated with nosocomial infection in horses is *Salmonella* spp.[116,117] Horses undergoing abdominal surgery are at a high risk of developing salmonellosis postoperatively.[35,118] In these situations it may be difficult to determine if the infection was truly nosocomial or if the horse was subclinically shedding the organism at the time of admission. Research has demonstrated that in the general horse population 0.8% to 1.8% of horses shed *Salmonella*.[116] From 1.4% to 20% of horses admitted to veterinary teaching hospitals have been estimated to be shedding *Salmonella*.[118] Pulsed-field electrophoresis testing during *Salmonella* outbreaks has shown that they are nosocomial.[119] Other bacteria that are reported to cause nosocomial infections in horses include *Clostridium* spp., *Pseudomonas, Enterobacter, Citrobacter, Proteus,* and *Klebsiella*.[118,120,121]

S. aureus is the most common cause of SSI in humans.[95] The development of methicillin-resistant *S. aureus* (MRSA) is an emerging veterinary and zoonotic pathogen of great concern to both the veterinary and human medical communities. In horses, the colonization rate with MRSA has been reported to be 2.3% of admissions.[122] Clinical MRSA nosocomial infections occurred in 0.18% of admissions.[122] Currently, the MRSA infection rate is low in horses. Strict attention to appropriate antimicrobial guidelines will hopefully ensure that the prevalence of MRSA does not increase as it has in human medicine.

PREVENTION AND MANAGEMENT OF SURGICAL SITE INFECTIONS

Many factors are involved in development of SSIs. It is a delicate balance between the type of bacteria, the degree of contamination, and the innate resistance of the patient. Fortunately many of these factors can be inexpensively and effectively altered by careful attention to detail by the surgeon and the surgical team as a unit (Table 7-6). An infection at the surgical site, even when successfully treated, normally has dramatically adverse effects on treatment costs and the cosmetic and functional outcomes. The financial cost of SSIs in horses has not been determined. In humans it is estimated that SSIs extend the length of hospital stay by an average of 9.7 days and increase the cost by $20,842 per admission, which amounts to more than $1.5 billion annually that is expended to treat SSIs. In horses undergoing complicated orthopedic procedures, an SSI significantly increased the length of hospitalization from 13.4 to 45.5 days.[4] Furthermore, an SSI increased the duration of antimicrobial therapy from 4.5 to 21.8 days.[4] The results of a SSI associated with other surgical procedures are likely to be less dramatic than those occurring with orthopedic procedures but will still be very detrimental to the case outcome. Therefore it is very important that surgeons are aware of SSIs and how they can reduce their effects.

Diagnosis of Surgical Site Infections

Clinical Signs

An important principle for SSIs is that the earlier the intervention, the better the chance of resolution. A careful physical examination will often reveal the early onset of infection allowing appropriate measures to be taken regarding further diagnosis and treatment. Clinically apparent general signs suggestive of SSI include a fever that cannot be otherwise explained, postoperative swelling that either increases or does not decrease, pain or heat on palpation of the surgical site, erythema, and drainage. Lameness is a useful clinical sign for detecting and monitoring orthopedic and synovial infections in the horse.[54] Palpation and manipulation of the surgical site may elicit a painful response when a SSI is present. Any early signs of infection should prompt further investigation to determine an appropriate treatment plan as required.

Clinical Pathology

Complete blood counts may reveal a leukogram suggestive of infection; however, this is rarely reliable. Neutrophil and lymphocyte counts are variably high, low, or normal in the face of infection and as a result are not particularly useful in the diagnosis of SSI.[123] A marked leukopenia (less than 5000 cells/μL) can be a sensitive indicator of possible nosocomial enterocolitis when coupled with other supporting clinical signs.[35]

TABLE 7-6. Interventions to Decrease SSI in the Horse

Timing	Interventions
Preoperative	Minimize surgical duration by careful planning
	Thorough preoperative exam and CBC/fibrinogen to detect underlying disease
	Remove gross foreign material (bath) before induction
	Remove hair immediately before induction
	Remove hair using clippers, do not use razors
	Perform emergency surgery only when necessary
	Delay surgery to treat distant sites of infection
	Pay strict attention to aseptic preparation/technique
	Minimize movement and personnel in operating room
	Ensure instrument availability, quality, and sterility for the procedure
	Use appropriate perioperative antimicrobials
Intraoperative	Double glove during draping and use orthopedic gloves for fracture repairs
	Open surgical instruments/implants as required during surgery
	Administer antimicrobials as appropriate
	Strictly adhere to aseptic scrubbing/technique
	Drape appropriately – drape to isolate enterotomy
	Adhere to Halsted's principles
	Place exit drain distant to surgical incision
	Use close suction drains and remove before 48-72 hr postoperatively
	Débride infected/devitalized tissues
	Lavage contaminated surgical sites
	Minimize foreign material incorporated in surgical site
	Maintain patient's body temperature
	Use expedient surgical procedure as appropriate
	Consider changing gowns and gloves for procedures longer than 2 hr
	Ensure appropriate perfusion and tissue oxygenation
	Select appropriate suture material and patterns
	Follow appropriate surgical technique
Postoperative	Protect surgical site with bandages—colic (incise or abdominal bandage)
	Use therapeutic antimicrobials as appropriate
	Minimize duration of hospital stay
	Provide thorough discharge instructions on wound care and suture removal

Acute phase proteins (APPs) are the result of a highly organized physiological response to inflammation.[124] Although not specific for SSIs, they are a very useful means of indirectly detecting and monitoring the inflammation that results from SSIs. The most commonly measured APPs in horses are fibrinogen (FB), serum amyloid A (SAA), and less commonly haptoglobin. FB is a soluble plasma protein synthesized by the liver with a wide reference interval in horses (200 to 400 mg/dL, 2 to 4 g/L).[124] The lengthy response period after an inflammatory stimulus, such as SSI, means that FB is a fairly insensitive APP. SAA, in comparison, has rapid and large changes (up to a hundredfold) in response to stimuli (less than 0.5 to 20 mg/L in normal horses) and is particularly suited for real-time monitoring of disease process in horses.[124,125]

Fluid samples from surgical sites or in adjacent synovial or pleural spaces are useful indicators of SSIs. The color, turbidity, total protein, cell count, and cell morphology can be determined to evaluate potential infection. Normal synovial fluid generally contains fewer than 500 nucleated cells/mL, with a predominance of mononuclear cells. A cell count greater than 30,000 cells/mL and a protein level of 4.0 g/dL with greater than 90% neutrophils is specific for infection.[126] Infected synovial fluid is usually turbid or flocculent, cloudy, and nonviscous. Newspaper print cannot be read through the samples with a cell count of greater than 30.0×10^9/L, which is strongly suggestive of infection.[126] However, care must be exercised in interpretation solely of cell counts. Recent injection or sampling of synovial structures alone will significantly elevate synovial cell count and protein without SSI. Peritoneal and synovial pH (normal 7.30 ± 0.06) can be decreased when sepsis occurs.[127] A difference in peritoneal and peripheral serum glucose of greater than 50 mg/dL has been shown to be a good indicator of septic peritonitis.[127] Another potential, although not very specific, indicator of synovial infection is synovial fluid lactate (normally less than 3.9 mmol/L), which will rise with infection (greater than 4.9 mmol/L).[128] Trends in synovial fluid lactate may be more useful in monitoring synovial infections than the absolute number.

Microbiology

The definitive diagnosis of a SSI is a positive bacterial culture, and sensitivity testing is extremely useful in guiding subsequent appropriate therapeutic choices. However, a negative culture does *not* preclude a diagnosis of a SSI.[54] Bacterial culture at the time of surgical closure has not been useful in identifying incisional contaminants in horses undergoing a celiotomy.[85] Bacterial culture from infected synovial structures is negative in

almost 50% of clinical cases, but this rate is improved to 73% when enrichment media are used.[40] The identification of bacteria with Gram stains only occurs in approximately 25% of cases of synovial infection.[40] Obtaining fluid samples for culture before administering antibiotics, or delaying administration for 24 hours, may improve isolation of the cause of the SSI. Blood culture media are excellent for aerobic culture and are superior to directly plating onto agar plates.[54] Use of a sterile vial with transport media or direct injection into an enriched medium (brain heart infusion [BHI] agar or thioglycolate) are preferred. After the site is aseptically prepared, the sample should be obtained from deep in the surgical site.[67] Aspirates from a pocket of fluid suspected to be infected can be effective samples to use for early diagnosis of SSIs. Tissue samples, such as synovial membrane, have been shown to be beneficial for improving culture results, but in our experience this is not always reliable.[129] Careful and expedient handling of the samples in coordination with the receiving laboratory will greatly improve the likelihood of a successful bacterial culture and resultantly an antibiogram. It is important to remember to submit samples for fungal culture, especially if there is a history of intra-articular injections or wounds, and to repeat attempts to obtain a culture if initially unsuccessful.

Imaging Techniques

Ultrasonography of a suspected SSI can be useful. It may allow identification of a pocket of infection that can be sampled for culture and sensitivity, facilitating earlier diagnosis of the causative agent. Ultrasonography also can guide accurate aspiration for surgical drainage to ensure maximal effectiveness.[28,130-132]

Radiographic signs of acute infection are often limited to increased soft tissue swelling or possibly separation of tissue planes.[54] Signs progress to include radiolucency developing adjacent to metal implants and periosteal proliferative changes unassociated with fracture healing. Even later radiographic signs include lysis extending into cancellous bone, medullary cavity, or both. The radiographic appearance of an SSI is often not reflective of the severity of the underlying infection.

Scintigraphy is not commonly used to diagnose SSIs but can be a helpful tool for identifying deep infections without typical external localizing signs. The use of radiopharmaceutical-labeled liposomes or white cells may offer a novel and useful technique for identifying problematic SSIs in the future.[133]

More advanced diagnostic imaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) may be beneficial in select cases of infection that have atypical presentations.[28,134,135]

Pathogenic Bacteria Associated with Equine Surgical Site Infections

Knowledge of the common bacterial infections that occur globally in addition to locally at each surgical facility is very useful in the prevention and treatment of SSIs. Monitoring and surveillance for SSIs is essential to allow scientific, evidence-based decisions to be made related to prevention of SSIs.

In long bone fracture repairs and arthrodeses, SSIs are mostly polymicrobial in origin (40%), the remainder being half gram-positive (32%) and half gram-negative (28%).[4] In two other orthopedic-related studies, polymicrobial infections constituted 60% and 19%, respectively.[34,40] In equine long bone fractures the most common gram-negative bacterial isolate was *Enterobacter cloacae* (24.5%), which is very similar to other studies, and the most common gram-positive bacterium was coagulase-negative *Staphylococcus* (21%).[4,32,34] Other bacteria that are commonly associated with orthopedic SSIs are *Pseudomonas, Streptococcus,* and anaerobes.[4,32,34,40] *S. aureus* is the most common (31%) isolate in postoperative synovial structure infections.[40] Mixed bacterial isolates are commonly obtained from SSIs after gastrointestinal, urogenital, and respiratory tract infections, which means that obtaining a representative culture is extremely important for developing a successful targeted therapy.[67] *Actinobacillus* species have been reported as a cause of SSI following soft tissue surgery.[136] Common skin isolates from the ventral midline before surgical preparation for celiotomies were *Bacillus, Staphylococcus, Micrococcus, Streptomyces,* and *Streptococcus* species.

Treatment of Surgical Site Infections

Rapid and accurate identification of a SSI is essential. Once an SSI has been identified, the treatment options are varied depending on the relative importance of the SSI to the outcome, the location of the surgery, the type of procedure performed, and possible implants used. The following are basic principles that always apply: (1) drainage of infected tissues should usually be performed with the aid of gravity, (2) devitalized and infected tissue should be débrided, and (3) appropriate therapeutic antimicrobial therapy should be initiated based on accurate culture and sensitivity results.[67,85,137,138]

A key consideration for both surgical and antimicrobial therapy is whether any implanted prosthetic material is infected.[137] The formation of a biofilm around a surgical implant can be extremely resistant to antimicrobial therapy, and removal of the implant may be required to resolve the infection.[4] Treatment of SSIs related to orthopedic implants has improved markedly over the last decade, primarily because of improved local delivery of antibiotics.[4,139-144] There is little doubt that improved outcomes are possible when extremely high doses of appropriate antimicrobials can be instilled and maintained close to infected tissues and implants. Systemically administered antimicrobials, even combined with drainage and lavage, fail so frequently that equine surgeons have enthusiastically embraced local delivery techniques.

Antimicrobial Prophylaxis Against Surgical Site Infections

Perioperative Antibiotic Therapy in Horses

The use of antimicrobials in veterinary medicine has been and will continue to be an extremely controversial issue.[145,146] The development of multidrug-resistant bacteria and their effect on human medicine has widespread health consequences.[147,148] Antimicrobials should be carefully selected, achieve effective tissue concentrations at the time of surgery, and act against likely pathogens. The intelligent and optimal use of antimicrobials is essential for effectively preventing SSIs while minimizing the development of antimicrobial resistance.

Antibiotic Classification

Antibiotics can be broadly classified as either bactericidal or bacteriostatic or by their mechanism of action (Table 7-7).

TABLE 7-7. Antibiotics Commonly Used in Horses

Antimicrobial	Mechanism of Action	Adverse Effects
BACTERICIDAL		
Penicillin	Inhibit cell wall synthesis by binding to penicillin-binding proteins, leading to cell lysis	Autoimmune hemolytic anemia, anaphylaxis, transient hypotension, increased large intestinal motility, cardiac arrhythmia
Cephalosporins	As for penicillin	Enterocolitis
Aminoglycosides	Inhibit protein synthesis by binding to 30S ribosomal subunit	Nephrotoxicity, neuromuscular blockade, ototoxicity
Fluoroquinolones	Inhibit bacterial DNA gyrase	Cartilage disorders in young (<3 yr) horses, oral ulceration
Metronidazole	Disrupt bacterial DNA by free radicals and unstable intermediate compounds after structural change once in target organism	Enterocolitis, inappetence
Trimethoprim/sulfonamide	Synergistic action to inhibit folic acid synthesis (sulfonamides block first step and trimethoprim the second step in folic acid synthesis pathway)	Idiosyncratic reactions
BACTERIOSTATIC		
Tetracyclines	Inhibit protein synthesis by reversibly binding to 30S ribosomal subunit	Nephrotoxicity, discoloration of urine and erupting teeth
Chloramphenicol	Inhibit protein synthesis by reversibly binding to 50S ribosomal subunit	Reversible aplastic anemia (use carefully; it may causes idiosyncratic anemia in humans)
Macrolides	Inhibit protein synthesis by reversibly binding to 50S ribosomal subunit	Intestinal prokinetic

Additionally, there are two general categories of antimicrobials: concentration dependent and time dependent. Both are commonly used in equine surgery to prevent SSIs but require an understanding of the principles of actions.

CONCENTRATION-DEPENDENT ANTIMICROBIALS

Concentration-dependent antimicrobials, such as aminoglycosides and fluoroquinolones, are reliant on the ratio of the peak plasma concentration of the antibiotic to the minimum inhibitory concentration (MIC) for the bacteria.[148] A ratio of greater than 10:1 or 12:1 is optimal for concentration-dependent antimicrobial effect.[148] This commonly results in a longer dosing interval with higher doses used at each point. Controlled dosing and dose intervals can be achieved by monitoring the peak and trough concentrations of the administered drug. Knowledge of the MIC for the commonly occurring bacteria at a surgical facility will allow calculation of the desired peak concentration (more than 10 times MIC). Renal clearance of the antimicrobials may be monitored by the trough concentrations immediately before redosing. If the trough concentration is not suitably low (e.g., below 2 μg/mL for gentamicin), the dose interval must be increased.[148]

TIME-DEPENDENT ANTIMICROBIALS

Time-dependent antimicrobials include the β-lactams and macrolides. These antimicrobials have a saturable concentration-dependent effect on bacterial killing. This means that at a certain point above MIC, there is no additional benefit to higher concentrations of the antimicrobial. Bacterial killing is optimized for these types of antibiotics by maintaining the concentration above the MIC for a longer period.[148] Time-dependent antimicrobials are commonly given more frequently and at lower doses to achieve this effect.

Prophylactic Antibiotic Use

Surgical antimicrobial prophylaxis refers to a very brief application period of an antimicrobial agent just before a surgical intervention. This practice has had an enormous effect on improving surgical outcomes historically.[3,148] Therapeutic antibiotics refer to the treatment of an established infection. As previously outlined, the development of an SSI is an interaction of the infectious dose, bacterial virulence, and the host's resistance. Prophylactic antibiotics are given to reduce the bacterial dose at the surgical site at the time of surgery and as a result reduce the incidence of SSIs.[149] Four basic principles must be followed to maximize the benefits of prophylactic antibiotics and avoid potential complications: (1) antimicrobials should only be used when the benefits of their use have been demonstrated by clinical trials or when an SSI associated with a surgical intervention would have catastrophic results,[3,39]; (2) a safe, inexpensive, and bactericidal antibiotic should be selected that covers the most commonly occurring contaminants of the operation[3,39,57]; (3) the time of administration is essential in achieving an effective tissue concentration at the time of surgery[39,57,148]; and (4) tissue levels of antimicrobials should be maintained throughout the procedure and at most for a few hours after the skin incision is closed.[39,57]

SELECTION OF PROPHYLACTIC ANTIBIOTICS

When selecting an antibiotic, pharmacokinetics, drug distribution, cost, and potential side effects should be considered.

Overall, antibiotic use should be restricted to patients in which the incidence of infection exceeds 5% without prophylactic antimicrobial use or when an infection would have devastating results.[148] Using the classification system outlined earlier, this generally means that all procedures that are not "clean" have an indication for antimicrobial prophylaxis.[57,148] The use of antimicrobials in clean surgery is controversial in both human and veterinary medicine.[39,57] In equine arthroscopy, the use of antibiotics is probably not indicated with the infection rate being reported to be 1.5%.[150] General clean orthopedic procedures with a reported rate of SSIs of 8% and clean-contaminated procedures with a rate of 52% to 57% are obviously clear indications for prophylactic antimicrobial use.[4,36] Similarly, abdominal surgery in the horse (emergency SSI of 39%; elective SSI of 7%) has good evidence to support the use of prophylactic antimicrobials.

A very important factor in the rational selection of antimicrobials is recognition and awareness of risk factors for SSIs. Furthermore, knowledge of the common causative agents of SSIs and the antibiogram commonly encountered at a surgical facility will also facilitate directed antimicrobial therapy.

TIMING AND DURATION OF ANTIBIOTIC ADMINISTRATION

The appropriate administration of prophylactic antimicrobials has been demonstrated repeatedly in a large number of studies.[57,148,149,151] Despite this common knowledge, compliance in human and veterinary medicine is truly staggering. In human studies, the appropriate administration of prophylactic antibiotics (1 to 2 hours before surgery) to achieve effective tissue levels at the time of surgery is only achieved in 55% to 61% of cases.[151] In a retrospective study of perioperative antimicrobial use in horses undergoing arthroscopic surgery, only 6.3% received penicillin within 60 minutes of the first incision.[147] The average time before surgery for administration of penicillin in this study was 142 minutes.[147] In humans, when the prophylactic antibiotic was administered 2 to 24 hours before surgery, the relative risk of SSI increased by 6.7 times; if it was administered more than 3 hours after the incision was made, the relative risk increased by 5.8 times.[152] Antibiotics should be administered within 1 hour before the skin incision.[137,149,151] Additional intraoperative doses of antibiotic are advised if the duration of the procedure exceeds 1 to 2 times the half-life of the antibiotic used.[57,149]

The prolonged administration of prophylactic antibiotics (longer than 24 hours) is of no benefit.[28,57,149] Within 24 hours of a surgical procedure, the surgical site is believed to be sufficiently sealed and resistant to microorganism entry so that antimicrobial use beyond this time should be unnecessary.[148] The common trend in human medicine has been to decrease antimicrobial administration from multiple days to 24 hours.[28,149] In fact, a single dose of antibiotics is currently thought to be as effective as administration of multiple doses for 48 hours. In equine surgery, the use of antibiotics can be substantially improved and has started to follow human medical trends.[148] Knowledge of risk factors, the likelihood of infection, and the potential consequences of a SSI will allow the rational selection and administration of prophylactic antibiotics.

POSTANTIBIOTIC EFFECTS

The postantibiotic effect (PAE) refers to the period of time after which the antimicrobial concentration has fallen below the MIC, during which growth of the target bacteria is suppressed.[153-155] The PAE depends on several factors: the antibiotic concentration, the duration of exposure to the antibiotic, the bacterial species, and the antimicrobial used. All antibiotics appear to be able to produce a PAE against gram-positive cocci, but β-lactam antibiotics induce almost no prolonged effects against gram-negative bacilli.[154,155] Aminoglycosides, fluoroquinolones, and protein-synthesis inhibitors exhibit the most pronounced PAEs. The greater the magnitude above the MIC, the longer the PAE is for concentration-dependent antimicrobials.[155]

PROPHYLACTIC ANTIBIOTICS USED IN HORSES

β-Lactam antibiotics

β-Lactam antibiotics, such as penicillin and cephalosporins, are commonly used prophylactic, time-dependent antimicrobials.[54,148] The effect of this group of antibiotics is saturable, and as such their effect is maximized by the percentage of the dosing interval above the MIC. At plasma concentrations of two to four times the MIC of a certain bacteria, there is no benefit to further increases in the antimicrobial concentration.[148] β-Lactam antibiotics are readily inactivated by bacteria that produce β-lactamase.

Aminoglycoside antibiotics

Aminoglycoside antibiotics inhibit bacterial protein synthesis by binding to the 30S ribosomal subunit, resulting in disruption of mRNA function and aberrant amino acid sequencing.[156] Commonly used aminoglycosides include gentamicin and amikacin. Aminoglycosides must be transported into the bacterial cell, which requires energy and oxygen; as a result, anaerobic bacteria are commonly resistant. Aminoglycosides are *concentration-dependent* antimicrobials and as such are reliant on the magnitude to which the bacteria are exposed.[54] A ratio of aminoglycoside concentration to MIC greater than 10:1 or 12:1 is optimal for concentration-dependent antimicrobial effect.[148,156,157] Additionally, higher concentration to MIC ratios potentiates the aminoglycoside PAE.[156] These pharmacodynamic properties have lead to extended-interval dosing strategies, or once-a-day dosing regimens.[148,154,156,158] In contrast to the concentration-dependent antimicrobial activity of aminoglycoside, the toxic effects depend on the duration of host tissue exposure, not the absolute concentration. The nephrotoxic and ototoxic effects of aminoglycosides are related to the plasma trough concentration immediately before repeated administration.[156] There are several benefits of extended-interval dosing: (1) concentration-dependent bactericidal activity can be maximized by optimizing the peak plasma concentration, (2) time-dependent toxicity is minimized, (3) duration of PAE is maximized, and (4) adaptive resistance is limited.[156] Bacterial resistance is commonly caused by downregulation of the necessary influx pathways, leading to decreased intracellular drug concentrations.

Trimethoprim-sulfonamides

Trimethoprim-sulfonamides are less commonly used than the previously mentioned antibiotics as prophylactic antimicrobials. However, trimethoprim-sulfonamides act synergistically to inhibit the synthesis of folic acid, a compound that is required for microbial DNA production. Formulations are usually at a ratio of 1 : 5 for potentiated sulfonamides and the trimethoprim respectively. Sulfonamides prevent the conversion of *para*-aminobenzoic acid (PABA) to dihydrofolic acid by substituting competitively for PABA; when sulfonamides are used alone,

they are bacteriostatic. Trimethoprim inhibits the subsequent conversion of dihydrofolic acid to tetrahydrofolic acid by inhibiting dihydrofolic acid reductase. The combined activity of trimethoprim-sulfonamide as a result inhibits two successive steps in the folic acid pathway, producing a bactericidal effect.

Special Routes of Administration and Dosages

As mentioned before, there is little doubt that improved clinical outcomes are possible when extremely high doses of appropriate antimicrobials can be instilled and maintained in proximity the infected tissue and implants. The advantages of local antimicrobial therapy include (1) the ability to expose the pathogens to extremely high concentrations of the drug, (2) the avoidance of adverse side effects because of high doses of systemic antimicrobials, and (3) the ability to deliver high doses of antimicrobials that would otherwise be economically unfeasible in large animals. It is important to note that appropriate antimicrobial selection based on culture and sensitivity results are essential to effective therapy and should be considered the gold standard.[28]

ANTIBIOTIC IMPREGNATED POLYMETHYL METHACRYLATE OR PLASTER OF PARIS

The use of *antibiotic impregnated polymethyl methacrylate (PMMA)* or *plaster of paris (POP)* are reliable tools for delivering high levels of local antibiotics to a site of infection.[28,54,159] The advantages of PMMA as an antibiotic delivery device are that its biocompatibility has been well studied, elution profiles for many antibiotics have been documented, and the material is readily available in a sterile, easily used form. The major disadvantages of PMMA are that it is not absorbable, and heat-labile antibiotics cannot be incorporated within it. Although it is easier to mix a powdered antibiotic with the PMMA, liquid injectable forms can also be used. The authors typically use 1 to 2 g of antibiotic for each 10 g of PMMA. Some authors suggest using 5% of the weight of the PMMA (i.e., amikacin, 0.5 g, and PMMA, 10 g) because of concern when PMMA sets.[28] Additionally it should be noted that addition of greater than 10% of antimicrobials weakens the biomechanical properties of the PMMA.[28] If a liquid antibiotic is used, the volume of liquid methacrylate is decreased by half the volume of the added antibiotic. The materials are mixed routinely and formed into cylinders or beads as the material becomes claylike. The ambient temperature of the room will affect the speed of this process. If the PMMA is to be put on a suture (e.g., No. 2 monofilament with multiple bulky knots) to facilitate later removal, that suture should be prepared before mixing. Another alternative frequently used is to simply make beads or cylinders that are not attached to suture and pack them in position. Removal of the PMMA is not necessary unless the plastic is interfering with function. Antibiotics that have been successfully used include gentamicin, amikacin, tobramycin, multiple cephalosporins, and enrofloxacin.[28,54] Premade beads can be resterilized by ethylene oxide (gas) sterilization. The major benefit of POP over PMMA is in situations where removal of PMMA will be particularly problematic. In addition, the plaster is very inexpensive and slowly degraded and absorbed by the body. The antibiotics leach at a fairly rapid rate; about 80% is eluted within the first 2 days.[28] The major disadvantage of POP versus PMMA is that the set-up time is slow in plaster. It is easiest to make the beads up aseptically well in advance and then keep them in a sterile container. They also can be sterilized using ethylene oxide but they become more brittle.

BOVINE COLLAGEN SPONGES

Other popular and easily used local antimicrobial delivery devices are bovine collagen sponges sold to augment surgical hemostasis in human surgery. The advantage of this material is that it is readily available as sterile packaged product, easily fits into any space, and is readily absorbed. We have not seen any allergic responses to its use in horses.

MISCELLANEOUS PRODUCTS

Other products have been used, and newer, improved materials will undoubtedly replace PMMA and POP. Polylactide derivatives, various hydrogels, alginates, polyanhydrides, calcium sulfate, chitosan, and fibrin are all being studied for the local delivery of antimicrobials. It seems likely that a variety of products with different mechanical properties, elution profiles, and absorption will eventually be available to clinicians.

REGIONAL LIMB PERFUSION

Regional limb perfusion (RLP) with antimicrobials is possible in situations where a peripheral vessel is accessible and an effective tourniquet can be applied to isolate the infected region. The major disadvantage of regional perfusion in postoperative cases is the condition of the tissues and the need to avoid vascular damage near the surgical site. Some antibiotics, notably enrofloxacin, will induce significant vasculitis when used for regional perfusions.[28]

If possible, a tourniquet is placed above and below the area to be treated. The key is to have an accessible peripheral vein distal to the proximal tourniquet. The latter should be placed after heavy sedation. If necessary, regional analgesia is used, but many horses will stand with sedation alone. A 25- to 27-gauge butterfly catheter seems to cause the least vascular trauma. Because repeated treatments are usually indicated, it is imperative to keep the vessel in the best possible condition. The application of topical anti-inflammatories such as diclofenac (Surpass) has been shown to decrease postinjection swelling and makes repeated treatments easier to perform.[139] In our opinion, repeated infusions using these very small butterfly catheters allow far more perfusions to be performed before the veins become difficult to use. Even well-placed indwelling catheters in the lower limb seem to cause more problems. Dosage varies, but around one third of a systemic dose diluted to about 30 mL is typical for the distal limb, 60 mL if the tourniquet is above the carpus or tarsus. We have been using systemic doses for regional perfusions without other antibiotic administration. The injection is administered slowly over a period of about 15 minutes, and the tourniquet is left in place for approximately 30 minutes, if possible. These very high doses given regionally subjectively seem to result in the best clinical responses, but more vascular irritation can be a complication with the more concentrated doses of antimicrobials. It is essential to the effectiveness of an RLP that the tourniquet is sufficiently wide and movement of the horse is minimized.[139]

Both intraosseous and intra-articular antibiotic administration are also used to maximize antibiotic levels at the desired site of action. Commercial intraosseous catheters (Cook) and homemade cannulated screws can be inserted for repeated intraosseous treatments. An even simpler technique is to drill a 4.0-mm hole into the medullary cavity at the desired location.

The male end of a Luer-tip extension set will fit snugly into the hole, allowing direct injection. Both intraosseous and intra-articular perfusions should ideally be administered under tourniquet for 30 minutes to maximize tissue levels.

Toxic Side Effects of Antibiotics

The use of antibiotics in horses is rarely associated with toxic side effects. A thorough understanding of the risk factors associated with the use of each type of antibiotics will help prevent these complications.

Antibiotic-induced diarrhea or colitis has been most commonly attributed to *Salmonella* species or *Clostridium difficile* in horses. Horses with colic are particularly susceptible to salmonellosis and are reported to have 4.2 times greater risk of developing salmonellosis than horses presented for other reasons.[160] Additionally, the use of parenteral and enteral antibiotics increased the risk 40-fold, whereas treatment with only parenteral antibiotics increased the risk by 6.4-fold compared to untreated horses.[160] This corroborates the clinical impression that oral antibiotics are associated with a higher incidence of antibiotic-induced diarrhea compared to parenteral administration. In horses undergoing elective arthroscopy, 6.3% of horses that had received antibiotics developed diarrhea within 7 days of surgery.[147] In most circumstances the diarrhea was mild and resolved readily; however, and especially in horses with abdominal surgery, the mortality rate can be as high as 40%.[147,148,161]

Nephrotoxicity is the most common side effect associated with aminoglycoside therapy in horses. The renal toxicity is linked to aminoglycoside accumulation in proximal tubule cells following uptake via the organic ion transport system.[162] This is a saturable process, and as a result the degree of accumulation and toxicity depends more on time than on absolute dose.[162] Because of this mechanism and the concentration-dependent antimicrobial killing effect of aminoglycosides, once-daily dosing routines have been developed to reduce nephrotoxicity and increase efficacy.[148] We routinely use an 8.8 mg/kg sid dose rate of gentamicin in horses undergoing elective and emergency procedures without adverse effects.

Neuromuscular blockade in horses is a potential, although very unlikely side effect of aminoglycoside administration in horses. The use of 6 mg/kg in horses anesthetized with halothane resulted in no significant neuromuscular blockade in horses.[163] Gentamicin has been shown to augment the neuromuscular blockade of atracurium under anesthesia, but the effect was minimal.[164]

Fluoroquinolones, such as enrofloxacin, have been linked to detrimental effects on tendon, cartilage, and bone in horses.[165-168] The detrimental effect of enrofloxacin is of greater severity in younger horses, so it should be used with caution in these situations.[168,169]

Emergence of Bacterial Resistance to Antibiotics

Antimicrobial drug resistance is a very serious issue for both human and veterinary medicine. The cost of antimicrobial resistance to the U.S. human health care system was estimated to be $100 million to $30 billion annually.[170] Unfortunately, the use of antibiotics in animals has been maligned in the lay press, mostly inappropriately, as a cause of antimicrobial resistance. The emergence of drug resistance has been seen in equine pathogens between 1992 and 1997, and in a German study there was a 75% increase in resistance of *E. coli* to tetracyclines, 80% increase to ampicillin and 90% increase to sulfonamides. Furthermore, for *S. aureus,* resistance increased by 50% to 60% to penicillins and by 400% to gentamicin.[171] Bacteria develop resistance to antibiotics through many and varied mechanisms. These include β-lactamase inactivation of penicillins and cephalosporins, enzymatic deactivation of aminoglycosides, and chromosomally based reduction in susceptibility to trimethoprim. The excessive and often inappropriate use of antimicrobials in both human and veterinary medicine must be avoided to ensure the long-term efficacy of antimicrobial prophylaxis.

Summary of Antibiotic Prophylaxis

The use of perioperative antimicrobials in horses must be based on educated and scientifically proven principles. A large number of studies evaluating the use of antimicrobials in surgical facilities, both human and veterinary, have unanimously demonstrated noncompliance to multiple established antimicrobial principles. To prolong the efficacy of our antimicrobial options it is imperative that these principles and guidelines be followed.

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CHAPTER 8

Regenerative Medicine

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The impetus for regenerative therapy in horses originated in part from promising results obtained in a clinical retrospective study in which autologous bone marrow concentrate was injected intralesionally into 100 horses with suspensory ligament desmitis.[1] The results of this large study indicated that this form of therapy was a safe, effective, and economical way to treat horses with this condition. Nearly 90% of the horses responded favorably, with a significantly higher percentage of treated horses returning to soundness without reinjury than had been reported previously with traditional treatments.[1] These promising results, however, were obtained comparing treated horses against data from the literature (i.e., historical controls), and no data were available regarding the cellular composition of the bone marrow used.

Since this study, many different approaches have been employed in equine practice to aid tissue healing. The majority of these have included the use of autologous *nonexpanded* material, such as conditioned serum, platelet-rich plasma, bone marrow concentrate, and adipose-derived progenitor cells (stromal vascular fraction). The advantages of these products include rapid turnaround times, lack of immunogenic concerns, and absence of federal regulatory control. More recently, the use of autologous or allogeneic products that have been *expanded* in a laboratory setting have gained in popularity. These include mesenchymal stem cells (MSCs) derived from adipose tissue, bone marrow, and neonatal tissues such as cord blood and tissue. These cell-rich products cannot be used as point-of-care treatments because they may take several weeks to expand. It is possible, however, to preserve these expanded products for multiple future allogeneic or autologous uses, if prepared by following rigorous laboratory protocols, which may require more stringent regulation as cellular preparations for medical use.[2]

In contrast to this relative abundance of therapeutic options, regenerative veterinary medicine is still lacking thorough scientific awareness and practical applicability. Although steps have been made in the understanding of the biology behind regenerative approaches in equine practice, there are still many areas that deserve in-depth investigation. For example, no precise recommendations can be made regarding the selection of a specific regenerative regimen for a given lesion. Prospective clinical trials are lacking that would help determine the appropriate timing of the treatment and the outcomes the veterinarian should expect following the treatment. Finally, regulatory bodies that would ensure that appropriate standards of care are followed when using regenerative approaches are still in their infancy in veterinary medicine. These and many other issues remain unresolved in defining the role of alternative approaches to healing equine injuries.

The purpose of this chapter is to review the concepts underlying the use of nonexpanded and expanded regenerative medicine therapies in horses, including blood-derived products and adult stem cells.

AUTOLOGOUS CONDITIONED SERUM

Interleukin-1 (IL-1) and tumor necrosis factor-alpha (TNF-α) are two primary proinflammatory cytokines that initiate and participate in the destructive cytokine cascade in osteoarthritis, leading to catabolic morphological changes within the joint, most notably, erosion of articular cartilage.[3,4] In the course of osteoarthritis, the number of available IL-1 receptors (types I and II) expressed increases, enhancing the detrimental effects of IL-1.[3,4] Therapeutic modalities aimed at diminishing the biologic activity of IL-1 through targeted inhibition of the interaction of IL-1 with its receptor have been proposed and investigated primarily in human but also veterinary medicine for over 30 years.[5] Some of these inhibitors are based on the delivery of IL-1 receptor antagonist protein (IL-1ra), which prevents the interaction between IL-1 and its receptor.[3] IL-1ra availability within the joint has been increased through direct injection of recombinant human IL-1 receptor antagonist protein,[6-8] gene therapy,[7,9-11] or "conditioning" of serum to stimulate production of IL-1ra.[4]

Autologous conditioned serum (ACS) is produced by conditioning certain blood components (primarily monocytes) to increase endogenous production of IL-1ra. Originally, ACS was produced by stimulating blood-derived monocytes with molecules such as immunoglobulin,[12,13] lipopolysaccharide (LPS), phorbol myristate acetate (PMA), IL-1, and TNF-α.[14] In the early 1990s, a clinically more applicable method for production of IL-1ra through exposure of whole blood to medical grade chromium sulfate–treated glass beads was discovered.[15] This method of conditioning was found to not only enhance production of IL-1ra, but also increase production of additional disease-modifying anti-inflammatory cytokines, including IL-4, IL-10, and IL-13.[15] In vitro experiments have established that a 10- to 1000-fold increase of IL-1ra to IL-1 must occur to block all of the available IL-1 receptors that are upregulated during osteoarthritis.[14] IL-1ra has been reported to be increased up to 140 times more than the other anti-inflammatory cytokines present in ACS.[16] The cytokine profile of ACS has been further expanded to include other anti-inflammatory cytokines (IL-6 and osteoprotegerin), growth factors (transforming growth factor-β1 [TGF-β1], fibroblast growth factor [FGF], hepatocyte growth factor [HGF], insulin-like growth factor-1 [IGF-1], platelet-derived growth factor [PDGF], vascular endothelial growth factor [VEGF]), and interestingly, proinflammatory cytokines such as IL-1β, oncostatin M, and TNF-α.[4] The concentrations of these cytokines have been shown to vary greatly among individuals, leading to wide standard deviations of cytokine concentrations in ACS.[14] Despite the controversial discovery of significant levels of proinflammatory cytokines in ACS, no major differences were found in proteoglycan metabolism of human osteochondral explants obtained from osteoarthritic knees incubated in media with or without ACS.[4]

The possibility of interrupting cytokine based cartilage destruction prompted the commercial development of the human-based product known as Orthokine[15] by Orthogen. Since 1998 Orthokine has been used in human medicine to treat osteoarthritis,[14,15] rheumatoid arthritis,[14,15] muscle injuries,[17,18] and spinal disorders.[14] The preparation is formulated by aseptically exposing whole venous blood to medical grade chromium sulfate–treated glass beads.[4] The blood is incubated at 37° C for 24 hours, then the serum is obtained after centrifugation and stored at −20° C. Six (2 mL) injections of ACS are given over the course of 3 weeks.[4]

Several studies evaluating the effects of ACS use in humans with osteoarthritis have been conducted. One study using validated patient-administered outcome assessments evaluated the long-term clinical effects of the intra-articular injection of ACS, hyaluronan, or saline. There was statistically significant improvement in the scores of those patients treated with ACS after 2 years.[14,19]

In the United States, Orthokine is marketed by Arthrex Vet Systems and is known more commonly in the equine industry as IRAP, the acronym selected to define IL-1ra protein. Indications for use in the horse include patients with synovitis, capsulitis, and mild to moderate osteoarthritis. There have been few controlled trials evaluating the use of ACS in horses. One study evaluated clinical (lameness, range of motion, response to flexion, synovial effusion), biochemical (synovial fluid analysis, glycosaminoglycans [GAGs], prostaglandin E_2 [PGE_2] , IL-1ra), and histological effects (synovium, cartilage) of intra-articularly administered ACS in an experimental model of equine osteoarthritis.[20] In this study, joints were injected four times, approximately 1 week apart, with either IRAP or placebo. No adverse reactions to the injections were noted. A significant increase in IL-1ra within the ACS-treated horses compared to unconditioned serum was seen.[20] In addition, mean synovial fluid IL-1ra concentration increased over time in treated joints. Interestingly, IL-1ra concentration was increased in both the osteoarthritic and the sham-operated joint, indicating a possible systemic effect because of an endogenous production of IL-1ra.[20] There was no significant difference in the degree of joint effusion, response to flexion, or response to joint manipulation, but on day 70, there was a significant improvement in the degree of lameness in the horses treated with ACS.[20] The degree of synovial membrane hyperplasia was also significantly decreased in those horses treated with ACS.[20] This was the first controlled study in horses to identify significant clinical improvement in osteoarthritic joints after ACS administration. ACS is currently being used clinically across the United States and Europe for various joint, tendon, and ligament injuries despite the lack of further substantial evidence regarding its use. Prospective controlled clinical trials are warranted to establish the effectiveness of ACS in treating naturally occurring osteoarthritis in horses.

PLATELET-RICH PLASMA

The intralesional and topical use of blood derivatives for the treatment of equine musculoskeletal trauma and cutaneous injuries has gained popularity and has been recently reviewed.[21-23] The use of blood components in trauma is based on the concept that the delivery of growth factors and proteins of the clotting system may optimize tissue healing. Studies have been aimed at identifying specific cytokines contained in these preparations,[24] assaying their content in platelet concentrate preparations, determining their in vivo and in vitro mechanisms of action, and standardizing the production methods.[25,26]

Platelet-rich plasma (PRP) is derived from centrifugation of whole blood after red blood cells and the buffy coat have been separated from the plasma, which is rich in platelets. PRP has been used successfully in a variety of human ailments, treating lateral epicondylitis (tennis elbow) with percutaneous injections,[27] enhancing early bone formation during lumbar spinal fusion,[28] and increasing the healing rates of chronic foot ulcers in diabetics.[29] Also, PRP gel is widely used in wound therapy and oral or maxillofacial surgery. The platelet-rich gel matrix is formed by coincubation of PRP with calcium ions or enzymes such as thrombin to activate the coagulation process.[29-32] The main idea behind the use of these products is to enhance tissue repair by delivering growth factors directly to the area of injury.

Platelets are a vital source of growth factors, chemokines, and cytokines, all of which are released during the reparative processes occurring in the early phases of tissue healing. It is thought that the delivery of PRP and platelet gel mimics and enhances the physiological processes of clotting and wound repair, thereby stimulating intrinsic tissue regeneration in which endogenous platelets are a vital component.[32,33] Furthermore, PRP may also stimulate chemotaxis of macrophages, angiogenesis, proliferation and migration of fibroblasts, and collagen synthesis, which are all vital components of tissue healing.[33] Following activation, the alpha granules contained within the platelet cytoplasm degranulate and release a series of growth factors. Some of these are known to participate in healing of soft tissues and include PDGF, IGF-I and IGF-II, TGF-β1, VEGF, FGF, and platelet-derived epidermal growth factor (PDEGF). Other growth factors also present in PRP, such as osteocalcin (Oc), osteonectin (On), fibronectin (Fn), and thrombospondin-1 (TSP-1), are more important in assisting bone and cartilage healing.[34-36]

Biology and Preparation of Platelet-Rich Plasma

PRP is thought to be advantageous in tissue healing because it has a direct regulatory effect on cellular processes such as mitogenesis, chemotaxis, differentiation, and metabolism.[37,38] As described earlier, growth factors released from PRP improve and enhance the healing of soft tissues and promote bone regeneration processes. The secretion of growth factors begins a few minutes after the introduction of PRP into the site of injury when the clotting cascade starts; over 95% of the growth factors are secreted within 1 hour.[39,40] Growth factors bind to the outer surface of cell membranes through specific transmembrane receptors. Studies have shown that adult mesenchymal cells, osteoblasts, fibroblasts, and endothelial cells express transmembrane receptors for growth factors contained in PRP.[33] Via specific signaling pathways, activated receptors induce the expression of genes responsible for a variety of cellular processes, including cell proliferation, tissue matrix and osteoid production, and collagen synthesis.[32,41] It is important to note that PRP does not act directly on the cell nucleus, thereby eliminating any possible risk of mutagenic activity and tumor formation.[42] This has been considered a risk with the use of other cell-based regenerative approaches such as treatment with embryonic stem cells. Under normal circumstances, platelets may continue to release growth factors for several days after reaching the injury site, because the half-life of platelets has been estimated to be 5 to 7 days.[42]

The regenerative potential of PRP depends on the levels of proteins and growth factors that are secreted by activated platelets. Attempts to quantify the concentration of growth factors released by activated platelets revealed that there is a substantial variability among species and individuals. Other factors may alter the content of growth factors in PRP at the time of delivery into tissues, including platelet enrichment technique and activation of platelets during processing. Platelet concentrates have also been shown to have a dose-dependent trophic effect on the proliferation of adipose-derived mesenchymal stem cells and tenocytes in culture. In fact, rates of proliferation were more pronounced in adipose-derived mesenchymal stem cells than tenocytes; proliferation began at 24 hours and was maintained for up to 72 hours after the introduction of PRP.[43]

There are many different systems on the market that produce PRP, with a high variability in the final products' characteristics, including platelet concentration, output volume, and fibrinogen concentration. These commercially available medical devices rely on two fundamental principles of platelet isolation: centrifugation and filtration. With the first method, specialized equipment has been designed to process whole blood via sequential centrifugation steps, which yields deliverable PRP with a known platelet concentration. An alternative method uses specifically designed filters with appropriate pore sizes that are adapted to whole blood collection devices and rely on gravity flow to isolate platelets.

Regardless of the technique used, it has been suggested that an optimal PRP formulation should concentrate platelets at least three to five times the baseline platelet count for that individual, or alternatively to a concentration of at least 1×10^6 platelets/μL.[44] Therefore, before each clinical use, platelets and the resultant concentrations of growth factors should be quantified. Unfortunately, commercially available PRP kits should be scrutinized carefully in equine practice because quality control standards used in people may not apply to horses. Centrifugation speeds needed to separate platelets are known to vary across species and likely influence the final composition of PRP.[45] Specifically, important interspecies differences have been found in blood viscosity, platelet density, and degree of platelet activation and aggregation.[44-50] Because there are few precise recommendations regarding PRP concentration and volume, clinicians should use the product with the highest platelet concentration possible.

In a laboratory setting, the preferred method for PRP preparation is the differential centrifugation technique. Venous blood is collected using commercially available blood collection bags containing acid citrate dextrose (ACD) solution, which is usually 10% of the volume of blood collected. ACD contains dextrose as an energy substrate to sustain cell viability; citrate, which chelates calcium ions, thus inhibiting the coagulation cascade; and sodium citrate, which is known to preserve platelet morphology and function.[51]

To obtain PRP, venous blood is drawn and separated into sterile 50-mL test tubes and initially centrifuged at low speed. The sedimentation of cellular elements during centrifugation is determined by their size and the difference between their density and that of the surrounding fluid. Therefore red blood cells and leukocytes settle more quickly than platelets and plasma. Plasma and platelets are carefully recovered above the buffy coat layer and maximally concentrated with subsequent centrifugations at higher speeds to achieve the desired volume. Quality control policies should be implemented in laboratories where a sample of PRP preparation is used for platelet count, aerobic culture, and growth factor analysis.

Platelet-Rich Plasma Applications in Horses

The repair process of tendon and ligament injuries, which are common in both human and equine athletes, is slow, and the quality of the repair tissue is inferior to the original tendon tissue. Consequently, healed tendons and ligaments are predisposed to reinjury.[52] Limitations in the healing potential of tendons and ligaments have been ascribed to a relatively poor vascularization and the need for essential growth factors to be present to guide the healing response.[52-54]

PRP is an attractive alternative to traditional means of therapy in soft tissue injuries in horses because it is known to contain specific growth factors that participate in tendon and ligament repair.[55-58] As one example, a study using a collagen-induced superficial digital flexor tendonitis model showed that serial treatments with IGF-1 (a growth factor found in PRP) improved cell proliferation, collagen content, mechanical stiffness, and sonographic appearance of treated tendons during 8 weeks of treatment.[59,60]

Drawing from this and similar experiments, a study was conducted to determine the temporal release of growth factors from PRP, bone marrow aspirate, and a commercially available freeze-dried, stabilized, platelet product (PP). The effects of growth factor release were tested by measuring gene expression patterns in tissue explants of equine superficial digital flexor tendon and suspensory ligaments. The authors pursued the study in part to determine the possibility of using PP as a readily available off-the-shelf product that would decrease preparation time and cost. The results of this study indicated that growth factor concentrations were proportional to platelet concentrations and that the highest platelet counts were found in PRP and PP, both of which had significantly higher TGF-β1 and PDGF concentrations compared to bone marrow aspirate. Interestingly, growth factor release was sustained in these tissue explants for 4 days without exogenous induction of platelet activation. Furthermore, gene expression profiles in both PP and PRP were more favorable than those obtained after exposure to bone marrow aspirate.[37]

Gene expression patterns, DNA, and collagen content were also evaluated in explants obtained from equine superficial digital flexor tendon and cultured in media containing blood, plasma, PRP, platelet-poor plasma (PPP), and bone marrow aspirate. Concentrations of growth factors TGF-β1 and PDGF were higher in PRP than all other blood products tested. Additionally, tendon explants cultured in 100% PRP had superior tendon matrix gene expression patterns without simultaneous stimulation of catabolic cytokines.[61] A similar *in vitro* study was aimed at evaluating the effect of blood-derived products on synthesis of suspensory ligament matrix. In contrast to the previous study in which PRP was found to be more anabolic in flexor tendon explants, this study indicated that acellular bone marrow and not PRP increased matrix molecule expression, without concurrent increased catabolic gene expression. This study seemed to highlight differences in the matrix metabolism between the superficial flexor tendon and the suspensory ligament and indicated that acellular bone marrow might be preferred over plasma, blood, PRP, or PPP as a biological means to enhance suspensory ligament regeneration.[62]

Additional evidence that different regenerative approaches may be indicated for different tissues comes from another study that compared effects of acellular bone marrow, PRP, and serum on cultured suspensory ligament fibroblasts. These products were tested to determine their ability to stimulate production of cartilage oligomeric matrix protein (COMP), a component of ligament healing response and a marker of appropriate ligament matrix (re)modeling.[63] The study found that although all preparations had increased COMP levels, exposure of the tissues to acellular bone marrow resulted in the greatest increases in both COMP and total protein synthesis by the suspensory fibroblasts. The conclusion drawn from this study was that treating suspensory desmitis with bone marrow aspirate may enhance healing by providing anabolic factors, which stimulate matrix production.[64]

Among the growth factors abundantly present in PRP, VEGF is a powerful stimulator of angiogenesis. In a model of surgically induced superficial digital flexor tendon injury, the effect of PRP on neovascularization was studied using color Doppler ultrasonography and immunological staining of factor VIII.[65] PRP induced significantly more neovascularization than the placebo treatment until at least 23 weeks after treatment. The authors of this study speculated that a prolonged increase in neovascularization might suggest a long-lasting effect of a single intratendinous injection with PRP.[65] Furthermore, in a similar but separate study, PRP-treated tendons had a better organization of the collagen network, a higher content of glycosaminoglycans, and a higher strength at failure when compared to placebo treated tendons.[26] Using different investigative approaches, these studies highlight a prolonged effect of PRP treatment, which may corroborate the clinical notion that in naturally occuring tendonitis and desmitis, frequent PRP injection is not necessary.

A single PRP treatment and a controlled exercise rehabilitation program were, in fact, recommended in nine Standardbred racehorses with moderate to severe midbody suspensory ligament desmitis. All nine horses returned to racing within a median time period of 32 weeks, competing at least once during the first and second years after returning to racing. Although only five horses raced during the third year from the injury, the authors suggest that combining intralesional PRP and a careful rehabilitation program afforded these horses an excellent prognosis for returning to racing.[66]

Platelet-Rich Plasma in Osteoarthritis

Although PRP has been used more frequently to treat soft tissue injuries, it is steadily gaining popularity in the treatment of joint disease. Initial evaluations of the intra-articular use of PRP in animal models and in people have ascribed some of its success to modulation of pain response via an anti-inflammatory effect.[67] It is also possible that growth factors in PRP aid cartilage repair, preventing the progression of osteoarthritis.[68,69] In a retrospective cohort study in humans with knee arthritis, PRP was significantly better than hyaluronic acid preparations in reducing pain and improving function.[70] Furthermore, in a prospective clinical trial, patients undergoing total shoulder arthroplasty demonstrated lower postoperative pain scores and improvement in functional internal joint rotation index.[71]

Guidelines for joint injection of PRP in horses have not been established. Currently no recommendations of volume, platelet concentration, and frequency of injection can be given, although anecdotal evidence suggests administration of up to 3 injections at 2-week intervals. A review of five horses with osteoarthritis in which this approach was used showed improvement in degree of lameness and joint effusion that was maximal 2 months after the last PRP injection and persisted up to 8 months.[72]

ADIPOSE-DERIVED STROMAL VASCULAR FRACTION

Proof that adult stem cells could be obtained from adipose tissue was initially demonstrated in 2001, when an adipose aspirate was found to contain large numbers of mesenchymal stemlike cells that could be induced to differentiate into adipogenic, chondrogenic, myogenic, and osteogenic lineages.[73,74] Since then, many horses with soft tissue injuries and joint disease have been treated with a nonexpanded adipose-derived autologous preparation called stromal vascular fraction (SVF) (data from Vet-Stem; www.vet-stem.com). This fat-derived mononuclear cell fraction contains MSCs, T regulatory cells, endothelial precursor cells, preadipocytes, and macrophages.[74,75] When compared to bone marrow, adipose tissue is easier to obtain and has a proportionally higher content of MSCs, which according to some may also be easier to expand *ex vivo* and cryopreserve.[76,77]

Primary adipose tissue–derived cells such as those found in the SVF preparation are an attractive option because they do not require the 3 to 4 weeks necessary to expand MSCs, or the laboratory costs and expertise needed to avoid contamination during *ex vivo* expansion. Additionally, because SVF is considered a minimally manipulated autologous preparation, it is unlikely to undergo scrutiny or require approval of the U.S. Food and Drug Administration. Unfortunately, there are few studies that have critically addressed the clinical merit of SVF in veterinary medicine. Nonetheless, the abundant use of these products has supported the anecdotal notion that SVF may have benefit in healing of tendon and ligament injuries without any ill effects locally or systemically.[78,79]

In a blinded placebo-controlled study, SVF was used to treat collagenase-induced tendonitis in horses. The results of that study showed an improvement in inflammatory cell infiltrate, collagen fiber uniformity, polarized collagen fiber crimping, overall tendon healing score, and collagen oligomeric matrix protein scores.[80]

Furthermore, the intra-articular injection of adipose-derived stem cells significantly improved lameness, range of motion, and overall quality of life in double-blinded, multicenter, randomized studies conducted in dogs with osteoarthritis of the elbow joint.[81] However, the results were different when a well-established model of osteoarthritis of the equine middle carpal joint was used, and the efficacy of intra-articularly administered SVF and bone marrow–derived MSCs was compared.[82] Although no adverse effects were noted, this study was unable to demonstrate significant differences in clinical, biochemical, and histologic parameters between horses in the placebo or treatment groups. The one exception in that study was that synovial fluid obtained from joints treated with bone marrow–derived MSCs had decreased production of PGE_2 when compared to fluid from placebo-treated joints.[82] The obvious discrepancy that exists between anecdotal clinical impressions and controlled studies regarding the use of SVF in horses should motivate

researchers and clinicians to conduct well-designed, multicenter, prospective clinical trials.

MESENCHYMAL STEM CELLS

Stem cells can promote tissue regeneration, regulate the inflammatory response caused by trauma, and reduce scar formation during the processes of tissue repair.[83-85] Clinicians, researchers, and members of industry have explored the option of harvesting stem cells from the horse to promote tissue healing and explore their commercial value as a therapeutic agent. As often occurs in the equine industry, this new and exciting treatment has found widespread clinical applications without the development of a corresponding thorough scientific understanding of equine stem cell biology. Although equine researchers and clinicians are expanding our understanding of stem cell use in the horse, most of the concepts that currently underpin their use are extrapolated from a wealth of information in the human literature. Even an incomplete review of this work allows one to conclude that scientists have approached stem cell biology from many directions. Researchers have dedicated themselves to understand the role of stem cells in the maintenance of homeostasis during physiological tissue turnover,[86-88] to elucidate their phenotypic and genotypic characteristics in a variety of species,[89-92] and to explain the mechanisms underlying the interactions of these cells with the host environment.[93-96] Furthermore, the potential therapeutic value of stem cells is supported by the multitude of completed and ongoing clinical trials aimed at resolving many pathologies, including cancer, graft-versus-host disease, multiple sclerosis, and many others (see clinicaltrials.gov, a service of the National Institutes of Health).

Stem cells are able to replicate through a process of self-renewal and differentiate into various mature cell types. These characteristics are important in organogenesis during embryonic development, in the maintenance of tissue homeostasis during the normal processes of cellular turnover, and in tissue regeneration following injury.[97] Stem cells are generally divided into two groups, embryonic and adult. Embryonic stem cells are considered pluripotent because they are able to differentiate into cells belonging to all three germinal tissues, including endoderm, mesoderm, and ectoderm.[97,98] They are derived from the blastocyst stage of the mammalian embryo and are capable of unlimited, undifferentiated proliferation *in vitro*. Embryonic stem cells have been isolated from equine embryos, cultivated up to the blastocyst stage, and phenotypically and genotypically characterized.[99-101] Equine embryonic stem cell lines have been determined to be pluripotent *in vitro*, but to date there are no *in vivo* studies to support this quality.[102,103] Various companies have begun to explore commercial opportunities associated with embryonic stem cells (i.e., Celavet, Oxnard, CA; ViaGen, Austin, TX), although ethical and moral concerns that have beleaguered human embryonic stem cell research and applications are likely to pose similar concerns within the equine industry.[2]

After birth, adult stem cells, also known as somatic stem cells, can be found in various tissues in an environment known as a "niche," the characteristics of which depend on the nature and type of tissue in which they are located.[104,105] Although the concept of a niche as a reservoir seems to be well established, there are several aspects of somatic stem cell biology *in vivo* that are unclear. For example, it appears that injury will activate quiescent MSCs and stimulate self-renewal, but aging and disease states cannot.[105,106] The signaling pathways responsible for preserving these cells within a niche in an undifferentiated state and those regulating their activation are poorly understood.[107-109]

Because they are relatively easy to harvest, the adult stem cells that were initially studied and used for their regeneration capacity were the hematopoietic stem cells, which are responsible for reconstituting the cellular fraction of blood. These cells have played vital roles in the treatment of diseases in many systems, especially of the hematopoietic system. They are primarily isolated from bone marrow but have also been found in umbilical cord blood and peripheral blood.[110-112] In addition to the hematopoietic cell fraction, the bone marrow serves as one of the main niches from which adult mesenchymal stem cells are obtained. These cells are often referred to as bone marrow stromal cells, which are established in the developing marrow cavity within sinusoids near the branching of terminal marrow arterioles.[113] Although neither *mesenchymal* nor *stromal* are terms that adequately describe the properties of adult stem cells, they have been used interchangeably[114,115] and the term *mesenchymal stem cells* (MSCs) will be used from this point forward in this chapter.

MSCs were first described as adherent, clonogenic, nonphagocytic, and fibroblastic cells. They were isolated from the adult bone marrow stroma and under appropriate experimental conditions were able to differentiate into a variety of connective tissues, including cartilage, bone, adipose tissue, and fibrous tissue.[113,116-118] MSCs have since been characterized as undifferentiated cells capable of asymmetric cell division, a process by which one daughter cell develops into a somatic cell type while the other retains stem cell identity within the niche tissue.[119]

Several studies have demonstrated that MSCs obtained from human, canine, rabbit, rat, mouse, and horse can differentiate both *in vitro* and *in vivo* into somatic mesenchymal phenotypes, including bone,[120,121] cartilage,[122] tendon,[123,124] muscle,[125] adipose tissue,[125] and hematopoietic-supporting stroma[126,127] and unexpectedly into neural cells,[128] cardiomyocytes,[129] and pneumocytes.[130]

Regulation of Inflammation and Modulation of Immune Responses

In addition to their capacity to differentiate into various cell lineages and their potential use in tissue repair, MSCs are potent regulators of immune responses. For example, MSCs inhibit T-cell activation, B-cell function, and dendritic cell maturation *in vitro* and efficiently protect against allograft rejection and experimentally induced autoimmunity.[131-134] Moreover, MSCs recently were proved to be highly effective in the treatment of a murine model of acute and chronic colitis.[135,136] In those studies, a single systemic injection of MSCs at the onset of the disease ameliorated clinical signs and histopathologic lesions, and they reduced mortality rates.

The results of several studies have shown that the beneficial effects of MSC therapy are not restricted to administration of autologous cells, but that allogeneic cells are just as efficient in improving outcomes of colitis, myocardial infarction, and renal disease.[137-139] These findings suggest that the immunosuppressive action of MSCs is not restricted by class I major histocompatibility complex, and that the infused MSCs are tolerated by the host's immune system.[140]

One recent study explored the location and tissue interaction of autologous and allogeneic MSCs following injection into the equine superficial digital flexor tendon.[141] Follow-up examinations revealed that viable fluorescently labeled MSCs had remained within the lesions created in the tendons for up to 8 weeks posttreatment and that no visible cell-mediated immune response to the allogeneic treatment could be detected histologically.[141] Although more research is needed, this report suggests that allogeneic MSCs may not trigger an immune response, opening the exciting possibility of using banked allogeneic cell lines in equine regenerative medicine.

An emerging body of data indicates that MSCs reduce proliferative responses by allogeneic lymphocytes and the synthesis of inflammatory mediators by stimulated lymphocytes cocultured with MSCs.[142,143] Furthermore, evidence suggests that a profound anti-inflammatory effect can be elicited by using the supernatant obtained from cocultures of MSCs and stimulated lymphocytes. For example, studies have shown that MSCs alone or cocultured with lymphocytes secrete IL-10, a well-recognized growth factor for regulatory T cells.[144,145] IL-10 can subsequently antagonize the effects of IL-12 during induction of inflammatory immune responses.[145] Similarly, TGF-β1, which also plays a role in T-cell suppression, is secreted into media in cocultures of human MSCs and immune cells and can be used as an acellular preparation to modulate inflammation.[146,147] The potential application of MSCs in horses to control inflammation has not been explored, although modulation of wound healing and control of sepsis, endotoxemia, and other common equine inflammatory conditions may be researched in the very near future.

MSC Isolation from Horses

MSCs have been isolated with success from equine bone marrow and adipose tissue.[93,148-150] Equine MSCs have been also successfully isolated from prenatal tissues, such as placenta, umbilical cord blood, fetal bone marrow, and blood.[151-154]

Although bone marrow has been the most popular source of MSCs in horses for research and clinical applications, harvesting adipose tissue as a source of regenerative cells is advocated because it is readily available in large quantities beneath the subcutaneous space. The procedure is also less painful and lacks the possible complications associated with bone marrow aspiration.[155] Investigations into the superiority of one source of MSCs over the other have conflicting conclusions. Several studies have indicated little difference in the regenerative and differentiation potential, growth kinetics, cell senescence, and efficiency of gene transduction of MSCs obtained from the two sources.[156,157] Yet other research has supported the notion that fat yields more MSCs than bone marrow (2% versus 0.002%), and that adipose-derived MSCs demonstrate faster rates of proliferation and have a greater immunosuppressive ability.[158,159] One possible explanation for the superior yield of MSCs from fat may be derived from recent evidence of a link between cultured MSCs and vascular density of the tissue of origin. MSCs were associated with the vasculature, validating the use of adipose tissue as a source of mesenchymal progenitor cells for clinical purposes.[160]

Attesting to the lower number of MSCs found in the bone marrow, a study showed that only 0.01% to 0.001% of mononuclear cells isolated from the bone marrow form fibroblast-like colonies,[161] and this number decreases with age.[114] This is likely to be the case in horses where the quality of MSC harvest seems to decrease in older animals. When cells are harvested, plated, and near confluence, they can be divided among several culture flasks for amplification. As this process continues, cells assume a more homogeneous phenotype and may continue to proliferate for up to 40 generations, which has also been found in equine embryonic stem cells.[103]

Equine bone marrow is commonly obtained from the sternebrae, and bone marrow–derived mesenchymal stem cells (BM-MSCs) have been expanded and characterized.[162] An adequate sample of marrow can also be collected from the cancellous portion of other bones, such as the crest of the ilium or the proximal humerus and tibia. When harvesting from the sternum, horses are restrained in stocks and sedated with a combination of detomidine hydrochloride (10 μg/kg) and butorphanol (20 μg/kg). After clipping the midline area over the sternum, the intersternebral spaces are identified by ultrasonography. Aseptic preparation and local infiltration of anesthetic solution allows a stab incision to be made with a No. 15 scalpel blade on the sagittal plane of the sternum. A 10-gauge Jamshidi biopsy needle is introduced through the stab incision and advanced until it contacts the chosen sternebra. The needle is subsequently further advanced 3 or 4 cm with slow rotational motions. Syringes containing anticoagulant are attached to the needle, and marrow aspiration is completed. Occasionally, hemorrhage occurs at the biopsy site and is usually controlled by applying pressure with sterile gauze.

Once transferred to the laboratory, the bone marrow aspirate is processed using several cell isolation techniques, including direct adherence to a plastic tissue culture plate or density gradient centrifugation followed by adherence. With either of these methods, MSCs are grown using specific essential growth media, typically containing fetal bovine serum, and "purified" from the mononuclear cell fraction of the marrow because of their selective adherence to the plastic. The MSC amplification process involves the regular removal of nonadherent cells to obtain a more homogeneous cell culture, a process that requires between 2 and 4 weeks, depending on the number of cells harvested and the desired final cell number. Of interest is that when MSCs are seeded at low density they demonstrate a classic spindlelike shape, but when they are near confluence they begin to proliferate in layers and become flat with irregular cellular margins.[163]

Intra-Articular Use of MSCs

Regeneration of articular cartilage presents unique challenges because of its lack of vascularity and innervation, which leads to an inefficient and slow intrinsic healing capacity. Frequently, the tissue that regrows following injury consists of scar tissue or fibrocartilage that lacks the necessary mechanical properties to withstand the normal physiological strain of the joint, resulting in further articular degeneration.

Equine MSCs have been chondrogenically differentiated in monolayer culture, micromass pellets, or matrices. The micromass is a three-dimensional tissuelike construct that requires a minimal number of cells to induce the differentiation of mesenchymal precursors along the chondrogenic and osteogenic pathways.[164,165] Various growth factors have been studied, including TGF-β1, TGF-β3, and FGF. TGF-β1 at concentrations of 0, 1, 5, or 10 ng/mL were added to equine BM-MSCs monolayer cultures. In these studies, cells and media harvested after

4 days in culture. The media and cells were analyzed for evidence that the cells were being pushed to a chondrocytic phenotype. TGF-β1 at 5 ng/mL was shown to increase cellular density, cell layering, nodule formation, collagen type II mRNA expression, and collagen type I mRNA expression over controls.[166]

A study evaluating the effects of TGF-β3 supplementation compared BM-MSCs and adipose derived MSCs from 11 horses. Both cell types were cultured as micromass pellets with TGF-β3 (10 ng/mL) and bone morphogenetic protein (BMP)-6 (10 ng/mL) supplementation.[167] Pellet cultures were harvested at 3, 7, 14, and 21 days and analyzed for cross-sectional size, tissue composition, Alcian blue staining for GAG content, collagen type II immunostaining, and transmission electron microscopy. BM-MSCs obtained hyaline-like cartilage morphology by day 14 with lacunae formation and rounded chondrocytes, but adipose-derived MSCs produced a mature fibroblastic morphology for the duration of the study. BM-MSCs had more intense GAG staining and showed superior chondrogenic potential compared with adipose-derived MSCs.[167]

Another study compared the differentiation capacity of adipose-derived and BM-MSCs from three horses. BM-MSCs and adipose-derived MSCs were culture-expanded in growth media supplemented with 1 ng/mL TGF-β1 to prime the cells, then trypsinized and encapsulated (10×10^6 cells) in an agarose or a self-assembling peptide construct molded into 1.6-mm thick flat slabs. TGF-β1 supplemented BM-MSCs cultures had 5-fold higher ^{3}H-proline incorporation, 28-fold higher ^{35}S-sulfate incorporation, and 10-fold higher GAG concentration compared to adipose-derived MSC cultures supplemented with TGF-β1.[150] BM-MSC cultures supplemented with TGF-β1 produced extensive synthesis of aggrecan-like proteoglycan monomers and increased type II collagen gene expression. Histological analysis of the TGF-β1 supplemented BM-MSCs cultures revealed a continuous proteoglycan and type II collagen–rich extracellular matrix.[150] The results of this report would attest to superior chondrogenic differentiation capacity of BM-MSCs over adipose-derived MSCs when seeded in a hydrogel culture.[150]

Equine BM-MSCs and umbilical cord blood–derived MSCs (UCB-MSCs) have also been compared in their capacity for chondrogenic differentiation while maintained in a micromass pellet. UCB-MSCs were found to produce larger pellets with hyaline-like cartilage morphology, higher concentrations of cartilage-derived retinoic acid–sensitive protein, and higher gene expression of collagen 21, aggrecan, and CD-RAP. Sox 9 (marker of chondrogenic differentiation) expression, however, was similar in BM-MSCs and UCB-MSCs. This suggested that UCB-MSCs possess a higher capacity for chondrogenic differentiation than BM-MSCs.[168]

Unfortunately, production of extracellular matrix (ECM) by chondrogenically differentiated cells is not perfect. For example, a study comparing cartilage production of bovine-derived articular chondrocytes with bovine MSCs seeded on agarose gels and cultured with TGF-β3 under chondrogenic conditions showed that BM-MSCs underwent chondrogenic differentiation, but the amount of ECM produced and the mechanical properties of the ECM produced by the BM-MSCs was inferior to that produced by the articular chondrocytes.[169] In contrast, a more recent study compared chondrogenic differentiation of harvested chondrocytes and BM-MSCs from foals and a skeletally mature adult horses.[170] Constructs seeded with immature and mature BM-MSCs produced tissue that was mechanically superior to constructs seeded with immature and mature chondrocytes. BM-MSCs also produced longer core protein and longer chondroitin sulfate chains than chondrocyte-seeded constructs independent of age. BM-MSCs also produced cartilage-like ECM that was mechanically superior to age-matched chondrocytes.[170]

The majority of studies evaluating the use of BM-MSCs in articular regeneration use three-dimensional scaffolds and growth factors to chondrogenically differentiate MSCs and promote ECM formation. However, a large body of research has been dedicated to the implantation of undifferentiated BM-MSCs in suspension and relying on the synovial environment to influence the BM-MSCs toward the chondrocytic phenotype.[171] Injection of BM-MSCs in suspension would be clinically superior to scaffold implantation because injection is not as invasive as arthrotomy. Reduced cost, reduced morbidity, and decreased recovery time are also important advantages. Furthermore, injections of BM-MSCs would theoretically treat the synovial environment as a whole rather than only the local environment of the articular cartilage defect. Unfortunately, little is known regarding the intra-articular behavior of MSCs *in vivo*. Many pertinent questions remain unanswered regarding the differentiation of MSCs into the surrounding host tissue; also unknown is the possible trophic effect they may have on the surrounding cells through release of growth factors or cytokines to produce extracellular matrix.[171] The answers to all of these questions are likely to be complicated when accounting for the various permutations of the osteoarthritic synovial environment.

In one of the first studies conducted to evaluate intra-articular use of BM-MSCs in a large animal model, an experimentally induced osteoarthritis caprine model with anterior cruciate ligament transection and medial meniscectomy was used.[172] Approximately 10×10^6 autologous BM-MSCs suspended in 20 mg of hyaluronic acid were injected intra-articularly 3 weeks after osteoarthritis was induced. This study found that the degree of cartilage destruction, osteophyte formation, and subchondral sclerosis were all reduced in the treated joints compared to the control joints, and meniscal restoration was observed 6 weeks after injection. After 20 weeks, the repaired menisci had large areas that stained positive for proteoglycan and type II collagen with the typical appearance of fibrocartilage.[172] This study highlighted the potential of a therapeutic intra-articular injection of BM-MSCs in large animals with concurrent femorotibial soft tissue disease.

One subject of debate is the engraftment of intra-articularly administered MSCs. Researchers have expressed concern that cells may not adequately adhere to the areas of defective cartilage, which would diminish their effectiveness. One group implanted fluorescently labeled MSCs suspended in a gelatin matrix into full-thickness chondral defects in a caprine model. They found that the MSCs were gradually lost from the implant and that large fragments of the gel could be found in deep marrow spaces.[173] In another study, human MSCs were implanted *in utero* in sheep and then followed to assess engraftment and survival.[174] It was found that MSCs underwent site-specific differentiation into chondrocytes, adipocytes, myocytes, cardiomyocytes, bone marrow stromal cells, and thymic stroma. However, cartilage engraftment was found to be inefficient. [174] Despite these concerns about articular cartilage engraftment, cell therapy has been positively correlated with improvements in clinical symptoms of disease.

Regarding the intra-articular use of MSCs in horses, there is substantial contrast between the paucity of research and clinical trials and the relatively numerous anecdotal reports of horses benefiting from injections of autologous MSCs (see data from Vet-Stem; www.vet-stem.com). It is important to note that there are no controlled clinical trials that evaluate the use of MSCs in naturally acquired equine joint disease. One study had approximately 12 million equine BM-MSCs suspended in a self-polymerizing autogenous fibrin matrix implanted into 15-mm cartilage defects in the lateral trochlear ridge of the equine femur.[175] Arthroscopic evaluation 30 days postinjection revealed significant improvement in arthroscopic scores for cartilage defects treated with BM-MSCs. Biopsy of the defects at 30 days revealed increased fibrous tissue with predominant production of type II collagen. Unfortunately, 8 months after injection, no significant differences were detected in arthroscopic scores and biopsy assessments for proteoglycan and collagen type II. This led to the interpretation that BM-MSCs helped early chondrogenesis but did not significantly improve the long-term outcomes.[175]

Following this study, a technique was developed in which the nucleated cell fraction from equine bone marrow was heavily concentrated, suspended in a fibrin construct, and used to treat full-thickness articular defects. Twelve weeks after treatment, concentrated fibrin-based bone marrow aspirates yielded positive arthroscopic scores that were also visible 8 months postinjection. In fact, treated horses continued to show significant improvement over control animals in gross, histological, and MRI grades.[176]

In contrast, and as reviewed more in detail in the section on stromal vascular fraction, intra-articular injection of BM-MSCs, adipose derived stromal-vascular fraction, and a placebo were compared in an established experimental model of equine osteoarthritis.[82] The only reported beneficial effect of BM-MSC treatment was a significant decrease in the PGE_2 concentration in synovial fluid in both the sham operated and treated joints, indicating a possible systemic effect of the BM-MSCs.[82]

Despite these results, a report was published on 162 equine cases treated with BM-MSCs from six equine referral centers. A total of 97 horses with 101 treated lesions were available for follow-up. Three horses that were injected intra-articularly had adverse reactions, but they improved and were able to return to work. Fifty-two of 61 (85%) horses with soft tissue injuries and 29 out of 40 (73%) with orthopedic injuries were able to return to work with mean follow-up time of 21 months. No significant association with outcome was found for age, sex, breed, discipline, or severity of lesion.[149]

Application of MSCs in the Treatment of Tendonitis and Desmitis

Tendon and ligament injuries in horses are a major cause of unsoundness in part because these injuries require prolonged healing times and are often the cause of reinjury and further loss of soundness.[177] As occurs in other tissues, tendon healing follows a sequence of events consisting of an acute inflammatory phase, a proliferative (fibroblastic) phase, and a remodeling phase, during which fibrous scar tissue replaces the originally well-arranged collagen network. The elasticity of a scarred tendon is vastly inferior to that of healthy tendon, which increases the risk of reinjury.[177] For more information see Chapter 83.

The use of MSCs as candidates for tendon and ligament regeneration has been recently reviewed.[57,178,179] MSCs have been considered as viable candidates to support the healing of soft tissue injuries because of their differentiation into tenocytes that may occur following *in vivo* transfer.[178-180] It is also possible that MSCs provide the injured area with growth factors that may guide the tissues toward an improved healing response.[181-183]

An original report described the implantation of expanded BM-MSCs into a superficial digital flexor tendon lesion in a horse. This report was the first to describe the expansion and intralesional injection of BM-MSCs using plasma as the vehicle. It also highlighted the need to expand the use of cell therapies in horses with tendonitis by designing carefully controlled clinical trials.[184] Similarly, racehorses with naturally occurring superficial digital tendonopathy were successfully treated via the injection of undifferentiated autologous BM-MSCs.[185] In this study, 90% of racehorses returned to previous levels of competition following treatment, without evidence of reinjury for more than 2 years, whereas in the non–MSC-treated control group, reinjury occurred in all horses after a median time of 7 months.[185]

Intralesionally administered BM-MSCs were shown to cause tendon regeneration in a collagenase-induced tendonitis model in Standardbred horses.[186] A similar tendonitis model was used to assess the potential of adipose-derived nucleated cell fractions for improving tendon repair. The study concluded that cell fractions improved the overall tendon architecture and fiber organization and supported the use of such a preparation to aid in the therapy for tendonitis.[80]

The reader should be aware of the difficulties arising from comparing adipose-derived nucleated cell fractions (discussed previously in "Adipose-Derived Stromal Vascular Fraction") and culture-expanded MSCs in the therapeutic approach to tendonitis and desmitis. Expanded cell cultures have the disadvantage of requiring several weeks of preparation before clinical use, although the most appropriate timing of any regenerative treatment remains subject of debate. Expanded MSCs offer the opportunity to manipulate the cells before introducing them into the injured tissue. As an example, a recent study evaluated the effects of MSCs as well as IGF-1 gene-enhanced MSCs on tendon healing *in vivo* using an equine collagenase model of flexor tendonitis.[187] The study reported that following treatment, tendons had improved biomechanical characteristics; this supports the use of MSCs alone and IGF-1–enhanced MSCs into tendonitis core lesions, although the differences between the two treatments were not significant.[187]

Only a few reports are available that describe the long-term outcome of horses with naturally occurring tendonitis treated intralesionally with MSCs.[179] One large study reviewed 168 horses with superficial digital flexor tendonitis treated with approximately 10×10^6 expanded BM-MSCs. That study found that the reinjury rate was significantly lower (13% and 18% for horses in full training) than had previously been reported for both race and sport horses.[179] Furthermore, another report of 120 cases where BM-MSCs were used to treat tendonitis and desmitis reported that nearly 80% of horses with superficial digital flexor tendonitis and over 70% of horses with suspensory ligament desmitis had returned to their previous level of performance.[188]

In an additional clinical study, adipose-derived MSCs grown in the presence of autologous platelet lysate were inoculated into injured equine flexor tendons after the cells had been dispersed in activated PRP.[189] A total of 14 out of 16 treated horses

showed a functional recovery and were able to return to their normal activity. The authors of this report attributed the success of the treatment in part to the biological effects of PRP, which acted as a scaffold, maintaining MSCs in the tendon defects and contributing to tissue healing.[189]

More recently, efforts have been made to promote MSC differentiation *in vitro* before implantation into tissue. Researchers have explored the possibility of genetically manipulating BM-MSCs by introducing growth factors such as BMP within the cells, thereby using them as a delivery vehicle for tissue regeneration.[190] BMPs are a group of related proteins in the TGF-β superfamily known for osteoinductive capacity. However, BMP-12, has been implicated in the biological processes leading to the formation of tendon and ligament-like tissue *in vivo* and has been shown to promote tenocyte differentiation *in vitro*.[191] As an example of such an effort in equine research, one investigator cultured superficial digital flexor tendon–derived tenocytes and BM-MSCs, either untreated or transduced with adenovirus encoding green fluorescent protein, adenovirus encoding BMP-12, or adenovirus encoding BMP-2.[191] Both cell types had increased BMP-12 gene expression, were viable up to 6 days, and became mineralized with BMP-2 but not BMP-12. The study concluded that BM-MSCs may be able to serve as a cell delivery method for BMP-12.[191] In a similar study, BM-MSCs were shown to differentiate into tenocytes following exposure to BMP-12, as assessed via the measurement of tendon-related markers such as tenomodulin and decorin.[192]

Safety Concerns

The most commonly cited concerns when considering MSC-based therapeutic approaches are development of tumor cells and the contamination of the injected cells with infectious biological and nonbiological substances during the laboratory cell process. MSCs are capable of forming teratomas, although this aspect of their biology has been poorly characterized in animal models. Veterinarians need to be acutely aware of the possible transmission of infectious diseases, especially with the more frequent application of allogeneic transplants of MSCs. Consideration should also be given to the possible introduction of biological components that may be immunoreactive to the recipient. These components may be derived from exposure of MSCs to culture media elements such as fetal bovine serum.[148,153]

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SECTION
II

Surgical Methods

Jörg A. Auer

CHAPTER
9

Instrument Preparation, Sterilization, and Antiseptics

David E. Freeman and Jörg A. Auer

In human medicine, national regulations demand that quality control and assurance be observed for reprocessing medical devices and instruments, including resterilization for aseptic surgical use. In the veterinary field, the veterinarian, practice manager, or head of the department is responsible for evaluating the risks, classifying the various risk areas, providing written standards and detailed instructions, and ensuring adequate documentation for quality control. Validated cleaning, disinfecting, and sterilization processes, supplemented by defined configurations for loading the washer-disinfectors and autoclaves, are essential for quality assurance.[1] Other requirements result from good veterinary practice.

It is particularly important to follow the manufacturer's instruction manual, because ignoring these instructions could lead to expensive and untimely replacements or repairs and also because incorrect reprocessing or product failure may endanger the patient or third parties.

Sterilization refers to the complete destruction or elimination of vegetative bacteria, bacterial spores, viruses, and fungi, by physical or chemical methods.[2] Antisepsis signifies the inhibition of the growth and development of microorganisms without necessarily killing them. Therefore, antiseptics can be applied to living tissues.[2] Physical methods of sterilization include heat (thermal energy), which is the most commonly used type in veterinary hospitals, and filtration and radiation, which are usually applied in the industrial preparation of sterile materials. Ethylene oxide is the most widely used method of chemical sterilization, but physical methods are considered to be more uniformly reliable.

INSTRUMENT PREPARATION AND PACKING

Cleaning

Instruments cannot be sterilized until they are completely clean, because steam cannot penetrate materials such as oil, grease, dried blood, and other organic material.[3-5] Ideally an assistant should wipe every instrument used during surgery, before it is replaced on the surgery table, to prevent blood drying on the instrument. Once the instruments are returned to the sterilizing room, they are prepared for autoclaving by immediately rinsing in cold water to remove any remaining blood and debris.

Water plays an essential role in cleaning and sterilizing instruments: it dissolves cleaners and other treatment agents; transmits heat and other mechanical forces; dissolves unclotted blood, dirt, and impurities; removes cleaning fluids and detergents; and is used for steam sterilization.[1] Experiments have shown that increasing water hardness reduces the removal of blood in cold water prewashes.[1] It is therefore of utmost importance to use good quality water, ideally fully demineralized or at least softened water, because high concentrations of dissolved particles in the water can induce or facilitate surface pitting, even in high-quality stainless steel. Chloride concentration of the water that is greater than 120 mg/L (equivalent 200 mg/L NaCl) at room temperature increases the risk of surface pitting.[1] Even water evaporation during the drying process leads to drastic increases in chloride concentration in water droplets that exceed the limit just mentioned.[1] Other substances, such as silicilates or silicic acids, may cause brownish, bluish, gray-black, or iridescent discolorations even when present in small quantities. However, these discolorations are harmless and do not facilitate corrosion.[1] Tap water occasionally contains rust particles from corroded water pipes, and these tend to adhere to the instrument surfaces during processing, causing rust spots that lead to subsequent corrosion.[1]

After prewashing, the instruments should be cleaned with a mildly alkaline, low-sudsing detergent or in an ultrasonic cleaner. Prerinsing in an enzymatic detergent solution is an acceptable alternative to manual cleaning.[1,6] Enzymatic detergents used for cleaning medical devices (Endozime, Enzol, Sterizyme, and MetriZyme) help remove proteins, lipids, and carbohydrates, depending on the formulation.[7] Many of the available enzymatic detergents have a minimum contact time of 2 to 5 minutes (Endozime AW Plus, Asepti-Zyme, Gzyme, Optim22, Adi-Zyme, and Klenzyme) or 10 minutes (MetriZyme), and the recommended temperature for most is room temperature.[7] For the majority of them, a maximum soaking time of 30 to 45 minutes is recommended.[7] No commercially available detergents combine cleaning efficiency with microbial killing, with the exception of a newly formulated hydrogen peroxide–based cleaning detergent (Hydrox).[7] The advantages of Hydrox are in the realms of protection of health care workers from infectious risk and reduced bioburden on instruments before sterilization or disinfection.[7]

Instruments containing narrow lumens, such as cannulas, flexible tubes, and egress portals, are difficult to clean. Therefore it is important to ensure that all internal and external surfaces are completely wetted by cleaning solutions. Care must also be

taken to see that the lumens are patent. Soft, lint-free cloths or towels, plastic brushes, or cleaning air guns should be used to clean these channels. Following cleaning, both the external surfaces and internal surfaces should be rinsed, ideally with fully demineralized water. After rinsing, the instruments must be dried carefully. The drying method of choice is compressed air drying, because it is gentle and extremely effective.[1]

Ultrasonic cleaners use high-frequency vibratory waves that clean through cavitation.[7] As the ultrasonic waves propagate through the liquid, this liquid is subjected to alternating negative and positive pressures as the compression and rarefactions of the sound waves pass. During the phase of negative pressure, numerous gas bubbles are formed and enlarge in the liquid. This formation of gaseous cavities in the liquid is called cavitation. During the subsequent phase of ultrasonic compression (i.e., phase of positive pressure), the pressure exerted on the newly expanded gas bubble compresses the same until the bubble collapses on itself, imploding with a consequent shock wave of energy. This shock wave of energy—a mini vacuum—provides the energy used to assist cleaning. Ultrasonic action effectively dislodges impacted debris from holes, jaws, box joints, channels, and complex surfaces, and it disrupts air pockets, ensuring thorough wetting during the cleaning process.[6] The instruments are loosely loaded in wire mesh trays for this process; all box locks are left open; complicated instruments, such as dynamic compression plate (DCP) drill guides, are taken apart; and instruments are thoroughly rinsed afterward to remove detached particles.[8] For ultrasonic cleaning, as used for the manual cleaning step, a nonfoaming enzymatic type of detergent solution is preferable (Endozime AW Plus, Asepti-Zyme, Gzyme, Sterizyme, Adi-Zyme, Klenzyme, and MetriZyme).[9] Ultrasonic cleaning times in modern equipment are approximately 3 minutes at frequencies of around 35 khz.[1]Following ultrasonic cleaning, the instruments must be thoroughly rinsed, preferably with fully demineralized water, to remove all cleaner residues. It is also advisable to treat hinged and articulated instruments with a special lubricant such as Sterilit Power Spray. These lubricants withstand sterilization and maintain proper functioning of the instrument during use.

Minimally invasive surgery instruments, rigid endoscopes, and high-frequency instruments must be disassembled for machine processing according to the manufaturer's instructions, and only those parts approved by the manufacturer may be cleaned ultrasonically. Camera units, optics, and optic cables should never be cleaned with this technology.[1]

Packing

All packs should be marked as to content, date of sterilization, and person responsible for their assembly, and the pack should be stored for times appropriate for the material and the method of storage (Table 9-1). For muslin wraps, double layers and two wraps are recommended for each pack. Alternatively, pima cotton can be used, which is a more effective barrier than muslin because of the smaller pore size. Pima cotton wraps can be reused approximately 75 times, after which so much fabric has been lost that no effective barrier against microorganisms exists.[5] Sterilization fleece, an interleaved security product, is the preferred synthetic wrapping material because of its superior durability and handling qualities and because its safe storage time is longer than that of fabrics.

Instruments can also be sterilized in stackable containers of aluminum composite material (Figure 9-1) that are dent resistant, are available in a variety of sizes, are easy to store and transport, and allow safe storage times of up to 1 year (Amsco Sterilization Container System). The aluminum composite increases the thermal conductivity of the container during drying to help ensure dry contents. The system is suitable for use in steam, ethylene oxide, and gas plasma sterilization. Three types of filters—cartridge (with internal chemical indicator), disc, or ceramic—are available for this system. A retainer ring maintains the filter in the filter access portal in the lid and base of the unit. Selection of the type of filter is determined by the sterilization cycle to be used, such as prevacuum steam, gravity steam, flash, ethylene oxide, or gas plasma.

Although pima cotton and sterilization fleece are well suited for instrument packs, these materials are rarely used to wrap single instruments. For this purpose, special sleeves have been

TABLE 9-1. Storage Times for Sterilized Packs

	Open Shelf	Closed Cabinet
Single-wrapped muslin (2 layers)	2 days	7 days
Double-wrapped muslin (each 2 layers)	3 wk	7 wk
Crepe paper (single-wrapped)	3 wk	8 wk
Heat-sealed paper and transparent plastic pouches	At least 1 yr	—

From Mitchell SL, Berg J: Sterilization. p. 158. In Slatter D (ed): Textbook of Small Animal Surgery. 3rd Ed. Saunders, Philadelphia, 2003.

Figure 9-1. Stackable Steriset sterilization container with the lid removed, showing a standard soft tissue instrument set. The instruments located in the bottom of the set are covered by a special paper sheet. On top of it, sponges, towel clamps, the cautery including its electric cord, two sterile light handles, and a sponge forceps are visible. The inside of the lid shows the two valves. In each of the valves, one of three types of filter systems (steam, ethylene oxide, or plasma gas) can be inserted, depending on the type of sterilization being carried out.

developed that are paper on one side and clear cellophane on the other. The sleeves come in different sizes to allow the packing of instruments of different sizes. Also, different sizes are required because the instruments should be double wrapped. The ends of the sleeves are heat sealed. The sharp points of all instruments must be protected by special paper or plastic covers. The paper side allows penetration of steam, ethylene oxide, or gas plasma, and the cellophane side provides a view of the contents (Figure 9-2). These single packs should be identified by date of sterilization and the person who packed it.

Autoclave Indicators

Autoclave indicator systems include *chemical indicators* that undergo a color change on exposure to sterilizing temperatures and biologic indicators, such as heat-resistant bacterial spores. An indicator tape on the outside of the pack provides no information about the sterility of the pack's contents, so an additional indicator should be placed in the center of the pack. Many of the currently available indicators of sterility are more reliable than simple physical indicators, such as tape, because they indicate that both temperature and time are sufficient for providing sterility (Figure 9-3), whereas tape merely indicates to the surgeon that the pack was subjected to heat. *Biological indicators,* however, provide more security that the contents of the pack are sterile. For most of the commonly used sterilization processes, special reference biological germs have been selected, such as *Bacillus stearothermophilus* for steam, formaldehyde, and hydrogen peroxide sterilization processes; *Bacillus subtilis* for ethylene oxide and dry heat sterilization processes; and *Bacillus pumilus* for radiation sterilization processes.[5,8,10] Depending on the type of sterilization process, a special resistance characteristic of biological indicators is required to prove the success of a defined sterilization process. During such a sterilization process, the spore population always decreases because of the exponential kill characteristic called reaction kinetics of first order. The population however, will never reach an absolute 0 value. Therefore modern definitions of goods declared sterile do not specify the absolute absence of biological activity, but determine aseptic conditions with certain probability, called sterility assurance level (SAL).[11]

Figure 9-2. A single wrapped catheter in a paper-cellophane sleeve. These wrappers may also be used for single instruments.

PHYSICAL STERILIZATION

Thermal Energy

Dry heat kills by a combination of oxidation and removal of water, whereas moist heat kills by the coagulation of critical proteins. Moist heat sterilization can coagulate and denature cellular protein at lower temperatures than those required by dry heat and thus can decrease the temperatures and exposure times necessary for sterilization.[3,4]

Exposure time and temperature required to kill microbes are functions of their individual heat sensitivities, which vary with type of organism and the environment to which they are accustomed.[4] For example, bacterial spores are more resistant than the vegetative form of the bacteria.[4] Recommended sterilization times and temperatures are designed to kill all microorganisms, even those that are heat resistant (Table 9-2). Minimum guidelines are an exposure time of 15 minutes at 121° C (249.8° F) and 15 psi or 2 atmospheres of pressure in a steam autoclave. Because microbial death occurs in a logarithmic fashion, exposure time is as important as temperature. The greater temperatures and water saturation attained by pressurized steam allow shorter sterilization times. Steam gives up its heat to materials to be sterilized by the process of condensation, and it is able to penetrate porous substances more rapidly than dry heat.[4]

The prion scare that surfaced a few years ago also had an impact on sterilization methods. Regimens had to be developed to ensure sterility of prion-contaminated medical instruments.[12,13] Although prions do not represent a major threat to equine surgery, it is worthwhile to become familiar with the proper techniques and protocols developed, in case a similar situation arises in our field.[12,13] Table 9-3 lists the appropriate regimens developed for the different prion sterilization techniques. The present European standards ask for a temperature setting of 134° C and a maintenance time of 18 minutes. This standard may be reversed, because the prion threat has drastically diminished. Such a change would be welcomed to increase efficiency in busy hospitals.

Figure 9-3. Example of a chemical indicator used to confirm that sufficient exposure conditions have been met in the center of the pack. In this 3M Comply (SteriGage) Steam Chemical Integrator, the chemical pellet contained in a paper, film, and foil laminate envelope melts and migrates as a dark color along the paper wick. The distance or extent of migration shown at the bottom (compare with unused, *top*) depends on exposure to steam, time, and temperature. The dark color should enter the Accept window for an Accept result.

TABLE 9-2. Exposure Times and Temperatures for Autoclave Sterilization Systems

Procedure and Conditions	Time (min)	Temperature	Comments
Heat-up time (prevacuum and pulse type)	1	Up to 120° C (250° F)	Timing of exposure begins when exhaust line reaches 120° C.
Minimum standard*	13	120° C	5-10 min destroys most resistant microbes; an additional 3-8 min provides a safety margin.
Present European standards	18	134° C (273° F)	Introduced because of prion scare; may be reversed
Emergency/"flashing" (prevacuum)†	3	131° C (270° F)	Instruments sterilized in perforated metal trays
Large linen packs (gravity-displaced)	30	120° C	—
Large linen packs (prevacuum)	4	131° C	—
Drying period	20	NA	—

*Times are given for gravity displacement autoclaves. Extra time is required for pack contents to reach sterilization temperatures (heat-up time).
†Emergency sterilization is best accomplished in prevacuum autoclaves, which have shorter heat-up times required.
From Southwood LL, Baxter GM: Instrument sterilization, skin preparation, and wound management. Vet Clin North Am Equine Pract 12:173, 1996 (with permission).

TABLE 9-3. Efficacy of Sterilization Processes in Inactivating Prions

Ineffective (≤3 $\log_{10}$ reduction within 1 hr)	Effective (>$\log_{10}$ reduction from 18 min to 3 hr)
Autoclave at standard exposure conditions (121° C for 15 min)	Autoclave at 121° C to 132° C for 1 hr (gravity displacement sterilizer) or 121° C for 30 min (prevacuum sterilizer)
Boiling	Autoclave at 134° C for 18 min (prevacuum sterilizer)
Dry heat	Autoclave at 134° C for 18 min immersed in water
Ethylene oxide	Hydrogen peroxide gas plasma (Sterrad NX)
Formaldehyde	Radiofrequency gas plasma
Hydrogen peroxide gas plasma, Sterrad 100S (ASP) (ionizing radiation)	Sodium dodecyl sulfate, 2%, plus acetic acid, 1%, plus autoclave at 121° C for 15-30 min
Microwave	Sodium hydroxide (NaOH), 0.09 N or 0.9 N, for 2 hr plus autoclave at 121° C for 1 hr (gravity displacement sterilizer)
UV light	Vaporized hydrogen peroxide, 1.5-2 mg/L

Note: The same process may be listed as both effective and ineffective because of differences in sterilant concentration, exposure time, temperature, etc., or differences in testing methods. All of these experiments were performed without cleaning.
Modified from Rutala WA, Weber DJ: Creutzfeldt-Jakob disease: Recommendations for disinfection and sterilization. Clin Infect Dis 32:1348, 2001; from Rutala WA, Weber DJ: Guide for disinfection and sterilization of prion-contaminated medical instruments. Infect Contr Hosp Epidemiol 31:107, 2010.

A recent experimental study that evaluated short-duration sterilization techniques in a resistometer (at 134° C, 273° F) used different models (threads, gap, empty tube, tube with insert, sliding surface, etc.) contaminated with test microorganisms to simulate situations encountered during sterilization. After 90 seconds in the threads model, no organisms could be isolated.[14] In the gap model, micoroorganisms could be isolated after 90 seconds but were inactivated after 180 seconds.[14] In the empty tube, tube with insert, and sliding-surface models, test microroganisms could still be isolated even after 5 minutes.[14] Repeating the same experiment in a test autoclave at 134° C after 90 and 180 seconds, viable microorganisms could only be detected in some of samples in the tube with insert model.[14] When the temperature was lowered to 132° C (270° F) at 2 and 4 minutes, only the 2-minute test with the tube with insert model revealed visible microorganisms.[14] These results indicate that a secure sterilization result can be guaranteed on various surfaces at 134° C with a maintenance time of 4 minutes, conditions that cover the vast majority of situations a practitioner faces daily.

Most autoclaves used in veterinary hospitals use steam pressure to drive air downward and out of the pressure vessel in a process called gravity displacement (Figure 9-4).[4] Air displacement by steam is critical to achieve condensation on all surfaces, and air reduces the temperature of steam at any given pressure.[4] Arrangement of trays or bowls within the autoclave must be such that air cannot be trapped by the downward

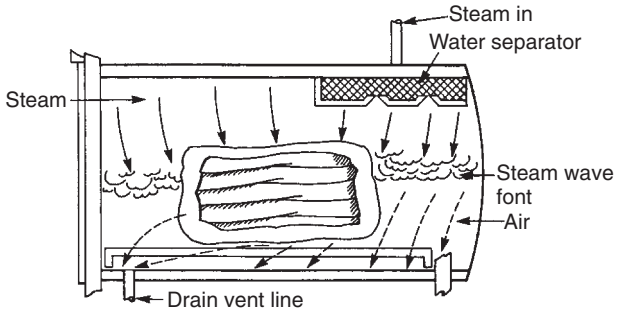

Figure 9-4. Schematic drawing of a gravity displacement autoclave, showing downward displacement of all air by steam in this system. (From Lawrence CA, Block SS: Disinfection, Sterilization, and Preservation. Lea & Febiger, Philadelphia, 1991.)

Figure 9-5. A typical sterilization unit in a large hospital, loaded and ready for use. Note that the contents are loosely arranged to facilitate access of steam around each item.

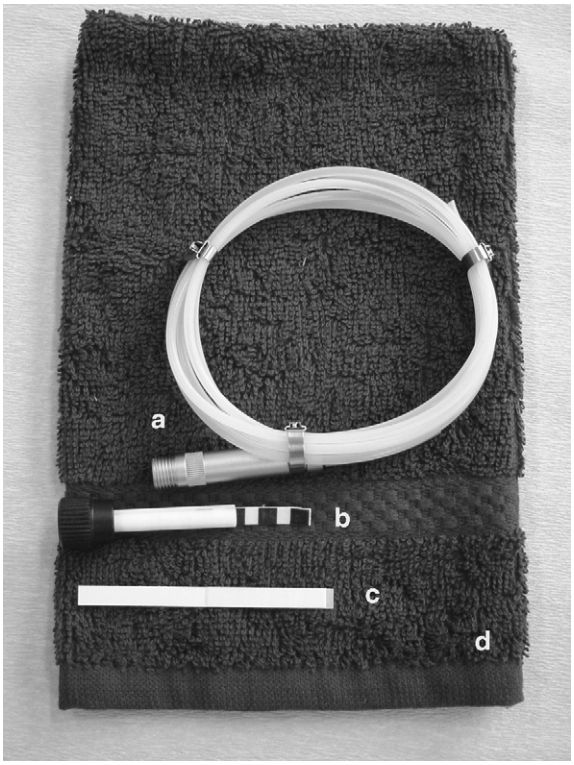

Figure 9-6. The gke Steri-Record Helix Bowie-Dick simulation (BDS) test kit. *a,* Metal test capsule attached to the polytetrafluoroethylene tube; *b,* lid of the metal capsule holding the integrating indicator (the *dark dots* indicate proper function); *c,* unused indicator strip; *d,* cloth container for the BDS kit.

progression of the steam, and bowls should be placed with their openings to the side or facing down.[8,10] Also, packs should be loosely loaded into the autoclave to ensure distribution and circulation of steam around each pack without the formation of air pockets between them (Figure 9-5).[10] Valves in cannulas should be left open to ensure adequate steam penetration.[15] Because air trapped in closed, impervious containers can inhibit steam penetration, items in glass tubes should be sealed with cotton plugs.[3-5,8] Many newer or more sophisticated types of autoclaves use a vacuum to displace air from the materials to be sterilized.[10] This allows shorter sterilization times but adds to the cost of the equipment. Other modifications use pulsed steam pressure and special valve systems to hasten air removal before sterilization. Prevacuum steam sterilizers evacuate air from the chamber before steam is admitted, so the time lag for complete air removal is eliminated and the problem of air entrapment is minimized.[4] This system is well suited for flash sterilization.[4]

It is also recommended that the steam sterilizer be periodically tested for functionality. The Bowie-Dick test can be used to prove that air removal and steam penetration were complete.[6] The Steri-Record (gke-GmbH) provides two simulation tests for different applications, depending on the sterilization programs used.[11,16] These Bowie-Dick tests simulate hollow devices, such as trocars, which require more demanding air removal and penetration conditions than porous cotton. The indicator systems consist of a process challenge device (PCD) with an indicator inside. One of the systems is the Helix-PCD, consisting of a polytetrafluoroethylene (PTFE) tube and a metal test capsule holding the integrated indicator (Figure 9-6). The second system is the Compact-PCD, consisting of an external plastic casing with a stainless steel coil inside that holds the indicator. To ensure proper functioning of the sterilizer, such a kit should be included in each sterilizer charge.

Filtration

Sterilization by filtration is used for air supply to surgery rooms (laminar flow ventilation), in industrial preparation of medications, and for small volumes of solutions in practice settings.[10,14] The laminar air filtering system for surgery suites is discussed in Chapter 10. For fluids, two types of filters are commonly used—depth filters and screen filters.[10,17] Screen filters function like a sieve to remove any microorganisms or particulate matter larger than the pore diameter of the screen.[10,17] Depth filters trap microbes and particles by a combination of random absorption and mechanical entrapment.[17]

Radiation

Sterilization by radiation is used in the industrial preparation of surgical materials that are sensitive to heat or chemical sterilization.[10] The facilities required for ionizing radiation render them unsuitable for use in veterinary hospitals.[10] Although radiation is suitable for items that cannot tolerate heat sterilization, it can change the composition of some plastics and pharmaceuticals.[10,18]

CHEMICAL STERILIZATION

Ethylene Oxide

Ethylene oxide (EO) is the most commonly used agent in chemical sterilization. Because it is a gas, it rapidly penetrates

TABLE 9-4. Requirements for Ethylene Oxide Sterilization

Variables	Range	Comments
Concentration	450-1500 mg/L	Doubling the concentration approximately halves the sterilization time.
Temperature	21°-60° C	Activity is slightly more than doubled with each 10° C increase.
Exposure time	48 min to several hours	Room temperature, 12 hr 55° C, 4 hr or less "Oversterilization" period allowed
Humidity	40%-60% (minimum, 33%)	Can be provided by vials of water or sponges

From Southwood LL, Baxter GM: Instrument sterilization, skin preparation, and wound management. Vet Clin North Am Equine Pract 12:173, 1996 (with permission).

packaging and items to be sterilized at temperatures tolerated by almost all materials. However, its use is limited by the size of the equipment, the time requirement, and concerns about toxicity. It is recommended for use only for items unsuitable for steam sterilization, including laparoscopes, light cables, and camera heads.[15,19] In fact, because of environmental concerns, EO sterilizers are now required by law to be retrofitted with abaters that reduce more of the exhausts to water vapor. Despite the use of abaters, the Environmental Protection Agency has outlawed the use of EO sterilizers altogether in some areas. Gas plasma sterilizers are a logical replacement choice (see later).

EO is an alkylating agent that kills microorganisms by inactivation of proteins, DNA, and RNA, and it is effective against vegetative bacteria, fungi, viruses, and spores.[20] It is supplied as a gas mixed with a carrier agent (Freon or CO_2) to reduce flammability.[5] Mixed with air or oxygen, EO is explosive and flammable.[5] Carbon dioxide is the preferred diluent because of environmental concerns about fluorinated hydrocarbon (Freon) release, although EO has a tendency to stratify from carbon dioxide in storage containers, which could affect sterilization.[5]

Sterilization by EO is influenced by gas concentration, temperature, humidity, and exposure time (Table 9-4).[21] The more sophisticated equipment for EO sterilization includes methods for temperature elevation to shorten sterilization times.[19] Spores require time for humidification to allow optimal killing by EO.[10,20,21] The humidity should not be raised by wetting the materials to be sterilized, because EO forms condensation products with water that may damage rubber and plastic surfaces. Also, the effectiveness of EO sterilization may be reduced below the lethal point by moisture left in needles and tubing.[22] Instruments need to be cleaned as described for steam sterilization.

Because EO penetrates materials more readily than steam, a wider variety of materials may be used in packaging items for sterilization and storage. Films of polyethylene, polypropylene, and polyvinyl chloride are commercially available, but nylon should not be used, because it is penetrated poorly by EO.[10,20-22] Positioning of packs is less critical than with steam, but overloading and compression in the sterilizer can prevent adequate penetration.[5]

After sterilization by EO, materials must be aerated to allow dissipation of the absorbed chemical (Table 9-5), because residual EO can damage tissues.[23,24] For example, inadequate aeration of endotracheal tubes sterilized by EO caused tracheal necrosis and stenosis in horses and dogs.[25,26] Although some EO chambers are equipped with mechanical aeration systems to reduce aeration times, those commonly used in veterinary hospitals use natural aeration in well-ventilated areas.[7] EO sterilization indicator strips should be used on the outside of surgery packs, and chemical or biologic indicators of EO exposure are used inside.[22] The 3M Comply EO chemical integrators demonstrate a color change and migration on an absorptive strip in response to all the critical aspects of EO sterilization, such as EO concentration, relative humidity, time, and temperature. Safe storage times are 90 to 100 days for plastic wraps sealed with tape, and 1 year for heat-sealed plastic wraps.[22]

TABLE 9-5. Average Minimal Aeration Times after Ethylene Oxide Sterilization

Material	Aeration Time*	
	Natural (days)	Mechanical (hr)
Rubber products	1-2	46
Latex	7	46
PVC ⅛ in (thick)	12	46
1/16 in (thin)	7	46
Polyethylene	2	46
Vinyl	3	32
Plastic-wrapped supplies	3	32
Implants	10-15 (recommended)	32

*Times are given for natural aeration and, where available, for mechanical aeration. Ethylene oxide sterilizers equipped for mechanical aeration produce significantly shorter aeration times (hours instead of days).
From Clem M: Sterilization and Antiseptics. p. 107. In Auer J (ed): Equine Surgery. Saunders, Philadelphia, 1992.

Exposure to EO can cause skin and mucous membrane irritation, nausea, vomiting, headache, cognitive impairment, sensory loss, reproductive failure, and increased incidence of chromosomal abnormalities.[10,23] Ability to detect the gas by smell is lost after prolonged exposure.[24] Ethylene chlorohydrin is a highly toxic degradation product of EO that is formed most readily in products that have been previously sterilized by radiation.[10,20,21] This risk is greatest with polyvinyl chloride products.[9]

Gas Plasma

Gas plasma sterilization (Sterrad Sterilization System) (Figure 9-7) allows short instrument turnaround time, has no recognized health hazards, and operates at a low temperature (less than 50° C).[9] An aqueous solution of hydrogen peroxide is injected into the chamber and converted to gas plasma by radio waves that create an electrical field.[5] In this field, hydrogen peroxide vapor is converted to free radicals that collide with and inactivate microorganisms.[8,9] Gas plasma is suitable for heat- and moisture-sensitive instruments (rigid endoscopy lenses and

Figure 9-7. Gas plasma sterilization unit (Sterrad) that uses H_2O_2 to generate free radicals, which inactivate microbes.

instrument sets, objective lenses for microscopes, nonfabric tourniquets, medication vials, insulated electrosurgery and cautery instruments, and metal instruments).[8,9] Also, the process does not dull the sharpness of delicate microsurgical instruments.[8] Gas plasma is unsuitable for flexible endoscopes, liquids, and items derived from plant fibers (paper products, linens, gauze sponges, Q-tip applicators, cast padding, wooden tongue depressors, gloves, and single-use items), because these materials absorb hydrogen peroxide and inhibit sterilization.[8] Very long narrow lumens, lumens closed at one end, folded plastic bags, and sheeting are unsuitable for sterilization by gas plasma.[9]

DISINFECTANTS

Several disinfecting agents have been developed for medical purposes and are widely used in the sterilization of inanimate objects, such as surgical instruments, endoscopes, hospital surfaces, and fixtures. They are well suited for complex surgical instruments and endoscopes that are heat sensitive. High-level disinfection refers to the use of a chemical sterilant for exposure times that are insufficient to achieve sterilization (elimination of all microorganisms *and* spores) but sufficient to inactivate all microorganisms (bacteria, fungi, viruses, and mycobacteria), *but not* all bacterial spores.[27] However, in typical use, high-level disinfection appears to provide the same efficacy as sterilization.[27] An ideal chemical sterilant as a high-level disinfectant should have broad-spectrum antimicrobial activity, rapid effect, compatibility with materials to be sterilized, and long reuse life and shelf life, nontoxic to human beings and the environment, odorless, nonstaining, free of disposal restrictions, and should be easy to use, resistant to organic material, readily monitored for concentration, and cost-effective.[27] Unlike antiseptics (Table 9-6), disinfectants are not intended for use on living tissue and actually can be harmful to tissues at the concentrations required

TABLE 9-6. Characteristics of Selected Antiseptics and Disinfectants

Agent	Trade Name	Action	Effects	Disadvantages
Isopropyl alcohol	Propanol	Protein denaturation	Bactericidal, effective against vegetative bacteria only	Poor against spores, fungi, viruses Cytotoxic in tissue
Propan-2-ol and Propan-1-ol	Sterillium	Protein denaturation	Bactericidal, fungicidal Effective against many important viruses	Cytotoxic in tissue
Ethanol 96% and Biphenyl-2-ol	Desderman pure	Protein denaturation	Bactericidal (MRSA), fungicidal Effective against many important viruses	Cytotoxic in tissues, avoid contact with the eyes, easily flammable
Glutaraldehyde	Cidex Omnicide Abcocide	Protein and nucleic acid denaturation	Bactericidal, fungicidal, viricidal, sporicidal	Long (10-hr) exposure time required for sporicidal effect Limited shelf life once activated Tissue irritant/toxicity
Chlorhexidine	Nolvasan	Cell membrane disruption and cellular protein precipitation	Bactericidal, fungicidal; variable activity against viruses	Not sporicidal
Povidone-iodine	Betadine	Metabolic interference	Bactericidal, viricidal, fungicidal	Poorly sporicidal Some inactivation by organic debris

MRSA, Methicillin-resistant *Staphylococcus aureus.*
Modified from Clem M: Sterilization and Antiseptics. p. 107. In Auer J (ed): Equine Surgery. Saunders, Philadelphia, 1992.

to achieve full efficacy. In fact, the broader the range of microbes it eliminates and the faster it acts, the more corrosive and toxic it is.[28]

Aldehydes

Because heat and moisture are damaging to certain instruments, such as endoscopes, arthroscopes, and laparoscopes, cold disinfection with glutaraldehyde, a saturated dialdehyde (Cidex, Omnicide 28, Abcocide), can be used for these items.[7] Olympus, Pentax, and Fujinon list glutaraldehyde as compatible with their endoscopes, but manufacturer recommendations need to be closely followed for all such instruments.[8] Although glutaraldehyde is effective against a wide range of susceptible organisms (see Table 9-6), Cidex is now classified as a disinfectant by the manufacturer, rather than as a sterilant, and therefore its use on arthroscopic and laparoscopic instruments is questionable.[29] Peracetic acid (PAA) would be preferable to sterilize these items, as discussed later.

Glutaraldehyde has broad-spectrum antimicrobial activity and is the most widely used chemical for the high-level disinfection of endoscopes and other such instruments.[27] Glutaraldehyde owes its biocidal activity to alkylation of sulfhydryl, hydroxyl, carboxyl, and amino groups, which alters microbial RNA, DNA, and protein synthesis.[27] The antimicrobial activity of glutaraldehyde is greatly enhanced in alkaline solutions (pH 7.5 to 8.5), although high pH hastens its polymerization and therefore limits its shelf life. To overcome this problem, glutaraldehyde is supplied as an acidic colorless solution that is activated at the time of use by adding an "activator" that converts it to a green (Cidex, Abcocide) or blue (Omnicide) alkaline solution with a sharp odor.[9,29] Acid glutaraldehydes also are available and do not require activation, but they lack the microbiocidal activity of alkaline preparations.[27]

Repeated use of an activated solution or placing damp instruments into the solution can dilute it to less than the effective concentration. Solutions should be reused only when the minimum effective concentration, as determined by the appropriate test strip, is assured, and when the pH and temperature are correct (Table 9-7). Solutions should be discarded after the specified reuse period has elapsed, even if the appropriate conditions have been met.

Antimicrobial activity of Cidex increases with increased temperature and decreases with organic matter.[29] Therefore presterilization cleaning and drying are important, and an enzyme-based presoak detergent can be used.[8] Instruments soaked in glutaraldehyde must be thoroughly rinsed with sterile water before they touch tissue, and gloves must always be worn when removing items from glutaraldehyde baths.

The potential hazards of glutaraldehyde for staff are considerable. Toxicity has been suspected in 35% of endoscopy units, with harmful or potentially harmful problems in 63% of these.[9] Direct contact with glutaraldehyde is irritating to skin and other tissues, and repeated exposure can result in sensitization and allergic contact dermatitis, rhinitis, and asthma.[25] Vapor may cause stinging sensations in the eye, excess tear production, redness of the conjunctiva, a stinging sensation in the nose and throat, nasal discharge, coughing, symptoms of bronchitis, and headache.[9] Glutaraldehyde is not ideal for chemical disinfection of instruments that are hinged, are corroded, or have deep or narrow crevices, and it should not be used for critical, single-use devices, such as catheters. Prolonged use of glutaraldehyde can corrode metals and some plastics.[30,31] As with all aldehydes, glutaraldehyde can fix proteins by denaturing and coagulating them, and this creates a biofilm on instruments that can make them difficult to sterilize.[9]

TABLE 9-7. Recommended Conditions for Use of Three Glutaraldehyde Preparations

	Cidex (Activated)	Cidex 7 (Long-Life Activated)	Cidexplus (28-Day Solution)
Concentration (%)	2.4	2.5	3.4
Maximal reuse period	14 day	28 day	28 day
AS A STERILANT			
Temperature (° C)	25	20-25	20-25
Minimal immersion time	10 hr	10 hr	10 hr
AS A HIGH-LEVEL DISINFECTANT			
Temperature (° C)	25	25	25
Minimal immersion time	45 min	90 min	20 min
AS AN INTERMEDIATE-LEVEL DISINFECTANT			
Temperature (° C)	20-25	20-25	20-25
Minimal immersion time	10 min	10 min	10 min

Sterilant conditions apply to surgical instruments and devices that penetrate skin or are used in sterile tissues; the longer times are required for spores. High-level disinfectants are used for semicritical devices that do not penetrate sterile tissues (endoscopes, anesthesia equipment). Intermediate-level disinfectants are used for noncritical devices that contact skin surface only. Recommendations for other glutaraldehyde preparations may vary—the manufacturer's advice should be followed.

From Southwood LL, Baxter GM: Instrument sterilization, skin preparation, and wound management. Vet Clin North Am Equine Pract 12:173, 1996 (with permission).

Ortho-phthalaldehyde (OPA; Cidex OPA) is a high-level disinfectant that contains 0.55% 1,2-benzenedicarboxaldehyde. OPA completely destroys all common bacteria in 5 minutes of exposure, does not produce noxious fumes, does not require activation, is compatible with many materials, does not coagulate blood or fix tissues to instrument surfaces, and is stable at a wide pH range (3 to 9).[9,27] Exposure to OPA vapors may be irritating to the respiratory tract and eyes, and it can stain linens, clothing, skin, instruments, and automatic cleaning devices.[9]

Succindialdehyde with dimethoxytetrahydrofuran and anticorrosion components (Gigasept FF) is recommended for flexible endoscopes and ultrasonic probes by well-known manufacturers (e.g., Fujinon, Olympus, Hewlett Packard, Acuson, Toshiba). It is a broad-spectrum cold-sterilizing or disinfecting solution with excellent material compatibility and a pH of approximately 6.5. It does not require activation additives and might be preferable when there is a desire to avoid formaldehyde or glutaraldehyde products.

Peracetic Acid

Peracetic acid or peroxyacetic acid (PAA) is an oxidizing agent that functions in much the same way as hydrogen peroxide, through denaturation of protein, disruption of cell wall permeability, and oxidation of sulfhydryl and sulfur bonds in

proteins, enzymes, and other metabolites.[24] PAA is available under numerous brand names with different chemical formulations (Nu Cidex 0.35%, STERIS 0.20%, Anioxyde 1000, and Sekusept Aktiv). The STERIS Corporation has marketed STERIS 20 Sterilant Concentrate, a 35% peroxyacetic acid concentrate, for use in the STERIS System 18 (Figure 9-8). An arthroscopic camera and telescope can be processed, rinsed, and dried in this system in a 20-minute cycle. It is routinely used to sterilize flexible endoscopes as well. A contact time of 10 or 15 minutes and a concentration of greater than 0.09% PAA are recommended for destruction of bacteria, fungi, viruses, and spores, if used manually.[9] Compared with glutaraldehyde, PAA has a similar or even a better biocidal efficacy and is claimed to be less irritating for staff and safer for the environment. PAA does not fix proteins and therefore does not create a biofilm. It has the ability to remove glutaraldehyde-hardened material from biopsy channels, and its activity is not adversely affected by organic matter.

Potential adverse effects are strongly linked to the pH value of the application solution, with minimal effects in a pH range of 7.5 or higher. PAA is less stable than glutaraldehyde, can be corrosive, and has a strong, vinegar-like odor. PAA has additional drawbacks when used on immersible instruments; it can cause serious eye and skin damage in a concentrated form, it can dull aluminum anodized coating, instruments treated with it cannot be stored, and it is expensive.[9,27] PAA is also a weak carcinogen.[28] Therefore, when using manual immersion methods, PAA should be used with adequate ventilation and personal protective measures. PAA also causes cosmetic discoloration of endoscopes, but without any functional damage, if used manually; the STERIS System 1 sterilizer does not have this problem, however, because adequate rinsing is automatic.

Hydrogen Peroxide

Hydrogen peroxide is an oxidizing agent that can be used as a high-level disinfectant.[27] It produces destructive hydroxyl radicals that attack membrane lipids, DNA, and other cellular components when used at recommended concentrations.[27] Its antimicrobial activity is very slow.[27,28] It is marketed as Sporox as a premix that contains 7.5% hydrogen peroxide and 0.85% phosphoric acid.[27] The minimum effective concentration (i.e., 6.0%) must be checked regularly. It is compatible with many tested endoscopes, but black anodized metal finishes can become discolored.[27] Hydrogen peroxide can be corrosive to flexible endoscopes.[28] It can enhance removal of organic matter, is easily disposed of, and is neither malodorous nor irritating.[27]

A solution of 0.5% hydrogen peroxide (Hydrox) has been shown to combine microbial killing with a cleaning efficiency on medical devices that is superior to that of many detergent solutions.[7] A new high-level disinfectant has been developed as an accelerated hydrogen peroxide (AHP) product obtained by blending with commonly used safe ingredients that dramatically increase the germicidal potency of hydrogen peroxide.[28] This AHP product, Accel HLD 5, is a blend of 2% hydrogen peroxide, anionic surfactants, nonionic surfactants, and stabilizers, that is odorless and has a pH of 2.5 to 3.0. In studies, it proved to be a broad-spectrum and fast-acting microbicide that is effective in the presence of soilage and safe to end users and the environment. It is considered to be compatible with flexible endoscopes.[28]

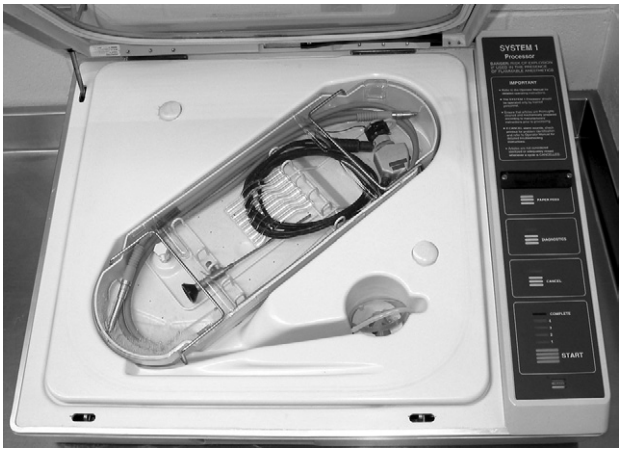

Figure 9-8. Peracetic acid sterilizer (Steris System 1), which is used to sterilize endoscopes, arthroscopes, and other equipment.

Electrolyzed Acid Water

At present, two types of electrolyzed acid water (EAW) are available—electrolyzed strong acid water with a pH of less than 3 (e.g., Cleantop WM-S) and electrolyzed weak acid water, with a pH of between 6 and 7.[9] EAW is produced by using water and salt under electrolysis with membrane separation. The process generates hydroxy radicals that have a rapid and potent bactericidal effect. Additionally, the low pH (pH 2.7) and high oxidation-reduction potential (1100 mV) are toxic to microorganisms.[9] EAW breaks the bacterial cell wall and degenerates various inner components of the bacterium (including chromosomal DNA). EAW is nonirritating, has minimal toxicity, and is safe and inexpensive, but the bactericidal effect is drastically decreased in the presence of organic matter or biofilm. Also, EAW is unstable, and the full disinfecting potential of EAW and its long-term compatibility for endoscopes remain to be examined.[9]

Sterilox, often referred to as superoxidized water, is a dilute mixture of mild oxidants at neutral pH derived from salt by electrolysis in a proprietary electrochemical cell.[9] The primary active species is hypochlorous acid, an extremely powerful disinfectant that is completely nontoxic in the low, clinically effective small concentrations produced in Sterilox. It is generated on site, as needed, and stored no longer than 24 hours. The active agents decompose slowly to harmless species.[9]

Chlorine Dioxide

Chlorine dioxide (e.g., Tristel, Dexit, and Medicide) is a powerful oxidizing agent and is active against nonsporing bacteria, including mycobacteria and viruses, in less than 5 minutes, and is rapidly sporicidal (10 minutes).[9] Chlorine dioxide is more damaging to instruments and components than glutaraldehyde[9]; it discolors the black plastic casing of flexible endoscopes and irritates the skin, eyes, and respiratory tract.[9] Chlorine dioxide emits a strong odor of chlorine and should be stored in sealed containers and handled in well-ventilated areas.[9]

Miscellaneous

The monomer of 2-butanone peroxide is a novel peroxygen derivative that has exhibited biocidal activity against several

bacteria and has good fungicidal and virucidal activities.[32] Results of toxicity assessment and material compatibility studies were favorable.[32] Peracetic acid (0.08%) plus 1.0% hydrogen peroxide, marketed as Cidex PA, can inactivate all microorganisms, but not bacterial spores, within 20 minutes.[27] It is unfortunately not endorsed by Olympus for any Olympus endoscopes. The product has required reformulation to improve its material compatibility.[27]

Peroxygenic acid (Virkon) owes its oxidizing activity to its three major components, potassium monoperoxysulfate (primary ingrediant), potassium hydrogen sulfate, and potassium sulfate. At a concentration of 1%, it is a low-level disinfectant, rapidly biocidal against vegetative bacteria and viruses, with some activity against yeasts and nontuberculous mycobacteria in suspension tests.[33] It has a limited spectrum of activity, because it cannot destroy endospores and molds within a practical time frame, and it is potentially corrosive. Although it is unsuitable as a disinfectant for medical devices, its biodegradability and low toxicity would make it a good environmental disinfectant. In veterinary hospitals, this disinfectant has received favorable reviews for use in footbaths and foot mats as a means of reducing bacterial contamination on the soles of boots and thereby potentially reducing the risk for spread of nosocomial infections.[34,35]

ANTISEPTICS

Antiseptics are intended for use on living tissue, whereas disinfectants are intended for use on inanimate objects and can harm tissue (see Table 9-6).[9] An agent can be an antiseptic at low concentrations and a disinfectant at higher concentrations.[9]

Alcohols

Alcohols are commonly used in veterinary medicine, but they are effective only against vegetative bacteria (see Table 9-6).[36] They have a mild defatting effect but they are inactivated by a variety of organic debris and have no residual activity after evaporation.[5,36] Alcohols have a higher and more rapid kill rate than chlorhexidine, and third best is povidone-iodine.[37] The bactericidal efficacy of 1-propanol can be regarded as superior to that of 2-propanol, and third best is ethanol.[38]

Either alcohol or sterile saline can be used to rinse the surgical scrub solution from the surgery site. Alcohol does not inactivate chlorhexidine gluconate *in vitro* and has no significant effect on its protein-binding property *in vivo*.[37] However, isopropyl alcohol rinse can inactivate hexachlorophene-based preparations (e.g., pHisoHex).[10,39,40] Alcohol is a commonly used rinse in veterinary hospitals[39] and is preferred over sterile saline when used for soaking sterile sponges in "community" jars, because it is more likely to maintain sterility of jar contents over the long term.[40] Isopropyl alcohol potentiates the antimicrobial efficacy of povidone-iodine by increasing the release of free iodine, so it should be used as a rinse after this surgical scrub.[41,42]

Chlorhexidine

Chlorhexidine diacetate (2%) and chlorhexidine gluconate (4%) have a rapid onset of action and a persistent effect[43] but variable and inconsistent activity against viruses and fungi (see Table 9-6).[8] Chlorhexidine binds to protein of the stratum corneum, forming a persistent residue that can kill bacteria emerging from sebaceous glands, sweat glands, and hair follicles during surgery.[44] Another approved antiseptic for preoperative skin preparation, 2% chlorhexidine gluconate plus 70% isopropyl alcohol (ChloraPrep), provided significantly more persistent antimicrobial activity on abdominal sites at 24 hours after application than either of the components used separately.[45]

Chlorhexidine has low toxicity as a skin scrub or as an aqueous solution for wound disinfection, oral lavage, and mucous membranes of the urinary tract.[43] Although it can be toxic to fibroblasts *in vitro*, *in vivo* lavage with dilute chlorhexidine (0.05%) is not harmful to wound healing.[42] However, the least known bactericidal concentration (0.05%) of chlorhexidine diacetate causes synovial ulceration, inflammation, and fibrin accumulation in the tarsocrural joints of horses.[46] Chlorhexidine (0.0005%) potentiated with 3.2 mM EDTA and 0.05 mM Tris buffer (hydroxymethylaminomethylamine) is 90% lethal to *Escherichia coli*, *Staphylococcus aureus*, and *Streptococcus zooepidemicus* and is not harmful to the synovium or articular cartilage of the tarsocrural joints of ponies.[47] Chlorhexidine (0.02%), like 1% povidone-iodine, promotes intra-abdominal adhesion formation and therefore should not be used for peritoneal lavage.[48]

Iodine Compounds

Inorganic or elemental iodine has a very broad antimicrobial spectrum compared with other agents (see Table 9-6) and a very short kill time at low concentrations, and organisms do not develop resistance to it.[49] Its undesirable characteristics are odor, tissue irritation, staining, radiopacity, and corrosiveness.[49] Iodophors are complexes of elemental iodine with a carrier, such as polyvinylpyrrolidone (PVP), which forms povidone-iodine (PVP-I_2; Betadine surgical scrub). The complex retains the bactericidal activity of iodine, while reducing tissue irritation and staining. Povidone-iodine is usually supplied as a 10% solution with approximately 1% available iodine, which is not equivalent to free iodine but must be converted to free iodine to become bactericidal.[49] However, iodine is so tightly bound to PVP that the standard 10% solution contains as little as 0.8 parts per million of free iodine.[46] This concentration may not be sufficient to kill bacteria, especially as some free iodine is readily neutralized by protein and by conversion to iodide *in vivo*.[49] However, dilution of the 10% solution of povidone-iodine liberates more free iodine than is present in the undiluted solution—thus the diluted solution is more bactericidal.[49] Contamination of 10% povidone-iodine solution by bacteria has been reported, apparently because it liberates an insufficient amount of free iodine at this concentration.[49] At least 2 minutes of scrubbing is required to release free iodine from povidone-iodine.[37] Addition of detergents, as in surgical scrubs, further reduces the release of iodine.[50] Inadequate release of free iodine from povidone-iodine causes some concern about its efficacy in skin preparation.[49]

Before application of iodophor compounds, hair should be removed and the skin well cleaned to remove organic debris that can reduce the bactericidal activity of the iodophor. However, when arthrocentesis sites in the midcarpal joint and the distal interphalangeal joint region of horses were not clipped of hair, a 5-minute surgical scrub with povidone-iodine followed by a rinse with 70% alcohol was as effective as the same

regimen on corresponding clipped sites.[51] Although a scrub with povidone-iodine, followed by a 24-hour soak in povidone-iodine solution, could reduce bacterial numbers on the surface of the equine hoof, especially if the superficial layer of the hoof capsule was removed, bacterial populations capable of inducing wound infection still remained.[52]

The toxicity of iodine-releasing compounds is low, although individual sensitivities can occur and some horses may develop skin wheals about the head and neck (e.g., at the laryngoplasty site). Undiluted povidone-iodine solutions have no effect on numbers of viable bacteria in wounds, and povidone-iodine surgical scrub can potentiate infection and inflammation.[47] The practice of lavaging the peritoneal cavity with povidone-iodine has been abandoned because of evidence that even dilute solutions can cause a sterile peritonitis in ponies and induce metabolic acidosis.[49,53] Although 0.1% povidone-iodine has been reported to be bactericidal and to have minimal deleterious effects on the equine tarsocrural joint, it was ineffective in the treatment of experimental infectious arthritis in horses.[54,55] Concentrations greater than 0.05% *in vitro* can disrupt neutrophil viability and migration.[56]

A one-step surgical preparation technique using DuraPrep Surgical Solution is as effective as a two-step povidone-iodine preparation.[41] The antimicrobial properties of the solution are the result of 70% isopropyl alcohol in an iodophor-polymer complex that forms a water-insoluble film with sustained chemical and physical barrier properties on skin.[41] In a study on skin preparation for ventral midline incisions in horses undergoing celiotomy, DuraPrep was as effective as povidone-iodine and alcohol in reducing colony-forming units (CFUs) up to the time of skin closure, and both methods had comparable rates of incisional drainage.[57] However, preparation time was significantly shorter for DuraPrep than with the routine skin preparation technique.[57]

Antimicrobial film drapes with adhesive backing (Ioban 2) contain an iodophor and come in different sizes that make them suitable for equine surgery. After the skin has been prepared with an accepted surgical scrub, it is rinsed with isopropyl alcohol and may need to be dried with a sterile towel to improve adherence.[57] In some clinics, the proposed surgery site is shaved with a size 40 blade to improve adherence beyond that achieved by clipping.[57] A medical grade adhesive spray can also be used (Medical Adhesive, EZ Drape Adhesive), but this is not essential. Adherence to smooth flat surfaces, such as the ventral abdomen, may be better than to the irregular contour of a joint. A tight adherence of the drape in areas of complicated contours can be achieved by applying the adhesive drape circumferentially while pressing the excess edges of the drape tightly together behind the limb on the side opposite the surgical site. Care should be taken that small pieces of the drape not be torn off and dragged into joints by arthroscopic instruments, to end up as free-floating objects in the joint cavity. Although iodophor skin preparations do not produce a radiopaque artifact on intraoperative radiographs, folds in iodophor-impregnated plastic drapes can produce confusing radiographic images.

The value of antimicrobial adhesive drapes is questionable.[37] In a study on human patients undergoing hip surgery, bacterial contamination of the wound at the end of surgery was reduced from 15% with conventional preparation to 1.6% by use of an iodophor-impregnated plastic adhesive drape (Ioban).[58] In a prospective randomized clinical trial on 1102 patients, isolates of normal skin organisms were less frequent when an iodophor-impregnated plastic incise drape was used in clean and clean contaminated abdominal procedures than when the drape was not used.[59] However, no difference was found between wound infection rates for patients on whom the iodophor drape was used compared with those patients on whom it was not used.[59,60] In a study on dogs that underwent elective ovariohysterectomy or stifle surgery, adhesive incise drapes did not reduce wound contamination based on number of CFUs counted for positive cultures from the surgical incisions.[61] Although it is logical that skin flora reduction might translate into reduced surgical site infections, that relationship has not been established in this or other studies.[62]

Chlorhexidine versus Povidone-Iodine

In tests with *E. coli* and *S. aureus* on canine skin, 2% chlorhexidine diacetate was superior to hexachlorophene and povidone-iodine in rapid removal of bacteria and in residual activity.[63] In another study, chlorhexidine and povidone-iodine were effective in reducing bacteria from the surgeons' hands, but the apparently greater residual effect of chlorhexidine (120 minutes) was not statistically significant.[64] Such a residual effect could be of value during long surgical procedures, in which rates of glove puncture could be as high as 17% and many perforations are unnoticed by the surgeon.[65] In one study, 4% chlorhexidine gluconate was found to be superior, on the basis of efficacy and prolonged effects, to 7.5% povidone-iodine throughout a 3-hour period after hand antisepsis.[66] Compared with iodine preparations, chlorhexidine preparations are less susceptible to inactivation by organic debris.[43]

Although chlorhexidine's wider range of antimicrobial activity, longer residual action, minimal inhibition by organic material, and greater tolerance by skin would render it superior to povidone-iodine, both agents perform comparably in the surgical setting.[37] In a prospective randomized study of 886 human patients, there were significantly fewer wound infections with chlorhexidine preparations for surgical hand washing and patient skin preparation than with povidone-iodine (hand washing and skin preparation) in operations on the biliary tract and in "clean" nonabdominal operations; however, there were no significant differences in a number of other types of surgery.[67] The authors concluded that "on the evidence of this study, there is no overwhelming case for using one compound rather than the other as an all-purpose preparation and scrub."[67]

The preceding results were confirmed in a study on cattle, which showed that povidone-iodine and 4% chlorhexidine gluconate scrubs rinsed with 70% isopropyl alcohol decreased skin microflora and had similar frequencies of surgical wound infection.[55] CFUs were lower with chlorhexidine and alcohol immediately after scrubbing, but there was no difference in residual effect between the two scrubs.[68] In an experimental comparison in dogs between povidone-iodine and 70% isopropyl alcohol rinse, 4% chlorhexidine gluconate with 70% isopropyl alcohol rinse, and 4% chlorhexidine gluconate with saline rinse, there were no significant differences in percentages of bacterial reduction immediately and at 1 hour after scrub, and the percentages of negative cultures and cultures with more than 5 CFUs.[39] However, when the study was repeated in a clinical trial on 100 dogs undergoing a variety of procedures, 4% chlorhexidine gluconate with 70% isopropyl alcohol rinse was not superior to povidone-iodine and was actually inferior in residual microbial activity.[40] However, this was based only on percentage of

negative cultures, and the overall postoperative infection rate was too low to allow a meaningful statistical comparison.[40]

In a recent study on human adults undergoing clean contaminated surgery in six hospitals, enrolled patients were randomly assigned to a skin preparation at the surgical site with a preoperative scrub with an applicator that contained 2% chlorhexidine gluconate and 70% isopropyl alcohol (ChloraPrep), or preoperative scrub and then the site painted with an aqueous solution of 10% povidone-iodine (Scrub Care Skin Prep Tray).[69] Chlorhexidine-alcohol was significantly more protective than povidone-iodine against both superficial incisional infections and deep incisional infections.[69] The benefit for the chlorhexidine-alcohol scrub was a 41% overall reduction in infection rates and elimination of 50% of *S. aureus* infections.[69] This is consistent with findings of other studies,[70] including one that demonstrated an approximately 50% reduction in catheter-associated infections after a chlorhexidine-alcohol solution compared with povidone-iodine.[71] However, in a single hospital study on general surgery patients, the lowest surgical site infection rate was obtained with iodine povacrylex in isopropyl alcohol (DuraPrep) compared with 2% chlorhexidine and 70% isopropyl alcohol (ChloraPrep) and with povidone-iodine scrub paint.[62] Both iodine preparations were superior to chlorhexidine in that study, which unfortunately suffered from some limitations in experimental design.[62] Therefore these conclusions must be considered in that context.

Based on the available evidence, chlorhexidine would appear preferable to povidone-iodine for preparation of surgery sites and the surgeon's hands. Povidone-iodine solutions are inferior to chlorhexidine for wound lavage.[72] Also, an undesirable side effect with povidone-iodine is a greater risk of skin reactions than with chlorhexidine preparations, as demonstrated in dogs[39] and observed in horses. However, chlorhexidine is more expensive than povidone-iodine.[8]

Hydroalcoholic Solution versus Chlorhexidine and Povidone-Iodine

A new study surveying 951 ACVS and 349 ECVS diplomates, with a return rate of 42.6%, revealed that 81.4% of the surgeons used chlorhexidine, 12.2% povidone-iodine, and 6.7% hydroalcoholic solution (Sterillium) for presurgical hand desinfection.[73] The same study reporting preliminary data revealed significant differences between the three products tested in immediate and sustained activities. The hydroalcoholic solution showed a significant reduction of CFUs after presurgical hand antisepsis compared to povidone-iodine and a significant reduction after 3 hours of gloving compared to the other two products. As a matter of fact, the hydroalcoholoic solution led to an additional reduction of CFUs during the 3-hour gloving period. This study shows that a solution consisting of 45% 2-propanol, 30% 1-propanol, and 0.2% mecetronium ethylsulfate is more effective in reducing bacterial counts on hands before surgery in a veterinary setting than are chlorhexidine and povidone-iodine soap. Nevertheless it is only a small, mainly European group that uses this effective hand antiseptic.

Sterillium is currently not available in the United States. However an ethanol-based product (Sterillium is propanol-based), Avagard (61% ethanol and 1% chlorhexidine gluconate), is currently only available in the United States. Very few trials have been performed with this product, although one study comparing the infection rates in pediatric urologic procedures found no differences between using Avagard and scrubbing with an antiseptic-impregnated hand brush.[74] In another study, the antimicrobial efficacy of the product was shown to be superior to 4% chlorhexidine scrub and 61% ethanol alone, both immediately after use and after 6 hours.[75] A crossover trial conducted by the prEN 12791, however, could not demonstrate the effectiveness of Avagard as a suitable surgical hand disinfection method because the product did not meet the requirements for either immediate or sustained effect in comparison to the reference alcohol.[76]

Octenidine

Octenidine dihydrochloride is a cationic antiseptic that belongs to the bispyridine class of chemicals. It has activity against Gram-positive and Gram-negative bacteria.[77] It was effective in oral hygiene, preventing plaque and gingivitis, as a whole body wash for methicillin-resistant *S. aureus* decolonization[78] and for skin disinfection of premature newborn infants.[79] Octenidine concentrations of less than 1.5 μM (0.94 μg/mL) reduced each microbial population by more than 99% within 15 minutes. *Staphylococcus epidermidis* was the most susceptible of the test organisms, and *E. coli* and *Candida albicans* were the least susceptible. Octenidine was more active than chlorhexidine against each test strain.

This antiseptic has not been established in veterinary medicine for skin preparation, but it is used for wound cleansing.

Phenols

Phenol, cresol, and other coal tar derivates, such as hexachlorophene (pHisoHex; see Table 9-6), are generally considered to be inferior to chlorhexidine and povidone-iodine.[10,37]

Hexachlorophene has a relatively slow onset of action but a prolonged residual activity, and it is not adversely affected by organic materials. Hexachlorophene-based preparations are inactivated by alcohol.[10,37] Use was largely curtailed after hexachlorophene was shown to be neurotoxic at levels obtained with dermal exposure.[80]

Quaternary Ammonium Compounds

Quaternary ammonium compounds, such as benzalkonium chloride, are cationic surfactants that dissolve lipids in bacterial cell walls and membranes.[81] Drawbacks to the group are ineffectiveness against viruses, spores, and fungi; formation of residue layers; and inactivation by common organic debris and soaps.[9]

Miscellaneous

Hydrogen peroxide is used to clean severely contaminated wounds, but it is a poor antiseptic and is mainly effective against spores, and concentrations lower than 3% are damaging to tissues.[82] Chloroxylenol, or parachlorometaxylenol, a synthetic halogen-substituted phenol derivative, and triclosan, a diphenyl ether, do not appear to offer any advantages over the more commonly used antiseptics in veterinary medicine.[37,65,83]

Current trends in surgical hand disinfection have evolved very rapidly in the last several years and now include alcohol-based and quaternary ammonium compounds using brushless

techniques. A complete discussion on these newer products and techniques can be found in Chapter 10, under "Surgeon's Skin."

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CHAPTER
10

Preparation of the Surgical Patient, the Surgery Facility, and the Operating Team

John A. Stick

Having the capacity for sound clinical judgment is the ultimate characteristic of the mature veterinary surgeon. To attain this capacity, the surgeon needs to be able to provide an accurate assessment of operative risk. This can be done only if there is thorough preparation of the surgical patient combined with knowledge of the primary problem, experience, and an open mind.

ASSESSMENT OF OPERATIVE RISK

When determining operative risk, each surgeon must consider the relative rewards and risks in treating a specific illness.[1] The surgical risks encompass not only the odds of surviving surgery but also the long-term prognosis, the potential for the develpoment of complications, and the patient's future use and quality of life.[1,2] Basic factors affecting operative risk include age, overall physical status, elective versus emergency operation, physiologic extent of the procedure, number of associated illnesses, and projected surgery time. Although the surgeon can informally consider all this information and make a guess as to the surgical risks based on experience in similar cases, formal assessment schemes are useful and their adoption is encouraged. Two main components comprise a logical formal determination of surgical risk: the primary disorder and the general health of the patient.

Primary Disease

Primary diseases with a tendency to progress rapidly and involve other body systems are associated with more risks than those that progress slowly and do not affect the patient's systemic health. The procedure's invasiveness and potential for complications are also considered in risk assessment.

Complications and the risk of death increase with the duration of surgery. The risk of surgery also varies with the system

involved. For example, diseases involving the gastrointestinal tract have a tendency to cause shock and sepsis early in their course. Elective orthopedic surgery has a much lower associated risk than nonelective general surgeries and major trauma. When emergency surgery is necessary, the surgical risk increases. When a disorder is fatal without surgery but has the potential for a surgical cure, surgery is likely to be recommended despite a high surgical risk.

General Health Assessment

Surgery and anesthesia are never without risks, and unexpected complications can occur even in the healthiest patient undergoing a minor procedure. However, the risks are increased by a variety of conditions. Risk is increased in the very young and the very old patient. Neonatal animals are predisposed to hypothermia, hypoglycemia, and infection. Morbidity and mortality increase with age in human and veterinary surgical patients.[2]

The effects of concurrent disease on an animal's general health are important determinants of surgical risk. Animals with normal physical findings and no history of cardiovascular, respiratory, renal, or liver disorders have a relatively low surgical risk. Additionally, the preoperative nutritional status is an important determinant of surgical risk (see Chapter 6). Cachectic animals may experience delayed wound healing and a higher incidence of postsurgical wound infection and susceptibility to multiple organ disorders.

The importance of establishing the physical status cannot be overstated. The American Society of Anesthesiologists (ASA) has created a classification system for human patients based on evaluation of their physical status, and the rankings can be used to determine surgical risk (Table 10-1). In humans, physical status was second only to albumin level in its accuracy in predicting survival and postoperative complications.[3,4] Similar findings were observed in high-risk canine surgical patients: 92% of canine patients assigned to ASA class II survived, compared with 73% in class III and 38% in class IV.[5] In fact, in a recent study on surgical site infections in 97 human hospitals, admission illness severity was significantly associated with higher mortality and increased length of stay and cost.[6]

Personal Relationships

A bond of communication, cemented with personal responsibility, is established between the surgeon and the client (usually the animal's owner), whenever a surgical procedure is being considered. The confidence of the well-informed client is based on a true understanding of the situation, which allows the client to participate in decisions regarding operative risks, outcomes, the process of postoperative recovery, and financial implications. Legal action is rare when a careful effort has been made by the veterinarian to achieve such understanding before a surgical intervention.

Veterinary surgeons should also appreciate the importance of an effective relationship with the referring veterinarian. In many situations, patients are referred to surgeons by veterinarians with valuable skills and expertise. It is important to understand the wishes and views of the referring veterinarian.

TABLE 10-1. American Society of Anesthesiologists Classification System for Physical Status and Recommended Tests for Each Class

Physical Status	Definition	Example	Recommended Laboratory Tests		
			Minor*	Major†	Prognosis
I	Healthy with no organic disease	Elective procedures not necessary for health (ovariohysterectomy)	PCV, TP, urine specific gravity	CBC, U/A, surgical panel‡	Excellent
II	Local disease with no systemic signs	Healthy nonelective surgery (skin laceration, simple fracture)	PCV, TP, urine specific gravity	CBC, U/A, surgical panel‡	Good
III	Disease causes moderate systemic signs that limit function	Heart murmur, anemia, pneumonia, mild chest trauma, moderate dehydration	CBC, U/A, surgical panel‡	CBC, U/A, biochemical panel§	Fair
IV	Disease causes severe systemic signs and threatens life	Gastric torsion, diaphragmatic hernia, severe chest trauma, severe anemia, or dehydration	CBC, U/A, biochemical panel§	CBC, U/A, biochemical panel§	Guarded
V	Moribund, not expected to live for more than 24 hours with or without surgery	Endotoxic shock, severe trauma, multiorgan failure	CBC, U/A, biochemical panel§	CBC, U/A, biochemical panel§	Grave
E	Emergency	Qualifier of previous classes	PCV, TP, urine specific gravity	Depends on facilities available	Variable

CBC, Complete blood cell count; *PCV,* packed cell volume; *TP,* total protein; *U/A,* urinalysis.

*Duration less than 60 minutes.

†Duration longer than 60 minutes or patients older than 7 years.

‡Surgical panel: urea, creatinine, alkaline phosphatase, alanine aminotransferase, glucose, sodium, potassium, chloride, and total protein levels.

§Biochemical panel: the full panel is the surgical panel tests plus bicarbonate, anion gap, calcium, phosphorus, cholesterol, total bilirubin, γ-glutamyltransferase, and albumin levels.

Differences in judgment must be discussed. Both the surgeon and the referring veterinarian should be aware of the expected course of treatment and the extent of the referring veterinarian's participation in postoperative care. This prevents communication errors and contradictory efforts.

True informed consent is attained when there is a full and frank discussion with the client in the presence of an appropriate professional witness.[1] The surgeon should record a summary of this encounter in the hospital chart. The surgeon should also record why the operation is needed, the operative risks, and the problems anticipated intraoperatively and postoperatively. When a condition is expected to have a clinically significant course beyond the duration of the early follow-up period, the client should be told how much continuing commitment will be needed.

PREPARATION OF THE SURGICAL PATIENT

History

The first step in the assessment of a patient is interviewing the owner to determine the animal's medical history, its overall health, and the impact of the presenting complaint.[2] At this time, the surgeon should determine the owner's wishes and expectations.

The patient's signalment should be reviewed to determine the potential for problems related to age, breed, and sex. Questions about the animal's general health and environment can contribute to making the diagnosis. The animal's intended use and the owner's expectations of its future performance are explored to gauge the future satisfaction of the owner with the proposed procedure. Past medical problems should be discussed, because they may influence the outcome.

Physical Examination

Despite a surgeon's natural tendency to focus on the presenting problem, a thorough physical examination should include an assessment of each system. A general physical examination determines the need for in-depth assessment and preoperative stabilization. It is this examination that is most likely to identify risk factors affecting surgical outcome.[2] The animal should first be examined for general demeanor, nutritional status, and gait. Temperature, pulse, and respiration rate are noted because the respiratory and cardiovascular systems are emphasized.

Finally, the affected area and related systems are evaluated. A physical status ranking, based on the American Society of Anesthesiologists classification system (see Table 10-1), should be assigned. This will allow a more accurate determination of what supplemental testing should be performed.

Supplemental Testing

Laboratory testing is not a substitute for the thorough examination, and all abnormal findings in the laboratory data should be interpreted in light of the initial physical findings. When abnormalities in the function of organs (e.g., the heart, kidneys, and respiratory system) are detected, testing may be expanded to include chest radiography, urinalysis, and biochemical profile. However, although preoperative tests that screen for clinically silent disease will not replace the physical examination, some basic laboratory data are recommended for use with the American Society of Anesthesiologists classification system for physical status (see Table 10-1).

Physiologic Preparation

In preparation for elective surgery, steps should be taken to correct physiologic deprivations. Surgical procedures in chronically anemic patients should be delayed until the anemia can be corrected. If fluid deficits exist, plasma or fluids should be administered in appropriate volume, concentration, and composition (see Chapter 3). Although not all volume and concentration deficits need to be corrected before the surgery, a significant fraction of the total deficit should be replaced to enhance the safety of the anesthesia, even in emergency patients. Nutritional replenishment supplementation should be provided for a patient that awaits an elective operation if deficits are obvious.

Infection is a major source of morbidity and a disconcerting source of mortality in some surgical patients. Badly injured or traumatized horses, and those that undergo an operation and survive despite the development of secondary shock and electrolyte disturbances, are at a very high risk for serious infection.

Infection rates of surgical wounds in horses are higher than those seen in people and dogs. Overall infection rates for equine orthopedic surgeries have been reported to be 10%, compared with 4.7% in people, and 5.1% in dogs and cats.[7-10] Infection rates for abdominal surgery in horses have been reported to be 25.4% and 30%.[8,9] Therefore a primary consideration in preparing the patient is antibiotic prophylaxis. Because equine patients do not live in particularly clean environments, antimicrobial drugs are frequently administered prophylactically even for elective orthopedic surgeries. For additional information on surgical infections and management of sepsis, see Chapters 7, 9, and 85.

Skin Preparation

The patient is the primary source of pathogens involved in surgical wound infections and therefore should be groomed before surgery, or even bathed if the hair coat contains a lot of organic material, and the tail should be wrapped.[10] Preparation of the surgical site should include hair removal and cleansing to remove dirt and oil and to reduce resident skin flora. The suggested procedure is to clip the entire surgical area using a No. 40 clipper blade, then scrub and apply an antiseptic solution. Additionally, wrapping the limb with a sterile bandage overnight reduces the chance of contamination in orthopedic cases. However, clipper blades used repeatedly without sterilization have high levels of bacterial contamination and therefore are a potential source of infection.[11] Sterilization of clipper blades between uses has been shown to decrease bacterial counts and would lessen this problem. A study in humans revealed that using razors for the close removal of hair caused significant injury to the skin and increased bacterial colonization by altering wound defense mechanisms and delaying healing.[12] Consequently, if the surgical site is to be shaved, it should be done immediately before surgery and not the night before.

Clipping should be performed whenever possible outside of the surgical theater, as should the initial skin preparations. For surgical sites on the mid to distal limb, the hair from the elbow and stifle distad should be clipped circumferentially.

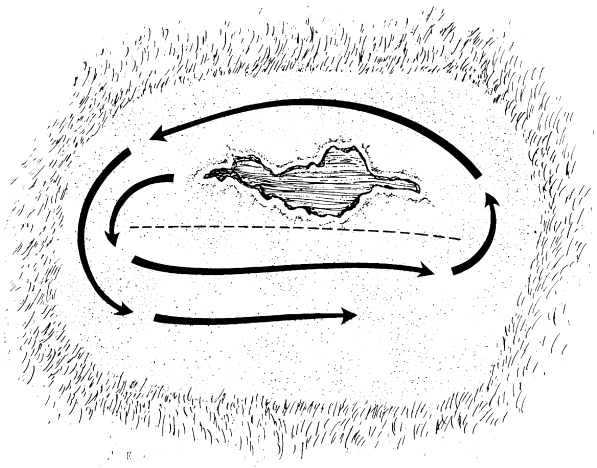

Figure 10-1. Proper method of skin preparation. In the initial preparation, the scrub begins at the anticipated incision site *(dotted line)* and moves outward in expanding concentric circles. The process is repeated until the sponges are free of visible soiling.

Additionally, the hair should be clipped 10 cm (4 inches) further proximad and distad relative to the intended surgery site to facilitate appropriate skin preparation and draping. If possible, only the final skin preparation should be performed in the surgical theater to prevent dust, dander, and exfoliated skin cells from contaminating the environment.

The optimal scrub time for maximal reduction of skin flora and lowest wound infection rates has not been determined for the horse. A povidone-iodine or chlorhexidine diacetate aqueous or alcohol solution used for 10 minutes, alternating with an alcohol rinse, is currently recommended,[13,14] and other scrub solutions are available (see Chapter 9). A recently available method of preventing infection is a cyanoacrylate-based microbial sealant (IntéguSeal Microbial Seal, Kimberly-Clark), which mechanically blocks migration of pathogens to the surgical wound.[15,16] It has been shown to reduce the pathogens commonly implicated in surgical site infections (SSIs) by 99.9% and improves the effects of povidine-iodine by fixing it on the skin and preventing it from being washed off. Another commonly used final application product that has a similar mechanism of action is DuraPrep Surgical Solution (iodine povacrylex [0.7% available iodine] and isopropyl alcohol, 74% wt/vol).

Surgical scrubs are applied to an area starting at the expected surgical incision and moving outward in expanding concentric circles, extending to the outer margins of the clipped area (Figure 10-1). This maneuver is repeated, alternating rinse solutions with the antiseptic until the sponges are free of visible soiling. Then a final application of the disinfectant is applied and left in place. In the distal limbs, the entire circumference of the limb is aseptically prepared, applying the scrub at the proposed surgical site and expanding distad and proximad, as just described.

Draping the Surgical Field

Ideally, barrier materials prevent the movement of debris and bacteria from nonsterile areas onto the surgical field for the duration of surgery. Bacterial penetration is time dependent, and colony-forming units (CFU) increase after 90 minutes of surgery.[17] Therefore drapes should be economical and easy to sterilize, and they should retain their barrier properties for at least 90 minutes, even after they are washed, sterilized, and reused. Woven fabrics that are intended for reuse consist of interlacing fibers that cross at right angles. The number of threads per square inch reflects the tightness of the weave, and the higher the number is, the tighter the weave and the more effective the barrier.

Reusable woven fabrics fall into two categories: cotton muslin with 140 threads per square inch, and pima cotton with tightly twisted fibers woven into 270 threads per square inch (58 threads/cm^2).[2] The cotton muslin is not a good barrier. It instantly allows passage of bacteria when wet (termed *strikethrough*), and dry penetration of bacteria may also occur because its pore size is 50 to 100 µm, which is large enough to allow bacteria (5 to 12 µm) to pass through.[18] On the other hand, pima cotton has a weave tight enough to prevent penetration by skin squames, but it readily allows penetration of bacteria when wet. A chemical treatment process, Quarpel, makes cotton fabric water resistant by providing a fluorochemical finish in combination with pyridinium or a melamine hydrophobe. This process renders pima cotton an effective barrier for up to 75 washings.[19] It is necessary to record the number of washings each piece of fabric undergoes to ensure that it is replaced before the barrier properties become ineffective.

A disadvantage of reusable woven fabrics is that they can sustain tears or punctures from towel clamps (therefore only nonpenetrating clamps should be used at the surgical site) and needles, which destroy their barrier function, although holes can be repaired with vulcanized fabric patches. These patches generally resist autoclave steam penetration, so this material becomes a less than ideal barrier.

Disposable materials are made from cellulose, wood pulp, polyesters, or synthetic polymer fibers formed into sheets and bonded together. The barrier properties of the various nonwoven materials differ a great deal.[17] Polymeric ingredients in these barriers tend to be more impermeable, but only those with a reinforced polyethylene or plastic film prevent moist and dry penetration at pressure points.[2] Although disposable drapes result in lower particle counts in the operating room (because of the lack of lint from cotton), the air bacterial counts are similar to those of reusable drapes. However, they are reported to decrease the number of bacteria isolated from the surgical wound by up to 90% compared with the cloth draping systems, and surgical wound infection rates decrease by a factor of up to 2½.[20,21] Because the difference between the two materials appears to be small, the choice is often based on economics and convenience. Even though the cost of single-use gown and drape sets is higher than for reusable sets, single-use sets provide the highest benefit rates. When large volumes of liquids are expected in the surgery (e.g., in colic and arthroscopic surgery), nonwoven disposable materials should be the material of choice for barrier drapes.[22]

Before moving the patient into the operating theatre, the patient should be covered with a clean drape and its feet should be covered with plastic bags or other water-impervious coverings to prevent contamination from the foot and distal limbs. After the patient is positioned in the room and the final preparation of the surgery site is completed, draping begins at the surgical site and moves outward.

Drapes are applied to all visible surfaces of the patient, providing a barrier to aerosolization of debris from nonsurgically prepared portions of the animal's skin. When applying drapes,

Figure 10-2. Four-quadrant draping method with separate drapes in each quadrant, leaving a rectangular area of the surgical field exposed. Note how the surgeon's hands are protected by the drape.

the surgeon's gloved hands are positioned on the side of the drape away from the animal's skin and are protected by curling the outer surface of the drape over the hands (Figure 10-2). The portion of the drape that is to be adjacent to the incision is positioned first and then moved peripherally to the desired location, never the reverse. It is desirable to drape closely, leaving no unnecessary skin exposed. Drapes are generally positioned in a four-quadrant method, with separate drapes in each quadrant, leaving a rectangular area of the surgical field exposed. It is recommended that this process be repeated to double drape the area immediately adjacent to the surgical site.

Self-adhering drapes are helpful when larger areas need to be exposed for topographic orientation and palpation. The goal of multiple layers of draping is to build a waterproof barrier that extends to cover the entire patient. When the distal limb is draped, the quadrant method may be used. However, providing access to the entire circumference of the limb is often preferred, especially during orthopedic procedures. In such a case, the foot is often covered with a rubber glove, and circumferential draping is applied, by wrapping first around the foot and then around the proximal limb. Next, a self-adherant sterile drape (Ioban 2, Ethicon, Somerville, NJ) is applied over the foot and the half sheet that has been applied to the proximal limb. Then an extremity sheet with a fenestration is passed over the foot and secured around the limb proximal to the surgery site (Figure 10-3).

Because there is a risk of contamination during draping, it is best to practice double gloving for the act of draping, removing the outer gloves immediately thereafter.

The surgical field is defined by areas above and level with the surgical wound (Figure 10-4). Even if draped, areas below the level of the wound should be considered contaminated and not part of the surgical field.

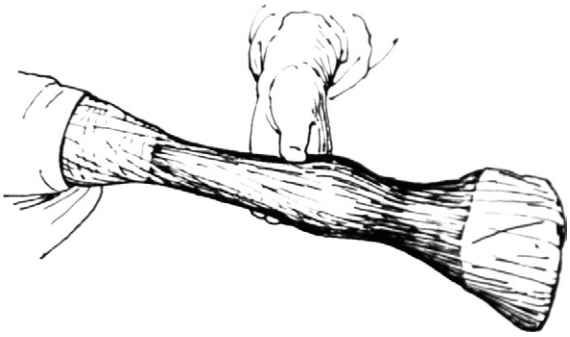

Figure 10-3. After drapes are applied circumferentially above and below the surgery site, they are covered with a self-adherant drape. An extremity sheet for fenestration can be passed over the foot to complete the draping.

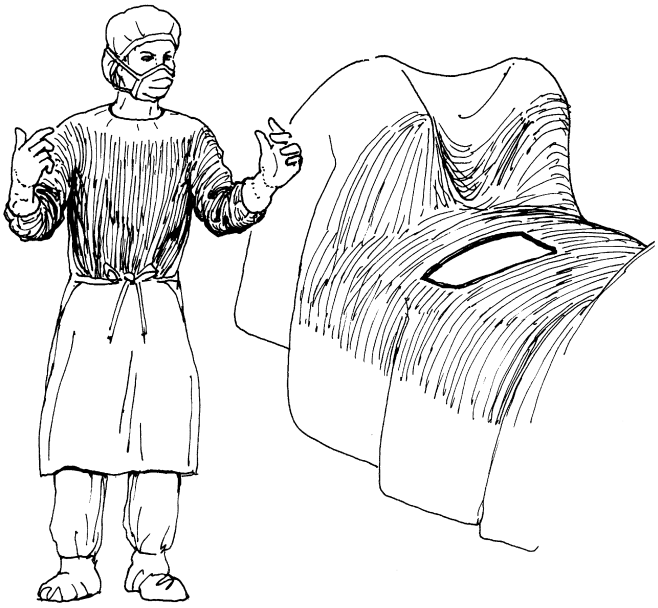

Figure 10-4. The surgical field is defined by the areas above and level with the surgical wound *(shaded area).* It is extended to include the front of the surgical gown from below the surgeon's shoulders to the waist *(shaded area).* Areas that are not shaded should be considered to be outside the surgical field.

THE SURGICAL FACILITY

With the increasing sophistication of surgical techniques and instrumentation available today, surgeries outside a proper surgical facility are becoming less common. If the procedure to be performed is expected to be lengthy, complicated, or sophisticated, use of a designated operating room is the standard of care. Surgical operating facilities should be equiped with separate induction, preparation, and recovery rooms. There should be a minimum of two surgical suites, so that clean procedures can be performed in one surgical suite dedicated to strict aseptic surgical procedures, and the other suite can be used for procedures on contaminated or infected sites (Figure 10-5). The surgery suite should be convenient to the work and have adequate room for the patient, anesthesia equipment and team, surgery team, and equipment. The average size of an equine operating room should measure 15 m^2 (135 square feet). Separate induction and recovery rooms should be available for each surgical suite. Floors and walls should be surfaced so that cleaning is efficient, and drains should be placed so that water does

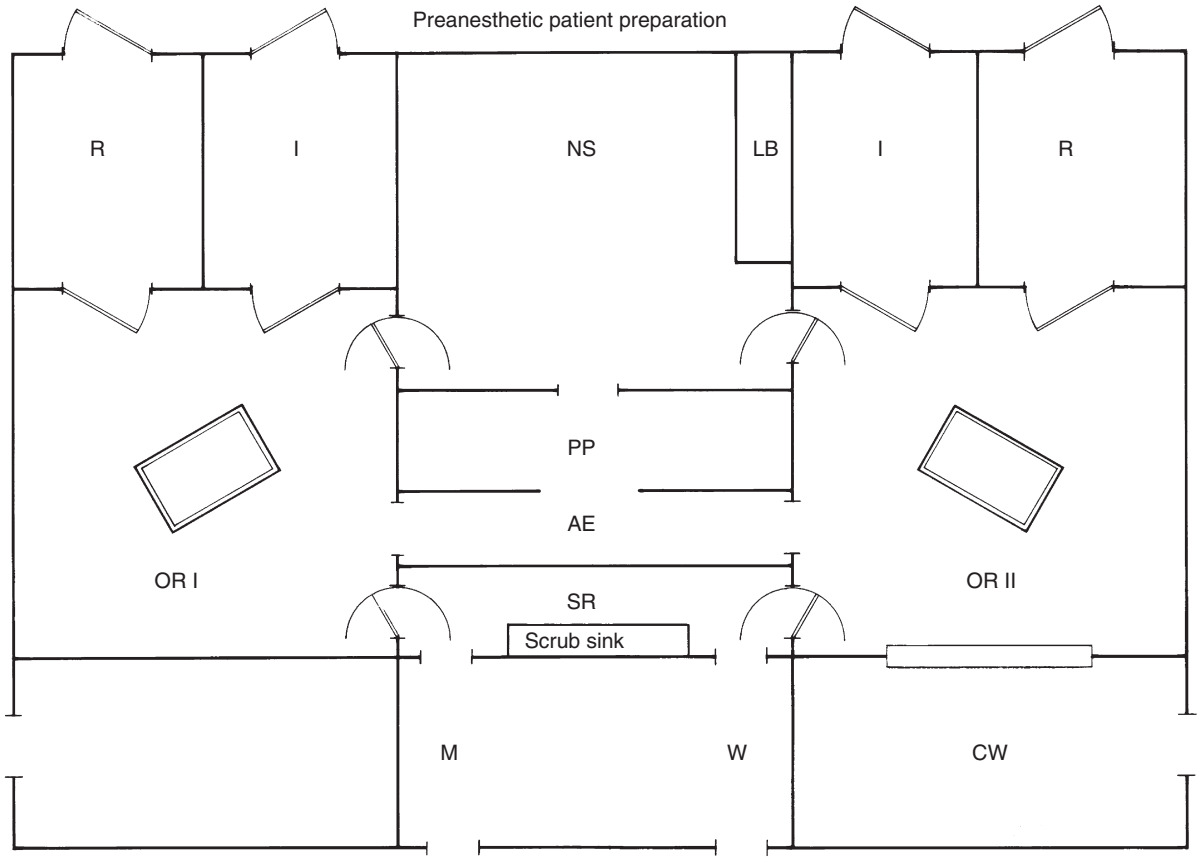

Figure 10-5. Suggested layout for an equine surgical facility. Separate rooms are provided for clean procedures and for contaminated or infected procedures, and a central station supplies both suites. *AE,* Anesthesia equipment; *CW,* client waiting room; *I,* induction stalls; *LB,* laboratory bench; *M,* men's dressing area; *NS,* nurses' station; *OR,* operating room; *PP,* pack preparation and storage; *R,* recovery stalls; *SR,* scrub room; *W,* women's dressing area.

not pool anywhere in the surgical suite after cleaning. Drains should be of sufficient diameter to remove the material and should contain a flushing system so that they do not harbor potentially dangerous mixtures of blood, feces, and bacteria.

One-way traffic should be maintained from the patient preparation area to the operating suite and then to the recovery room. After induction of anesthesia, the patient should be properly positioned on the surgery table and prepared for aseptic surgery, and then the table with the horse should be transported into the surgery suite. The suite should not be a high-traffic area, and proper surgical attire, including caps, boots, mask, and surgical caps, should be worn over the scrub suit when in the operating theater.

A room temperature of approximately 20° C (70° F) with a relative humidity of 50% provides a comfortable environment.[23] Air within the operating room should be under low positive pressure, so that when the doors open, air flows out of the room rather than into it. A minimum of 25 air exchanges per hour is recommended if the air is recirculated, and 15 air changes per hour if the air is exhausted to the outside. In selected human surgery suites, especially those used for joint replacement, laminar air filtering systems are installed to reduce the number of airborne microorganisms in the surgery suite. The filtering system measures about 3 × 3 m (Figure 10-6). The air is directed in a vertical flow of 0.5 m/sec initially through a rough, then through a fine, and ultimately through a high-efficiency particulate air (HEPA) filter. The ultraclean air reaches the surgical field and is directed around the patient to the floor. From there it is aspirated into exhaust outlets located all around the walls at the ceiling. The air is recirculated through the filtering system. Ideally, the outline of the filtering system is marked on the surgery room floor, which facilitates the positioning of the surgery site, the instrument tables, and surgeons within the field (Figure 10-7). Such ultraclean filtering systems are rarely found in equine hospitals and may not be necessary.

The door should be wide enough to allow the surgery table with the horse and other large equipment, such as the digital capture C-arm and radiography machines, to pass through easily. Electrical outlets should be located waist high or suspended from the ceiling so they do not become wet during cleaning of the room. Ideally, several locations for hooking up the anesthetic gases and the exhaust pipes of the anesthetic machine should be available. Also, devices should be placed in the wall to allow the application of traction pulleys for reducing fractures. Some provision should be made for emergency lighting, either by battery units or with an emergency generator that starts automatically when needed. At least one surgery light in each room should be wired to the emergency system. All cabinets should be recessed into the wall so that the floor can be adequately cleaned after each surgery. Viewing windows are desirable in operating rooms. This is done not only for direct viewing from a doctor or nurse's station but also so the public can view surgeries from an outside hall. Another option is the installation of a closed-circuit video camera system, which can

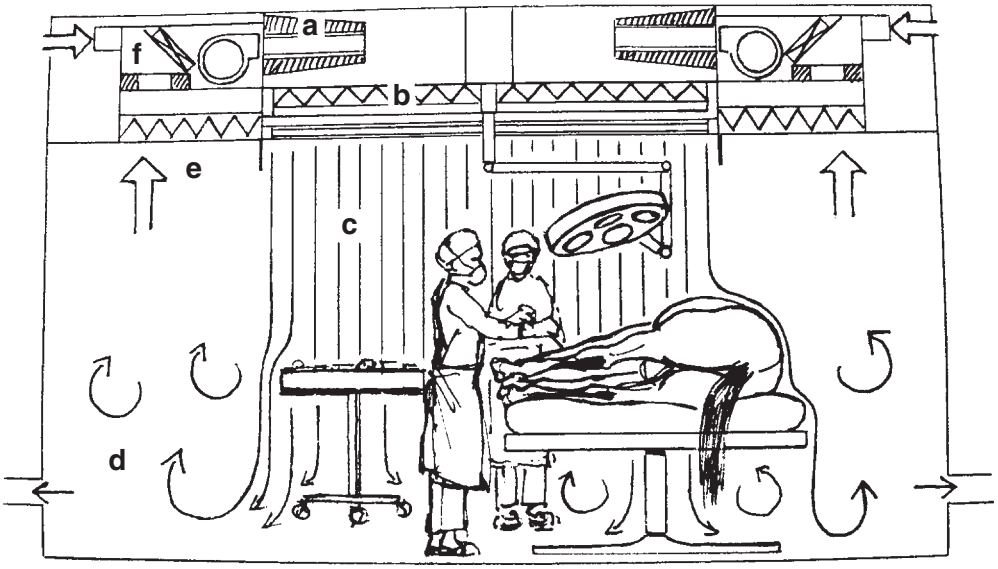

Figure 10-6. Schematic drawing of a laminar air filter system. The surgical site, the surgeons, and the instrument tables must be situated in the field of filtration. *a,* Blower to force filtered air through the pores in ceiling. *b,* Laminar air filter in the ceiling (frequently illuminated). *c,* Laminar air stream gently falling towards the floor. All objects within the field are surrounded by this air. *d,* Once at or near the floor, the air is directed toward the periphery, and some of it is lost through doors and other openings. The rest is gently pulled up toward a filter system mounted along the walls in the ceiling. *e,* After being extensively filtered and mixed with clean air from outside, the air is directed through the blower *(f),* again and reentered into the cycle.

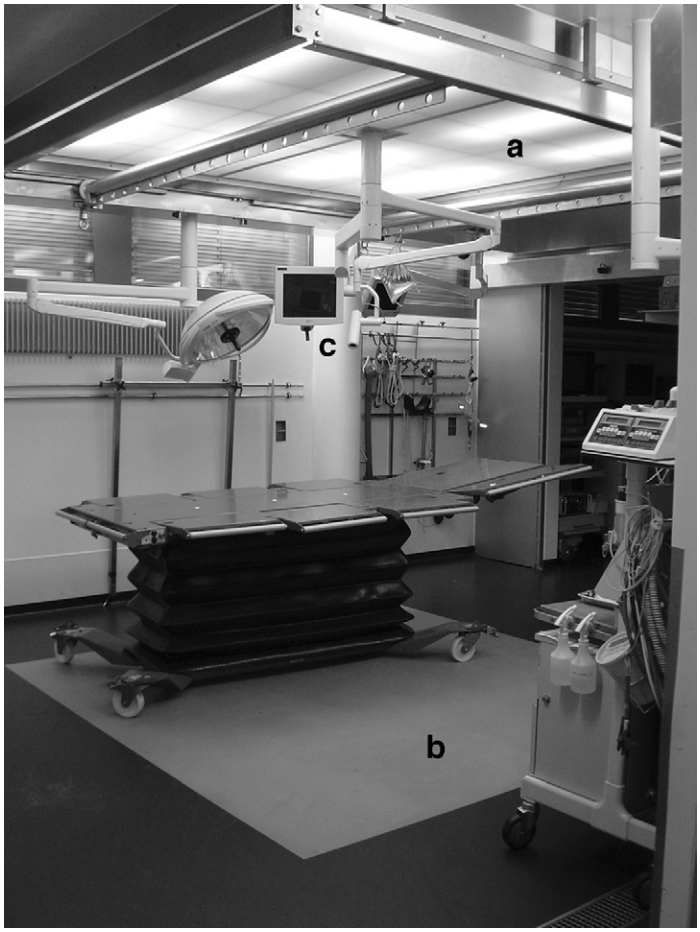

Figure 10-7. View into the aseptic surgery suite of the Equine Hospital, University of Zurich. *a,* The laminar air filter is illuminated. *b,* The grey area marked on the floor delineates the extent of the filter field. The surgery site, the surgeons, and the instrument tables need to be located within this field during surgery. (A mobile Haico, surgery table [Loimaa, Finland] is shown in the room). *c,* A movable video camera *(left)* is mounted on the ceiling together with a video screen.

be operated by the owners or students from an observation room distant from the surgery facility.

Biosecurity and infection control practices are becoming more important considerations when designing the surgcial facility, especially if the surgical caseload is large.[24] Ultimately the success and reputation of a surgical practice can depend on having surgical personnel trained in infection control with awareness toward SSI and the impact of antimicrobial-resistant microbes such as methicillin-resistant *Staphylococcus aureus* (MRSA) and *Salmonella* ssp.[6,25,26] Sometimes the facility itself will prevent optimal minimization of SSI if the design does not support easy cleaning and proper storage of waste materials. It may behoove the director of the facility to enlist the advice of an infection preventionist in planning the facility, then training the personnel, and developing an effective infection control program.[27] Infection control programs should include monitoring, surveillance, hygiene, disinfection protocols, and education.[28]

THE OPERATING TEAM

Scrub Attire

The operating team consists of the people performing the surgery and administering the anesthesia, nonscrubbed assistants, and observers within the operating room. All individuals, regardless of their role in the surgical intervention, contribute to operating room contamination and potential infection of the wound. Therefore scrub suits, caps, masks, sweat bands, shoe covers, gowns, and gloves are worn to prevent shed particulates and microorganisms from reaching the surgery site.

Scrub suits usually have separate pants and shirts and should be clean, comfortable, and dedicated to the operating room (Figure 10-8) Many blended cotton materials are available for this purpose. Although the design is relatively standard, optimally sized garments will cover the surgeon effectively from neck to ankle while leaving the arms exposed. The bottom of the scrub shirt is tucked into the pants to prevent shedding of hair, skin cells, and bacteria between the top and the pants. For those not needing to gown and glove for the procedure, long-sleeved cuffed jumpsuits are also quite useful, because they provide a barrier against shedding of skin debris and microorganisms. The scrub suit should not be worn outside the surgery suite without being covered by a clean laboratory coat, and it should be laundered after each case or at least daily. This scrub clothing should be steam sterilized weekly to ensure removal of the microorganisms that can remain after routine laundering. Alternatively, bleach can be added to the laundry cycle to reduce the number of bacteria.

Air in an operating room contains approximately 250,000 particles (bacteria, lint, and skin squames) and 11 to 13 bacteria per cubic foot.[29,30] These particles and bacteria increase with the number of people and level of activity in the room, the amount of uncovered skin area, and the amount of talking. Bacterial levels in excess of 400 per cubic foot may be seen in a busy operating theater. Therefore barrier apparel is worn to minimize these numbers and their effect on surgical wound rate.

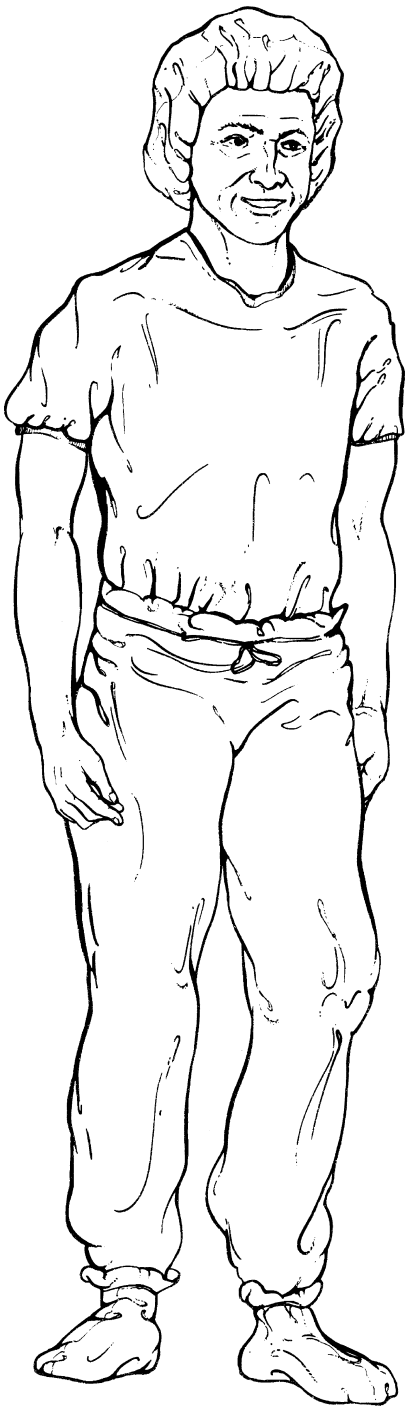

Figure 10-8. Scrub suit recommendations. The scrub shirt should be tucked into the pants. Although not always practical, the pants legs may be tucked into boots or shoe covers. Peripheral personnel may wear long-sleeved tops with elastic cuffs to further limit transmission of skin debris.

Head Covers

Human hair is a major source of bacteria. Because the uncovered hair of the surgeon, who stands over the incision, is frequently a major source of surgical wound contamination, head covers are worn to reduce the shedding of hair and bacteria. All people in the operating room should wear head covers—caps, hoods, or bouffants (Figure 10-9). These are available in reusable cloth and disposable nonwoven material and should cover all the hair on the head, including moustaches and beards. The reusable head covers should be washed after every

Figure 10-9. Headcover styles are shown in order of increasing barrier capability, from left to right. Surgeon's caps, bouffant caps, and hoods offer protection against shedding hair and debris into the surgical wound. Coverage by the old style surgeon's cap is obviously limited compared with the other types.

procedure (up to a total of 75 times and then, like the reusable drapes, discarded).

Gowns

Gowns provide an aseptic barrier between the skin of the operating team and the patient. The gown should be water resistant as well as comfortable and breathable. It should not produce lint. Gowns are packaged individually and folded so the interior back region is outermost, allowing this area to be handled without contaminating the gown's exterior surface. Once the gown is donned, the sterile surgical field extends only above the waist (see Figure 10-4).

Gowns, like the draping materials, can be made of either reusable woven fabric or nonwoven disposable material. The most effective barrier gown contains some type of polyester or plastic film over a breathable material. Preventing strikethrough when becoming wet is an attribute that is mandatory for surgical gowns. Gore-Tex gowns with double-layered barriers in the elbows, chest, and abdominal areas have become popular because they are comfortable and meet the necessary criteria. Gore-Tex is a barrier material consisting of an expanded film of polytetrafluoroethylene between two layers of fabric with a maximal pore size of 0.2 μm, which resists strikethrough by water and bacteria.[31] It allows evaporation of perspiration, which increases comfort for the surgeon. Gore-Tex fabrics are more durable than Quarpel-treated pima cotton and will retain barrier quality characteristics for up to 100 washings.

Gloves

Surgical gloves are made of natural rubber latex and are provided in a sterile, single-use package. Gloves should fit tightly, because gloves that are loose will impair dexterity, but they should not be so tight that the surgeon's fingers lose sensitivity. Modified cornstarch is preapplied to most gloves for easier application, and therefore the outside of the gloves should be rinsed before patient contact.[32] Cornstarch is referred to as "absorbable powder." Magnesium silicate (talcum) powder is no longer used in powdered gloves because it potentiates latex allergies and causes granulomas in patients even when gloves are thoroughly rinsed. However, even absorbable powdered gloves contribute to natural rubber latex allergies (which cause contact dermatitis), and the powder acts as an airborne carrier of natural latex proteins (which can cause respiratory allergies). Therefore most surgical gloves are treated with multiple washings to reduce the latex proteins. If allergies to latex develop, powderless latex gloves are available, which are chlorinated during manufacturing to decrease their tackiness. (However, these gloves do not store well, and inventory should be monitored to prevent use of these gloves beyond the expiration date, because failure becomes common.) Alternatively, vinyl gloves are available to eliminate this problem, although their performance is less desirable (i.e., dexterity is reduced).

The accepted industry standard for surgical gloves is that 1.5% contain punctures before use.[33] One study found that 2.7% of latex and 4.1% of vinyl gloves leak when filled with water. By the end of surgery, up to 31% of gloves have perforations, and when double gloves are worn, 16% to 67% of the outer gloves and 8% to 30% of the inner gloves contain perforations. Holes are most common on the thumb and index finger of the nondominant hand. Closed gloving techniques are preferred over open techniques because the surgeon's skin will not contact the outside of the gown cuff. If soiling of the gloves is expected or extra protection is needed during a surgical procedure (i.e., during most orthopedic procedures), many surgeons elect to apply and wear a second pair of gloves. Cuffs of the surgeon's gown should be completely covered, because cuff material is not impervious to water penetration. The use of plastic safety sleeves often helps when the surgeon's hands and arms may be submerged, such as during colic surgery. Extra-thick gloves are available for orthopedic surgeries, where there is an increased risk of puncturing the gloves from sharp bone spikes and implant materials.

Face Masks

Facial coverings are not effective bacterial filters. When properly fitted, they redirect airflow away from the surgical wound and in doing so reduce the potential for surgical wound infection

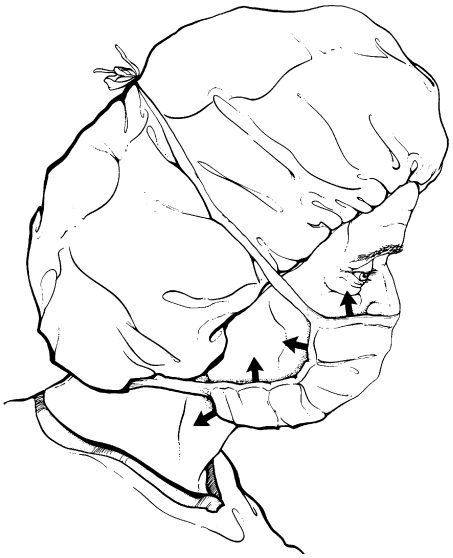

Figure 10-10. Potential leakage sites of the standard surgical mask *(arrows).* Transmission of contaminants around the edges of the mask can be limited by properly conforming the nosepiece to the nose and tying the mask snugly.

(Figure 10-10). Despite clinical reports that facial coverings do not reduce surgical site infections, the use of a face mask is considered mandatory during surgery. Tie-on face masks are tied over the head first, the wire on the top of the mask is fitted tightly over the surgeon's nose, and the lower ties are pulled around and tied behind the neck.[2] The mask should fit tightly around the sides of the face and over the tip of the chin. Cup masks with elastic bands provide a better fit and offer less chance of bacterial contamination.

Disposable surgical face masks are recommended over washable gauze because of improved efficiency and comfort. Masks should be worn by all personnel entering the surgery room at any time. Failure to wear masks even when surgery is not in progress promotes contamination of the surgical area. Masks should not be removed and replaced, pushed on the top of head, dangled from the chin, or tucked in a pocket. Each of these common practices risks contamination of scrub clothing with bacteria from inside the mask, which may be transmitted to the patient. The effectiveness of masks and other barriers in a surgery room should probably not be relied on for more than 2 hours.[2] A frequent change of masks, caps, and other apparel is warranted when this time period is exceeded. Bearded surgeons should wear a hood that covers all facial hair in addition to a face mask, which alone is insufficient.

Foot Covers

Disposable shoe covers are usually made of light nonwoven material and sometimes have polypropylene coatings to avoid strikethrough. Shoe covers help keep the surgeon's feet dry and thus more comfortable during surgical procedures, but they are not believed to be useful in reducing the soil brought to the operating room floor in an equine surgery suite, because of the obvious soiling that occurs with this type of patient. Therefore, shoes dedicated to the operating room are a better option for reducing environment contamination; these shoes should never be worn outside the surgery area without shoe covers, which are then removed before reentering the surgery suite.

Surgeon's Skin

A surgeon's hands have higher bacterial counts and more pathogenic organisms than the hands of other medical personnel because of increased exposure to scrub solutions (which irritate the skin) and contaminated wounds.[34] The objective of a surgical hand scrub is to remove gross dirt and oil and decrease bacterial counts, and just as importantly to have a prolonged depressant effect on transient and resident microflora of the hands and forearms. Surgical scrub protocols are based either on scrubbing time or on stroke counting. Principles of the scrub procedure are standard. Fingernails are kept short, clean, and free of polish and artificial nails (chipped nail polish and polish worn for more than 4 days foster an increased number of bacteria on the fingernails, even after a surgical hand scrub).[35] All surfaces of the hands and forearms below the elbow are exposed to antiseptic scrub. Special attention is paid to the area under the nails. The ideal scrub time is controversial, but 2 to 5 minutes seems to be safe and effective, depending on the agent used. Ten-minute scrubs are no longer used because they do not result in additional reductions in bacterial counts (and in one study, counts were increased) and are more irritating to the skin. A 2-minute scrub results in bacterial count reduction similar to that of a longer scrub.[36] It is currently recommended that soft brushes or sponges be used for the first scrub of the day, but subsequent scrubs can be brushless.

The residual activity of antisepsis is widely accepted as being useful in the preoperative disinfection of the surgeon's hands. The residual activity of either chlorhexidine gluconate or alcohol chlorhexidine is reported to be superior to that of aqueous povidone-iodine against resistant bacteria. Therefore, for procedures lasting less than 1 hour, aqueous povidone-iodine is acceptable, but if the procedure is going to exceed 1 hour, either alcohol chlorhexidine or chlorhexidine gluconate is the antiseptic of choice.[10] The primary objective of surgical hand disinfection is destruction or maximal reduction of the resident flora; the secondary objective is elimination of the transient flora. Surgical hand disinfection with alcohol-based hand rubs, many of which contain emollients, is growing in popularity over surgical hand washes made of an antiseptic-based liquid soap, because the rubs have a rapid and immediate action, do not require water or a scrub brush, are considerably faster than the traditional hand scrubs, and cause less skin damage after repeated use.[10] Recent meta-analyses of human studies show that alcohol-based antiseptics or rinses and products, including povidine-iodine combinations, and alcohol rubs between scrubs are the most effective method of hand preparation.[25,37,38]

One large veterinary clinic in Europe (University of Zurich) uses a combination of three products in sequence. A disinfectant solution, Bactolin (Bode Chemie, Hamburg, Germany), is applied with a soft brush or foam pad as the initial wash. Then the hands are dried, and 10 mL of Sterillium (Bode Chemie, Hamburg, Germany) is applied for 3 minutes. Postoperatively, Baktolin Balm is applied for rehydration. Sterillium contains 2-propanol (45%) and 1-propanol (30%), and mecetronium ethyl sulfate (MES), a nonvolatile quaternary ammonium compound with skin soothing and mild antiperspirant effects. Manufacturer's claims are exceptionally good skin protection and

skin care, even with long-term use; efficacy against a broad range of microorganisms and viruses (bactericidal, fungicidal, tuberculocidal, virus inactivating); and excellent residual effect. The manufacturer also claims that this preparation permits penetration deep into the stratum corneum of the skin, where it forms a defensive barrier against organisms that emerge with perspiration.

Bacterial examination after disinfection was conducted in two ways. The volunteer rubbed the distal phalanges of one hand (randomly selected) for 1 minute in a petri dish containing 10 mL of tryptic soy broth (TSB) supplemented with neutralizers (immediate effect). The other hand was gloved for 3 hours for the assessment of the sustained effect. After removal of the glove, sampling was done as for the immediate effect. From the sampling fluid, two 1-mL and two 0.1-mL aliquots were seeded, each in two petri dishes with solidified TSB. A 1:10 dilution of the sampling fluid in TSB was prepared, and two 0.1-mL aliquots of this were seeded as described earlier. Dishes were incubated at 37° C for 24 to 48 hours. For each dilution the mean number of colony-forming units (CFUs) scored in duplicate dishes was calculated. This was multiplied by the dilution factor to obtain the number of CFUs per milliliter of sampling liquid.[25] The examination technique described earlier has confirmed that rubbing the hands with an antiseptic is significantly more effective than scrubbing with brushes.[39] Hand rubbing with 0.2% chlorhexidine and 83% ethanol (Hibisoft) suppressed the number of bacteria and prolonged sterilization for more than 3 hours. In a study conducted according to two European standards for bactericidal efficacy, all alcohol-based surgical hand rubs (Sterillium and Softa Man) and the hand washes, chlorhexidine (Hibiscrub), and povidone-iodine (Betadine) fulfilled the requirements of a bacterial suspension test.[40] However, only the hand rubs met the requirements of the *in vivo* test of efficacy on resident skin flora, and chlorhexidine failed that test. In another study on surgical hand scrubs, Sterillium was superior to Hibiscrub and alcoholic gels in terms of skin tolerance and microbicidal efficacy.

A 1% chlorhexidine gluconate solution and 61% ethyl alcohol with moisturizers (Avagard) is currently in use as a hand cleaner in the United States. Advantages claimed are greater preservation of the skin's own moisture, pliability, and integrity; rapid microbial kill; and activity against a wide range of organisms, including MRSA. It has been shown to have residual activity comparable to that of chlorhexidine gluconate alone and greater than that of aqueous povidone-iodine. A similar product, 0.5% chlorhexidine gluconate plus 70% isopropanol (Hibisol), has greater residual activity against clinically significant test organisms than chlorhexidine digluconate skin cleanser (Hibiscrub), povidone-iodine surgical scrub (Betadine), or 60% isopropanol.

Despite growing evidence in favor of alcohol-based hand rubs for preoperative preparation, many surgeons remain reluctant to switch from an antiseptic soap to an alcohol-based hand rub. Large-animal surgeons pose a considerable challenge to methods employed in human hospitals, because they so often handle heavily contaminated areas on their patients before surgery. Therefore thorough prewashing is strongly encouraged before using alcohol-based hand rubs.

Additionally, recently developed microbicide products containing substituted phenolic and quaternary phospholipids have 30-second kill times and are used in 2-minute brushless scrubs (Techni-Care). These products are less irritating to the skin and are being used in several hospital applications, even including the direct applications to infected and open wounds. All of these products are recommended to be used with a skin balm after surgery to prevent the surgeon's skin from drying with multiple uses.

Staffing the Surgery Area

Equine surgery requires a team of at least three people. A surgeon, an anesthetist, and a dedicated surgical technician form the minimal operating team for most efficient operation and least risk to the patient. The properly trained anesthetist allows the surgeon to concentrate entirely on the surgical procedure and must be able to restrain patients effectively, place catheters, calculate drug dosages, and be familiar with various sedative and anesthetic agents and regimens. The surgical technician becomes an extension of the veterinary surgeon and usually is more adept than the surgeon in the support areas.

An operating room supervisor is important regardless of the size of the facility. The supervisor is responsible for ordering and stocking all supplies, maintaining a surgery log, and recording all controlled substances and their use. The dedicated surgical technician can fill this role. Additionally, a surgical assistant is invaluable, and technicians with the proper basic training skills and attitude can be acceptably competent in a relatively short time with minimal training, rounding out the team to a perfect four.

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CHAPTER
11 Surgical Instruments

Jörg A. Auer

Instruments are the verterinary surgeon's best friends. It is therefore important to be surrounded by the "best friends possible." In other words, for a specialist in veterinary surgery, it is very worthwhile to acquire top-quality instruments and to take good care of them. The veterinary surgeon can choose from an abundant selection of instruments manufactured predominantly for human surgery. However, the number of instruments specially designed for veterinary applications is steadily increasing. Practically speaking, the instruments used by a surgeon are determined by a combination of economics, predicted use, specialty considerations, and personal preference. The costs involved in the purchase of instruments are substantial and demand a clear understanding of manufacturing procedures, maintenance, and potential applications during surgery.[1-4]

Surgical instruments are offered by a large number of manufacturers, all competing for the same customers. There are still no international standards for instrument quality. Therefore, caution must be exercised before purchasing instruments at bargain prices. When costs for replacement of prematurely worn-out instruments are combined with the frustrations encountered during surgery because of poorly functioning equipment, the higher costs of high-quality instruments are justified. On the other hand, some disposable instruments intended for human surgery can be used repeatedly by veterinary surgeons, which reduces costs for such high-tech instruments considerably.

MATERIALS

A description of the different compositions of stainless steels used for manufacturing instruments is found in Chapter 75. Here, only some general comments referring to instrument materials are made.

High-quality stainless steel has become the material of choice for most surgical instruments. In its various forms, hardened, corrosion-resistant stainless steel exhibits a number of desirable instrument characteristics, such as elasticity, tenacity, rigidity, ability to hold an edge, and resistance to wear and corrosion. Variation in the carbon content of the steel results in changes in the handling characteristics of the material to meet special needs. Currently, most stainless steels used for instrument manufacturing contain a high content of carbon. Although high-carbon stainless steel is resistant to wear and allows the instrument to hold its sharp edge, tungsten carbide inserts have been introduced to replace stainless steel cutting and gripping surfaces (Figure 11-1).[5] These inserts are even harder and more resistant to wear, prolonging the life of the instrument considerably. The bond between these inserts and the body of the instrument represents a potential problem area, because it may loosen

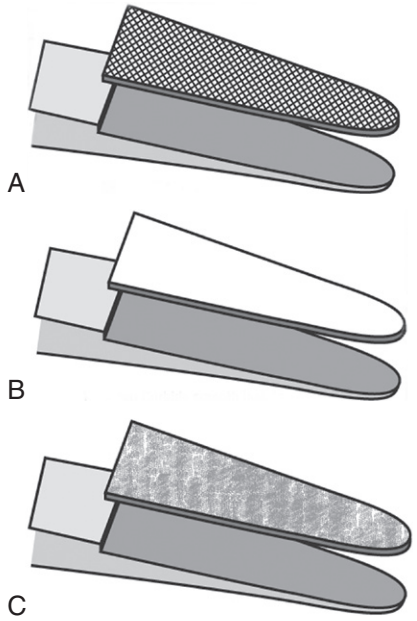

Figure 11-1. Three different types of tungsten carbide inserts for instruments. **A,** Serrated inserts. **B,** Smooth inserts. **C,** Tungsten carbide dust inserts. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

through frequent use and repeated sterilization.[6] Although the inserts on the needle holders can be exchanged, tungsten carbide cutting surfaces in scissors cannot be replaced once they are damaged; instead, a new instrument has to be purchased. Some manufacturers offer a reduced price, if the original instrument was purchased through them.[5]

The fine edges and working surfaces required for microsurgery have led to the use of titanium alloys for this specialty instrumentation. Titanium alloys can be produced with excellent corrosion resistance and temperature strength. The brittleness of such alloys complicates the manufacturing process and dictates particular care during use and maintenance. Manufacturers' recommendations for cleaning and sterilization of titanium alloy instruments should be closely followed. Recently, titanium nitrite–coated scissors became available. The glide quality is considerably improved by electrophysical smoothing, which results in fewer abrasions and reduced wear. The coating renders the surface three to five times harder, which multiplies the lifetime of the cutting edges and makes the instrument extremely resistant to scratches and other damage. The use of this material is also reflected in the increased cost of manufacturing and therefore also in the purchase price.

Before the wide availability of low-cost stainless steel, many instruments were manufactured from chrome-plated carbon steel. The chrome plating provided corrosion resistance unavailable with carbon steel alone. Unfortunately, the chrome plating itself is susceptible to early deterioration from frequent rough use and exposure to acidic solutions. Failure of the chrome coating exposes the underlying carbon steel, allowing oxidation and rust formation. Although deteriorated instruments can be refurbished and replated, replacement with higher-quality, longer-lasting stainless steel instruments is more cost-effective and is strongly encouraged.

Corrosion resistance can also be improved by the process of passivation. This process uses nitric acid to remove foreign materials from the stainless steel surface, while covering it with a thin coat of chromium oxide. Both actions contribute to corrosion resistance of surgical stainless steel. Polishing provides a very fine instrument surface, further increasing corrosion resistance.

One popular surface finish for increased corrosion resistance is a dull satin finish. Created by abrasion or sandblasting techniques, the satin finish reduces light reflection and thus eyestrain. A black finish, which serves a similar purpose, is also available. Gold electroplating of instrument handles does little to improve working surfaces but is generally recognized as a symbol of high-quality instrumentation.

Instrument companies use various colors on the handles to designate different quality of instruments or cutting edges. Sontec Instruments uses plain handles to represent standard quality instruments, gold handles for tungsten carbide (TC; see Figure 11-5) inserts, gold handles with an additional gold stripe or black anodized handles for power-cut blades (PC), which are the sharpest cutting edge available, and one gold and one black anodized handle for tungsten carbide inserts with power-cut blades.

Hundreds of different instruments are available today, and it is impossible to know them all by name, function, and design. Frequently, instruments are modified and manufactured under different names. In this chapter, instruments are discussed in groups according to function, and differences within the groups are mentioned where appropriate.

INSTRUMENTS FOR GENERAL SURGERY

All surgeons must be familiar with all basic instruments, which will aid in the selection of the appropriate instrument for a specific procedure and expedite communication during surgery. The parts of a typical surgical instrument are identified in Figure 11-2. Specialty instruments will be covered in subsequent chapters where applicable. Instruments that fall into more than one category are described only once.

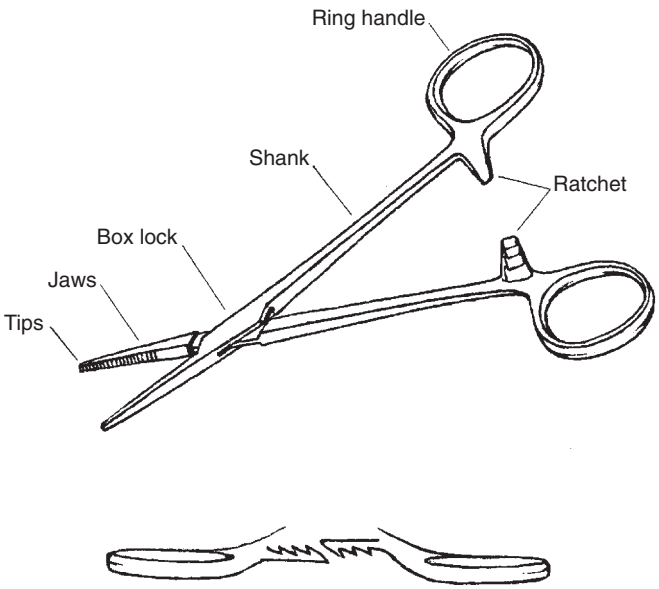

Figure 11-2. *Top:* Labeled parts of a typical surgical instrument. *Bottom:* End-on view of the ratchet mechanism. The ratchets should be slightly separated when the jaws are closed.

Scalpels

Steel Scalpels

Scalpels are available with detachable blades, as disposable units with blades attached, and as reusable units with blades attached. In most clinics, scalpel handles with different detachable disposable blades are used (Figure 11-3). The No. 3 scalpel handle is the most frequently used and comes in different shapes (see Figure 11-3, *A-C*). Most surgeons prefer the No. 10 blade; the No. 15 blade is a smaller version in a similar shape (see Figure 11-3, *E*). The No. 11 blade is frequently used for stab incisions during arthroscopic surgery, and the No. 12 blade is used for periosteal stripping (see Figure 11-3, *E*). The No. 4 handle (see Figure 11-3, *D*) accepts larger blades such as No. 22 to 24 (see Figure 11-3, *F*) and is used in less delicate surgical procedures, such as debulking granulation tissue and resecting large wounds with proud flesh. A detachable blade should not be used in joints or deep within heavy connective tissues, where they could break off and be lost from view. The primary advantage of disposable blades is that replacement blades are consistently sharp.

The reusable scalpel with attached blade has a single advantage over the disposable units: the blade will not detach when used in heavy connective tissue, within joints, or in deep tissue planes, where visibility and access for removal are poor. Ethylene oxide or gas plasma sterilization (see Chapter 9) is recommended, because heat and chemicals will dull the reusable blade.

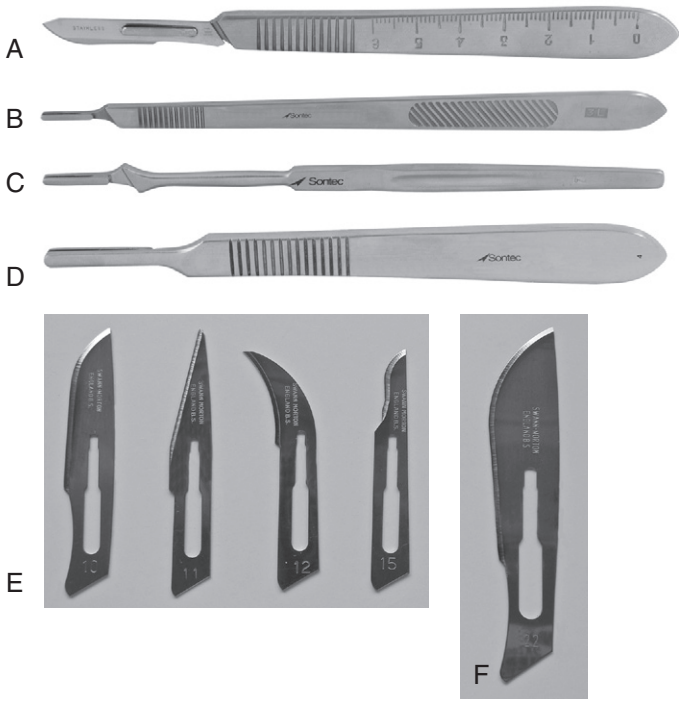

Figure 11-3. Different types of scalpel handles and blades. **A,** Knife handle No. 3, fits surgical blades 10-15. **B,** Knife handle No. 3, long, fits surgical blades 10-15. **C,** Knife handle No. 4, fits surgical blades 20-25. **D,** Knife handle No 7, fits surgical blades 10-15. **E,** Different scalpel blades for the No 3 scalpel handles (f.l.t.r.): Nos.10, 11, 12, 15. **F,** No 22 scalpel blade fits scalpel handle. No. 4 (there are additional modifications of the blade available).

Disposable Scalpels

Disposable scalpels with nondetachable blades are frequently used in the field or for bandage removal. In a surgical procedure that requires no other instruments, such a scalpel may be used instead of opening an entire set of instruments.

High-Energy Scalpels

High-energy cutting instruments include the electrosurgical scalpel, the plasma scalpel, the water scalpel, and various forms of lasers. Although their energy sources differ, they share a common cutting mechanism of action. Energy is focally transmitted to tissue, and the effect depends on the water content of the tissue. The result is vaporization of cells along the line of energy application, a variable degree of thermal necrosis of the wound edges, and a relatively bloodless incision. Electrosurgical incisions are by far the most frequent applications of high-energy cutting.

Electrosurgery uses radiofrequency current to produce one or more of the following effects: incision, coagulation, desiccation, or fulguration of tissues. Most modern electrosurgery units use controlled high-frequency electrical currents ranging between 1.5 and 7.5 mHz.[6] The predominant effect depends on the waveform of the current. Continuous undamped (fully rectified, fully filtered) sine waves provide maximal cutting and minimal coagulation, and they produce the least amount of lateral heat and tissue destruction.[6] On the other hand, interrupted damped (partially rectified) sine waves maximize coagulation and minimize cutting capabilities. Modulated, pulsed (fully rectified, nonfiltered) sine waves allow simultaneous cutting and coagulation, or "blended" function. The magnitude of the selected effect is directly proportional to the duration and power (in watts) of the applied current.[6]

Because most modern units can be used with unipolar and bipolar instruments, adequate electrical grounding of the patient is required for the unit to function properly in the monopolar mode (Figure 11-4). The desired function (cutting or coagulation) can be selected by activating a button on the handle. Cutting and coagulation tips are available and can be exchanged as desired. Frequently, needles are used for cutting tissue because of their limited contact area, which reduces the amount of tissue necrosis. Correct technique dictates that the tissue be placed under tension and that the contact area of the point be minimized to prevent adjacent tissue destruction. Skin and fascia incise easily, whereas muscle and fat are more easily incised using a cold scalpel. Units can also be used to coagulate vessels less than 1 mm in diameter (see Chapter 12). Coagulation time should be minimized to limit the amount of tissue destruction. The bipolar forceps for direct coagulation of smaller vessels speeds up hemostasis, because the vessel can be grasped directly by the bipolar forceps, bypassing the initial placement of a hemostatic forceps.

Scissors

Surgical scissors are available in various lengths, weights, blade types (curved or straight), cutting edge types (plain or serrated), and tip types (sharp-sharp, sharp-blunt, and blunt-blunt). The two most commonly used operating scissors for tissue dissection are the Mayo and the Metzenbaum scissors (Figure 11-5). The sturdier Mayo scissors, available in 14- to 40.5-cm (5½- to

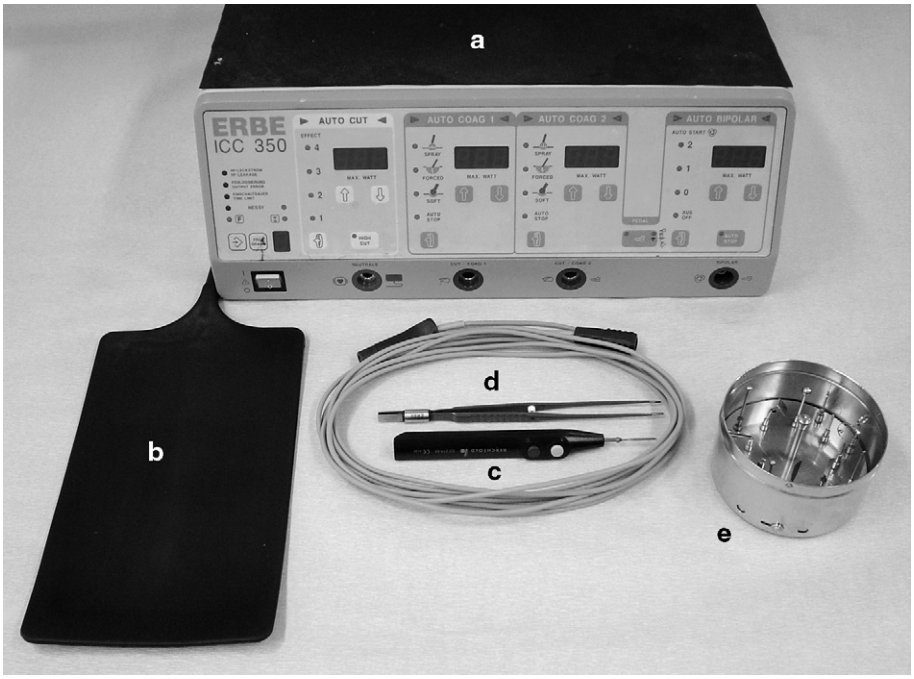

Figure 11-4. Electrosurgical instrumentation; *a,* electrocautery unit with capacity for monopolar modes of cutting and coagulation, and for bipolar coagulation mode; *b,* patient grounding plate; *c,* monopolar handpiece with thin knife; *d,* bipolar electrode forceps with connection cable; *e,* exchangeable electrodes for the monopolar handpiece.

16-inch) lengths, should be used for cutting connective tissue. Metzenbaum scissors are reserved for delicate soft tissue dissection and should not be used for dense tissue dissection. They are available in 11.5- to 40.5-cm (5- to 16-inch) lengths.

Specially designated and marked suture scissors are used during surgery to cut the sutures. It is important to use only the suture scissors for cutting sutures, because this job rapidly dulls the blades, making them less effective for soft tissue dissection.[7] The Olsen-Hegar needle holders are equipped with cutting edges (see later) to cut sutures, which obviates the need for a special set of suture scissors. Suture removal scissors (Figure 11-6, *A*) are lighter in weight, and they have a sharp, thin point and a concave lower blade that facilitates blade placement underneath the suture, which reduces suture tension as it cuts. Wire-cutting scissors (see Figure 11-6, *B*) have been designed specifically for wire suture removal and are typically short and heavy and have serrated blades.

Of the bandage scissors, the Lister (see Figure 11-6, *C*) and the all-purpose utility scissors are the best known. The lower blade of these scissors has a blunt tip that allows it to be inserted underneath the bandage without damaging the patient's skin. The all-purpose scissors comes with a needle destroyer and a serrated blade (see Figure 11-6, *D*). The serrated blade reduces bandage material slippage during cutting. Both scissors can be autoclaved.

As a general rule, scissors should be used only as intended by their design. Misuse dulls the edges and causes blades to separate, rendering them ineffective. Properly functioning scissors should open and close with a smooth, gliding action, and their tips should meet when closed. Scissors should be sharpened only by a qualified person. Incorrect blade sharpening causes the metal to overheat and lose temper, and the cutting edges to become soft, resulting in loss of a sharp edge. Scissors with tungsten carbide inserts maintain sharpness longer. The insert can be replaced when dull.

Needle Holders

A needle holder is selected on the basis of the type of tissue to be sutured, the needle and suture material used, and personal preference. The grasping surfaces of the needle holders are cross-hatched with a central longitudinal groove that facilitates the holding of curved suture needles. The two most commonly used needle holders are the Mayo-Hegar and the Olsen-Hegar (Figure 11-7). The Olsen-Hegar is a combination of needle holder and scissors and is available in lengths between 15 and 30 cm (6 to 12 inches). It allows the surgeon working without an assistant to place, tie, and cut suture material swiftly. Its major disadvantage is the occasional inadvertent and premature cutting of suture material, which occurs usually from inexperience with the instrument. The Mayo-Hegar needle holder (see Figure 11-7, *B*) has approximately the same shape as the Olson-Hegar, minus the scissors, and is available in lengths between 14.5 and 19.5 cm (5¾ and 7½ inches). Both needle holders are available in various jaw widths. The choice of jaw width is based on the size of the needle. Narrow jaw widths are recommended for small needles to prevent needle flattening as the ratchet is tightened, whereas wider jaws prevent larger needles from rotating as they pass through dense tissue.

The Mathieu needle holder (see Figure 11-7, *C*) is also popular in equine surgery. It lacks finger holes and has an open box lock that is released by further closing of the handles and is available in lengths between 14 and 20 cm (5½ and 8 inches). Unfortunately, this can occur when a firm grip is applied to the

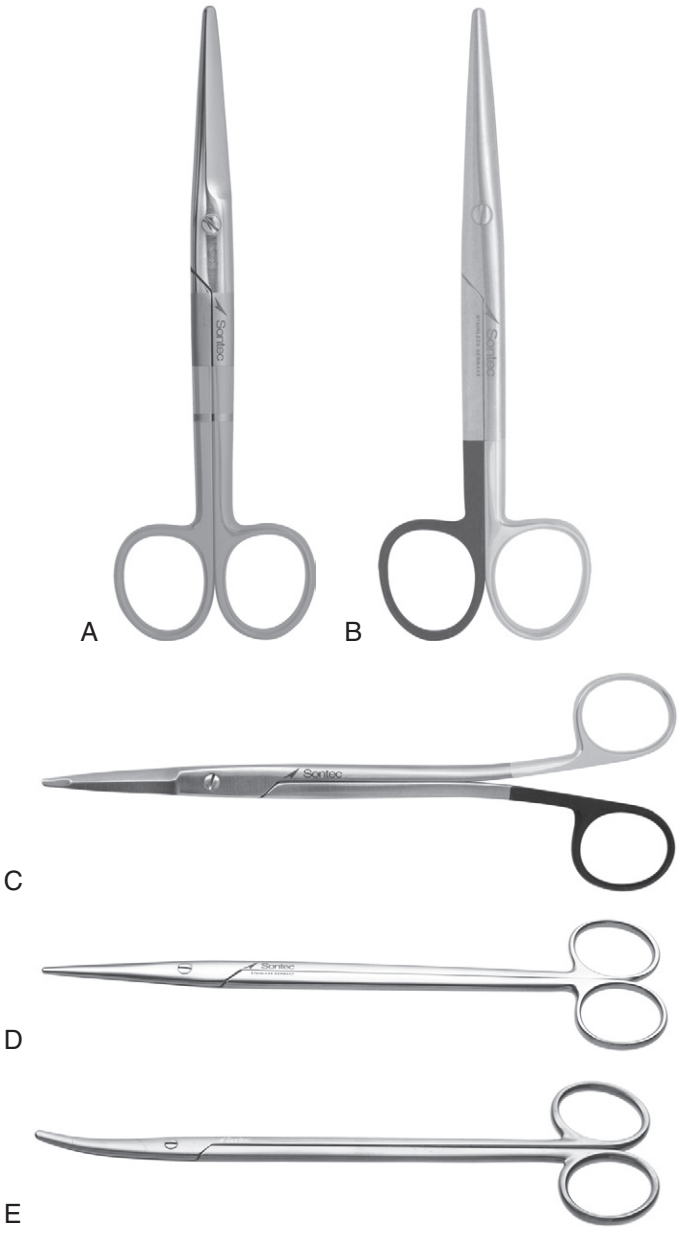

Figure 11-5. Scissors. **A,** Mayo scissors, power-cut, straight. **B,** Mayo Stille scissors, tungsten carbide, power-cut, straight. **C,** Freeman-Kay scissors, TC with ergonomic spread handle. **D,** Metzenbaum scissors, classic model, long style straight. **E,** Metzenbaum scissors, titanium nitrate coated. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

instrument while passing a needle through resistant tissue, which restricts its use somewhat. The efficient use of this needle holder requires practice.

The needle holder is the instrument that receives the most use and, through its constant metal-on-metal action, the most wear. It is advisable to purchase good-quality needle holders with tungsten carbide (TC) inserts that facilitate needle grip and instrument durability. The inserts lack a longitudinal groove and are designed with pyramidal teeth to provide a nonslip grip on needles. Instrument life can be prolonged by choosing the appropriate needle for the size of the needle holder. The lock box will be damaged if the instrument is used

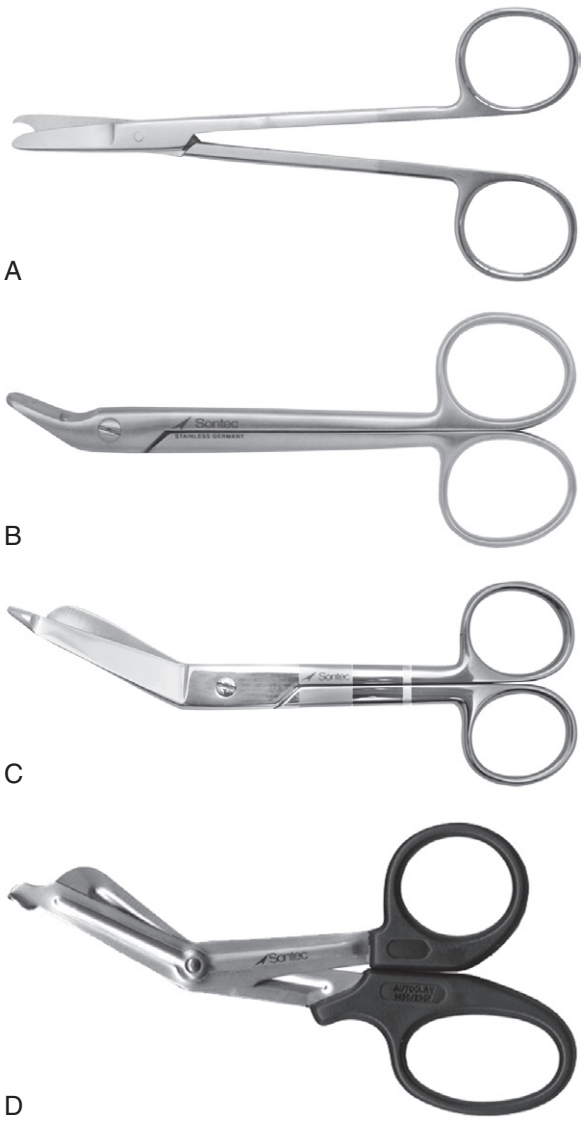

Figure 11-6. Specialty scissors. **A,** Littauer stitch scissors. **B,** Wire suture scissors tungsten carbide serrated. **C,** Lister bandage scissors. **D,** Utility scissors. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

to grasp too large a needle. Repair or replacement is necessary if the needle can be rotated by hand when the instrument is locked at the second ratchet position. New needle holders will hold an appropriately sized needle securely when locked in the first ratchet tooth.[1]

Forceps and Clamps

Forceps and clamps are available in many designs, each intended to perform specific functions or tissue manipulations. They range from simple thumb forceps to instruments containing various hinge configurations and ratchet locks. The selection of appropriate forceps for inclusion in surgical packs can greatly facilitate some maneuvers. Improper use can compound tissue trauma during surgery, increasing inflammation and delaying healing. Also, improper use may alter the shape of the jaws, rendering them useless for the intended application.

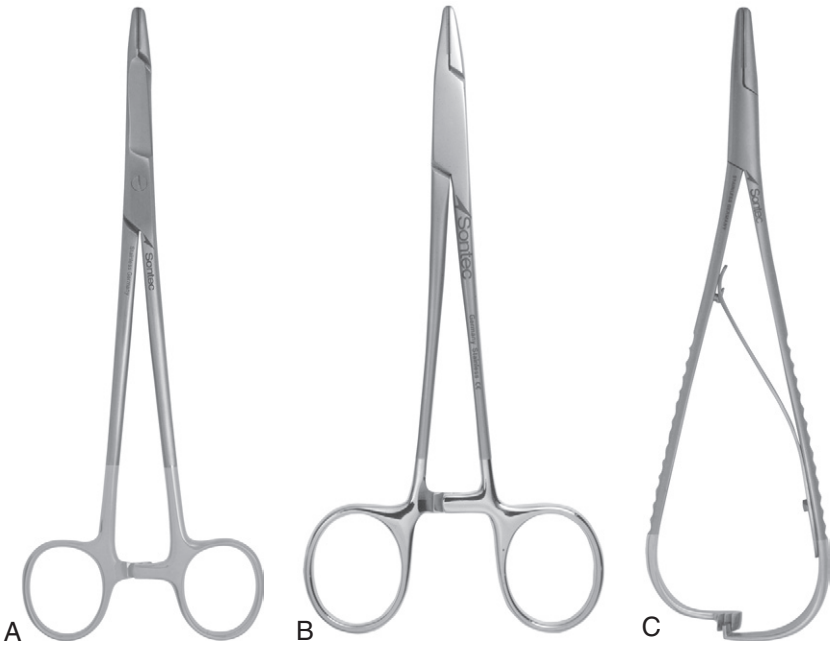

Figure 11-7. Needle holders. **A,** Olsen-Hegar tungsten carbide (TC) serrated. **B,** Mayo-Hegar NH TC inserts. **C,** Mathieu TC serrated straight. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

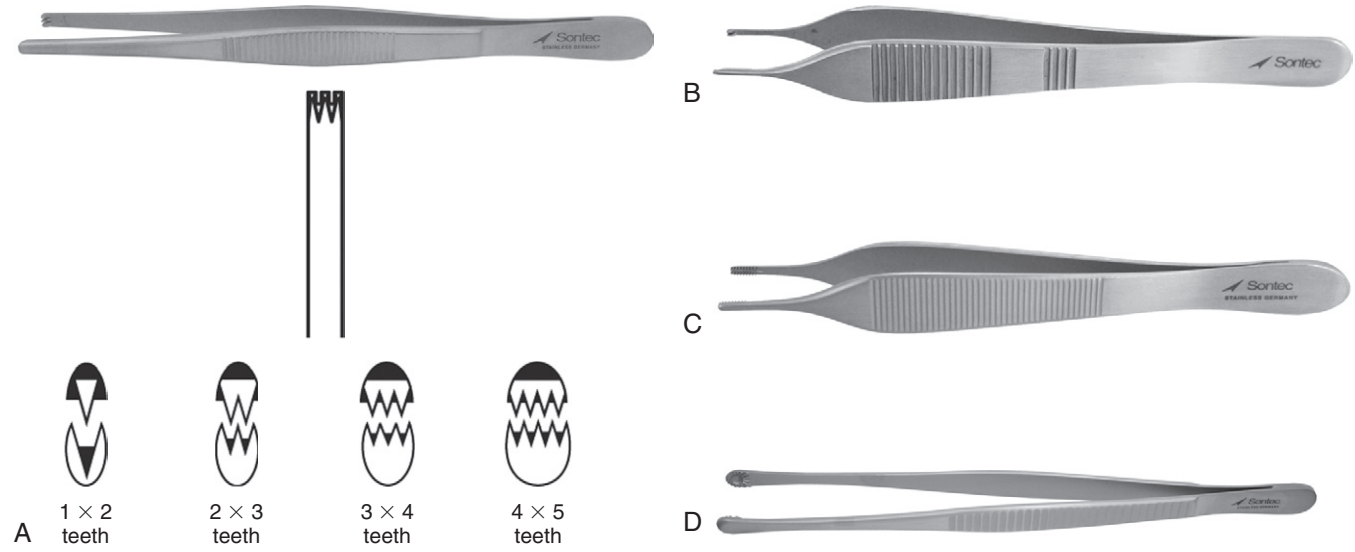

Figure 11-8. Thumb forceps. **A,** Tissue forceps with teeth, cross section details. **B,** Adson tissue forceps cross-serrated platform. **C,** Brown-Adson tissue forceps. **D,** Russian tissue forceps. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

Thumb Forceps

Thumb forceps (Figure 11-8) are designed to grasp and hold tissues and small objects, such as suture needles, and thus they serve as an extension of the surgeon's fingers. They consist of two blades attached at the proximal end, and the tips come together to hold tissue as finger pressure is applied on the blades. The outer surfaces of the blades are grooved to increase digital purchase. Thumb forceps are distinguished by the configuration of the tips. Forceps with smooth tips (without grooves or teeth) crush tissues because a considerable amount of force is necessary to gain purchase on the tissues. These smooth-tipped forceps are called traumatic (or anatomic) thumb forceps and should not be used for surgery.

A variety of serrated and toothed (or surgical) thumb forceps are available. The serrations and teeth allow a secure hold on tissues with minimal digital crushing pressure. The most aggressive of the thumb forceps is the rat tooth or tissue forceps (see Figure 11-8, *A*), which is available with 1-to-2 to 4-to-5 (see Figure 11-8, *A*) interlocking tooth patterns and comes in lengths between 11.5 and 30 cm (4½ and 12 inches). They are used primarily for manipulating skin and tough connective tissue. The Adson forceps has a 1-to-2 toothed tip but affords precise control of instrument pressure (see Figure 11-8, *B*). The Adson forceps is used to grasp thin skin and light fascial planes. The Brown-Adson forceps has two longitudinal rows of small, fine, intermeshing teeth (see Figure 11-8, *C*). The tooth configuration provides a broad but delicate tissue grip and facilitates grasping of the suture needle. The Russian forceps, which is not so frequently used, is very sturdy (see Figure 11-8, *D*). It has a broad, round tip with a grooved perimeter and a concave center. This

thumb forceps has a grip that is considered less traumatic than the Adson and Brown-Adson forceps, because pressure on the tissues is spread out over a larger area and it lacks teeth, making it less likely to tear or puncture tissue. The DeBakey (Figure 11-9) and Cooley forceps lack teeth but are still considered atraumatic forceps because of the serrations in the tips. These forceps are designed with longitudinal grooves and fine, horizontal striations that grip tissue without injury. They are considered ideal for vascular, thoracic, and intestinal surgeries. The DeBakey and Cooley serrated groove patterns are also available on hemostatic forceps.

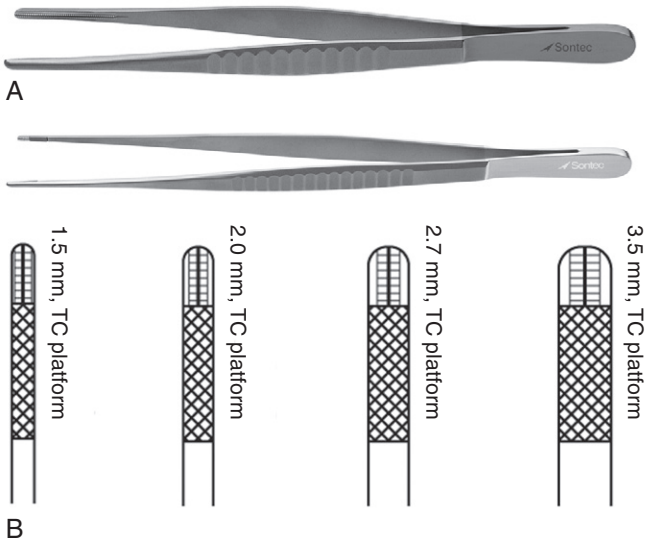

Figure 11-9. Specialty forceps. **A,** DeBakey tissue forceps flat handle. **B,** DeBakey needle pulling forceps with tip illustrations. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

Hemostatic Forceps

Hemostatic forceps are crushing instruments, designed to collapse vessels until hemostasis occurs or until electrocoagulation or ligation is accomplished (Figure 11-10). Most of these forceps have transverse grooves on the inner jaw surface that increase tissue purchase. The Halstead mosquito forceps (see Figure 11-10, *A*) are the smallest and most frequently used of these. They are available in standard and delicate configurations,

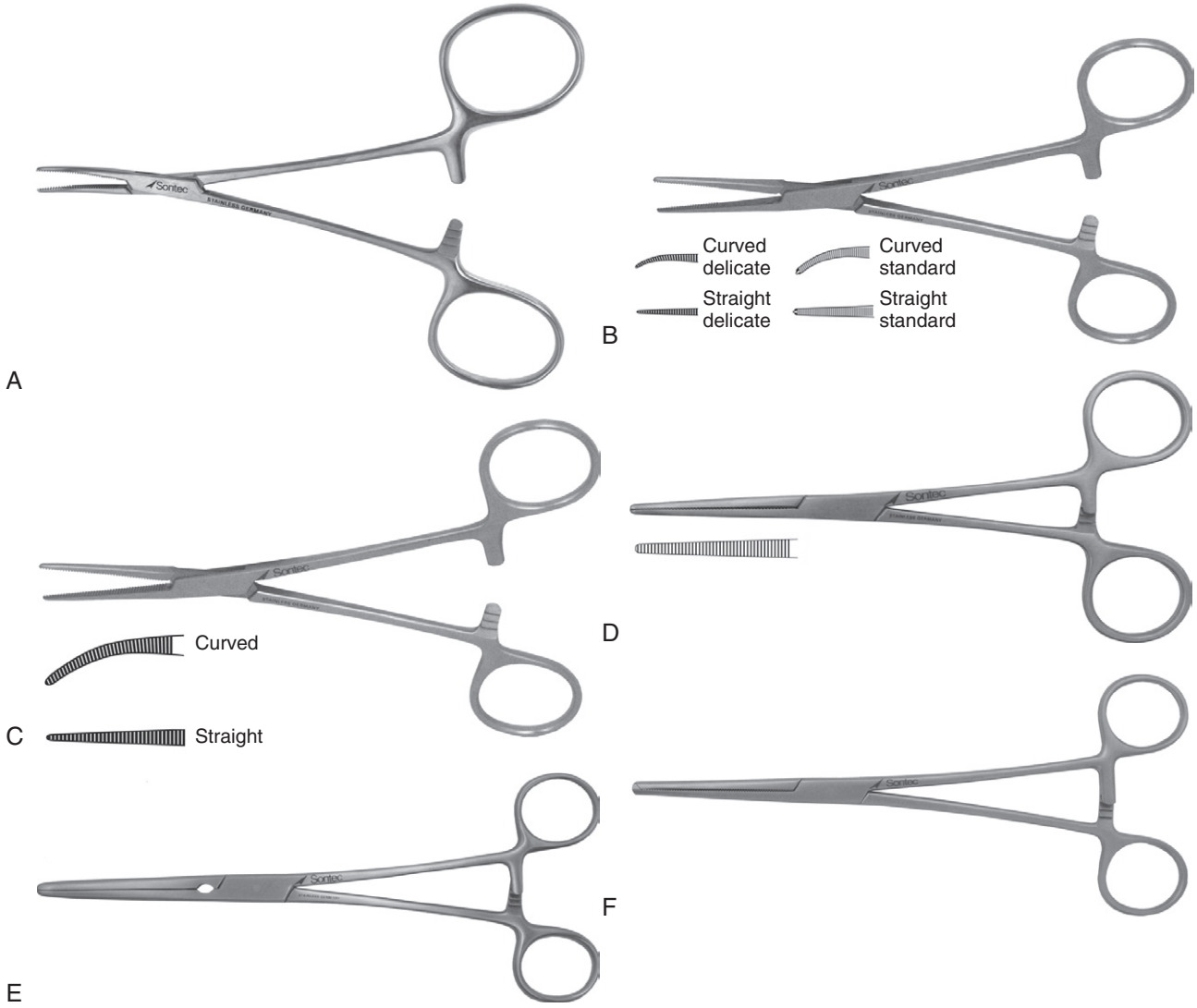

Figure 11-10. Hemostatic forceps. **A,** Halstead mosquito standard. **B,** Kelly straight with the details. **C,** Crile, curved with details. **D,** Rochester-Pean with details. **E,** Rochester-Carmalt. **F,** Rochester-Ochsner. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

as well as in 9- and 12.5-cm (3½- and 5-inch) lengths, with thin or standard width, curved or straight jaws. They should be used only on small vessels. The Kelly and Crile forceps (see Figure 11-10, *B* and *C*) are sturdier hemostatic forceps. These instruments are available in a standard 14-cm (5½-inch) length, with curved or straight jaws. The two differ in that the transverse grooves are restricted to the distal half of the jaw on the Kelly forceps, whereas the entire surface is grooved on the Crile forceps. Both are used for manipulating larger vessels.

To clamp large tissue bundles and vessels, Rochester-Pean forceps (see Figure 11-10, *D*) are recommended. They have deep transverse grooves over the entire jaw surface, are available in 14- to 30-cm (5½- to 12-inch) lengths, and come with straight or curved jaws. Rochester-Carmalt forceps (see Figure 11-10, *E*) are made to assist in pedicle ligation. Their jaw grooves run longitudinally with a few horizontal cross-striations at the tips. The groove design facilitates removal during ligation. Rochester-Ochsner forceps (see Figure 11-10, *F*), available in 16- to 25-cm (6¼- to 10-inch) lengths and with curved or straight jaws, have transverse grooves and 1-to-2 interdigitating teeth located at the jaw tip to help prevent tissue slippage. Rochester-Ochsner forceps are considered traumatic and should be reserved for use on tissue that is to be removed.

Tissue Forceps

Tissue forceps (Figure 11-11) are available in many shapes and sizes, and for a variety of uses. Doyen-DeBakey intestinal forceps, when properly used, are the least traumatic to tissue (see Figure 11-11, *A*). They are manufactured with slightly bowed, flexible jaws with longitudinal serrations. The longitudinal serrations allow easy removal from the intestine. The instrument is available in 13- to 33-cm (5- to 13-inch) lengths with straight or curved jaws, and it can be obtained with a wing nut to secure the tips in a clamping position, which is especially useful for longer forceps. The tips of the jaws should just meet when the ratchet's first tooth is engaged. The instrument will traumatize tissue if the ratchet is closed too tightly.

Allis tissue forceps vary in length between 14 and 25 cm (6½ and 10 inches) and form 4 × 5 to 5 × 6 teeth at the tip (see Figure 11-11, *B*). Designed to grip tissue, the teeth are oriented perpendicular to the direction of pull. The teeth can be traumatic, especially when excessive compression is applied to the handles, and this forceps should be used only on heavy tissue planes or on tissue that is to be excised.

Babcock intestinal forceps, like the Allis tissue forceps, pull in a direction that is perpendicular to the tissue, but the Babcock forceps are considered less traumatic (see Figure 11-11, *C*). The instrument is available in lengths from 16 to 30 cm (6¼ to 12 inches) and has tip configurations that vary from standard to micro tip, to closed jaws, to TC.

Sponge forceps are used to grab sponges and clean or swab specific tissues or cavities (see Figure 11-11, *D*). They are available as straight or curved instruments of 18- to 24-cm length (7 to 9½ inches) with serrated or smooth fenestrated, oval tips.

Hemostatic and tissue forceps should regularly be inspected for instrument wear and damage. When the instrument is closed, the jaws should align perfectly and the teeth, if present, should interdigitate. When clamped on tissue, the instrument should not spring open.

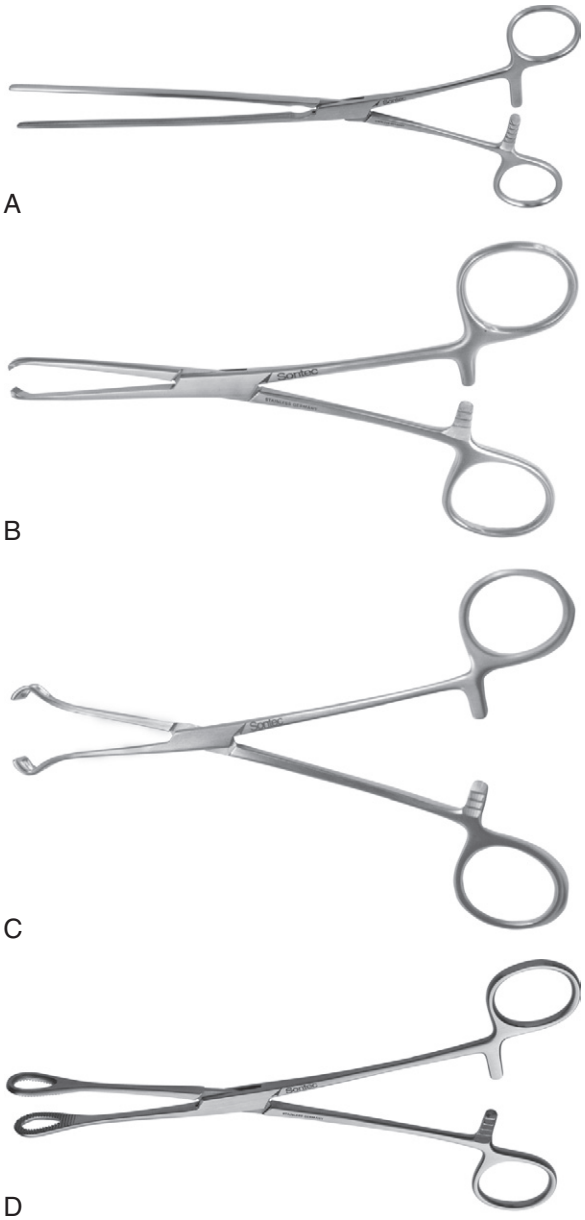

Figure 11-11. Tissue forceps. **A,** Doyen-DeBakey intestinal forceps straight. **B,** Allis tissue forceps. **C,** Babcock tissue forceps. **D,** Foerster sponge forceps, serrated straight. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

Clamps

Satinsky clamps have atraumatic longitudinally grooved jaws that contain two bends. They vary in length from 17 to 28 cm (6¾ to 11 inches) (Figure 11-12). This type of clamp is mainly used for vascular surgery, because it provides a good view of the vessel held in the clamp.

Retractors

Soft tissue retractors are designed to spread the wound edges to facilitate exposure of the surgical field. A classification used by many manufacturers includes the finger-held, the hand-held, and the self-retaining retractors. All three types require an adequate length of incision to prevent tissue tearing when retraction is used. The finger-held and hand-held retractors require a surgical assistant.

Finger-Held Retractors

Senn, Volkman, Meyerding, Farabeuf, and Parker retractors are typical representatives of this group (Figure 11-13). The Senn retractor (see Figure 11-13, *A*) is available with either blunt or sharp retractor prongs at one end and a right-angled fingerplate on the other. It is used to retract skin and superficial muscle layers, but is less useful for retracting a large muscle mass. The Volkman finger retractor (see Figure 11-13, *B*) is available with sharp or blunt retractor prongs and a single-ring handle. The Parker retractor (see Figure 11-13, *C*) are larger, with deeper, flat blades on both ends that allow the retraction of more tissue.

Figure 11-12. Satinsky clamp.

Hand-Held Retractors

Common hand-held retractors are the Army-Navy, Hohmann, Kelly, and Meyerding retractors (Figure 11-14). Army-Navy retractors are available in a standard 21.5-cm (8½-inch) length (see Figure 11-14, *A*). They have double-ended retracting blades of two different lengths, which allow the surgeon to select a blade according to tissue depth. Hohmann retractors are available in 16.5- to 24.5-cm (6¼- to 9¾-inch) lengths, and with blade widths from 6 to 70 mm (see Figure 11-14, *B*). The blade has a blunt projection that is useful in exposing bone while retracting the muscle in orthopedic and reconstructive procedures. The Kelly retractor (see Figure 13-14, *C*) has a loop handle and broad blade that projects at a right angle relative to the long axis of the instrument with a rounded, bent-down tip.

Figure 11-13. Finger-held retractors. **A,** Senn retractor. **B,** Volkmann retractor. **C,** Parker retractor. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

Figure 11-14. Hand-held retractors. **A,** Army-Navy retractors. **B,** Hohman retractor with an 18-mm blade. **C,** Kelly retractor. **D,** Meyerding retractor. **E,** Lahey retractor. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

Meyerding retractors are available with three different blade widths and depths (see Figure 11-14, *D*). The largest blade is 9 cm (3½ inches) wide and 5 cm (2 inches) deep. The Lahey retractor (see Fig 11-14, *E*) has a smooth handle and a rectangular narrow blade that provides good tissue visibility.

Self-Retaining Retractors

The Gelpi, Weitlaner, Balfour, and Finochietto retractors (Figure 11-15) are representatives of the available self-retaining retractors. The Gelpi retractor has a grip-lock mechanism that maintains tension on its two outwardly pointed tips (see Figure 11-15, *A*). The instrument is available in sizes ranging from the 9-cm (3½-inch) pediatric size to the 20-cm (8-inch) standard size. The larger version is available with ball stops to prevent excess tissue penetration. There are two other variations on this retractor: a sturdy retractor for more robust tissues, and a deep angled version, which has longer shanks from the point of the angle to the tip.

Weitlaner retractors range in size from 10 to 24 cm (4 to 9½ inches) and are available with 2-to-3 or 3-to-4 outwardly pointed blunt or sharp teeth (see Figure 11-15, *B*). A hinged Weitlander retractor (see Figure 11-15, *C*) is also available in sizes between 14 and 20.5 cm (5½ and 8 inches) containing

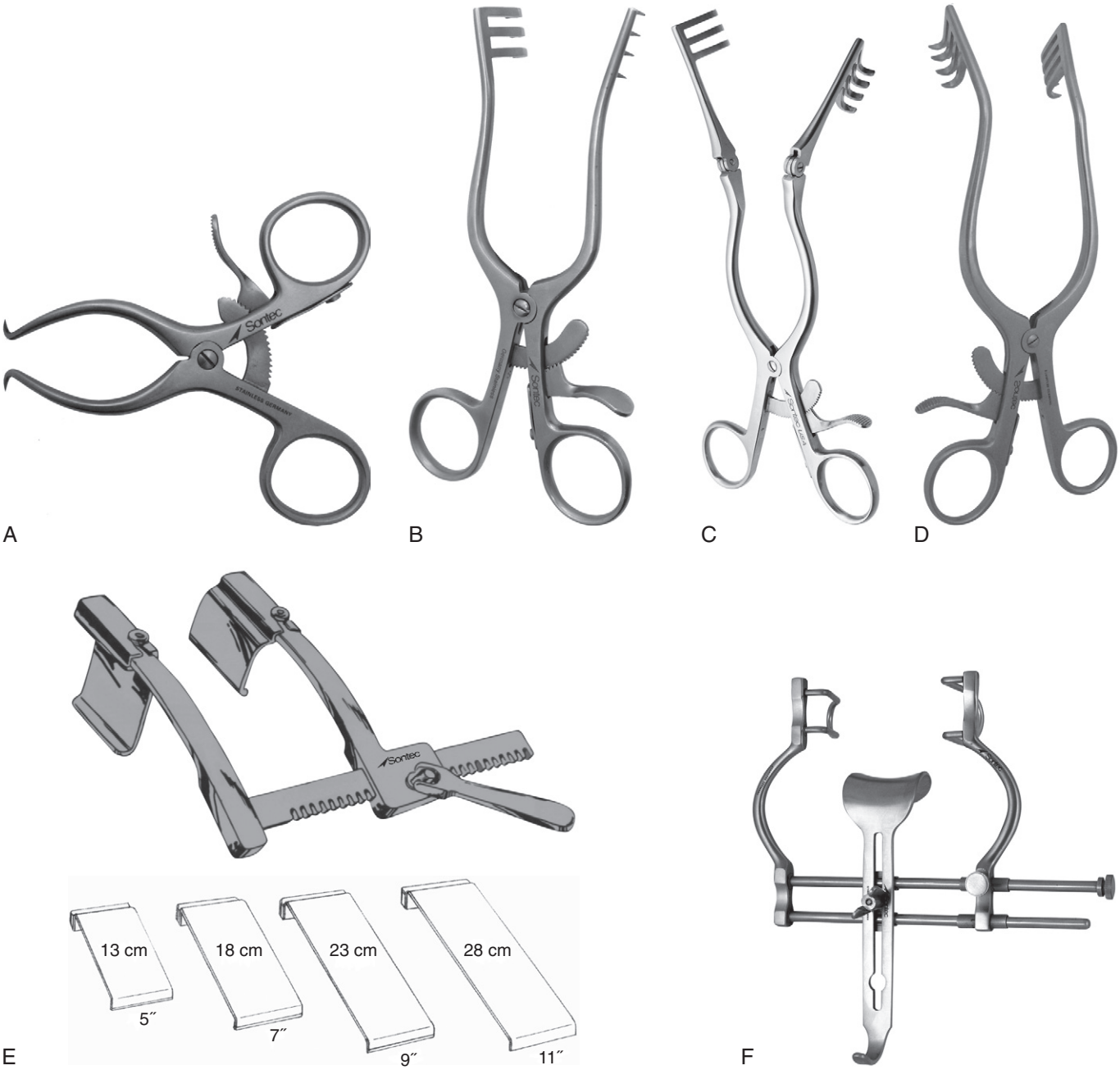

Figure 11-15. Self-retaining retractors. **A,** Gelpi retractor. **B,** Weitlaner retractor. **C,** Weitlaner retractor with hinged blades. **D,** Adson Cerebellar retractor. **E,** Aanes retractor/speculum with the different blades. **F,** Balfour retractor. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

3 × 4 blunt or sharp prongs that allows placement of the prongs deep in the tissues. The Adson Cerebellar retractor (see Figure 11-15, *D*) has 4 × 4 sharp prongs that has either straight or angled arms, the latter, again, to facilitate seating the instrument deep in the incision. The Aanes retractor/speculum (see Figure 11-15, *E*) is a modification of the Finochietto retractor with the benefit that the blades can be exchanged for deeper exposure. The blades range from 13 to 28 cm (5 to 11 inches). The Balfour self-retaining abdominal retractor is available in 10- to 36-cm (4- to 14-inch) spreads and with 6.5- to 10-cm (2½- to 4-inch) deep, solid, and fenestrated side blades (see Figure 11-15, *F*). These retractors are distinguished as pediatric, adoloscent, and elite retractors and are used to allow vision into the depth of a body cavity, such as the abdomen.

Towel Clamps

Several types of towel clamps are available (Figure 11-16). Backhaus towel clamps are the most commonly used (see Figure 11-16, *A*) and are available in 9- and 13-cm (3½- and 5¼-inch) sizes. The Jones towel clamps (see Figure 11-16, *B*) are spring-loaded and available in smaller sizes as 6- and 9-cm (2½- and 3½-inch) instruments. The Lorna-Edna towel clamps are non-penetrating and therefore ideal for securing suction lines and cables to drapes (see Figure 11-16, *C*). These towel clamps are available as 10- and 14-cm (4- and 5½-inches) sizes. Penetrating towel clamp tips should meet when closed, and they should be sharp and free of burrs.

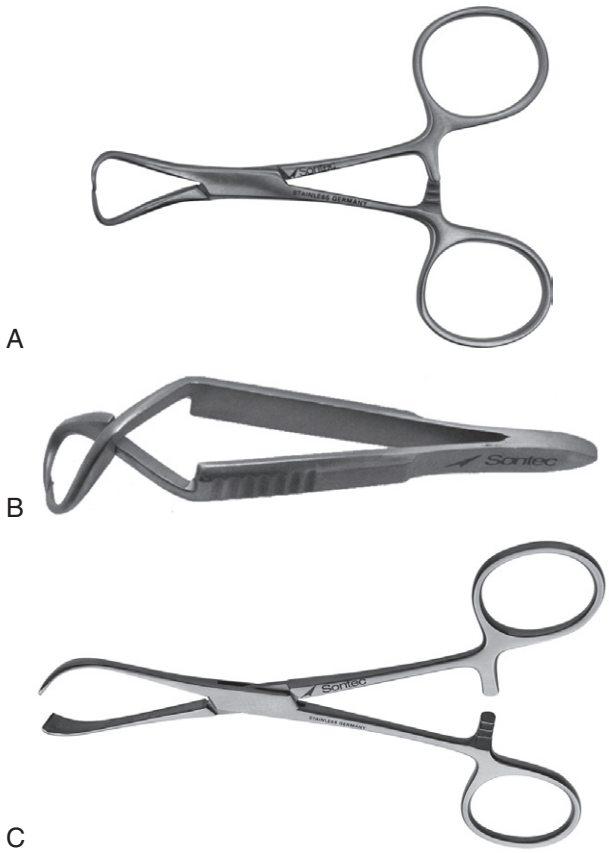

Figure 11-16. Towel clamps. **A,** Backhaus towel clamps. **B,** Jones towel clamps. **C,** Lorna-Edna towel clamps. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

Suction Tubes

There are three basic types of suction tubes available (Figure 11-17). The Yankauer tip is relatively large, allowing the removal of large volumes of blood or fluid from the surgical site (see Figure 11-17, *A*). The Frazier-Ferguson suction tube is available with a curved or straight tube (see Figure 11-17, *B* and *C*). It has diameters ranging from 4- to 15-French. The suction intensity of these tubes can be varied by placing the index finger over the hole on the handle. Both models are available in stainless steel and in disposable plastic. The Poole suction tube has multiple ports along the tube, making it ideal for use within the abdomen, where single-orifice tubes are easily plugged by omentum (see Figure 11-17, *D*).

ORTHOPEDIC INSTRUMENTS

A wide variety of instruments are available for orthopedic surgery. Those presented here are used outside the realm of fracture repair. For information regarding instruments used for reconstruction and fracture treatment, the reader is referred to Chapter 76.

Rongeurs

Rongeurs have opposed cupped cutting jaws that allow precise removal of bone, cartilage, and fibrous tissue (Figure 11-18). Most contain either a single- or a double-action mechanism and curved or straight jaws. The double-action rongeurs are stronger and have a smoother action. Ruskin rongeurs (see Figure 11-18, *A*) are available with 2-, 3-, 4-, 5-, or 6-mm wide jaws in straight, slightly curved, curved, and full curve shapes

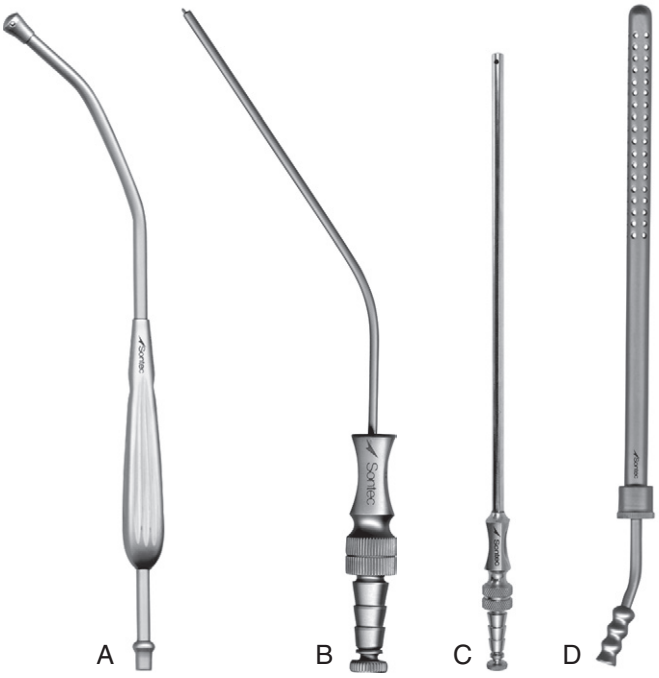

Figure 11-17. Suction tubes. **A,** Yankhauer suction tube. **B,** Frazier suction tube, angled. **C,** Frazier suction tube, straight. **D,** Poole suction tube. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

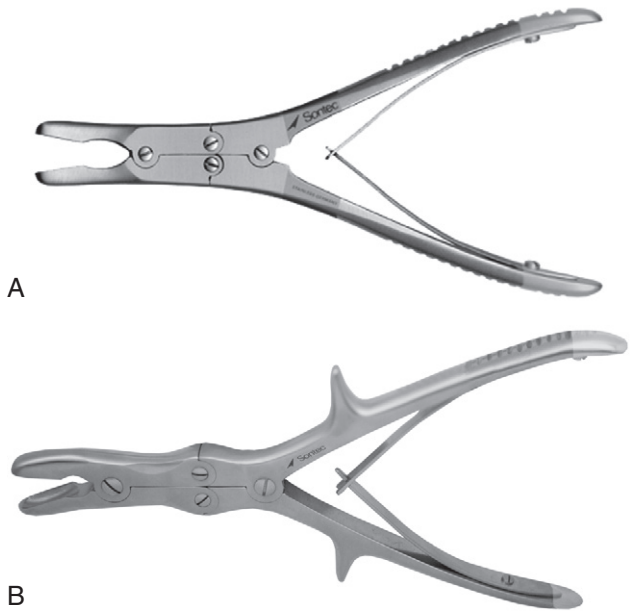

Figure 11-18. A, Ruskin rongeur. **B,** Stille-Luer duckbill rongeur. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

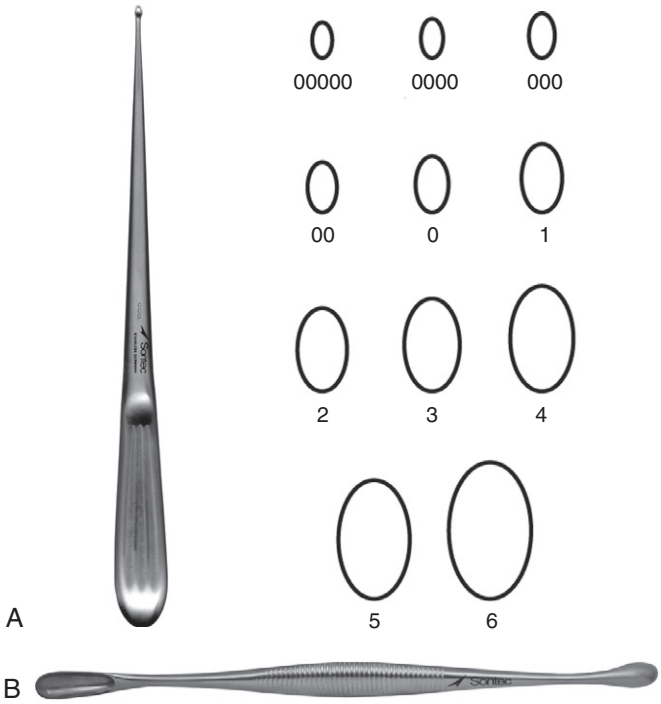

Figure 11-19. Curettes. **A,** Burns curette with details of cup sizes 00000 to 6. **B,** Volkman curette. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

and are 15.5, 18.5, and 23 cm (6, 7¼, 9 inches) long. They are available in black as well. The slightly larger Stille-Luer Duckbill rongeurs (see Figure 11-18, *B*) are available with straight or curved jaws in width-length combinations of 6 × 13, 6 × 15, and 8 × 18 mm.

Curettes

Curettes are easily recognized by their cuplike structure (Figure 11-19). The sharp, oval, or round edges are useful for removing diseased bone, cartilage, debris, and damaged tissue from dense tissue surfaces. Their shape also makes them ideal for harvesting cancellous bone grafts. A wide variety of sizes and types of curets are available. The Burns curettes (see Figure 11-19, *A*) have a straight or angled single oval cup at the end of a grooved handle, whereas the Volkman curettes (see Figure 11-19, *B*) are double-ended, having an oval cup on one end and an oval or round cup on the other.

Periosteal Elevators

As their name suggests, periosteal elevators are designed to elevate periosteum and muscle attachments away from bone. Common elevators include the single-ended Adson, McIlwraith, and Foerner elevators and the double-ended Freer elevators (Figure 11-20). The Adson elevator (see Figure 11-20, *A*) is available with either a blunt or sharp, and a straight, curved, or full curve tip. The McIlwraith elevator (see Figure 11-20, *B*) has only a sharp tip, and the Foerner knife elevator (see Figure 11-20, *C*) is the sharpest of all, designed to free the attachment of the interosseus ligament from the proximal sesamoid bone. The double-ended Freer elevators (see Figure 11-20, *D*) are narrow and have one end that is blunt and one that is sharp.

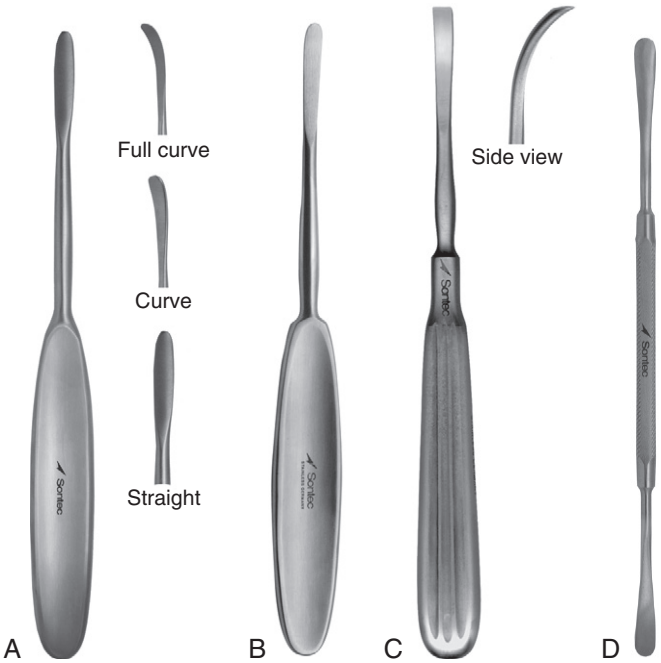

Figure 11-20. Periosteal elevators. **A,** Adson periosteal elevator with details. **B,** McIlwraith periosteal elevator. **C,** Foerner knife elevator. **D,** Freer periosteal elevator. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

Bone-Cutting Instruments

Osteotomes, chisels, and gouges are all hand-held instruments that are used in combination with a mallet (Figure 11-21). Osteotomes (see Figure 11-21, *A*) are double-beveled at their cutting tip, and chisels are single beveled. The cutting widths vary from 2 to 38 mm (1 to 15 inches). The chisel (see Figure 11-21, *B*) tends to move away from the beveled edge. Therefore it needs to be applied at a somewhat steeper angle relative to its axis. This allows the chisel to move along the bone surface on its beveled edge. If the chisel is reversed, it tends to dive into the bone, leaving sharp edges on the surface. The chisel is the preferred instrument to remove exostoses, but when the direction of bone cutting needs to be more precise, it is better controlled with an osteotome. Common types for these three cutting instruments are Army-Navy, Hibbs, and Smith-Peterson. Gouges (see Figure 11-21, *C*) are easily distinguished by their concave shape. They are available in 4- to 30-mm (1½- to 12-inch) widths.

The mallet can be solid stainless steel or have an aluminum handle and a stainless steel head. Polyethylene-capped stainless steel heads are quieter and prevent the production of metal particle flakes during striking. There is a mallet available with a stainless steel head on one side that can be exchanged for a nylon head (see Figure 11-21, *D*).

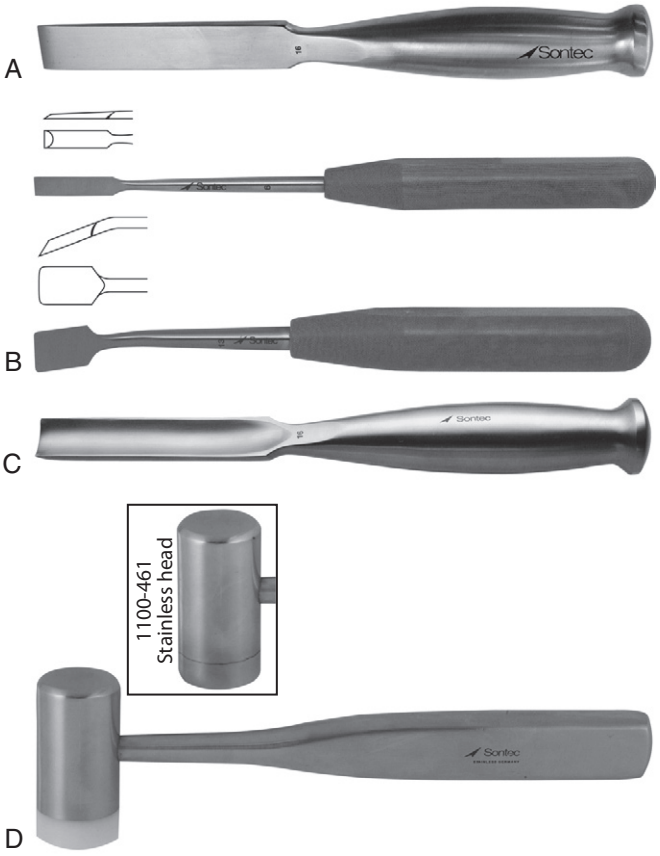

Figure 11-21. Bone cutting instruments. **A,** Smith Peterson osteotome. **B,** Chisel, also called elevator/raspatory; straight *(top);* curved *(bottom).* **C,** Smith Peterson gouge. **D,** Sontec bone mallet with removable stainless steel and nylon head. (Reprinted with permission from Sontec Instruments, Inc. Centennial, CO. 2010.)

Bone-cutting forceps can be single- or double-action and straight or angled. The Liston bone-cutting forceps (Figure 11-22, *A*) are representatives of single-action, and Ruskin-Liston (see Figure 11-22, *B*) and Stille-Liston are double-action bone-cutting forceps.

Bone Clamps

Bone clamps or bone-holding forceps come in a variety of shapes and sizes and are used for fracture reduction. Verbrugge, Kern, Stefan bone clamps are typical representatives thereof (Figure 11-23). The Verbrugge bone-holding forceps is curved to the side with one arm longer than the other, contains a speedlock, and is available in sizes from 15 to 29 cm (5¾ to 11¼ inches). Modifications of this forceps are a swivel jaw (see Figure 11-23, *A*) and a reverse jaw configuration that is more suitable in specific situations. The Kern bone-holding clamp has symmetric, straight, strong jaws and a ratchet at the end to maintain the bone-holding force (see Figure 11-23, *B*). It comes in sizes between 12 and 33 cm (4¾ and 13 inches) and is well suited for equine long bone fracture reduction. The Stefan bone-holding forceps comes in sizes between 15.5 and 24 cm (6 and 9½ inches) and contains a speedlock. The jaws are rounded and sturdy (see Figure 11-23, *C*). The bone-reduction clamp has two pointed and thin jaws (see Figure 11-23, *D*). It comes with either a speedlock or a ratchet; an extra-long ratchet is also available. This is the most frequently used bone clamp.

Cerclage Wire Instruments

Instruments used for application of cerclage wires include flat-nosed pliers, pointed pliers, and wire twisters (Figure 11-24). A universal flat-nosed plier/wire cutter is shown in Figure 11-24, *A*. The wire cutter is mounted on one side and cuts wires to

Figure 11-22. Bone-cutting forceps. **A,** Liston bone-cutting forceps. **B,** Ruskin-Liston bone-cutting forceps. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

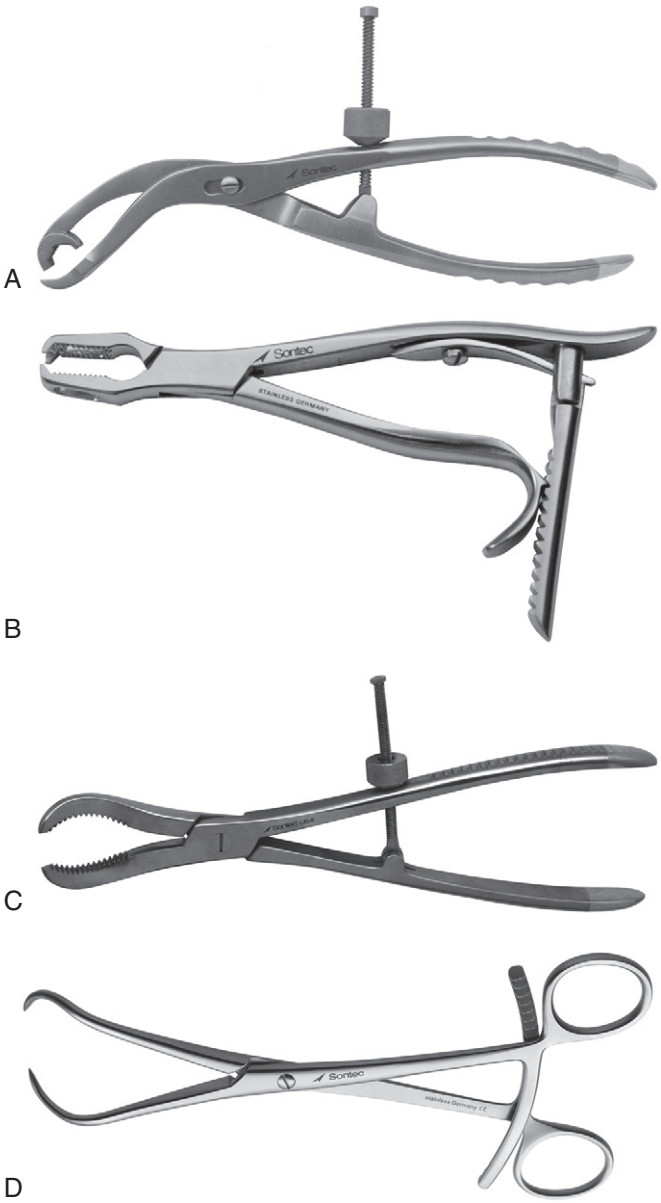

Figure 11-23. Bone clamps. **A,** Verbrugge swivel jaw bone clamp. **B,** Kern bone clamp. **C,** Stefan bone-holding forceps. **D,** Bone-reduction clamp with extra-long ratchet. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

1.6 mm (⅔ inch). The universal pliers are pointed and allow excellent maneuvering of the wire in many different situations (see Figure 11-24, *B*). The Axel wire twister (see Figure 11-24, *C*) feeds each wire through a hole on the side of the blade and fixes the wires by closing the ratchet at the end. The instrument is subsequently pulled axially away from the bone while twisting the instrument evenly around its axis. This action twists the wire ends around each other. The same can be achieved by grabbing the wire ends with the flat-nosed pliers.

Pin Insertion and Pin-Cutting Instruments

Pins are not frequently applied in horses, but occasionally the need arises. Aside from a drill, the Jacobs chuck is the most

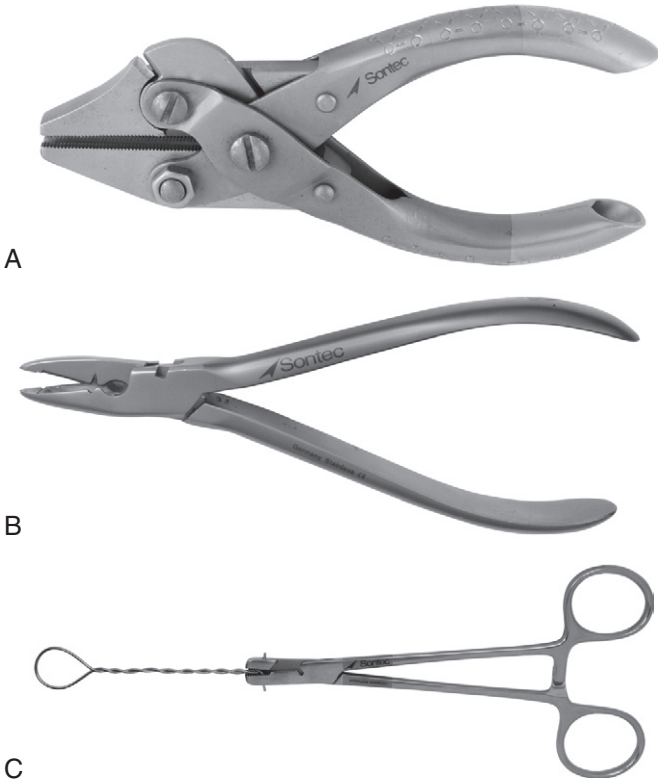

Figure 11-24. Cerclage instruments. **A,** Pin puller/side cutter. **B,** Waldsachs universal pliers. **C,** Axel wire twister. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

versatile instrument for such an occasion (Figure 11-25, A). It accepts pins up to a diameter of 0.6 mm (¼ inch). An extension can be applied to its back end to protect the surgeon from the sharp pin end protruding behind the end of the chuck. The small pins can be cut with a sturdy wire cutter (see Figure 11-25, *B*). Larger pins must be cut with a pin cutter (see Figure 11-25, *C*).

Trephines

Two types of trephines are available, Galt and Michele (Figure 11-26). Both are T-shaped and capable of drilling a cylinder of bone. The Galt trephine (see Figure 11-26, A) can cut bone at the end of the shaft and along the outside perimeter of the shaft. It is available in graduated sizes from 1.25 to 2.5 cm (½ to 1 inch) in diameter and has an adjustable central trocar. The trocar centers the trephine and stabilizes it until a circular trough is cut in the bone. The Michele trephine is available in graduated inner diameters of 0.6 to 3.1 cm (¼ to 1¼ inch). It contains a graduated scale along its shaft, allowing the penetration depth to be measured. It cuts through bone on the end of the shaft only. The plug cutter trephine is similar to the Michelle trephine but has a saw blade–like front rim that is better suited for equine bone (see Figure 11-26, *B*). This trephine is available with diameters ranging from 3 mm inside/5 mm outside up to 22 mm inside/25 mm outside.

MICROSURGICAL INSTRUMENTS

At present, reconstructive vascular and neural surgeries are rarely performed in equine patients. The exceptions are

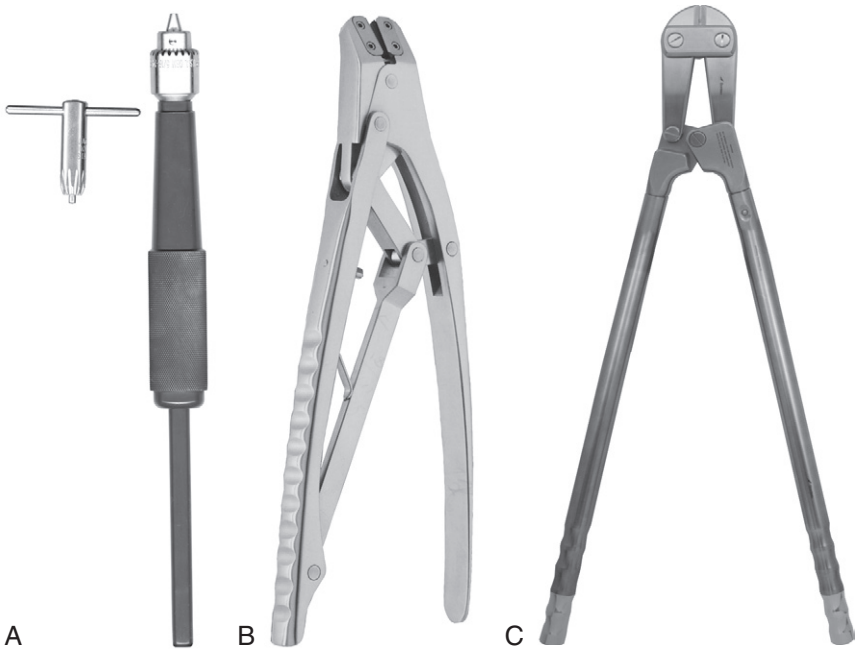

Figure 11-25. Pin insertion and cutting instruments. **A,** Jacobs pin chuck. **B,** Big gold-cut Hercules wire cutter. **C,** Pin cutter. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

thrombectomies, which may be performed with the help of catheters (see Chapter 13). Because horses are rarely used as research animals, microsurgical techniques play a minor role in this species. The microsurgical instruments used for ocular surgery can be found in Chapters 55 and 57.

INSTRUMENT MAINTENANCE

Proper care maintains long-term instrument serviceability. Instruments should be cleaned immediately after use. Sharp and delicate instruments should be separated from other instruments that may damage them. When washing them by hand, it is best to use warm water, a neutral pH detergent, and a soft bristle brush. Ultrasonic cleaners are more effective than hand washing; however, the manufacturer's recommendations for type of water, such as deionized or distilled, and detergent used should be followed (see "Cleaning" in Chapter 9). If cleaning cannot be done immediately, instruments should be submerged, in the open position, in a solution of water and neutral pH detergent. Hard water, saline solution, and nonneutral pH detergents (dishwashing liquids) should be avoided, because surface discoloration, corrosion, and poor mechanics of the joints may result.[7] Once cleaned, instruments should be rinsed with deionized or distilled water. Instruments with a working action should then be treated with an instrument lubricant (instrument milk). The lubricant, which often includes a rust inhibitor, should not be rinsed off. Instruments are then dried and stored or resterilized.

A German group called Arbeitskreis Instrumentenaufbereitung (Working Circle Instruments Reprocessing) offers on its website (www.a-k-i.org) valuable information on the handling and use of surgical instruments. Several brochures can be downloaded and among those, *Green Brochure* discusses the handling of surgical instruments.

Proper care of the instruments also must include the use of high-quality cleaning products. It is wise to use top-quality products, such as those offered by the Ruhof Corporation and distributed for veterinarians in the United States by Sontec Instruments, Centenial, CO, because these products significantly extend the life of the surgical instruments.

Instrument refurbishing programs are available through most instrument manufacturers. In addition to resharpening cutting edges and replacing tungsten carbide inserts, instruments are cleaned, polished, and refinished to retard corrosion. Refurbishing generally costs less than replacement.

IDENTIFICATION

Instruments are frequently marked to identify their owner or the instrument set they belong to. Various identification methods are available. Commercially available engraving should be avoided, as should any other method that damages the surface of the instrument. Surface damage, with removal of the corrosion-resistant coating, will shorten instrument life. Electrochemical etching units are acceptable as long as they are properly used. After etching, the instrument must be thoroughly rinsed to neutralize the acid etching fluid.

Autoclavable plastic tapes for instrument identification are available in different colors and are easy to apply. Color coding with tape does not harm the instrument's surface. All instruments belonging to a specific set can be marked with the same color. This is helpful in large clinics, where different surgical teams work parallel to each other with different instrument sets. During cleaning and resterilization, instruments belonging to different sets may be mingled. The color coding allows easy and efficient separation. Poorly applied tape, however, may begin to peel off, creating crevices that could harbor debris and

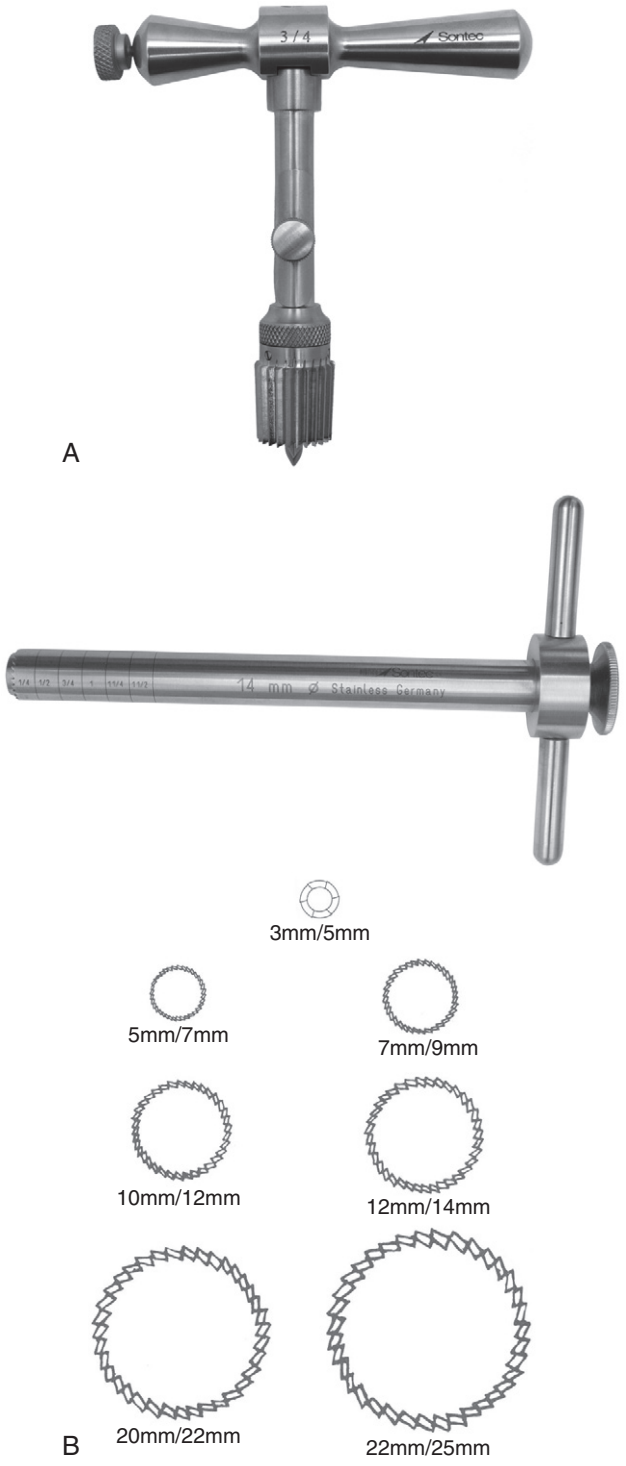

Figure 11-26. Trephines. **A,** Galt trephine. **B1,** Plug cutter with obturator. **B2,** Plug-cutter tips. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

microorganisms. Proper selection of a tape marking system should include considerations of color, durability, and adhesive properties to ensure a long life once applied to the instrument. Higher-quality marking systems are frequently marketed through instrument manufacturing companies.

PACK PREPARATION AND STORAGE

Tightly woven linen drapes have long been used to package instrument sets. Their disadvantages include short shelf life and the cost of laundering for reuse. Because microorganisms can fairly rapidly penetrate linen wrappers, it is prudent to double wrap the sets with linens. Safe storage times have been established[7] (see Table 10-1).

A variety of paper products have been developed to replace linen wrappers. Although these products share some of the disadvantages of linen, they offer longer safe storage times, because the sterilization process closes the pores within the sheet. As a result, these paper products cannot be reused and are therefore disposable, so laundry expenses are avoided. On the other hand, disposal costs and the burden on the environment through exhaust gases (e.g., CO_2) from incinerators rise. Many of the newer paper wrappers handle like linen.

Both paper and linen prevent visualization of the instruments within the pack. In the case of sets, this is not a problem because the contents are known. However, if instruments are separately wrapped, visualization is important. Therefore special wraps that consist of a sheet of paper on one side and a clear plastic sheet on the other have become popular. The plastic side allows the instrument to be seen inside, and the paper side allows steam or ethylene oxide to penetrate the package. Sharp points of instruments have to be covered by plastic or paper caps to prevent inadvertent damage to the paper layer. These wrappers are available in tube rolls in several sizes, and most of them contain sterilization indicators (see Chapter 10). The ends are heat sealed. Safe storage time is extended with this type of wrapping, but the paper side is still susceptible to microorganism penetration when wet. The best effect is achieved by double wrapping the instruments. Regardless of the type of wrapping chosen, the instruments should be loosely packed with the jaws slightly opened to allow circulation of steam, ethylene oxide, or gas plasma (Figure 11-27).[7] All instrument packs should be dated and labeled for easy identification, as well as for resterilization if they are not used within the safe storage time frame. For prolonged storage life, the packs may be placed within a plastic envelope or into a glass closet. It is equally important to have the initials of the person wrapping the set marked on the set or pack. This allows direct communications with this person should an instrument be missing during the surgery.

Lately, reusable metal sterilization containers enjoy renewed popularity, after having almost disappeared in the late 1980s

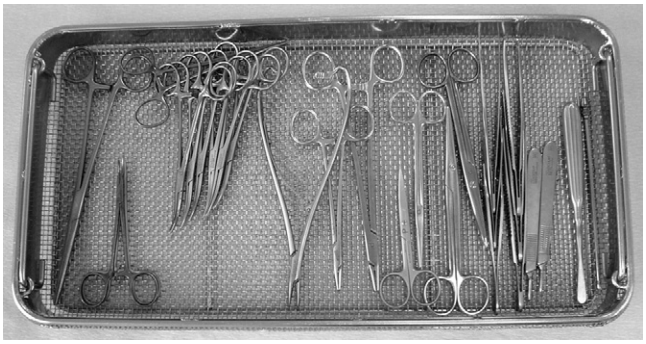

Figure 11-27. Example of a standard soft tissue set. The instruments are neatly arranged in a logical sequence.

(Steriset Containers, Wagner GmbH, Munich, Germany) (see Figure 9-1). These containers are used for holding surgical instrument sets or textiles during vacuum steam sterilization procedures and for maintaining sterility of the contents during storage and transport under hospital conditions. They operate with either filters or valves. The filter units are single-use filters or reusable textile filters with known service life spans. SteriSet valve containers have a closed base and permanent stainless steel pressure-sensitive valves in the inner lid. The sterilization valves react to the change in pressure during the sterilization process. During the vacuum phase, the valves open upward, and the air and steam mixture can escape from the container. During the pressurization phase, the valves open inward and allow steam to enter the container. The system is automatically flushed and sterilized by the hot steam rushing through the valve with every sterilization cycle. Outside the sterilizer (i.e., during storage or transport), the valve is closed and serves as a barrier to microorganisms.

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CHAPTER
12

Surgical Techniques

Jörg A. Auer

Surgery can be defined as goal-oriented violence to tissue, and therefore considerations related to minimizing tissue damage are an important part of adequate preoperative planning and proper surgical technique.[1] Since time in surgery is directly proportional to tissue damage, adequate preparation for each surgery is the best prevention of unnecessary delays that prolong surgery. Before embarking on an unfamiliar or complicated surgical task, the operator should plan the procedure step by step, from skin incision to closure. This chapter describes those aspects of surgical manipulations that are basic to the performance of any procedure—namely, the different techniques of incision, excision, and dissection of tissue; the methods of surgical hemostasis and tissue retraction and handling; and surgical irrigation and suction. Adherence to the basic principles of state-of-the-art surgical technique, described by Halsted, minimizes tissue trauma and blood loss and decreases the risk of wound dehiscence, resulting in a better overall surgical result.[2]

William Stewart Halsted (1852-1922) was one of the most influential human surgeons of his time. He taught at Johns Hopkins Hospital in Baltimore and was the first one to list basic principles for aseptic surgery. These priniciples became known as "Halsted's Principles" and are as follows: (1) apply strict asepis during preparation and surgery, (2) assure good hemostasis to improve conditions for the procedure and limit infection, (3) avoid the formation of dead space, (4) minimize tissue trauma through careful handling thereof, (5) maintain blood supply, (6) avoid undue tension on tissues, and (7) carefully adapt the corresponding tissue layers.[1] Additional information on tissue handling will be discussed later in this chapter.

BASIC MANIPULATIONS OF SURGICAL INSTRUMENTS

Incising or cutting into tissue represents the initial step of every surgical intervention. The instruments used for this procedure and the manner in which they are applied provide the surgeon with the means to vary the type of incision and its effects on the surrounding tissue. The scalpel and scissors are the basic instruments for incising or excising tissues. Separation along tissue planes is usually accomplished through blunt or digital dissection. Electrosurgery and laser surgery complement the instruments used for incisions and excisions.

Scalpels

Steel Scalpel

The steel scalpel with disposable blades is the instrument most frequently used to incise skin and other soft tissues. It is prudent to apply the blade to the scalpel handle with the help of a needle holder or similar instrument to prevent inadvertent puncture of the surgery gloves or, even worse, cutting of the surgeon's fingers.

There are three ways to hold the blade handle: the pencil grip, the fingertip grip, and the palm grip.[3] With the pencil grip, very precise cuts can be performed. The distal end of the scalpel handle is grasped between the thumb and index finger and rests on the middle finger, while the tip of the middle finger contacts the patient (Figure 12-1). The surgeon's hand also rests lightly on the patient and the fingers are moved rather than the entire arm, which allows better control of the blade. This grip works best for short incisions where precision is important.[4] Contact with the patient controls precisely the depth of penetration. The disadvantage of this grip compared with the others is the relatively steep angle with which the scalpel is held, thereby decreasing contact of the cutting edge with the skin.

For the fingertip grip, the tips of the third, fourth, and fifth fingers are placed underneath the handle, while the tip of the thumb is placed on the other side. The index finger rests on the top surface of the blade to carefully control downward pressure (Figure 12-2). This grip is useful for long straight, curved, or sigmoidal incisions, because it places the long surface of the blade against the tissue, providing greater cutting surface, better control of the blade angle, and optimal control of incision depth. The blade movement originates in the shoulder, with the entire arm participating in directing the incision.[5]

The palm grip is not commonly used. Some surgeons prefer it for standing flank incisions. It provides the strongest grasp of the scalpel. The scalpel is grasped with the fingers and palm wrapped around the handle, while the thumb is placed on the top edge of the blade to create downward pressure (Figure 12-3). The small finger is rested on the patient to steady the hand.

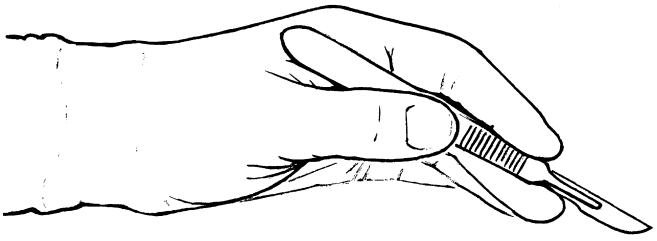

Figure 12-1. The pencil grip for holding a surgical scalpel.

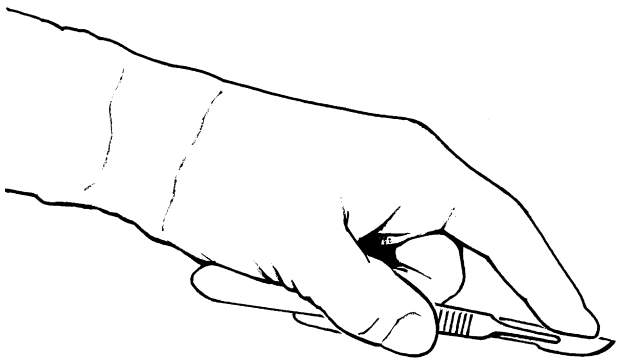

Figure 12-2. The fingertip grip for holding a surgical scalpel.

Electro Scalpel

Proper cutting technique with the electro scalpel differs markedly from that with the steel scalpel. A modified pencil grip is used to hold the instrument almost perpendicular to the tissue surface to be cut, to minimize the area of energy contact at the point of incision. The use of a needle scalpel further minimizes the contact area. The handpiece is held between the thumb and the middle fingertips, leaving the index finger free to activate the trigger button of the handpiece. The best effect is achieved when an assistant streches the skin or tissue to be transected.

Scissors

Operating scissors cut tissues by moving edge contact between two blades that are set slightly toward one another.[6] This action is most effective near the tips of the instrument, dictating their use for precise tissue cutting. Tissues that are too thick or too dense to be cut with the tips of the scissors should be separated with either a larger pair of scissors or a scalpel blade. The blade near the hinge should not be used for cutting, because the tissues are crushed more than cut, resulting in additional trauma.

As shown in Chapter 11, many scissors are available either with straight or slightly curved blades and with long or short handles. The mechanical aspect of scissor cutting is best achieved with straight blades. Therefore straight-bladed scissors should be used in dense tissues. Curved scissors provide a more comfortable positioning of the surgeon's hand and better visualization of the tips in deeper planes, but these instruments are less efficient in cutting tissues.

The classic tripod grip provides the best functional result. The tip of the thumb and last digit of the third finger are placed in the rings of the scissors, while the index finger stabilizes the instrument along the shaft toward the tip of the blades. The wide-based tripod grip or thumb-ring finger grip involves the last digit of the fourth finger instead of the last digit of the third finger (Figure 12-4). This grip is best suited to surgeons with large hands. The tripod formed by the thumb, third or fourth finger, and index finger creates a stable and powerful base for cutting.

Suture scissors are usually held in the classic tripod grip to cut the sutures at the designated spot. Because it is the surgeon who is responsible for the lengths of the suture ends, an

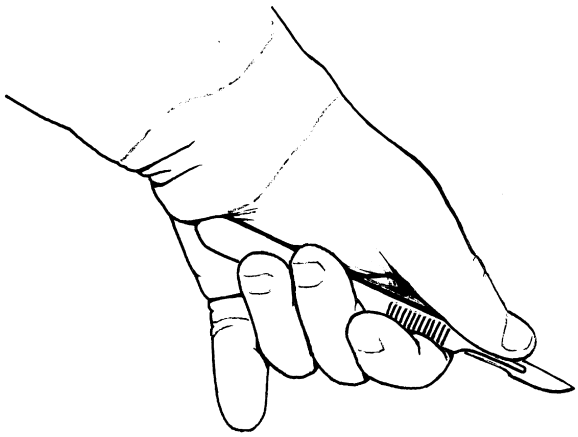

Figure 12-3. The palm grip for holding a surgical scalpel.

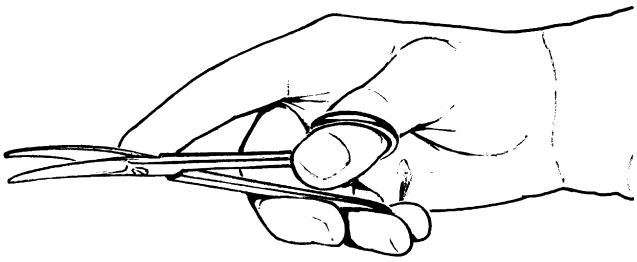

Figure 12-4. The tripod grip for holding surgical scissors.

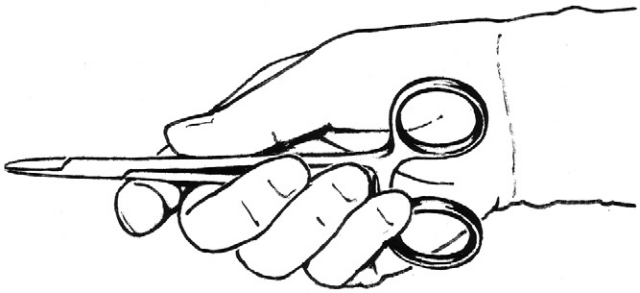

Figure 12-5. The palm grip of a needle holder.

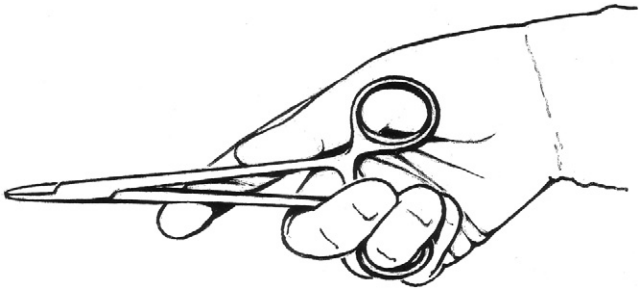

Figure 12-6. The thenar grip of a needle holder.

adequate length must be presented to the assistant so that the scissors can be applied at the desired spot.

A surgeon working without an assistant may use an Olsen-Hegar needle holder with built-in suture scissors (see Figure 11-7, *A*), or the suture scissors can be held in the same hand as the needle holders, in the manner described for handling multiple hemostats (see later).

Needle Holders

There are three methods for holding needle holders. One is the classic tripod grip just described for scissors. The greatest advantage of the classic tripod or thumb–ring finger grip is that it allows precision when releasing a needle. Although slower than the palm or thenar grip, it is preferred when tissue is delicate or when precise suturing is required.

The palm grip is useful for rapid instrument manipulation in closure of tissue when precision is not essential; however, the palm grip is not universally accepted as proper technique.[3] With the palm grip, also referred to as the modified thenar eminence grip, the surgeon places the instrument in the palm of the hand with the one ring resting against the thenar eminence of the thumb but no finger placed in one of the rings of the needle holder (Figure 12-5). The index finger stabilizes the instrument along the shaft. The lock mechanism is disengaged by lateral pressure applied to the instrument using the thenar eminence. The tips of the instrument may be opened and closed by adduction and abduction of the thumb. This method of manipulation is useful for rapid closure, because it allows the needle to be more easily grasped, extracted, and readied for the next pass.[7] It is also advantageous for suturing robust tissue that requires a strong needle-driving force; however, the needle cannot be released and regrasped after guiding the needle through tissue without changing to another grip, making suturing less precise.[8] Please note that left-handed surgeons cannot "palm" right-handed instruments because the boxlock closes rather than opens with pressure.

The thenar grip, where the upper ring rests on the ball of the thumb, and the ring finger is inserted through the lower ring (Figure 12-6), allows the needle to be released and regrasped for extraction without changing grips. Although it allows mobility, releasing the needle holder by exerting pressure on the upper ring with the ball of the thumb causes the needle holder handles to pop apart, and some needle movement occurs during this process.

The pencil grip, where the index finger and thumb rest on the shafts of the needle holders, is used with very delicate needle holders (Castroviejo) used in ophthalmic surgery and microsurgery.

A needle holder grips the suture needle along its shaft so that the needle is perpendicular to and near the tip of the instrument. The needle is usually grasped midshaft, but it can be grasped closer to the needle tip for greater precision.[7,8] The needle is passed through tissue by rotation of the surgeon's hand, always following the curve of the needle. Care should be taken to advance the needle so that it protrudes from the tissue enough to allow the needle holder or tissue forceps to grasp it far enough behind the tip to prevent dulling or bending the needle. When using the needle holder, the surgeon may pronate the hand for greater precision or supinate the hand for greater speed.[7]

Forceps

Thumb Forceps

Thumb tissue forceps are used to manipulate and stabilize tissue during incising and closing. Thumb forceps are usually held in in a pencil grip with the nondominant hand. When not in use, they may rest in the palm.[7] If the surgeon's hand becomes fatigued, the natural tendency is to switch to a palm grip. This grip is less precise and more likely to incite unnecessary tissue trauma.

When closing deep tissue layers, thumb forceps are useful for retracting superficial layers during needle placement, starting on the far side of the incision (Figure 12-7). As the needle is passed, the forceps moves to the layer being closed, exposing the exit point. The process continues with the tissue forceps being used to grasp tissue layers in opposite order on the near and far side of the incision.[5]

Hemostat Forceps

Mosquito and other tissue forceps used for hemostasis are held in the classic tripod grip to grasp the vessel to be ligated. When

a surgical assistant is not available and several hemostats have to be applied, time can be saved by introducing the ring finger through the left ring of several such instruments and holding them in the palm of the right hand, while applying a hemostat to a vessel in the tripod grip with the same hand (Figure 12-8). By arranging the hemostats so that the tips point toward the thumb, the instruments can one by one be rotated into the tripod grip and applied to a bleeding vessel.

Tissue Forceps

The most commonly used tissue forceps in equine surgery are towel clamps, mosquito forceps, Allis tissue forceps, Ochsner forceps, and Carmalt forceps. All these forceps are applied to tissues with the tripod grip. Towel clamps (except the Lorna-Edna clamps [see Figure 11-16, *C*]) are useful during some procedures for tissue manipulation, even though their primary purpose is to secure drapes on the patient. Towel clamps attached to skin edges provide an atraumatic method of retraction for exposing deeper tissues. Because Allis tissue forceps and Ochsner forceps are traumatic and crush the tissue, they are best reserved for securing tissue that can be excised.

TISSUE INCISION AND EXCISION

Slide Cutting

The skin is usually incised with a scalpel, because this is the method that is least traumatic and most conducive to primary healing. The incision should be made in one smooth pass of the scalpel through the skin, using the slide-cutting technique, transecting the dermis without cutting deep fascial tissue. The surgeon's free hand should stabilize and stretch the skin being incised (Figure 12-9). When skin is properly transected, the edges will retract. In a longer incision, it may be necessary to reposition the free hand to put tension on the skin along the entire incision. During this repositioning, the scalpel should not be lifted from the tissues. Each time the scalpel leaves and returns to the tissue, a jagged edge is created that will adversely affect healing (Figure 12-10).[9]

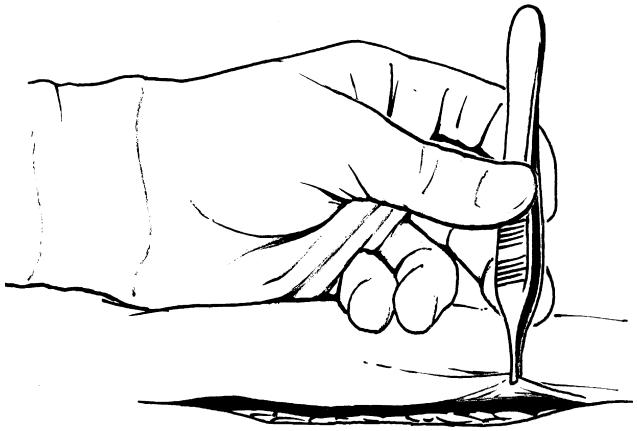

Figure 12-7. Proper technique for holding and using thumb forceps.

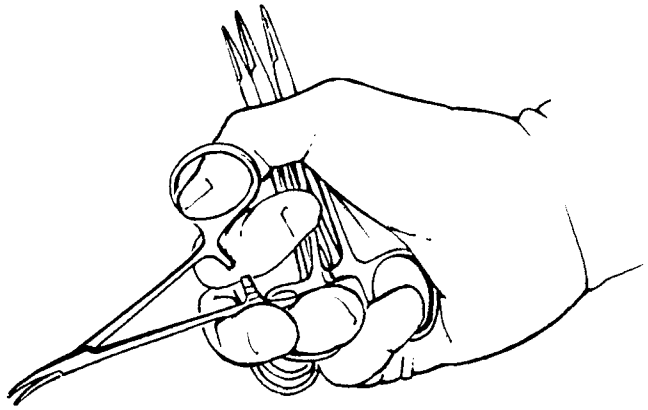

Figure 12-8. Several mosquito forceps are held in the surgeon's palm, allowing effective sequential application to a number of vessels.

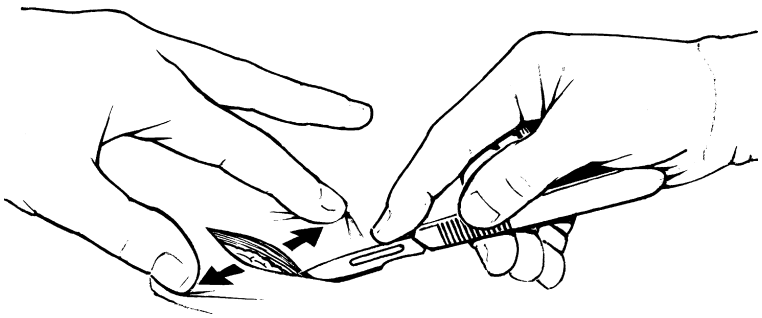

Figure 12-9. Stabilizing and stretching the skin between the thumb and index finger facilitates the incising of the skin.

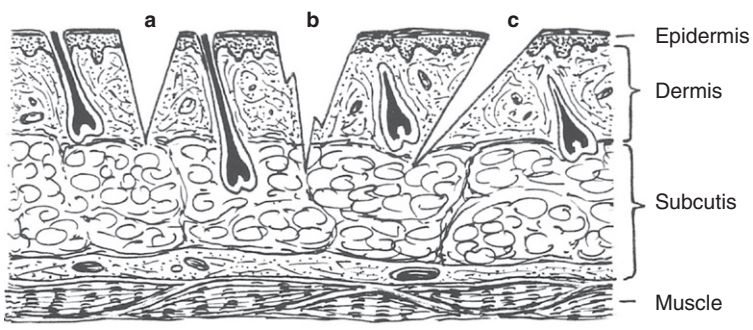

Figure 12-10. Skin incisions. *a,* Correctly performed incision. *b,* Timid slide cutting resulted in jagged incision edges. *c,* Slide cutting with a sideways-angled blade resulted in an obliquely angled skin incision.

Stab or Press-Cutting Incision

Stab or press-cutting incisions are generally performed with the scalpel held vertically in the pencil grip (Figure 12-11). A stab incision results when the bursting threshold of the tissue being incised is exceeded. Press cutting is applied to initiate incisions into hollow, fluid-filled structures, such as the bladder. For this technique to be effective, the tissue to be entered should be under tension. Press-cutting incisions are also used frequently during screw fixation of an anatomically reduced condylar fracture of the third metacarpal/metatarsal bone (MCIII/MTIII) or of the proximal phalanx. The scalpel is held in a pencil or palm grip, perpendicular to the surface of the tissue. The tissue is entered with a slight thrust, and the incision is extended carefully by pushing the cutting edge of the scalpel through the tissue. With this technique, depth control is poor, but it can be improved by using the index finger as a bumper (Figure 12-12), effectively limiting penetration of the blade to a predetermined depth.[7] Press cutting with an inverted blade (Figure 12-13) elevates the tissues to be transected and provides more safety for deeper structures, while preventing fluid from exiting a fluid-filled structure or organ.

Two rarely applied techniques are the sawing (or push-pull slide cutting) and the scalpel scraping techniques, the latter of which is used for separation of fascial planes or for subperiosteal dissection and elevation of muscles.[7]

Scissor Incision

The scissor tips are often used to transect tissues. Before this technique is used, the tissue to be incised must be isolated from underlying tissues using blunt scissor dissection (see later). This isolates the tissue structures to be cut. Some tissues can be effectively transected by partially opening the scissors, holding the blades motionless relative to each other, and pushing them through the tissue. Allowing the scissors to slide through the tissue creates a clean, atraumatic incision. This method is appropriate for opening fascial planes over muscles or subcutis, or for opening tissue planes in which the start and finish points of the incision are well defined.

Electroincision

Because lateral heat production during electroincision increases with the duration of trigger activation and tissue contact time,

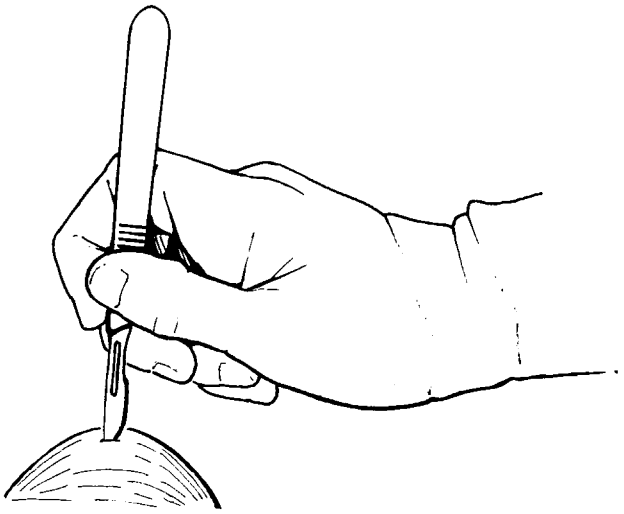

Figure 12-11. Stab or press cutting into a hollow organ.

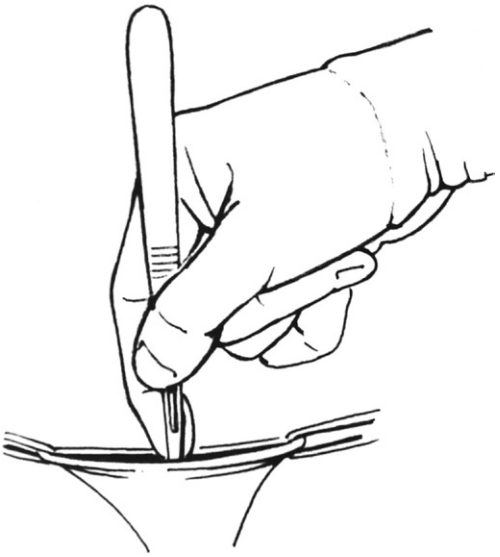

Figure 12-12. Bumper cutting into a structure elevated and stretched between two Allis forceps.

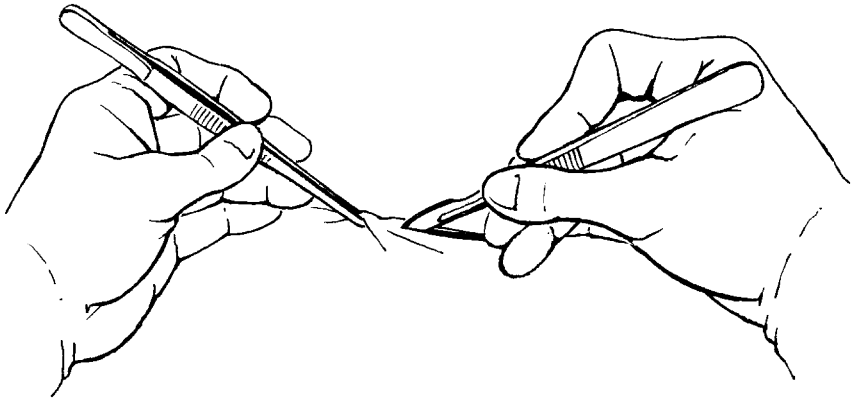

Figure 12-13. The technique of inverted-blade press cutting facilitates blade control.

the blade is moved at a speed of about 7 mm/sec.[7] Only one tissue plane is cut at a time, using only the tip of the blade. Depth control with the electro scalpel is less precise than with the cold scalpel. Because the electrode cuts all tissue it contacts, visual control is of paramount importance. Electrosurgical incision should not be used in areas with poorly defined anatomic planes. Thermal necrosis at the wound edges can be reduced and depth control can be improved by using the lowest setting on the controls that allows clean cutting.

The electrode should be cleaned frequently to ensure proper function. Charred tissue that accumulates at the tip of the electrode acts as an insulator and decreases effective cutting. Three undesirable effects are associated with a charred electrode: (1) higher power is required to incise tissues; (2) current is dispersed to a larger area of tissue, diminishing control; and (3) thermal necrosis of the wound edges is increased.[2] If the buildup of charred material at the tip is rapid or excessive, the power setting may be too high or the cutting speed may be too slow.[10]

Advantages reported for electrosurgical incisions over those made with a steel scalpel are (1) reduction in total blood loss; (2) decreased need for ligatures, and thus reduction in the amount of foreign material left in the wound; and (3) reduced operating time.[11,12] These advantages come at the expense of delayed wound healing and decreased resistance of wounds to infection. Controlled experiments revealed that there is no overall difference in epithelial healing between incisions made with the electro scalpel and those made with the steel scalpel. However, a difference in the initial response of the connective tissue was recorded.[5] Electro incisions of the skin heal primarily, but there is a definite lag time in reaching maximal strength. Because of this delay, skin sutures or staples should remain in place an additional 2 to 3 days if the incision was made with an electro scalpel. Electrosurgical incisions should be avoided in the presence of cyclopropane, ether, alcohol, and certain bowel gases because of the risks of ignition and explosion.[7]

Tissue Excision

Most tissues are excised primarily by scalpels or scissors. Skin, hollow organs, contaminated subcutaneous tissues, and neoplastic tissues are best excised with a scalpel. This is performed by a single passage of the scalpel along or around the periphery of the tissue to be removed. However, repeated passes or a sawing action with the scalpel may be necessary to complete excision of the tissue. This is especially true for thick, dense tissue or en bloc excision. Precise excision of tissue deep within surgical wounds or body cavities is best performed with scissors.

BLUNT DISSECTION

Blunt dissection is used to reduce or prevent the risk of damaging deeper vital structures during a surgical approach. The technique is performed digitally or with surgical scissors. Blunt dissection is generally carried out along natural tissue planes or parallel to tissue fibers. Excessive dissection and undermining should be avoided, because creation of dead space impedes wound healing and potentiates infection. If scissors are used—blunt scissors work best—the tips are placed in a closed position into the tissue, and the jaws are opened parallel to the tissue fibers or along natural tissue planes (Figure 12-14). Forceps can be used to stabilize the tissue during dissection. When digital dissection is applied, the gloved index finger of each hand is placed side by side in the same tissue plane and pulled in opposite directions to stretch and separate the tissue, thus increasing surgical wound exposure.

Scissors are useful for dissecting tissues, especially the subcutaneous tissue. The plane of dissection is parallel to the skin, along the incision edges. Limited dissection underneath the skin allows further retraction of the skin away from the center of the incision and facilitates visualization of deeper tissues. Scissor dissection is less useful, and potentially dangerous, in deeper dissections, where vessels or nerves could be severed before they are seen.

SURGICAL HEMOSTASIS

Proper hemostasis prevents the surgical field from being obscured by blood, and it decreases the potential for infection. Hemostasis minimizes blood loss and postoperative hematoma or seroma formation, which may delay healing or potentiate wound dehiscence. Additionally, excessive or uncontrolled hemorrhage can lead to anemia or hypovolemic shock.[7] Therefore the goal of hemostasis is to prevent blood flow from incised or transected vessels. This is accomplished primarily by interruption of blood flow to the involved area or by direct closure of the vessel walls.[13] There are mechanical, thermal, and chemical techniques to achieve hemostasis.

Mechanical Hemostasis

Pressure

Using the fingers or the hand, pressure can be applied directly over the site of a major vessel, or over a major vessel at a site remote from the wound. Oozing from small vessels is best controlled by direct pressure using sterile gauze. Although this is the least traumatic means of vascular hemostasis, it is not adequate for medium-sized and larger vessels, which require some other means of hemostasis.

Gauze packing is used to control hemorrhage from open body cavities (such as the nasal cavity, paranasal sinuses,

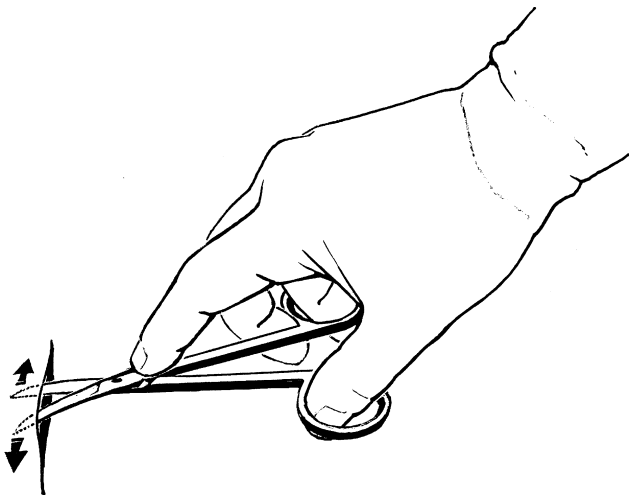

Figure 12-14. Blunt dissection of subcutaneous tissue can be performed by spreading the jaws of the scissors in the tissues.

urogenital tract, and defects created in the hoof wall or sole) and from large body wounds. Hemorrhage is controlled through pressure, allowing time for clot formation. The gauze can be soaked in iced or chilled saline solution, or diluted epinephrine can be added to a diluted antiseptic (e.g., povidone-iodine) or saline solution to help control the bleeding. Several gauze rolls tied together may need to be used to effectively pack large defects. The end of the packing is best secured to the body to ensure its presence at the time of removal.

Ligatures

Hemostats can be applied to small, noncritical vessels and held there for a few minutes. The vessel tissue trapped in the jaws is crushed, effectively occluding the vessel.[13] A combination of vasospasms and intravascular coagulation maintains hemostasis when the clamp is released. To facilitate these events, the vessel can be stretched or twisted before the instrument is released. If bleeding from a critical vessel needs to be controlled, atraumatic hemostatic clamps can be used to limit damage and allow repair.

Suture ligation is commonly used to control bleeding from larger vessels. Absorbable suture material is preferred over nonabsorbable material, because the latter can result in extrusion or sinus tract formation.[13] The number of ligatures required to maintain occlusion depends on vessel size and the material used. A simple circumferential ligature is generally used for small vessels (Figure 12-15, *A*), whereas pulsating or large vessels, such as arteries, should be ligated with two ligatures, a circumferential followed by a transfixation ligature placed more distally (Figure 12-15, *B*). In most situations, a hemostatic clamp is applied to the vessel before ligation. The clamp's crushing effect facilitates ligature placement and vessel occlusion. The following steps for proper use of hemostatic forceps should be kept in mind[4]:

1. The smallest forceps that will accomplish the needed hemostasis should be used.
2. Only the minimum amount of tissue should be clamped—preferrably only the vessel itself.
3. The tip of the instrument should be used rather than the middle or the base.
4. The mosquito forceps should be applied to small bleeding vessels perpendicular to the cut surface.
5. Other forceps should be applied perpendicular to the long axis of the vessel to be ligated.
6. The mosquito forceps should be applied to surface bleeders so that they come to rest lateral to the incision, with the concave part of the curved blades pointing down. In deeper locations, such as in the abdominal cavity, the forceps should be placed with the tips pointing upward.
7. The assistant should pick up the hemostat and direct it with the tip pointing toward the surgeon.
8. The hemostat should be held in the nondominant hand. One ring is held between the index finger and the thumb, and the other ring rests on the middle and ring fingers (Figure 12-16).
9. At the time of the final tightening of the first half hitch around the vessel, the surgeon should give the assistant the sign to release the hemostat.
10. The assistant should release the hemostat by pushing up with the middle and ring fingers while pressing down with the thumb, carefully releasing the ratchet mechanism of the hemostat.
11. Before releasing the hemostat, the instrument should be directed into the incision to relieve tension on the vessel and prevent it from slipping out of the ligature before the ligature is completely tightened.
12. The surgeon should apply a second half hitch over the first one, forming a square knot.
13. Then, the assistant should cut the suture ends at the level indicated by the surgeon, with the suture scissors held in the dominant hand.
14. If double ligation is indicated, clamps should be placed at each ligature site, approximately 2 to 3 mm apart. Once the vessel is clamped, a circumferential ligature should be placed around the vessel adjacent to the proximal hemostat. As the ligature is tightened, the clamp is released. The ligature should fall into the area of the vessel crushed by the clamp. The distal clamp should be released and replaced with a transfixation ligature.

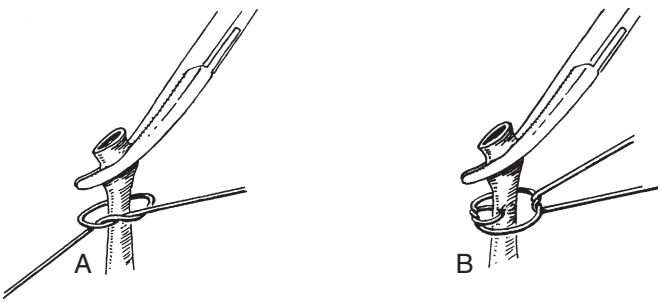

Figure 12-15. Circumferential **(A)** and transfixation **(B)** ligatures.

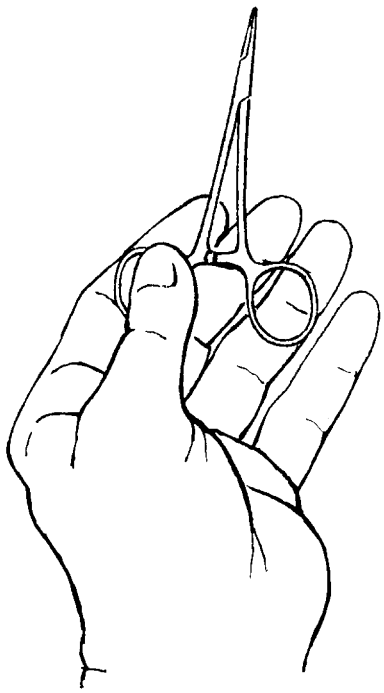

Figure 12-16. The hemostat is held in the nondominant hand. One ring is held between the index finger and the thumb, and the other ring rests on the middle and ring fingers. Pressing the rings toward one another releases the hemostat handle lock.

Large pedicles are preferably divided into smaller units, and each is separately ligated. After ligating the last unit, a suture is placed around the combined units and tied as one pedicle ligation. This is called the "divide and conquer" method (Figure 12-17, *A*).[7] The three-forceps method (Figure 12-17, *B*) involves initial clamping of the pedicle with three parallel forceps, 1 to 1.5 cm apart, incorporating the entire pedicle. The pedicle is transected between two such forceps, leaving one side with one forceps and the other with two forceps. A loose ligature is applied around the entire pedicle with the two forceps between the base of the pedicle and the first forceps. The forceps closest to the pedicle base is then partially taken off, leaving a strand of crushed tissue behind. The ligature is now solidly tightened, making sure that it comes to lie over the crushed line of tissue. While the surgeon tightens the ligature, the assistant carefully removes the forceps completely. If the pedicle is too large, insufficient hemostasis is often achieved with this technique.[7] In such cases, the divide and conquer technique should be used.

Ligation of vessels obscured by perivascular fat accumulation, such as occurs in the omentum, may be a challenge because occasionally the vessel is traumatized by trying to blindly pass a needle around the vessel. In these cases, the blunt end of the needle can be used to place the suture around the vessel. This part of the needle pushes the vessel aside if it is in its path rather than penetrating it. Subsequent ligation of the vessel is routine (see Figure 37-28).

Staples

Vascular staples, which can be used to occlude vessels up to 7 mm in diameter, are an alternative to suture ligation. They offer the advantage of speed and precision in placement. A specially designed instrument (the Ligate and Divide Stapler [LDS, US Surgical]) first applies two vascular staples that are crimped around the vessel simultaneously and then divides the vessel between the staples (see Figure 16-14). In cases of extensive intestinal resection with multiple mesenteric arcades, time is saved using this instrument. Disadvantages of staples are expense and potential failure when used on large vessels.

Surgical Repair

Management of lateral wall defects in vital vessels can be very difficult. Suturing the defect is recommended, incorporating the tunica adventitia and tunica media—the major holding layers within the walls of large vessels.[5,10] Fine suture material (4-0 to 6-0) is recommended, using a continuous pattern with bites placed close together. If a vessel is inadvertently lacerated parallel to its length, closure with the help of a simple continuous or interrupted suture pattern may reduce the vessel diameter such that effective blood supply to afferent tissue or drainage from the efferent tissue is no longer ensured (Figure 12-18, *A*). In such a case, closure of the laceration perpendicular to the long axis of the vessel increases the vessel diameter to ensure circulation (Figure 12-18, *B*).

Esmarch System

The Esmarch and pneumatic tourniquet systems are excellent methods of temporarily occluding blood flow to a distal extremity (Figure 12-19). They are used to maintain a bloodless operative field. An inflatable pneumatic cuff is placed around

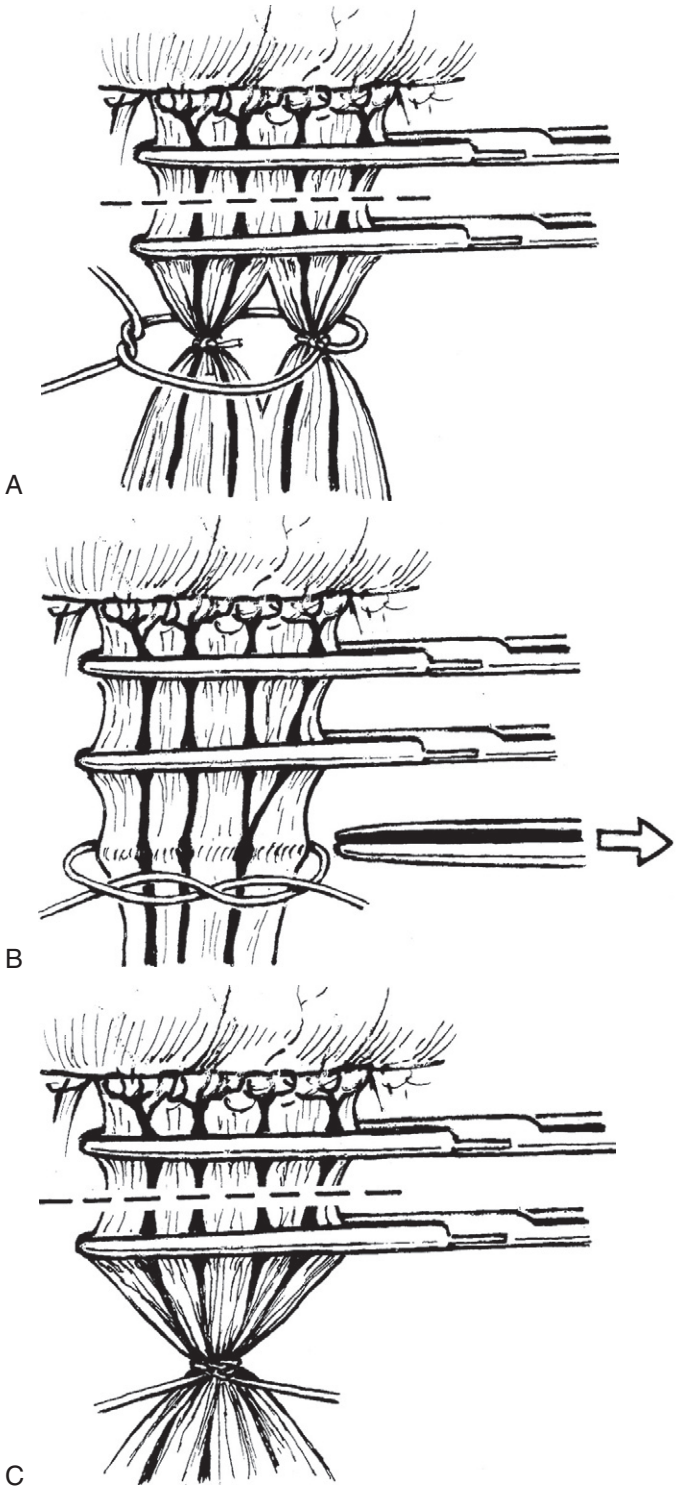

Figure 12-17. Ligation of large bundels of tissues. **A,** Divide and conquer technique. **B** and **C,** Three-forceps technique. The third hemostat has been removed *(arrow)* and in its place a ligature is applied **(B)**. The bundle is separated between the two remaining hemostats and ligatures are applied at the location of the hemostats or immediately adjacent to them (on the distant hemostat side relative to the division line) **(C)**.

the limb, 10 to 15 cm proximal to the surgical site, before preparing and draping the surgical site. If the cuff is applied proximal to the carpus or the tarsus, a gauze roll is placed on the medial and lateral sides of the limb over large vessels underneath the tourniquet to facilitate blood flow occlusion. Starting over the hoof and proceeding proximally, a long latex rubber bandage is tightly wrapped around the limb, overlapping the previous turn by 50% to force the blood from the limb. Once the Esmarch bandage reaches the level of the pneumatic tourniquet, the cuff is inflated above systolic pressure to occlude blood flow into the limb (approximately 600 mm Hg) (see Figure 12-19). Subsequently, the Esmarch is removed, beginning again at the hoof until the pneumatic cuff is reached. Nonpigmented skin will appear blanched. The tourniquet is generally left on the limb for no longer than 2 hours. When the procedure takes longer than that, the tourniquet should be partially deflated for 2 to 3 minutes, followed by reapplication of a sterile Esmarch bandage and reinflation of the tourniquet.

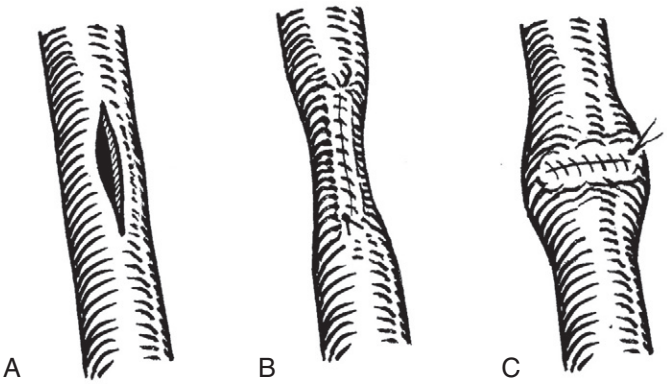

Figure 12-18. A, Surgical repair of a lacerated blood vessel. **B,** Application of a suture pattern parallel to the long axis of the vessel may decrease the lumen of the vessel resulting in its clotting. **C,** Application of a suture pattern perpendicular to the long axis of the vessel enlarges the lumen but also relatively shortens it.

Thermal Hemostasis

Electrocoagulation is a commonly used method of hemostasis. Heat generated from high-frequency alternating electrical current traveling between two electrodes denatures proteins inside cells.[11] Tissue damage from heat occurs between 3000 and 4000 Hz. Electrosurgical units can generate currents ranging between 1.5 and 7.5 MHz, and if the current applied is too high, the intracellular fluid boils instantly, potentially causing the vessel to explode without achieving coagulation.[12]

Electrosurgical units can produce different types of currents. A partially rectified waveform achieves the most effective hemostasis.[11] Vessels up to 2 mm in diameter can be coagulated in two ways. Obliterative coagulation is performed by direct contact between the handheld electrode and the vessel. This causes the vessel wall to shrink, occluding the lumen by thrombosis and coagulum formation.[11,14] Alternatively, hemostasis can be achieved by coaptive coagulation. In this method, the vessel is initially occluded by a hemostatic forceps. The electrode of the electrosurgical unit then contacts the occluding instrument, which conducts the energy to the vessel, inducing its permanent occlusion. This technique allows precise electrocoagulation of a vessel.

Cryogenic hemostasis, as the name implies, refers to coagulation caused by rapid freezing of vessels. The technique of cryosurgery is discussed in detail in Chapter 14.

Chemical Hemostasis

Occasionally, epinephrine is used to control hemorrhage. Epinephrine is a potent α-adrenergic agonist that causes peripheral vasoconstriction.[15] A solution of 1:100,000 to 1:20,000 is used to control superficial bleeding of mucosal and subcutaneous tissues.[13] Gauze packing soaked with a dilute epinephrine solution is an effective way to control bleeding.

Intravenous injection of 10% buffered formalin at a dosage of 0.02 to 0.06 mL/kg body weight diluted 1:9 in physiologic saline solution has been shown to be effective in controlling diffuse bleeding.[15] The exact mechanism of action is unknown, but it may be the result of induction of coagulation on the endothelial cell surface. Close monitoring of the patient during application is recommended. This technique is applied to stop bleeding after castrations, colic surgeries, and surgical interventions of the upper airways.

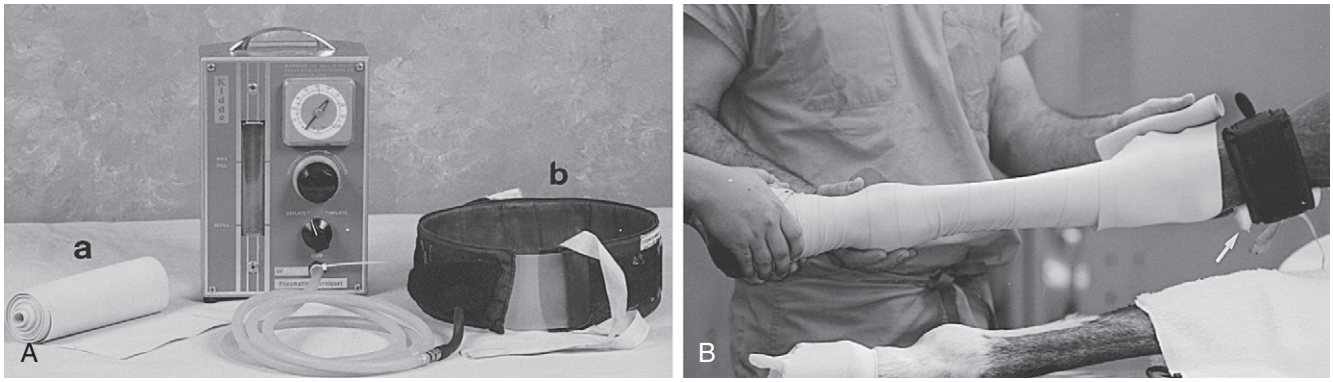

Figure 12-19. A, An Esmarch bandage *(a)* and pneumatic tourniquet *(b)* used for occluding blood flow in a limb. **B,** Application of an Esmarch bandage and a pneumatic tourniquet. Gauze rolls are placed over vascular pressure points under the tourniquet *(arrow)*.

Physical Hemostasis

Soluble sponge materials control hemorrhagic oozing by promoting clot formation. Various types of hemostatic materials include gelatin foam, oxidized cellulose, oxidized regenerated cellulose, and micronized collagen (see Chapter 4 for more details). While these materials press against the wound surface, the material's interstices provide a scaffold for clot organization.[5] These materials are most beneficial for low-pressure bleeding and in friable organs that cannot be readily sutured.[7] The materials are nontoxic, but they will delay wound healing and can potentiate infection because they are absorbed by phagocytosis.[2]

Bleeding from the bone can be controlled with the help of bone wax, which consists of purified and sterilized beeswax. The wax is physically packed onto the bone to block oozing of blood from cut cortical and cancellous bone. The material is relatively nonirritating, but it will remain in contact with the bone for years.[7]

TISSUE RETRACTION AND HANDLING

Retraction

Unnecessary tissue trauma induces inflammation, which can delay healing. Therefore incisions should be made only long enough to allow adequate exposure. However, trying to complete a surgical intervention through small incisions, which are currently trendy, often results in excessive trauma of the wound edges. Such manipulations delay wound healing. Therefore the proper length of incision is the goal of a good surgeon. Gentle manipulation of tissue with respect to blood supply, innervation, and hydration is essential for atraumatic surgical technique. To achieve this, instrument retraction may be preferred over direct hand retraction in selected situations.

Handheld retractors are designed with a single handle and blade to be used as an extension of the assistant's hand. Alternatively, self-retaining retractors are designed with a locking mechanism on the handles to keep the blades in an open position. The blades of the retractor are placed within the incision and opened until the tissues on each side are spread maximally. Occasional repositioning or relaxation of the instrument blades, in conjunction with padding (i.e., moist gauze sponges placed between the blades of the retractor and tissue), minimizes tissue damage.

Careful retraction and stabilization of nerves and neurovascular bundles with Penrose drains or umbilical tape should always be considered in place of metallic retractors.[13] This both facilitates atraumatic manipulation of the vessels and nerves and prevents inadvertent traumatization.

Careful and atraumatic tissue handling is as important as applying aseptic technique during surgery. Rough handling of the tissues may induce inflammation and subsequent delayed wound healing.

Tissue Handling

An incision heals from side to side, not from end to end. Therefore the incisions should be long enough to facilitate a clear view of the surgical site. Inadequate exposure may increase tension on the tissues through overzealous retraction, jeopardize hemostasis, increase the risk of traumatizing a nerve or vessel, and delay healing.

Sharp dissection should be carried out with sharp instruments. The use of dull scalpel blades and dull and worn-out scissors only increases tissue trauma. Whenever possible, natural tissue cleavage planes should be followed during dissection; this prevents inadvertent transection or tearing of fibrous tissues that heal poorly, if at all. Excessive undermining of tissues should be avoided, because it leads to the formation of dead spaces, which allow hematoma and seroma formation.

Most tissues should be handled with appropriate instruments; fingers should only be used for blunt disection. In small wounds, the introduction of a surgeon's finger prevents adequate evaluation of the deeper structures. Probing with a thin instrument allows simultaneous observation and manipulation. Tissue forceps are available for just about any manipulation necessary. Hemostatic forceps should only be applied to tissues that will be excised, because the tissues between the jaws are crushed and devitalized. Allis forceps are designed to hold tissues. However, excessive compression of the tissues in the clamp should be avoided. Stabilization and retraction of tissue may be accomplished with methods that do not involve tissue forceps. In selected situations, the assistant's fingers may be used for temporary occlusion of bowel to facilitate an enterotomy without additional trauma. Alternatively, a pair of self-retaining Doyen clamps may serve the same purpose.

Stay sutures can be used in a variety of situations—for example, to stabilize vessels and bowel. These sutures can be placed through very small amounts of tissue and still allow manipulations without tearing of the structure being repaired. Handheld and self-retaining retractors can be used in many surgical procedures to facilitate certain manipulations.

Nerves and vital vessels should be spared whenever possible. Once they are isolated, they should be manipulated with great care. The identification of these structures with the help of a Penrose drain is atraumatic and effective.

SURGICAL IRRIGATION AND SUCTION

Surgical Irrigation

Operative wound lavage has been associated with reduced rates of postoperative infection for both clean and contaminated wounds in direct proportion to the volume of irrigation solution used.[16,17] This phenomenon has been attributed to the removal of surface bacteria and debris from contaminated wounds, dislodgement and removal of bacteria and exudate from infected wounds, and dilution and removal of toxins associated with infection.[18] An additional benefit of wound lavage and suction is the moistening of tissues to counteract the dehydrating effects of air and surgical lights. Wound lavage removes blood from the surgical site, which also improves visibility. Various types of lavage solutions, delivery systems, and suction devices have been developed for various body regions (e.g., body cavity, skin), wound types (e.g., traumatic, surgical), and degrees of contamination or infection.

The ideal lavage solution is sterile, nontoxic, isoosmotic, and normothermic.[18] Sterile 0.9% physiologic saline, lactated Ringer solution, and Plasmalyte) are examples of available solutions that approach these criteria. Antibiotics are often added to a lavage solution as prophylaxis against possible infection or if contamination has occurred. Even though some effect has been reported, conclusive evidence that this technique is superior to saline lavage alone is lacking.[7,19] Infection implies bacterial penetration of tissues, and adequate blood and tissue

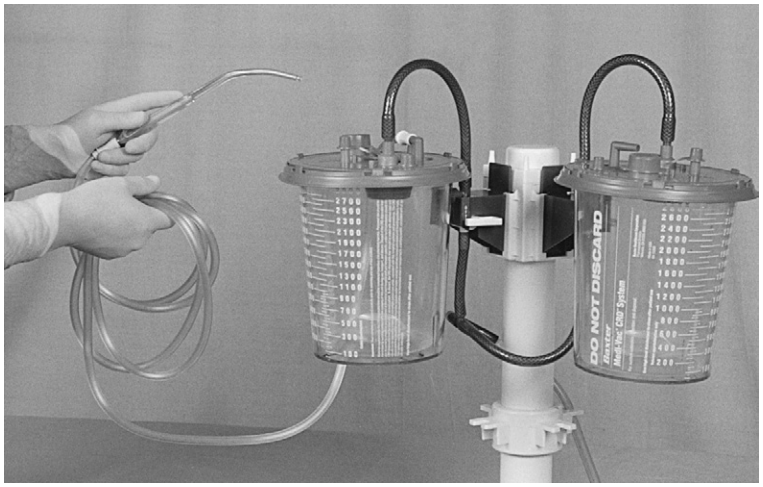

Figure 12-20. A suction tip is connected to sterile tubing to evacuate fluid from the surgical site into a reservoir.

concentrations of antibiotics via systemic administration are required for effective bacterial destruction.[7] Some antibiotics, such as tetracycline, are irritating when applied to exposed tissue or peritoneal surfaces and should be avoided.[20] Antiseptics such as povidone-iodine and chlorhexidine may be added to lavage solutions.[21]

Fluid delivery systems used for irrigation vary with location on the body and degree of contamination or infection. Lavage of body cavities is accomplished by flooding the cavity with large volumes of sterile solution, followed by suctioning to remove the fluid. Common methods involve pouring the sterile solution from the bottle or a bowl into the cavity or using a system capable of delivering large volumes of fluid at low pressure (referred to as diuresis).

Alternatively, traumatic and surgical wounds of the limbs are usually lavaged with the solutions under pressure. This is especially important if contamination or infection is present, because it dislodges bacteria or debris.[7] A bulb syringe or a 60-mL dose syringe is adequate for keeping tissues moist and removing débrided tissue particles in some circumstances, but automated systems that deliver a high volume of lavage solution at pressures not exceeding 10 to 15 pounds per square inch should be used on heavily contaminated tissues (additional information on wound lavage techniques can be found in Chapter 26). Alternatively, a sterilized squeeze hand pump can be inserted into a sterile fluid bag, and a fluid spray of the desired intensity can be applied to the selected tissue by squeezing the handle.[20]

Suction

Suctioning efficiently removes blood and fluid from the surgical site. A suction tip attached to sterile tubing connected to a suction pump that delivers a vacuum of 80 to 120 mm Hg is recommended (Figure 12-20).[8] When gentle suction is indicated, such as in deep incisions where exposure is limited, a Frazier tip is used. This tip has a side-hole port near the handle, which can be used to vary the amount of suction by either leaving the port uncovered or covering it with the index finger. When suctioning a large volume of fluid, a Yankauer suction tip with a single port can be used. The multifenestrated sump type of design of the Poole tip makes it ideal for use in body cavities, where a single-port tip will plug or injure viscera.[7] Figure 11-17 shows these special tips.

Figure 12-21. Proper technique for holding a curette.

CURETTAGE

Curettage refers to the removal of a growth or other tissue from the wall of a cavity or other surface with a curette. Curettage can be used in all types of surgical interventions, but it is mainly applied in orthopedic procedures. Débridement of sequestra, excess bone production such as periosteal exostoses, damaged or diseased articular cartilage, and subchondral bone during an articular procedure (arthroscopy or arthrotomy) represent some surgical procedures that may involve curettage. It is important to note that normal cortical bone cannot be removed with a curette; however, periosteal new bone formation or necrotic bone is easily removed with this instrument. Therefore, when initially efficient progress in bone removal is followed by a sudden increase in difficulty, the level of underlying normal bone has been reached.

The curette can also be used to remove necrotic soft tissue and debris from wounds, such as the tissue covering the bone after removal of a bone plate. The curette is used in an axial rotational motion (using its cuplike design at the instrument tip) to scoop out tissue, or with a pulling motion to scrape tissue from the surgical site. The handle of the instrument is grasped in the palm of the dominant hand and the index finger is placed on the shaft of the instrument to help stabilize the tip against the tissue (Figure 12-21).

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CHAPTER
13

Minimally Invasive Surgical Techniques

Andrew T. Fischer Jr., Joanne Hardy, Astrid B. Rijkenhuizen, and Jörg A. Auer

The evolution of minimally invasive human surgery that reached critical mass in the 1980s has been matched by a parallel development in minimally invasive surgical techniques in the horse. The evaluation of joints by arthrotomy, which was common until the mid 1980s, has been replaced by arthroscopy for almost all indications. Laparoscopic surgical techniques have continued to replace previous open techniques, such as cryptorchidectomy, ovariectomy, and inguinal hernia repair. In some cases, new techniques have been developed that were not previously available in the horse (e.g., testicle-sparing mesh repair of the inguinal ring). Thoracoscopic techniques are also continuing to evolve but at a slower pace because of the infrequency of surgical disease of the equine thorax. Hybrid procedures (laparoscopically assisted removal of cystic calculi, laparoscopically assisted nephrectomy, etc.) have also been developed that incorporate the improved visualization realized with laparoscopy but still maintain the tactile feedback of open surgery. The three major applications of rigid endoscopy (laparoscopy, arthroscopy, and thoracoscopy) share common surgical techniques and basic equipment. This chapter describes specialized equipment unique to each application and the basic procedures.

Additional minimally invasive surgery techniques include embolization and thrombectomy, which can be conducted through catheters introduced into vessels. These are effective procedures for treating disorders that a few years ago could be attempted only with great risk to the patient. Furthermore, computer-assisted surgery has only recently been introduced into equine surgery and may play a major role in orthopedic surgery of the future. It allows the accurate insertion of implants through small stab incisions, obviating the need for open approaches.

In addition to smaller surgical incisions, minimally invasive surgical techniques are characterized by vastly improved visualization. This has led to improved surgical outcomes and better overall understanding of regional anatomy. Minimally invasive techniques continue to evolve and replace previous open techniques as more surgeons become comfortable with them and as more thought is devoted to their development.

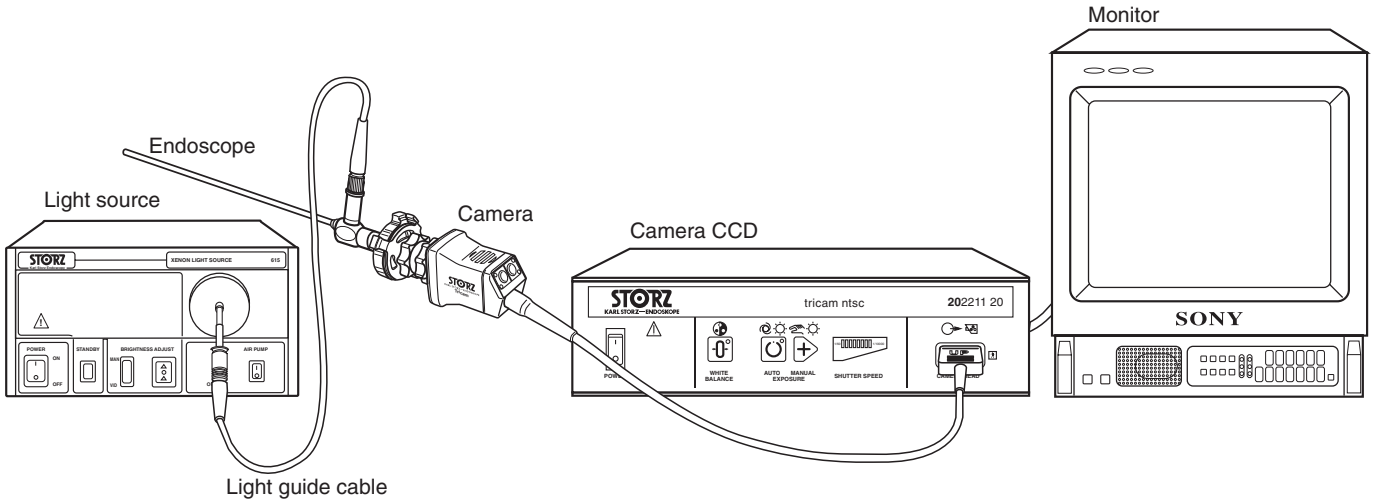

Figure 13-1. Basic equipment set up for minimally invasive surgery, consisting of light source, light cable, video camera with camera processor, and monitor. *CCD,* Charged capacitance device.

ENDOSCOPY

Andrew T. Fischer

Equipment

Illumination

Most minimally invasive procedures require a means of illuminating the body cavity and a telescope for viewing the target organs.[1-3] The supply of light into the patient's body cavity was a limiting factor until the development of cold light sources, which allowed high-intensity illumination of the cavity without danger to the patient or surgeon from excessive heat. The next major limitation of early arthroscopy and laparoscopy was the lack of video imaging equipment, which prevented an assistant from participating in the surgery. Without the aid of an assistant, the only procedures that could be performed were those that could be accomplished with one hand. Beam splitters were developed to share the image on the surgical telescope, but they were unwieldy and they markedly decreased the amount of light, resulting in a poor image. As video cameras were developed and refined, arthroscopy, laparoscopy, and thoracoscopy became popular. With time, the complexity of procedures markedly increased, as did the number of surgeons performing them.

Currently, light sources are capable of providing intense illumination to the selected cavity (Figure 13-1). Most manufacturers produce light sources with 300 watts of output from xenon bulbs. Xenon light sources are preferred, because they offer more lumens per watt than halogen light sources, and the light is whiter, offering more accurate reproduction of colors. A flexible fiberoptic or liquid light cable is needed to transmit the light from the light source to the telescope. Light cables are available in many lengths, but a 10-foot cable is generally preferred for equine endoscopy. A fiberoptic light cable must be checked regularly for broken fibers and must be well maintained by thorough cleaning. Poor illumination of the cavity can frequently be traced to a light cable with many broken bundles. However, a liquid light cable does not have this problem. Although a bit more expensive, they are quite durable and not subject to fiber bundle breakage.

When the light source is on and the light cable is connected to the light source, it is important that the distal end of the light cable or the telescope does not contact the patient, drapes, or any other combustible material, because burns may occur or fires may start as a result of the heat produced at the tip.

Figure 13-2. Laparoscopic and arthroscopic trocar/cannula assemblies. Note the pyramidal tip of the laparoscopic trocar and the conical tip of the arthroscopic obturator.

The three areas of rigid endoscopy all use a trocar and cannula assembly to first enter the body cavity (Figure 13-2). The cannula protects the telescope after insertion and has stopcocks allowing fluid infusion or gas insufflation for distending the cavity. The cannula has seals to prevent leakage of fluid or gas through it. Safety trocar cannulas may be used when entering the abdomen or thorax. These trocars rely on the tissue resistance encountered when inserting the trocar through the body wall to retract the safety shield and expose the blade system. Once the insufflated abdomen is entered, there is a loss of resistance and the safety shield snaps back over the blade, protecting the underlying viscera.

Telescopes

A high-quality surgical telescope is very important for all endoscopic procedures (Figure 13-3).[1-3] The Hopkins rod lens system provides more light transmission for illumination of the cavity and a wider field of view than traditional optical systems. Light is provided by optical fibers that surround the lens system. Telescopes of 5 mm or less in diameter provide adequate light and visualization for arthroscopy but not for laparoscopy or thoracoscopy. The reasons for this are that the cartilage covering the articular surfaces of the bones in the joints is bright and reflective, and the cavity is smaller, requiring less light for visualization. The most common telescope size used in equine laparoscopy and thoracoscopy has a 10-mm outside diameter. The large size allows adequate light transmission with good visualization. The standard length for human laparoscopes is approximately 30 cm, but a specially designed 57-cm laparoscope is available for equine use. The standard length for arthroscopes is 15 to 25 cm with an extra-long 4-mm diameter arthroscope of 35 cm. The distal ends of endoscopes are designed with different lens angles. The most commonly available distal angles are 0, 25, or 30 degrees of visualization. The zero-degree telescope allows more light transmission into the body cavity but does not offer the panoramic view that the 30-degree telescope provides. Panoramic visualization, which facilitates triangulation techniques, is accomplished by rotating the scope (not possible with the zero-degree telescope). For special procedures, a 70-degree arthroscope is available, but it is rarely used.

Video Equipment

A video camera that connects to the telescope is necessary to ensure aseptic surgical technique and allow assistance during surgery.[1-3] Most cameras contain either one or three chips—the charged capacitance devices (CCDs) used in the camera. Three-chip cameras have one chip for each of the primary colors (red, green, and blue) and generally offer better resolution than single-chip cameras. Newer video cameras have an increased light sensitivity, which is very helpful for laparoscopy and thoracoscopy in horses. Zoom features, gain changes, and multimedia image capture may also be offered as options on the various cameras. The video camera should be connected to a good-quality monitor in the direct line of sight of the surgeon. In some cases, it is helpful to have multiple monitors for the benefit of the assistant surgeon. The choice of cable connections affects monitor image—digital video cables offer the highest resolution. Multimedia digital capture of video-assisted surgery is becoming standard procedure and can be accomplished with personal computers, stand-alone video documentation systems, or video recorders incorporating hard disk storage and DVD burners. Many hospitals have central digital storage systems, also known as picture archiving and communication systems (PACS), that allow real-time collection of digital images into an electonic medical record.

Fluids and Gases

Arthroscopic, laparoscopic, and thoracoscopic procedures all require the creation of an optical cavity separating the joint capsule or body wall from the contents of the cavity, which facilitates a thorough visual exploration.[1-3]

Adequate visualization during arthroscopy is accomplished by the use of fluid or gas distention of the joint. Fluids used for joint distention are pH-balanced polyionic solutions such as lactated Ringer solution or Plasmalyte. If electrosurgical instrumentation within the joint is going to be used, fluids specially formulated for this are needed. Fluid distention is usually achieved with pressure or manually controlled pumps, but another possible driving force is gravity. Excessive fluid pressure is associated with extravasation of the fluid, resulting in marked subcutaneous edema and poor visualization because pressure on the skin and subcutaneous tissues compresses the joint capsule. Fluid extravasation can be minimized by making the skin incision slightly larger than the joint capsule incision. Gas insufflation may be used when the joint surfaces must remain dry during arthroscopy (e.g., when inserting cartilage grafts

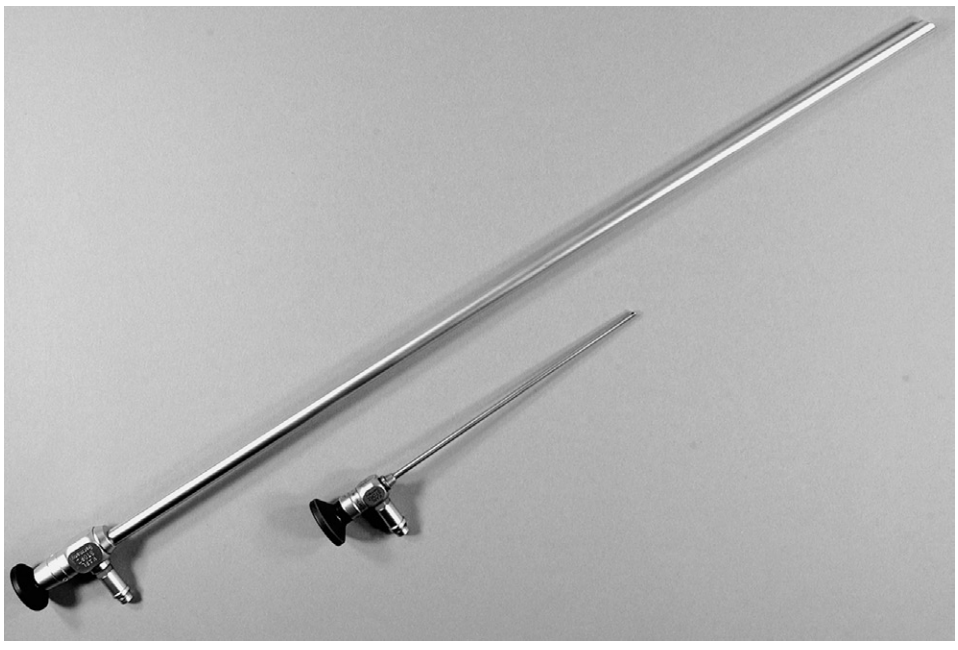

Figure 13-3. Laparoscopic and arthroscopic telescopes.

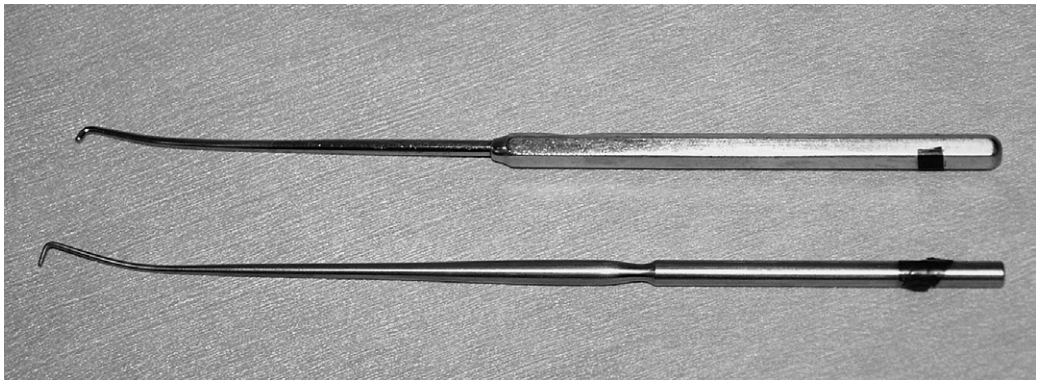

Figure 13-4. Arthroscopic probes.

or injecting gels into subchondral bone cysts). The pictures obtained with gas insufflation are clearer and truer to actual intra-articular colors. The insufflation technique is identical to the one described for laparoscopy.

The abdominal cavity requires insufflation for optimal viewing, which is accomplished by controlling the flow of gas into the patient's cavity. The insufflator should have settings that limit flow rate and pressure in the cavity to be examined. Insufflators for equine use should have flow rates that can exceed 10 L/min, and 20 L/min is desirable. Insufflators with slower rates require too much time for adequate initial inflation or reinflation of the cavity if it becomes deflated during manipulations. Initially the rate of gas flow into the patient is limited by the smallest diameter in the circuit, which is typically the insufflation needle. Needles such as the Veress needle have flow rates of less than 3 L/min, whereas teat cannulas can accomplish flows of 6 to 7 L/min. Once the laparoscopic trocar is inserted, the limit on flow rate is usually the insufflator.

The most commonly used gas for insufflation is carbon dioxide. Other inert gases have also been used in human medicine. The patient's abdominal pressure is usually 15 mm Hg or less. Higher pressures are associated with increased patient discomfort and respiratory compromise and are not necessary for visualization. Insufflation is less commonly used in thoracoscopy because the lung tends to collapse when air enters the thorax passively. In the rare case where insufflation is necessary during thoracoscopy, 5 mm Hg is usually adequate. The use of high intrapleural pressures is unnecessary and painful; high pressure decreases cardiac return and interferes with ventilation. Selective bronchial intubation may be performed for thoracoscopy in cases requiring general anesthesia.

Surgical Instruments

ARTHROSCOPY

The basic instruments necessary for arthroscopy include probes, rongeurs, grasping forceps, chisels, mallet, curets, periosteal elevator, flush cannula, and a bone awl (some of these instruments are described and depicted in Chapter 11).[3] Probes are used to evaluate looseness of fragments, determine stability of cartilage, and manipulate structures, testing their integrity or improving visualization (Figure 13-4). Multiple rongeurs may be used in a single surgery, and the choice is dictated by the operative target. Ferris-Smith rongeurs are available in different sizes and jaw angles (straight, angled up, and angled down),

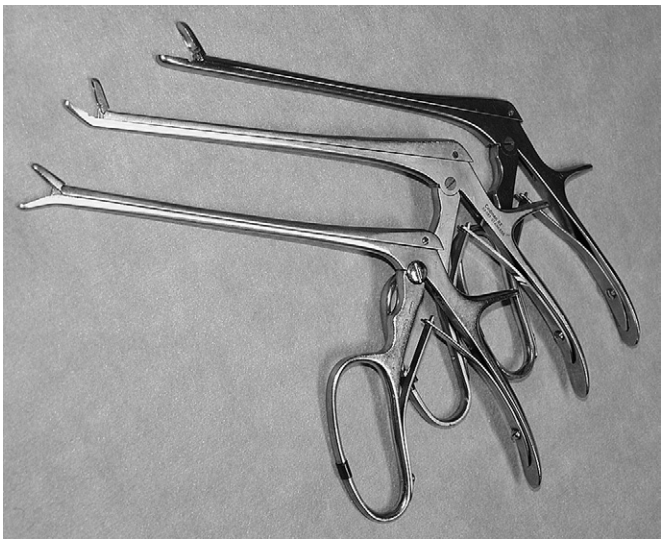

Figure 13-5. Ferris-Smith rongeurs with different cups.

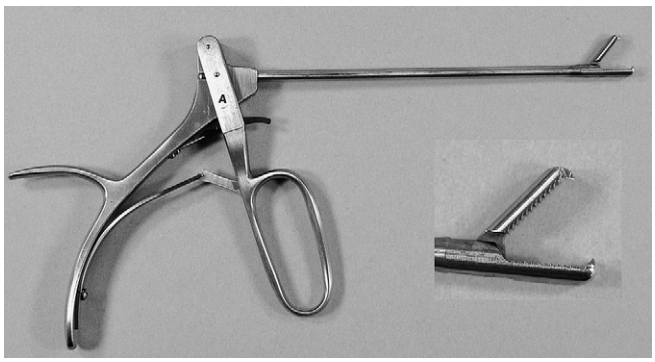

Figure 13-6. Grasping forceps.

and an assortment should be available in each surgical pack (Figure 13-5). Grasping forceps with small teeth in the jaws are preferred over rongeurs to to remove fragments from the joint (Figures 13-6 and 13-7). The EASY CLEAN line of rongeurs (Sontec Instruments, Inc., Centennial, CO) represents a new technology that allows cleaning between the two bars of the rongeur. It is anticipated that all rongeurs will be manufactured

Figure 13-7. The Maxi Grasper with specially designed jaws with radically enlarged multiple teeth and an oval gap between the jaws. Even when fully closed the jaws can securely hold the chips being removed. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

Figure 13-8. The EASY CLEAN Cushing rongeur representing the new wave of rongeur technology entering the surgical market. The wave shape facilitates cleaning between the two bars, extending the life of the instrument. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

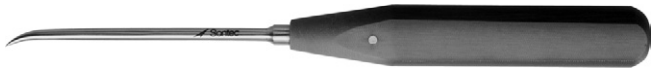

Figure 13-9. Peterson Micro Bone Pick for microfracturing the subchondral bone. (Reprinted with permission from Sontec Instruments, Inc., Centennial, CO. 2010.)

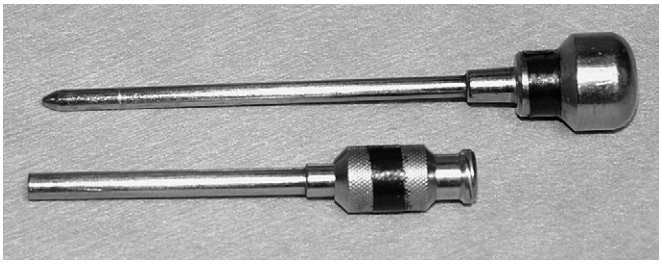

Figure 13-10. Arthroscopic flush cannula.

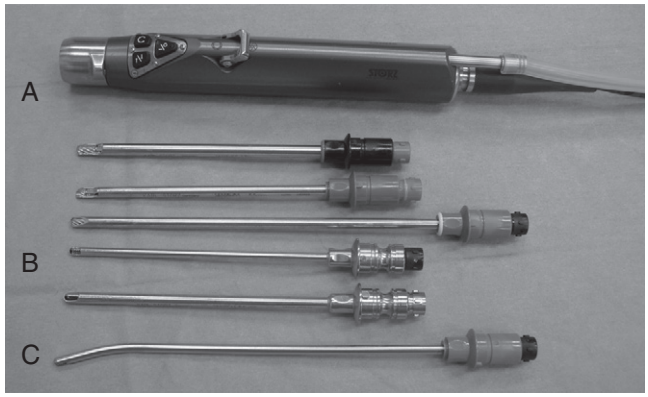

Figure 13-11. Motorized equipment. **A,** Handpiece with suction tube attached. **B,** Three synovial resectors. **C,** Three burrs.

with this technology. A recently developed suction arthroscopic rongeur facilitates removal of small chips and fibrin debris that has been disconnected from its origin (Figure 13-8). A 5-mm cannula that can be attached to a suction pump removes the debris immediately, obviating the removal of the rongeur after each piece has been detached. This speeds up surgery and reduces irritation at the incision site by constantly removing and reintroducing the rongeur into the joint. Chisels, osteotomes, and periosteal elevators are used to elevate osteochondral fragments with or without the use of a mallet. Curettes are used to débride cartilage edges and remove devitalized bone (see Chapter 12). Several different sizes and angles should be available to maximize access to the base of the defect. Bone awls (Figure 13-9) are used to produce microfractures in the subchondral bone plate, which are thought to improve cartilage adhesion after bone débridement of the articular surface (see Chapter 80). Flush cannulas are useful for lavaging the joint and removing any remaining bits of cartilage or bony debris (Figure 13-10). Motorized equipment (Figure 13-11) is useful for synovectomy, meniscectomy, tendon débridement, and removal of cartilage flaps. Different blades are used according to the structure being débrided.

LAPAROSCOPY, THORACOSCOPY, AND UROGENITAL PROCEDURES

The basic instruments used for laparoscopy include probes, Semm claw forceps, scissors, Babcock forceps, and biopsy forceps.[1] Probing organs provides tactile feedback regarding the consistency of the target and can be used to evaluate organ attachments. Semm claw forceps provide good security when grasping tissue that is to be removed from the patient (Figure 13-12). Atraumatic forceps such as Babcock forceps allow tissue manipulation without injury and are useful in exploratory laparoscopy or thoracoscopy (Figure 13-13). Endoscopic scissors are used for dividing tissue after adequate hemostasis has been obtained (Figure 13-14). Biopsy forceps are used for visceral biopsy (spleen, kidney, liver, and other solid organs) or tumors. Vessel sealing devices, such as LigaSure (Covidien, Mansfield MA) and SurgRx EnSeal (Ethicon, Somerville, NJ), have become one of the primary tools for controlling bleeding during minimally invasive procedures (Figure 13-15). These devices are modifications of previous bipolar electrosurgical units, which incorporate tissue impedence monitors that automatically adjust the current and output voltage, allowing lower settings to be used with improved outcomes.[4] The collagen and

Figure 13-12. Semm claw forceps used for tissue removal.

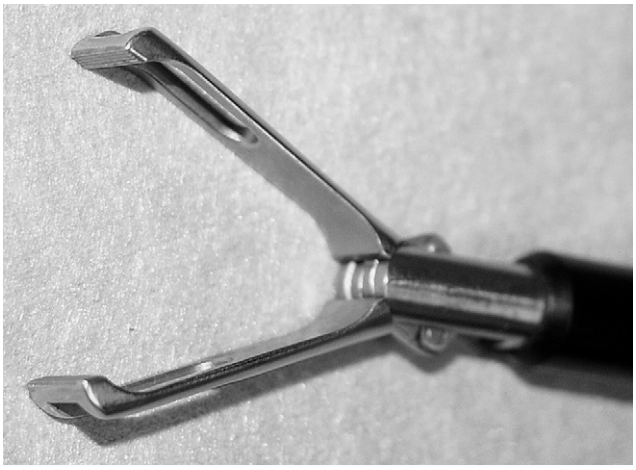

Figure 13-13. Babcock forceps used for atraumatic tissue manipulation.

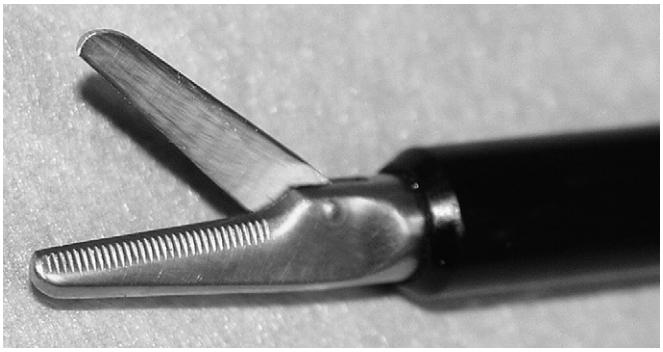

Figure 13-14. Scissors used for laparoscopic surgery. Note the increased length and size needed for efficient cutting.

elastin in the vessel wall are denatured by the high current and low voltage. This denaturation coupled with the mechanical pressure from the device causes coagulum formation, resulting in permanent sealing of the vessel. These devices allow permanent fusing of vessels up to 7 mm in diameter and tissue bundles without dissection within 2 to 4 seconds. The seals are capable of withstanding pressures up to 3 times normal systolic blood pressure. The feedback-controlled response system automatically discontinues energy delivery when the seal cycle is complete. The I-Blade Technology (Ethicon, Somerville, NJ) provides uniform compression forces along the clamping device and in the center of the blade that separates the tissues. The clamps are designed such that the tissue in between is trapped by atraumatic teeth before it is separated and uniformly clamped. Subsequently, separate electrical circuits permanently seal the tissues in each hemisphere of the jaw (Figure 13-16). Special insulation materials used for the clamps ensure that the tissues surrounding the jaws of the clamp are sealed only to a

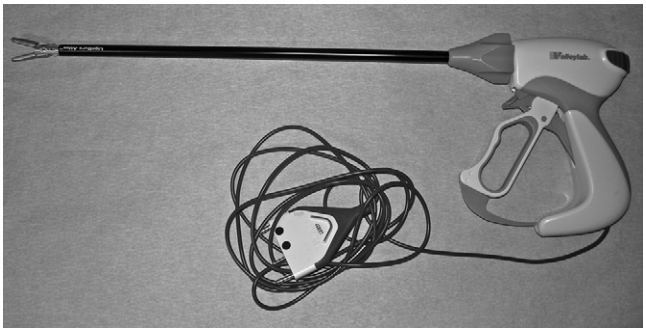

Figure 13-15. LigaSure electrosurgical instrument.

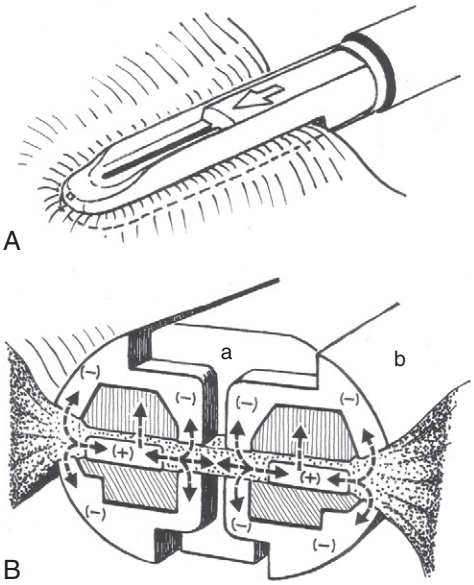

Figure 13-16. Schematic representation of the EnSeal Clamp. **A,** The EnSeal Clamp in action. The *arrow* indicates the direction the I-Blade separates the tissues before sealing the vessels. **B,** Close-up view of the electrical circuits crossing the tissue from the positive poles to the negatively charged surroundings. *a,* The I-Blade is partially advanced. *b,* The jaws are shut aided by the I-Blade.

distance of about 1 mm. Other hemostatic devices such as endoscopic staplers, electrosurgical units, ultrasonic scalpels, and different types of lasers are routinely used and will be discussed in appropriate chapters.

Triangulation Technique

Arthroscopic, laparoscopic, thoracoscopic, and urogenital endoscopic surgical procedures all share the common technique of triangulation. Triangulation refers to the placement of telescope and instruments through separate portals so that they converge on the operative target. Mastering the technique of triangulation is essential to becoming competent in minimally invasive endoscopic techniques. The visual target should be in front of the surgeon, with the monitor directly behind the visual target. The surgeon should be able to look from the operative field on the monitor to the surgical site on the patient by only looking up or down. The camera must be oriented so that true vertical and horizontal axes are maintained; this facilitates proper movement of the surgical instruments toward the surgical target (Figure 13-17). Triangulation techniques should be learned with training boxes before surgery is attempted on clinical cases. In general, the diagnostic evaluation in all minimally invasive surgeries should be performed before instrument portals are established, because they can collapse the optical cavity and interfere with visualization. An exception to this occurs when instrumentation must be introduced to manipulate viscera to facilitate exploration. Once the diagnostic exploration has been accomplished, additional instrument portals are established for the surgical procedure. The details for specific procedures are found in subsequent chapters and specialized texts.[1-3]

Basic Laparoscopic Technique

Standing Laparoscopic Surgery

Feed is withheld from the horse for approximately 24 hours before the procedure. Access to water is generally not restricted. Tetanus prophylaxis and routine perioperative antibiotics are provided. Nonsteroidal anti-inflammatories are also typically administered. The horse is restrained in standing stocks and the tail is tied to prevent contamination of the operative field. The head is either supported with a well-padded stand under its muzzle or tied up in an approximately horizontal position. Both flank regions are prepared for aseptic surgery. Sedation and analgesia typical for standing procedures is administered. Some surgeons prefer the use of bolus injections, whereas others use constant rate infusion techniques. For additional information on sedation and analgesia for surgical procedures that require standing for long periods, the reader is referred to Chapter 22. Once the horse is adequately prepared, local anesthesia is infiltrated at the site of desired trocar introduction. It is important to infiltrate both the subcutaneous tissues and muscle layers. The site of the first trocar is typically placed through the crus of the internal abdominal oblique muscle, midway between the last rib and the iliac crest. A 1.5-cm incision is made through the skin, and the trocar assembly that is large enough to accommodate the desired laparoscope is used. At insertion, the trocar is aimed toward the contralateral coxofemoral joint. If the peritoneum has not been penetrated at this point, a 30-degree laparoscope is used to ensure final penetration with a quick thrust. Insufflation with CO_2 is then started to a pressure of 10 to 15 mm Hg. Additional instrument portals are established under direct visualization to prevent damage to underlying structures. Once the desired surgical procedure has been completed, the abdomen is deflated and only the skin is sutured with simple

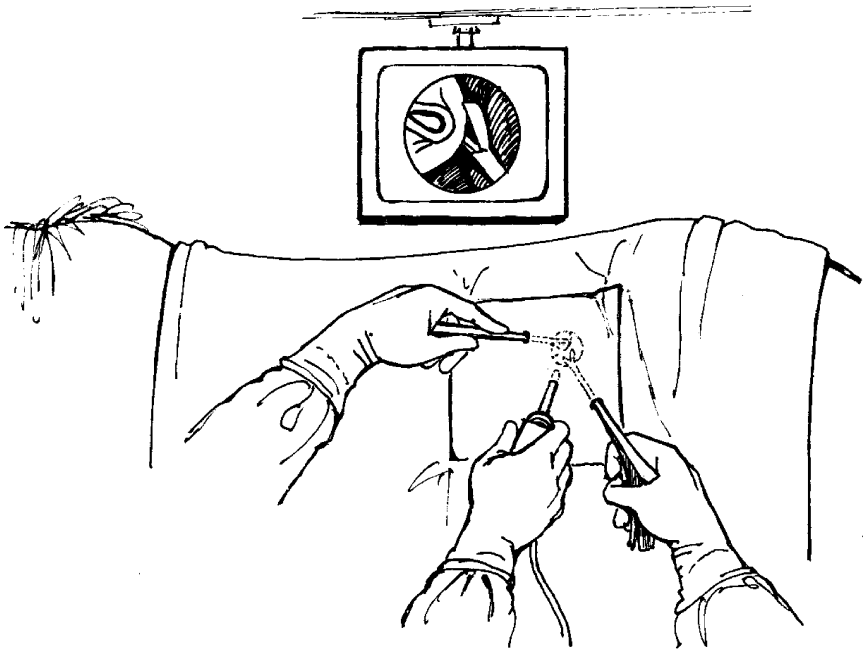

Figure 13-17. The proper use of the triangulation technique.

interupted sutures. Some authors prefer to insufflate the abdomen before inserting the trocar, but this is not necessary and may lead to insufflation of the retroperitoneal space, obscuring visualization for the rest of the procedure. At the end of the procedure the skin incisions and portals that are 10 mm or larger are closed in different layers; smaller ones are only closed with skin surtures.

Dorsally Recumbent Laparoscopic Surgery

Preoperative preparation for dorsally recumbent laparoscopic surgery is the same as that used for standing surgery. The horse is anesthetized and placed in dorsal recumbency and secured to the operating table to prevent the horse from shifting if it is tilted into Trendelenburg position (head down). The ventral abdomen is prepared for aseptic surgery. A 1.5-cm incision is made through the umbilicus and a teat cannula is inserted into the abdominal cavity. CO_2 insufflation is started. When the intra-abdominal pressure reaches 10 to 15 mm Hg, a trocar assembly large enough to accommodate the desired laparoscope is inserted into the abdomen. Safety trocars may also be used. Additional instrument portals are established under direct visualization. Skin closure is performed as described earlier.

Effects of Abdominal CO_2 Insufflation in Standing and Recumbent Horses

Abdominal insufflation with CO_2 is commonly used to create an optical cavity in horses that are undergoing laparoscopy for either standing or recumbent procedures. CO_2 insufflation causes a mild inflammatory reaction within the abdominal cavity, which is seen by an increase in peritoneal WBCs and should be remembered if serial abdominocentesis is necessary for evaluation of the horse's original problem.[1,5] Increasing the horse's intra-abdominal pressure with CO_2 does have effects on cardiopulmonary parameters, with more significant alterations noted in horses in dorsal recumbency.[1,5,6] Pneumoperitoneum in horses undergoing standing laparoscopic surgery had no significant effect on cardiopulmonary parameters.[5] Horses undergoing laparoscopic cryptorchidectomy in Trendelenburg position were noted to have a decrease in pH and an increase in $PaCO_2$ and mean arterial pressure, and these changes persisted while the horse was in Trendelenburg position but returned to baseline upon return to normal dorsal recumbency. PaO_2 decreases throughout the procedures but does not reach levels classified as hypoxemia, and it does not improve upon return to normal dorsal recumbency and normal intra-abdominal pressure.[6] Heavier horses have a greater change in pH, $PaCO_2$, and PaO_2 than lighter horses subjected to Trendelenburg position and abdominal insufflation.[6] Though cardiopulmonary parameters certainly change during laparoscopic procedures, little clinical effect has been noted. Positive pressure ventilation and blood gas analysis capabilities are suggested for use in horses undergoing laparoscopy under general anesthesia, particularly if Trendelenburg position is to be employed.

EMBOLIZATION

Joanne Hardy

Arterial embolization refers to catheter-directed delivery of particulate material for the purpose of embolizing selected arteries. Microcoils are a popular embolization material. They have been used for occlusion of normal and abnormal vasculature and for creating ischemia of neoplastic tissue (Figure 13-18). In dogs, coil embolization has been used to treat vascular occlusion of patent ductus arteriosus, occlusion of portosystemic shunts, and epistaxis; it has also been used in experimental treatment of cerebral aneurysms.[7-19] In horses, coil embolization has been used to occlude branches of the common carotid artery, usually involved in guttural pouch mycosis.[20-23] More recently, nitinol vascular plugs have been used for arterial embolization procedures in both dogs and horses.[24,25] The use of emulsions for embolization of tumors to create ischemia and reduce tumor size has also been described.[26] Chemoembolization refers to selective intra-arterial delivery of chemotherapeutic agents with particulate material to embolize arteries supplying blood to a tumor.[27] Numerous studies describe its use in humans and dogs, using various chemotherapeutic agents.[28-32]

Surgical Technique

Catheter-directed embolization involves accessing a peripheral artery, where an introducer is inserted. A catheter is then directed, under fluoroscopic guidance, within the artery until the tip of the catheter is located at the desired site of embolization. Accessing the proper site requires knowledge of local vascular anatomy and variances among individuals. Navigation through the arterial tree is facilitated by a gliding guide wire inserted within the catheter. Once the site of embolization is reached, the embolization material is delivered. The catheter and introducer are removed, and hemostasis at the arterial puncture site is achieved by direct pressure or suturing.

The sizes and materials used for embolization techniques are very specific, and correct selection of product characteristic for the desired purpose is essential. For example, catheters made of polyvinyl chloride (PVC) or vinyl do not allow the coils to glide within the catheter, resulting in occlusion of the catheter.

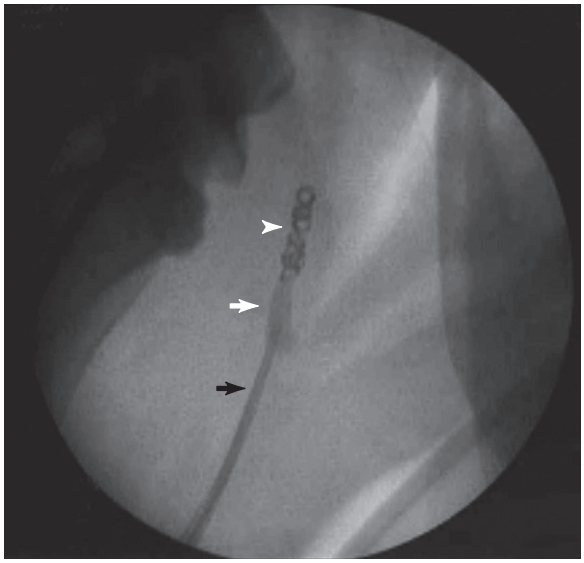

Figure 13-18. Fluoroscopic image of embolization coils *(white arrowhead)* occluding the internal carotid artery of a horse affected with guttural pouch mycosis. Note the position of the catheter *(black arrow)* within the artery, and injection of contrast material demonstrating arterial occlusion *(white arrow)*.

Similarly, selection of too small a coil diameter allows the coil to travel farther into the arterial vasculature, where it might embolize an undesired vessel. For details on use of this technique to control bleeding from guttural pouch mycosis, see Chapter 46.

THROMBECTOMY

Astrid B.M. Rijkenhuizen

Thrombectomy is performed in chronic arterial occlusive disease of the aorta and its caudal arteries, also referred to as aortic-iliac thrombosis (TAI). At first the symptoms are only induced by exercise, but in a later stage they also occur at rest. They signal ischemia in the hind limb tissue because of insufficient perfusion. The disease is progressive with a gradual onset. The clinical signs are related to the degree of vascular occlusion, the presence of collateral circulation, and the rapidity of the onset of the occlusion.[33,34] Affected horses could be asymptomatic or show only vague performance complaints. The most common manifestation is a predictable exercise-induced lameness that ceases with a resting period of 5 to 10 minutes. Patients who are forced to train "through the pain" show a more severe lameness and might require significantly more time for the symptoms to resolve. After physical activity, absence of sweating, retarded vein filling, and hypothermia of the distal extremity of the affected limb(s) can be observed.

Occasionally a thrombus embolizes from a proximal source and acutely occludes a distal peripheral artery. After training, acute coliclike signs can develop (pawing, straining, sweating, lying down and rolling), mostly combined with a severe lameness. The diagnosis is based on history, clinical presentation, rectal palpation combined with ultrasonography, and scintigraphy.[35-42] Information on the onset of ischemic symptoms, the duration of symptoms, the characteristics of pain, and any alleviating factors are helpful. The absence of a palpable pulse in an extremity is probably the most common physical finding. Rectal ultrasonography is used to recognize the thrombus in the aorta and the internal and external iliac arteries. Doppler-based ultrasonography renders both an anatomic and a functional assessment of the femoral artery condition in the inguinal region and is used to estimate the severity of a the arterial occlusion.[11] The femoral artery is visualized in the femoral triangle, which is bordered caudally by the pectineus muscle and cranially by the sartorius muscle, over a distance of approximately 15 cm (6 inches). In unilateral cases, the unaffected hind limb can be scanned as a reference.

To monitor the development of hypoxemia in the affected hind limb the oxygen pressure in venous blood samples before and after a workload can be measured.[37] The samples are taken from the right and left saphenous veins as far proximally as possible, at the level of the stifle joint. Samples are collected anaerobically in heparinized 2-mL syringes, which are immediately sealed so they are airtight and then immersed in melting ice. Within 15 minutes after the first sample is taken they are tested in a blood gas analyzer (ABL 505, Radiometer, Copenhagen, Denmark).

Treatment with exercise programs and pharmacologic therapy with sodium gluconate, with or without fibrinolytic enzymes, anticoagulants, and vasodilatators, have thus far been unsuccessful.[34,38,39,41,43,44] Promising results can be obtained by restoring blood supply to the ischemic regions through vascular surgery such as thrombectomy. For this purpose a Fogarty graft thrombectomy catheter (length of 50 cm [20 inches], a closed diameter of 4 mm, and an expanded diameter of 16 mm) is used to improve the blood flow to the limb.

Surgical Technique[37]

The horse is anesthetized and positioned with the hind quarters in dorsolateral recumbency with the affected limb close to the table and the head and neck in lateral recumbency. The uppermost hind limb is secured in flexion and abduction. The incision (approximately 10 cm) is made medially over the saphenous vein where its course changes from superficial to deep. Subsequently, the vein and the surrounding muscles are bluntly separated and the femoral artery is identified. Just before the artery is clamped 20 mL of a heparin solution (Heparin Leo (LEO Pharma A/S, Ballerup, Denmark), 250 IU heparin/mL physiologic saline) is injected into the femoral artery in distad and proximad directions. Careful blunt dissection allows mobilization of the artery and placement of ligatures and two vascular clamps (aortic forceps, DeBakey-Morris) proximally and distally to prevent excessive loss of blood during surgery. Small arterial branches of the femoral artery are ligated. A transverse arteriotomy is made and the blood flow is tested by loosening the vessel clamp and letting the ligature slip. Visible thrombi are loosened from the arterial wall and removed with forceps (Figures 13-19 and 13-20). The Fogarty catheter is subsequently inserted into the femoral artery in collapsed form, directed proximally, and positioned beyond the thrombi. The catheter has a flexible wire coil at the distal end that expands when retracted to form a double-helix ring (Figure 13-21). The sliding knob on the handle of the catheter is retracted slowly, which causes the wire loops to expand partially and carry the thrombi along as the catheter is withdrawn. This procedure is repeated with the diameter of the coil more expanded until no resistance during withdrawal of the catheter is felt and no more thrombi are retrieved. By removing this blockage, blood flow is restored from the proximal side. When indicated, an additional thrombectomy is performed distal to the incision. Before closure of the artery, blood is allowed to flow freely for a short period to remove detached thrombi and air. The incision in the femoral artery is sutured using a simple continuous pattern of monofilament polypropylene (USP 5-0). Fascia and subcutis are closed with a simple continuous suture; then the skin is closed using an intradermal continuous pattern.

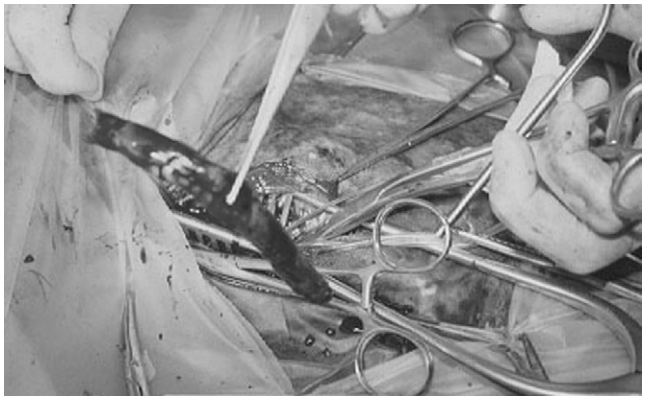

Figure 13-19. A thrombus is removed with the help of a forceps.

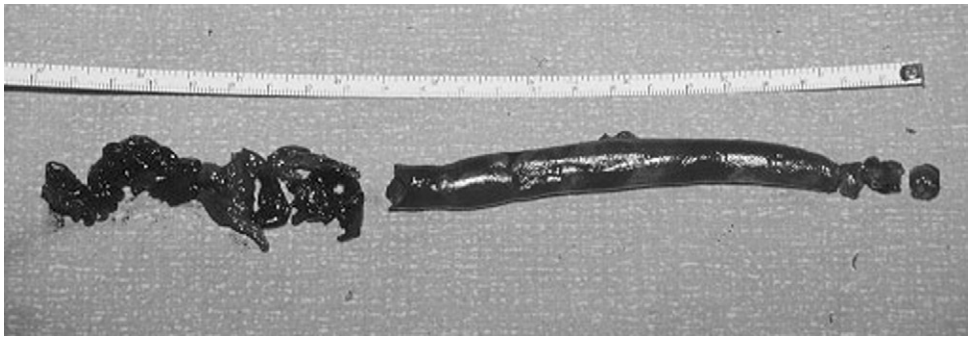

Figure 13-20. Removed thrombi.

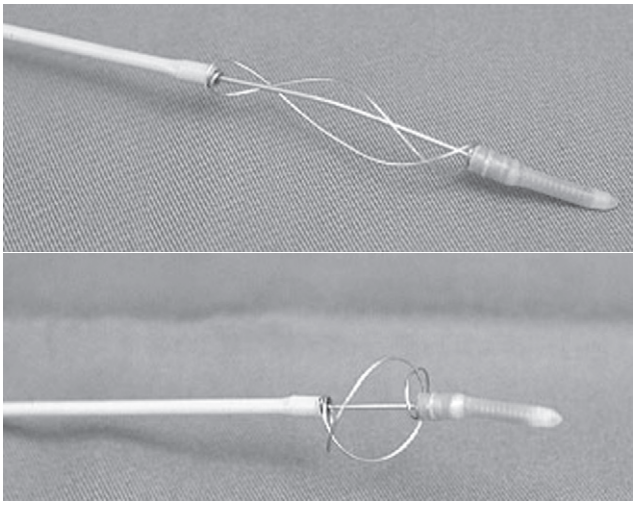

Figure 13-21. Fogarty catheter in closed *(top)* and expanded *(bottom)* positions.

Anticoagulation is initiated intraoperatively just before the arteriotomy through the administration of 100 IU heparin/kg or 50 IU/kg low-molecular-weight heparin (Dalteparin Natrium Fragmin, Pfizer, New York) intravenously. This is followed postoperatively by the administration of 50 IU heparin/kg or low-molecular-weight heparin subcutaneously once plus Carbasalate calcium 5 mg/kg (Ascal, MEDA Pharma, Solna, Sweden) or acetylsalicylate (5 mg/kg) orally once daily for at least 3 months. If there is diffuse intraoperative bleeding, heparin administration can be omitted or delayed, or low-molecular-weight heparin can be used, which lowers the risk of bleeding.[45]

Hand-walking is advised immediately after surgery. Light exercise can be initiated at 2 weeks postoperatively.

A severe complication is the appearance of TAI in the contralateral limb after surgery as a result of thromboembolization induced by clot fragments. Postanesthetic myopathy is seen in 24% of the cases in the affected limb.[46] This condition is assumed to be primarily caused by local hypoxemia of various muscle groups.[44] Horses with TAI that have preexisting hypoxemia before surgery are therefore at high risk for this complication. Providing adequate padding, positioning the horse correctly, preventing hypotension, and limiting surgical time are extremely important in the surgical management of these patients.[44]

The prognosis after surgical intervention is reasonable. In a recent study where 17 horses had been operated on, 65% of the horses regained athletic activity and 53% performed at their previous level.[46] No association could be made between the duration or the severity of the clinical signs and the clinical outcome. The success of this procedure depends on the length of time that the thrombus developed and adhered to the arterial wall.[46]

COMPUTER-ASSISTED SURGERY

Jörg A. Auer

Internal fixation of fractures is usually planned on the basis of a radiographic study. Occasionally, computed tomography (CT) is used to determine the exact course of the fracture line as it courses along the bone. Such a study allows the surgeon to determine exactly where to implant screws. Recently, intraoperative CT imaging has been introduced into equine surgery to aid in complicated and difficult-to-approach fractures, such as abaxial fractures of the distal phalanx.[47] With the help of radiodense markers, the ideal positioning of the implant can be preplanned and accurate measurements taken. Nevertheless, the actual result depends greatly on the surgeon's skill at inserting the implants according to the preoperative plan. Computer-assisted surgery (CAS) allows the surgeon to accurately implement the preoperative plan and to implant screws at the desired location and at the correct angle relative to the fracture plane.[48] CAS has been shown in numerous publications to improve accuracy in the placement of screws and other devices in humans.[49-51]

Technical Equipment

The equipment is composed of instruments with passive infrared light-emitting diodes (LEDs), the Vet*GATE* navigation system, (ARTORG Center for Biomedical Engineering Research, University of Bern, Bern, Switzerland) and the Arcadis Orbic 3D C-arm (Siemens Healthcare, Erlangen, Germany) (Figure 13-22). These instruments together define a fractured bone in three dimensions, and they allow real-time planning and observation of the implantation of the screw in three planes simultaneously.[52,53]

Arcadis Orbic 3D provides higher power and faster scan times in addition to enhanced image quality. Its 3D image data is acquired with 50 or 100 images recorded with a 1024 × 1024 resolution and is calculated in only 30 or 60 seconds, respectively. The acquisition can be repeated as often as necessary to include any anatomical changes that may occur in the operating field during surgery. The Arcadis Orbic mobile C-arm is suited for intraoperative 3D imaging of bones and joints of the upper and lower extremities, and the cervical region. It is important that the region to be scanned is either freely accessible or

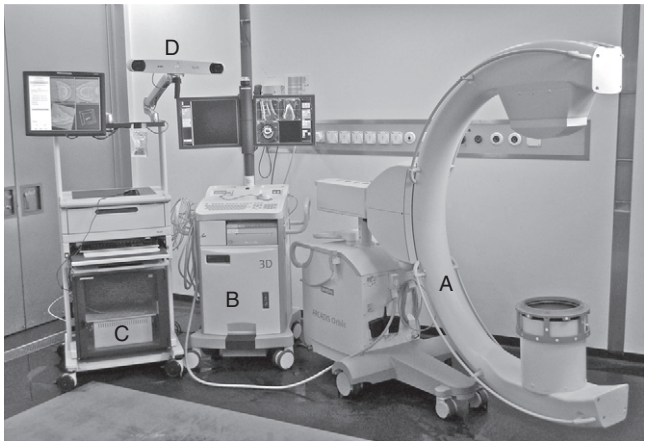

Figure 13-22. Equipment used for navigation. *A,* Arcadis Orbic 3D C-arm (Siemens AG, Munich, Germany). *B,* The corresponding computer with monitor. *C,* The Vet*GATE* computer system with monitor (ARTORG Center for Biomedical Engineering Research, University of Bern, Bern, Switzerland). *D,* The two-eyed navigation camera (Polaris Spectra, Northern Digital Inc., Waterloo, Ontario, Canada) on which the preoperative planning is performed and subsequent surgical guidance is viewed.

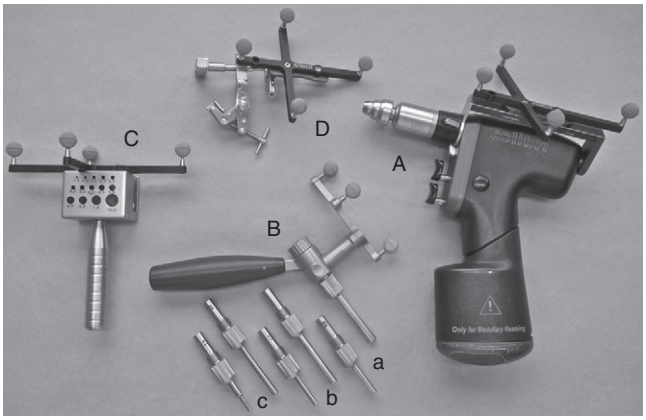

Figure 13-23. The instruments used for navigation equipped with passive light-reflecting balls mounted in different configurations. *A,* Battery-powered Colibri drill (Synthes, West Chester, PA). *B,* The drill guide handle with different sizes of exchangable drill guides (*a*, 5.5/4 mm; *b*, 4.5/3.2 mm; *c*, 3.5/2.5 mm). *C,* Calibration bloc with different sizes of holes. *D,* Foot clamp for attachment to the hoof capsule.

positioned on a carbon pad, which allows transmission of x-rays. The isocentric design of the Arcadis Orbic 3D (see Figure 13-22) features a 190-degree orbital movement. Further, both the patient and clinician can benefit from time and radiation dose savings through the isocentric design. Arcadis Orbic 3D can be equipped with Vet*GATE* 3D, an interface for direct 3D navigation. This interface combines the imaging capabilities of Arcadis Orbic 3D with high-precision surgical navigation, eliminating the need for manual alignment of the anatomy to the 3D images. This results in increased accuracy of surgical navigation and optimized clinical work flow. The 3D image data record compares the real patient anatomy through a matching free registration automatically. The C-arm incorporates a digital imaging chain from image acquisition to image processing and documentation. All image information is saved and administrated with a resolution of 1024 × 1024 pixels. With its electromagnetic brakes and ergonomic handles, the system is extremely easy to use.

Surgical Technique

First, the dynamic reference base (DRB) (Figure 13-23) is securely attached either to a Schanz screw, which was previously inserted into each of the main fragments of the bone involved. An alternative option available for the hoof is a studded clamp that can be attached to the hoof capsule (see Figure 13-23). Subsequently, the fractured bone is isocentrically positioned between the two components of the C-arm. Positioning is assisted by two laser beams positioned at 90 degrees to each other. The position of patient and table must allow movement of the C-arm over a 190-degree arc without interference (e.g., the surgery table, foot stands). The C-arm and the DRB must be located in the identifiable range of view window of the navigation camera. Over a 1-minute period, the C-arm takes 100 still radiographs (high-resolution mode) over an arc of 190 degrees, which are processed into 256 single pictures. The same number of radiographs can be taken at half the time (i.e., 30 seconds), but in lower resolution. The radiographic images can be viewed in three planes oriented at right angles to each other (in the horizontal, sagittal/parasagittal, and frontal planes).

The Vet*GATE* system consists of a two-eyed navigation camera (see Figure 13-22), a computer unit with sophisticated 3D software (see Figure 13-22), the instruments (e.g., power drill, awl), and a calibration unit that allows the surgeon to navigate within the system and to calibrate the instruments under aseptic conditions during surgery (see Figure 13-23). The data collected with the Arcadis Orbic 3D, the predecessor, the SIREMOBIL Iso-C 3D, is subsequently transferred to the Vet*GATE* computer (see Figure 13-22), where the future location of each screw is planned on the screen and marked in length and size. The Vet*GATE* system is then changed to the real-time navigation mode to guide the surgeon during the actual implantation. This is carried out by observing the computer screen and matching the drill and subsequently needed instruments with the planned image in three planes, similar to an arthroscopic technique (Figure 13-24). Once the location is matched, drilling is initiated. As soon as the drill bit crosses the fracture plane, which can be seen on the screen, the drill bit is changed to prepare the thread hole (see Figure 13-24). Insertion is then routine.

Three-dimensional navigation systems such as the Vet*GATE* in combination with the Arcadis Orbic 3D have great potential to be a real advantage for the precise and accurate implantation of lag screws in fractures in the horse. It has been used successfully in a limited number of clinical cases. Three controlled studies on cadaveric limbs were conducted with the predesessor of the Vet*GATE* system to evaluate the value of the system.[54-56] It could be shown that 3D navigation significantly improve accuracy compared to conventional surgery with C-arm assistance, especially with screw insertion into the distal sesamoid bone. The two studies conducted with the Vet*GATE* system showed significant improvements in accuracy compared to the Surgi*GATE* (Praxim-Medivision, Grenoble, France, no longer exists) system.[52,53] A significant part of the improvement is attributed to the use of a navigated drill guide with exchangable guides for the different sizes of drills and taps used to prepare the screw holes. With the Surgi*GATE* 1.0 system, the drill guide was not equipped with active LEDs. Therefore even the slightest

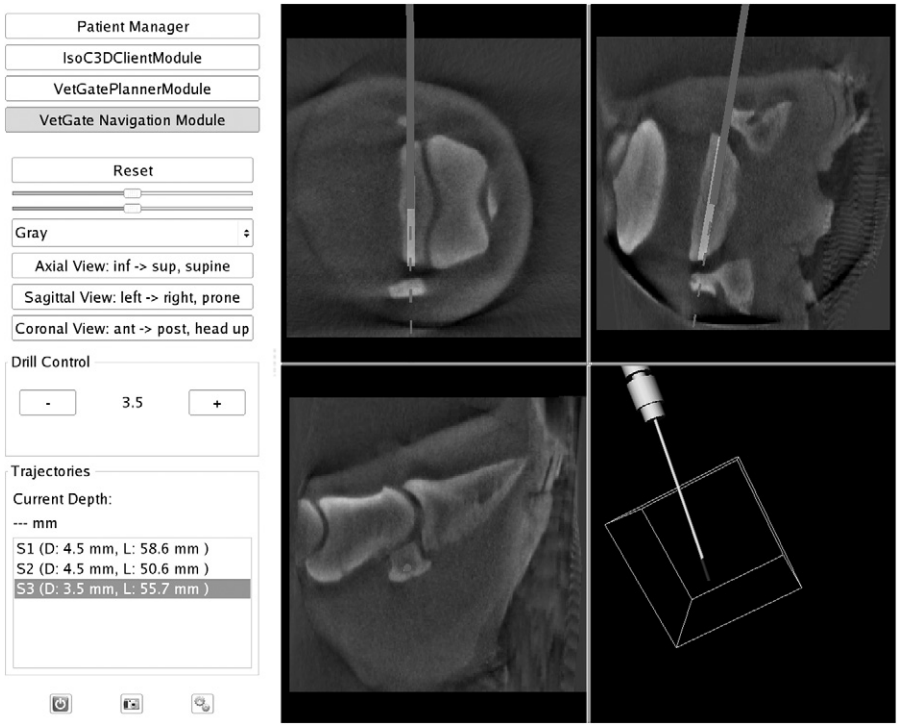

Figure 13-24. Screen shot of the navigation monitor during the insertion of a 3.5-mm screw into the distal sesamoid bone. Drilling is performed while constantly observing the monitor screen. The *lighter line* shown in the distal sesamoid bone represents the preplanned location of the screw within the bone. The *darker line* represents the drill bit and shows of the depth of penetration into the bone (in this case, half way). *Top two pictures* show the drill over the preplanned line in different projections. *Bottom left*, Only a round circle of the drill bit size can be seen on this lateral projection. *Bottom right*, A computer animation of the actual situation, where the drill *(lighter thick line)* is following the preplanned screw location *(dark thick line)* in space. The preplanned screw length measures 55.7 mm *(bottom line, far left)*.

bending of the drill bits resulted in inaccuracies in implant placement.

Other indications for CAS include fractures of the distal, middle, and proximal phalanx; condylar and saucer fractures of MCIII and MTIII; tarsal and carpal fractures, and cystic lesions of the various bones.

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CHAPTER
14

Cryosurgery

John A. Stick

PRINCIPLES OF CRYOBIOLOGY

Mammalian cells are destroyed when cooled to a temperature of –20° C (–4° F).[1] Primary injury begins with the formation of ice crystals, both intracellular and extracellular. The cell's outer membrane becomes ruptured by intracellular crystals, and ice formation outside the cell dehydrates the cellular environment, resulting in lethal electrolyte concentrations and pH changes. When organelles are damaged, the cell loses its ability to regulate ion permeability and cell death ensues. Secondary injury from freezing occurs from vascular stasis. As the permeability of vessels is increased, loss of plasma causes local hemoconcentration. Damaged endothelium in arterioles and venules induces thrombus formation of the vessels, and infarction of frozen tissue occurs within hours of freezing. The cryogenic lesion is a volume of coagulation that closely responds to the extent of the induced ice ball.

Rapid freezing results in the greatest intracellular concentration of ice. Thereafter, slow thawing of the tissue results in recrystallization, during which small crystals enlarge, producing

more cell damage. To ensure that all target tissue receives a lethal dose of cold, a second freeze/thaw cycle is used. Because precooled tissue freezes faster than normal tissue, repeating this cycle causes necrosis of the target tissue more consistently.

Variations in vascularity, noncellular structure, and water content cause tissues to respond differently to cryonecrosis. Dry tissues (e.g., the cornea) do not readily form ice crystals and therefore do not respond to cryotherapy very well. The cellular components of peripheral nerves are destroyed by freezing, but because the fiber scaffolding of the epineurium is not damaged, regeneration is possible.[2] Tissues near major blood vessels or in highly vascular areas are difficult to freeze rapidly and tend to thaw quickly without loss of function.[3] The use of epinephrine or temporary regional vessel occlusion may be necessary to ensure proper treatment in those tissues. Future developments in cryotherapy may include the use of nanoparticles to improve freezing efficiency. The basic principle is to deliver these particles into target tissues to maximize the freezing heat-transfer process, to regulate freezing scale, to modify ice-ball formation, to enhance ice-ball margin ultrasonographic imaging, and thus to prevent healthy tissues from being frozen.[4]

Immune responses directed against tumor cells have been documented after cryosurgery, and cryoablation-induced anticancer immune reaction is a well documented phenomenon in people and other animals.[5,6] Although this has not been proved clinically in horses,[7] numerous case reports suggest secondary tumor regression does occur as a result of cryosurgical treatment of a primary tumor.[8,9]

Although liquid nitrogen, nitrous oxide, and carbon dioxide are all cryogens used in veterinary medicine, liquid nitrogen is the most versatile and therefore the most commonly used. Liquid nitrogen has a boiling point of –195.8° C (–320.4° F). Cryogens, usually stored in liquid form in Dewar flasks (Figure 14-1), can be delivered as a spray or used by super-chilling a probe. Two types of probes are used: hollow probes and solid probes. When hollow probes are used, liquid is circulated through the probe and exits under pressure through a small opening. When solid probes are used, they are chilled by immersion into the liquid cryogen.

Indications

Cryosurgery does not require a sterile field. Therefore, it is a good choice for the treatment of benign and neoplastic cutaneous lesions. It can also be used in the mouth and in ocular surgery. By far the most common tumor that is treated with cryotherapy is the equine sarcoid. However, a plethora of skin conditions amenable to surgery can be treated by cryotherapy (see Chapter 29). Because there is frequently no need for general anesthesia of horses afflicted with skin lesions, cryosurgery has an advantage over other types of surgical extirpations—it frequently can be done on an outpatient basis.

INSTRUMENTATION

Sprays

Self-pressurizing spray guns (Figure 14-2) deliver a combination of vapor and droplets of liquid cryogen and are a most effective

Figure 14-1. Insulated Dewar flasks are used to store liquid nitrogen. This tank is fitted with a special adaptor lid and spray gun attachment. Note the pressure gauges used to regulate the liquid nitrogen.

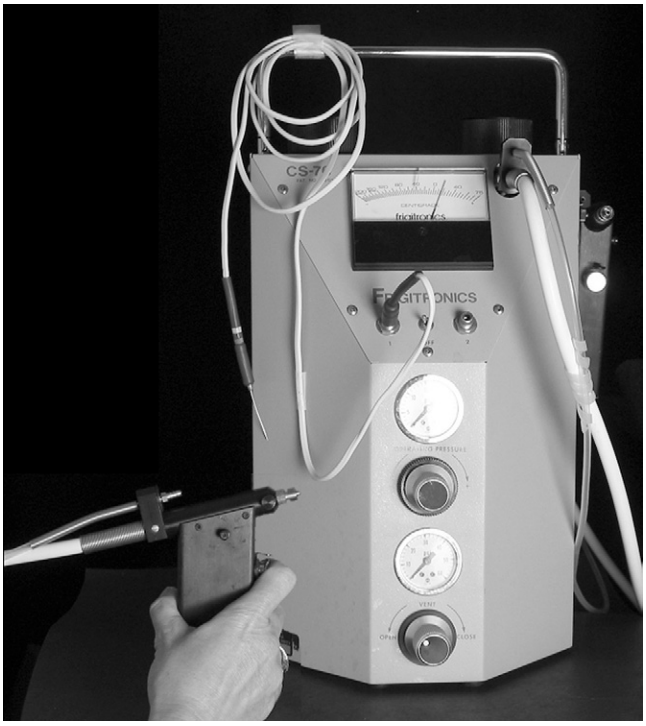

Figure 14-2. Special container used to deliver liquid nitrogen through a self-pressurizing spray gun. A thermocouple needle is to the left of the pyrometer, which is used to measure the temperature achieved beyond the limits of the targeted tissue. This single-channel monitor allows the needle to be placed into the tissue adjacent to the deepest portion of the target. When the temperature reaches –20° C, all unwanted tissue is destroyed.

method of cryogen delivery. As liquid nitrogen contacts the tissue, it evaporates, or changes from the liquid to the gas phase. This has been shown to remove a greater amount of heat from treated tissue than is achieved with probes. The volume and size of the spray droplet are controlled by the diameter of the needle orifice (Figure 14-3) and the trigger in the pressurizing gun. The surgeon can gauge the volume of the cryogen so that the wetting conforms to the shape of the tumor's surface. However, care must be taken to prevent excess liquid cryogen from running off onto surrounding skin. It is common to pack the surrounding area with Vaseline-impregnated sponges to prevent this runoff. Alternatively, a spray cup can be used that has the advantage of controlling runoff. A cup size (Figure 14-4) is chosen that fits over the tumor, and as the spray is applied, droplets form a liquid pool over the tumor.

Probes

Hollow probes are cooled by circulating a liquid cryogen through them. Hollow probe freezing is easiest to control, but the rate at which it cools an area is slow compared with the rate achieved by spray and solid probes. Hollow probes can be used for either contact or penetration freezing, depending on the configuration of the probe (Figure 14-5). During freezing, traction can be used to lift the tumor away from underlying structures as an ice ball is extended to the monitored limits.[10] Penetration freezing can be performed in larger lesions where a core biopsy specimen is removed from the center of the tumor (Figure 14-6) and the cryoprobe is placed within the mass. Contact freezing with solid probes is a very efficient manner of delivering cryotherapy to variously sized tumors, based on the size of the probe (Figure 14-7). As multiple probes are placed within the liquid nitrogen (Figure 14-8), they can be removed

Figure 14-3. Two examples of needles that attach to the spray gun to deliver liquid nitrogen sprays directly onto the tissue to be frozen. The volume and size of the spray drop is determined by the diameter of the needle orifice.

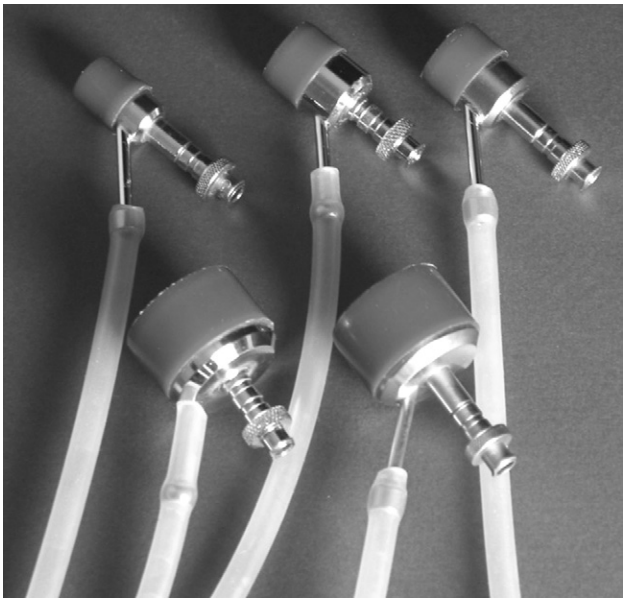

Figure 14-4. Spray cups come in a variety of sizes, so the cup can be fitted over a tumor, and as the spray is applied, droplets form a liquid pool contained by the cup. This prevents runoff generated by the spray method.

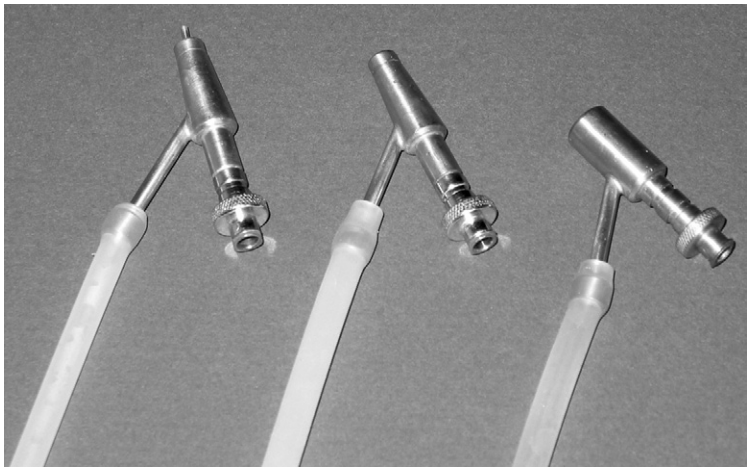

Figure 14-5. Hollow probes come in a variety of shapes and can be used for either contact or penetration freezing.

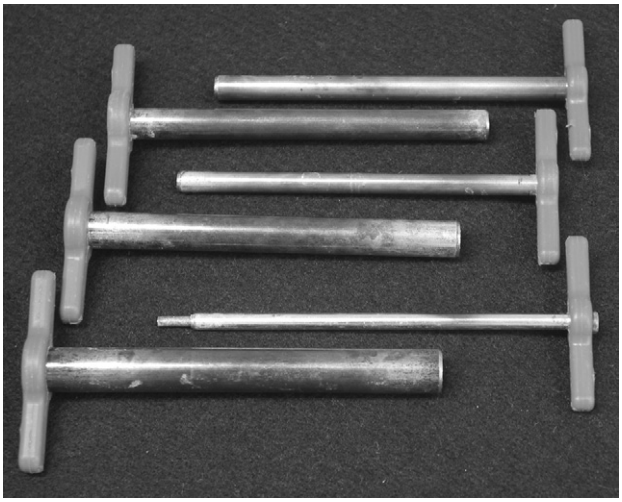

Figure 14-6. Core biopsy instruments used to remove the center of a tumor so that the same size of hollow spray probe can be inserted into the center of the tumor to perform penetration freezing.

Figure 14-7. Solid probes come in a variety of sizes, each fitted with a separate plastic handle that does not become chilled as the probe is immersed in liquid nitrogen. Various sizes and shapes allow these probes to become a heat sink when pressed onto the surface of the tumor.

and used to freeze tumors quite rapidly—a large advantage when multiple tumors need to be frozen in the same patient.

CRYOSURGERY TECHNIQUES

When using either contact or penetration cryotherapy, monitoring the depth of freezing can be done either by subjective inspection or by objective measurement of temperature changes. Subjective assessment is made by visual inspection or palpation of the ice ball. The outer edge of the ice ball is about 0° C (32° F), which is inadequate for tissue destruction. Seventy-five percent of the tissue within an ice ball is destroyed by freezing. The depth of contact freezing is estimated to be slightly less than the radius of the ice ball. Pyrometers can be used to measure the temperature achieved beyond the limits of the target tissue. Single- or multiple-channel monitors are available (see Figure 14-2). Needle probes are placed into the tissue adjacent to the deepest portion of the target. When temperatures of −20° C (−4° F) are recorded, all unwanted tissue is destroyed. Alternatively, ultrasonic evaluation of the margin of the ice ball

Figure 14-8. A special container is used into which liquid nitrogen is poured and the contact probe is submersed to attain the proper temperature before applying it to a tumor.

can be an accurate method of determining the extent of the freeze. If the tumor has a distinct ultrasonic appearance, it enables more accuracy in controlled freezing.[11,12]

COMPLICATIONS

Normal biologic reactions to freezing include swelling, bleeding, necrosis, depigmentation, and odor of varying degrees. Swelling occurs within hours of freezing because of increased vascular permeability and vasodilation. This is usually self-limiting and resolves in 48 hours. When lesions are biopsied or ulcerated and undergo cryotherapy, vasodilation after freezing can cause hemorrhage to become more obvious and may become cosmetically objectionable to an owner. Therefore, some form of hemostasis should be used during the biopsy procedure and on ulcerated lesions.

Necrosis occurs in 14 to 21 days. The wound contracts and epithelializes under a dry eschar that forms over the necrotic tissue. When the eschar sloughs, it usually reveals healthy granulation tissue or recurrence of the tumor. Because melanocytes and hair follicles are destroyed by freezing, the skin will show depigmentation and will not regrow hair. Owners need to be advised of this prior to treatment.

Offensive odors accompany necrosis of large tumors: cleansing of the area daily and excision of the necrotic tissue may be

necessary to ameliorate this problem. Freezing cortical bone causes cell destruction and reduces the strength of the bone by 70%. Spontaneous fractures have been reported months after cryotherapy treatment. Additionally, bone tumors do not respond well to cryotherapy, although aneurysmal bone cyst recurrance has been suppressed in people with cryotherapy used as an adjuvant to curettage.[13] Auricular cartilage does not respond well to cryotherapy either and can result in shortening or deformity of an ear. Therefore, cryotherapy should be used on skin tumors in the ears with caution.

FUTURE DIRECTIONS

The origins of cryotherapy in human medicine began in the 1960s, but enthusiasm for its use in cancer treatment dissipated in the 1980s. However, technologic advances in three areas have led to a renaissance in the interest in cryotherapy.[12] These advances are (1) intraoperative ultrasonography, as a technique for monitoring the tissue freezing process, (2) improved cryosurgical equipment, such as vaccuum-insulated small-diameter probes supercooled to –200° C, and (3) advances in instrumentation in mimimally invasive surgery. Additionally, the discovery that cryotreated tumor tissues are biophysically altered to allow enhancement of chemotherapy transport has sparked interest in combined cancer therapy.[14] Although these techniques are unlikely to be adopted into equine surgical practice anytime soon, because cancer is not a predominent problem in horses, some of these advances will make it into the hands of the equine surgeon as minimally invasive techniques become more commonplace.

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CHAPTER 15

Lasers in Veterinary Surgery

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Lasers expand surgical capabilities by facilitating minimally invasive surgery, by reaching areas that would otherwise be completely inaccessible, and by interacting with tissue in ways impossible with conventional instruments. Procedures previously requiring hospitalization, general anesthesia, and prolonged convalescence may be accomplished in an outpatient visit. However, lasers are not the most appropriate method for some procedures and the "fit" should not be forced.

Laser is an acronym for light amplification by stimulated emission of radiation. The excitation of a contained medium (for which the laser is often named) produces coherent electromagnetic radiation: light. The coherent beam remains intact almost indefinitely instead of diverging and can be manipulated by lenses. Lasers are typically monochromatic (a single wavelength or "color"), which determines interaction with tissue (Figure 15-1).[1]

FUNCTION OF LASERS

Surgical lasers produce a range of wavelengths (Figure 15-2) with varying tissue interactions, and understanding this is required to predict the laser's effect upon tissue. Behavior is determined by the degree to which the tissue absorbs the particular wavelength of laser energy. The more a tissue absorbs laser energy, the less it penetrates into the tissue and the more profound is the effect that is concentrated on the surface. Although deeper penetration allows controlled coagulation (denaturation of protein) of a larger volume of tissue, it may put associated deeper structures at risk of being injured. Complete lack of absorption of a wavelength by a tissue allows complete passage, thus affecting only a deeper tissue. Interaction between laser light and a tissue that preferentially absorbs that wavelength (apart from surrounding tissue allowing selective coagulation/necrosis of that tissue) characterizes the

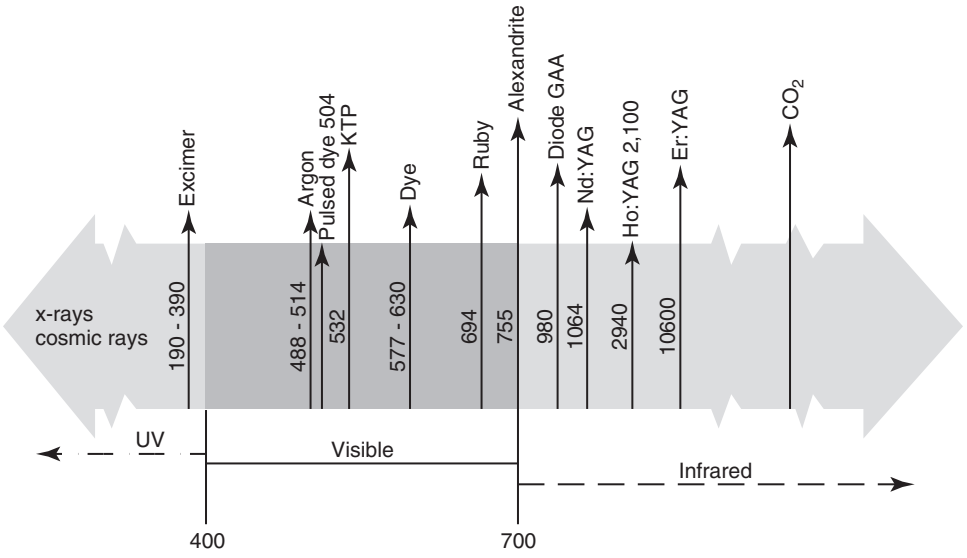

Figure 15-1. Wavelengths of surgical lasers. Ultraviolet wavelengths are generally absorbed by protein, whereas the visible and infrared wavelengths are generally absorbed by water or pigmented melanin or hemoglobin. Wavelengths in common veterinary use are in gray. *Er,* Erbium; *GAA,* gallium aluminum arsenide; *Ho,* homium; *KTP,* potassium titanyl phosphate; *Nd,* neodymium; *YAG,* yttrium aluminum garnet.

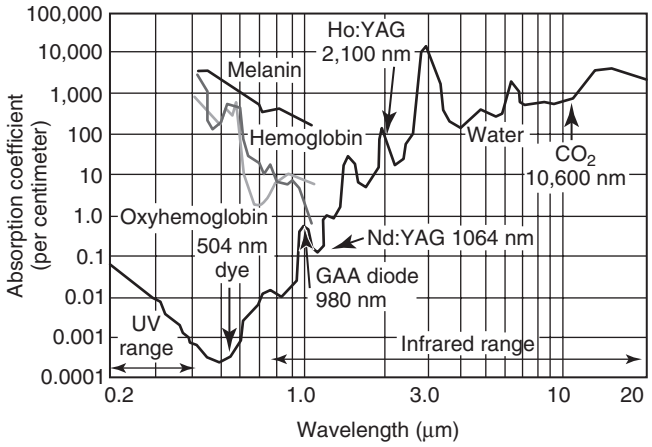

Figure 15-2. Tissue absorption of common surgical laser wavelengths. The visible spectrum is beneath the visible range. The near-infrared GAA Diode and Nd:YAG lasers are highly absorbed by dark pigment. However, note the increased absorption of the GAA Diode on the water curve compared to the Nd:YAG laser. The Ho:YAG and CO_2 lasers are both highly absorbed by water. *Er,* Erbium; *GAA,* gallium aluminum arsenide; *Ho,* homium; *Nd,* neodymium; *UV,* ultraviolet; *YAG,* yttrium aluminum garnet.

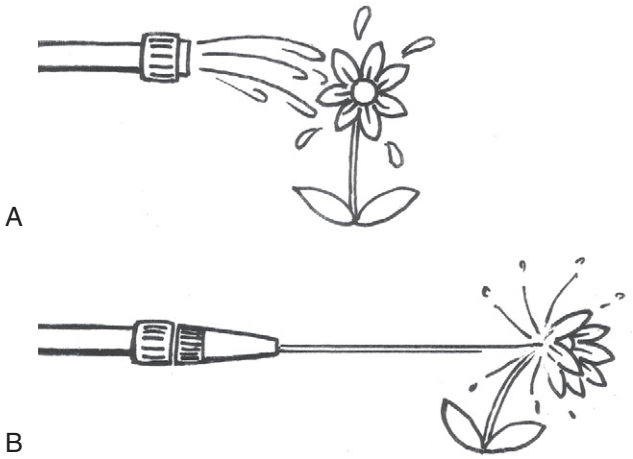

Figure 15-3. Power density profoundly affects rate of tissue effect and collateral heating of tissue. Both water hoses transmit identical flows of water. **A,** The wider aperture of delivery in the top image produces no mechanical effect on the flower. **B,** The narrower aperture in the lower image produces a jet of water that can disrupt the flower.

principle of *selective photothermolysis.*[2,3] By heating the target tissue above physiologic temperature but below that which would produce overt sloughing of tissue, the lesion will regress.

To "create" selective photothermolysis, pharmaceutical agents that absorb light of a particular wavelength may be administered systemically or locally. After these agents localize in a target tissue, such as a tumor, *photodynamic therapy* can be applied by using a laser emitting a wavelength that the agent concentrated in the tumor can absorb.[4-6]

A laser's effect upon tissue is due to optical and thermal interactions.[7] *Optical* interaction is the true result of absorption (or lack of it) of electromagnetic energy and usually results in a *thermal* effect when absorbed by tissue. Depending upon amount, heat may "boil" the cytosol, thereby vaporizing the tissue into the smoke plume, or simply denature tissue proteins. Optical interaction is the "true" effect of laser physics of the particular wavelength. When the optical interaction cannot achieve the desired effect, the irradiation is sometimes "artificially" converted to heat before applying it to tissue, which causes the energy to be absorbed at the tip of the fiber, thus producing heat and a profound surface effect on the tissue while minimizing penetration to deeper structures. Energy is measured in *joules* or calories (1 joule = 0.24 calories). Lasers are rated by power or the rate at which they can deliver energy, which is expressed in *watts* (W) (1 W = 1 joule/second). The total amount of energy delivered per unit area is *fluence,* expressed in Joules/cm^2, which depends upon time of exposure as well as *power density*. Power density (W/cm^2) is a critically important value that expresses the amount of energy delivered per unit area of tissue. Similar to water at a constant flow in a hose, laser energy delivered through a wider aperture will have a less profound effect than the same amount of laser energy delivered through a narrower aperture (Figure 15-3). Power density is varied by changing the output of the laser, by changing spot size of the laser beam delivered to the tissue (Figure 15-4), by changing the distance from the delivery device to the

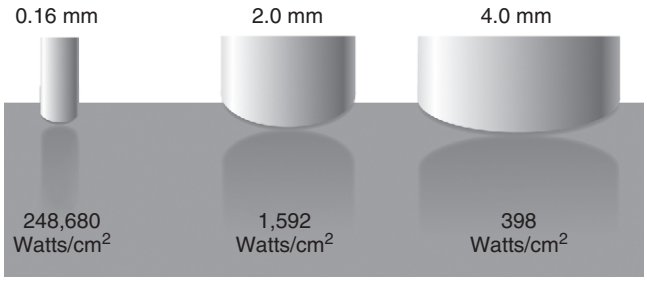

Figure 15-4. Power density decreases with the square of the increase in spot size, which in turn increases with distance from the surface. The beams depicted are all CO_2 laser beams from machines set to 50 W. The power densities shown below each demonstrate the profound reduction in tissue effect by increasing spot size. Moving the handpiece away from the tissue increases spot size.

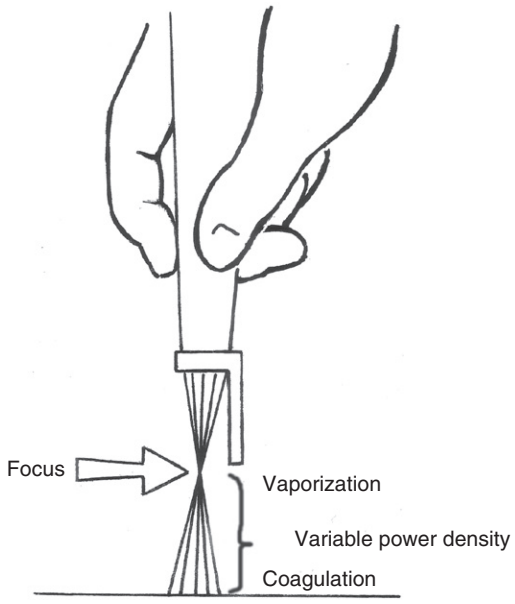

Figure 15-5. Focusing handpiece that would be used on an articulating arm of a CO_2 laser. The *arrow* points to the spot of maximum focus for creating a precise incision with minimal effect on margins of wound. The stylus contacts the tissue to fix the focal point and provide a feel on the tissue for making the incision. Below that point, power decreases with distance from the end of the stylus. Slight defocus allows vaporization of tissue with a relatively high power density, and more distance reduces power density to coagulation of tissue protein.

tissue (Figure 15-5), or by changing the delivery device. Power density (PD) varies with the square of the spot size and is calculated by the following formula, where s is the spot size in millimeters and W is the power setting of the laser.

$$PD(W/cm^2) = W/\pi[(0.1s)/2)]^2$$

Practically speaking, delivery of identical fluence values in different periods of time will produce different results. An acceptable full-thickness skin incision would be created with a 10 W laser beam delivered as a 0.4 mm spot size advanced along the skin for 5 seconds. (It just penetrates the skin completely.) If the rate of advancement is doubled (total time halved), the incision will be shallower because the total laser energy (fluence) has been halved. Conversely, if the original

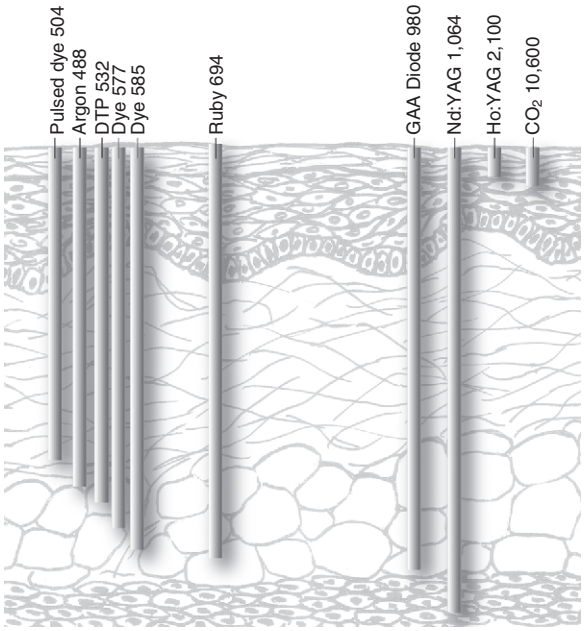

Figure 15-6. Absorption length of various wavelengths of surgical lasers in unpigmented skin. Wavelengths commonly used in veterinary medicine are in dark gray; wavelengths (nm) are stated beside the names. The far-infrared Ho:YAG and CO_2 lasers are highly absorbed by water; therefore, they penetrate minimally into skin, whereas the near-infrared Nd:YAG or GAA Diode lasers are absorbed more by the darker pigments of the deeper layers. *DTP,* Diagnostic and therapy systems for psychology; *GAA,* gallium aluminum arsenide; *Ho,* homium; *Nd,* neodymium; *YAG,* yttrium aluminum garnet.

time were doubled (slowed), the depth of the incision would increase beyond the skin. The same principle applies to any type of laser exposure.

The objectives of laser surgery fall broadly into three categories: incision/excision, ablation, and coagulation of tissue. Which of these occurs depends upon power density and absorption length of the laser, which in turn influence the rate of heat generation in tissue (Figure 15-6).[7] Incision/excision and ablation result in cell disruption and "vaporization" of tissue into smoke. Coagulation here refers to denaturing of tissue proteins, which grossly appears as blanching and contraction of tissue.

Excision is simply incising to dissect and remove tissue, and ablation refers to vaporization of tissue into smoke. Highly concentrated laser energy (i.e., high power density) is required to efficiently cut tissue with minimal heating of surrounding tissue. Because laser energy has no mass to separate tissue, tension on the tissue is necessary to separate the incised surfaces; without tension, excess heat will accumulate and the margins will be jagged and eventually necrotic. Collateral heating of tissue can be a substantial contribution to wound dehiscence because it produces a zone of necrosis along the wound margin. A small zone of necrosis has no effect on an open wound after resecting a mass, but it profoundly affects healing of a primarily sutured incision. Adequate power density to incise quickly[8] is critical to create a precise incision with healthy adjacent tissue to achieve primary wound healing.

A CO_2 laser in continuous mode at 50 W delivered with a 0.16-mm focused spot size yields a power density of 248,880 W/cm^2; a waveguide-delivered CO_2 laser at 8 W through a 0.4-mm

ceramic tip delivers approximately 6,300 W/cm^2. The former will produce an incision more efficiently, but it should be moved quickly across the tissue to limit penetration to the skin. The latter will produce an acceptable incision if tension is adequate to separate tissue and the waveguide is passed once and quickly across the skin. The skin defect will be 0.24 mm wider than the former with a perfect incision, which is clinically insignificant. With a small spot size, a single pass with efficient movement across the tissue and adequate tension on the tissue, 5,000 W/cm^2 is a minimally sufficient power density to avoid collateral thermal necrosis (Figure 15-7).[8] However, most experienced surgeons apply a significantly higher power density and work efficiently (Table 15-1). While learning, the tendency is to reduce the power setting and move tentatively or in multiple passes causing the laser to remain on the tissue longer and increasing the width of the wound and collateral heating. Incisions often dehisce because the margins necrose from thermal injury.

Reports of laser research should be examined closely to detect flawed methods. Incisions created with the CO_2 laser were reported to have reduced tensile strength upon healing, with more necrosis and inflammation compared to steel (scalpel) incisions, but the laser incisions were created using a power density of 1,990 W/cm^2, which resembles comparing a scalpel to electrosurgery.[9]

Laser energy is often delivered in *continuous mode* (i.e., uniform throughout application of the energy to tissue); some lasers have no other mode available. However, *pulsed modes* tremendously increase efficiency and minimize collateral heating of tissue. The principle is that spikes of laser energy at 200 Hz or more increase power density substantially while the interruptions allow tissue to cool slightly, which minimizes diffusion of heat into adjacent tissue.[10-12] A CO_2 laser in continuous mode at 50 W delivered with a 0.16-mm focused spot size yields a power density of 248,880 W/cm^2. In its pulsed mode,

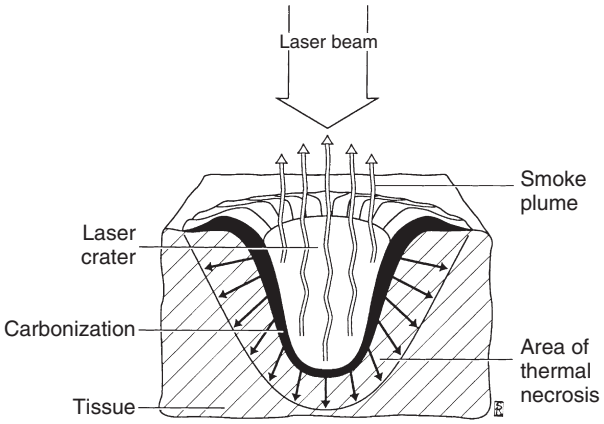

Figure 15-7. Range of tissue changes from laser beam. With sufficient power density, a laser beam has a central area of tissue vaporization/ablation shown by the crater in this drawing. A layer of carbonization occurs when tissue that has been significantly heated cools to produce char. The area of thermal necrosis is where tissue is heated beyond physiologic limits and sloughs later. The goal of incisive surgery is to use adequate power density to create as little carbonization and thermal necrosis as possible.

TABLE 15-1. Common Laser Techniques

Laser	Description	Capacity	Accessories	Preference for Skin Incision	Comments
GAA diode laser	Quartz fiber delivery	25-50 W	600 and 1000 µm quartz fibers. Handpiece to hold fibers.	1000 micron fiber sculpted down to approximately 600 µm at the tip.	25 W is insufficient for noncontact vaporization. 600 µm fiber too fragile for general surgery. Sterilize fibers for aseptic procedures.
Nd:YAG laser	Quartz fiber delivery	100 W	Gas cooled	Conical sapphire tip	Impractical to own both diode and Nd:YAG lasers.
CO_2 laser Articulated arm delivery	125-mm focusing handpiece. Minimum spot size 0.16 mm	Minimum 30 W	Computerized pattern scanner very useful for partial-thickness ablation of skin tumors or corneal tumors	30-50 W pulsed mode. Better hemostasis in continuous mode if wound is to be left open.	Sterilize handpiece and use sterile sleeve for aseptic procedures
CO_2 laser Waveguide delivery	0.25-4 mm (spot size) tips	15-40 W		Super pulse available	No lens focus of laser beam. Power density varied with distance, power setting or changing diameter of tip.
Laser smoke evacuator	Many brands available		Spare filters		Performance drops off quickly when filter fills. Sterilize hose for aseptic procedures.

GAA, Gallium aluminum arsenide; *Nd*, neodymium; *YAG*, yttrium aluminum garnet.

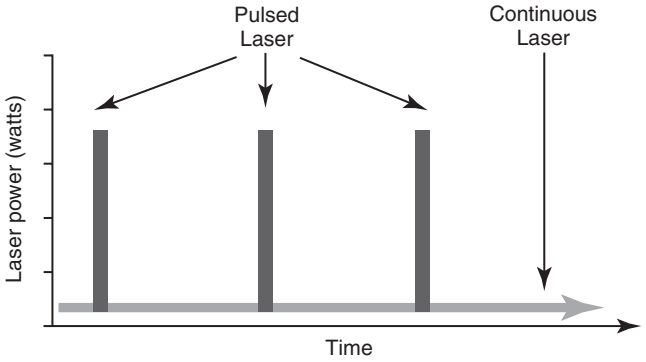

Figure 15-8. Pulsed laser energy compared to continuous laser energy. Pulsing higher power densities for short durations (vertical bars) produces a more efficient tissue effect with less collateral tissue heating compared to a continuous beam (horizontal bar) emitting the same average power (fluence). The tissue cools slightly between the pulses.

400-W power spikes provide power densities of 1,990,446 W/cm^2 while producing a fluence that is no more than with the continuous delivery (Figure 15-8). The technique depends upon the interval between laser exposure to avoid exceeding the thermal relaxation time of the tissue, which is the time required to cool 50% of the heat applied without conducting heat to the surrounding tissue. By supplying a second pulse before the tissue cools further, potential char is vaporized and tissue debris is evacuated as smoke or steam. This feature produces a skin incision with less collateral thermal injury than from a continuous wave.[13]

Laser energy can be delivered to the tissue in a *noncontact* or *contact* manner. As the term implies, with noncontact, nothing touches the tissue except the laser light, imparting a purely optical interaction. Sapphire tips touch tissue to deliver intense heat, and tips of quartz fibers produce varied interactions depending upon wavelength.

LASERS COMMONLY USED IN VETERINARY SURGERY

Carbon Dioxide Laser

The CO_2 laser is the classic instrument of general surgery. With only optical delivery, it has the convenience of having no fibers to stock or maintain (Figure 15-9, *A*). More CO_2 lasers than any other wavelength are in use in human and veterinary surgery.[14] This laser emits energy at 10,600 nm in the far infrared range; water absorbs this wavelength so completely that energy penetrates only 0.03 mm into tissue.[14,15] The ability to precisely control the effect makes the CO_2 laser safe for application to tissue overlying critical anatomic structures. Corneal squamous cell carcinoma can be ablated down to stroma without a deeper effect. However, heat can be conducted into normal tissue beyond the laser effect, which is of particular concern when applying laser to a thin structure, such as the cornea or an ear. A computerized scanner considerably reduces this risk (see later). When using continuous energy without a scanner, an ice pack on the back side of an ear is helpful.

When 50 W of energy is administered through a 125-mm handpiece to focus through a lens to a 0.16-mm spot size, the power density is 248,680 W/cm^2, which incises skin with 0.1 mm of collateral tissue effect. Microscopically, the "incision" actually has removed tissue; the narrower the spot size, the more natural the closure. Without changing settings, the handpiece can be retracted to defocus the laser beam to a 2-mm spot (1592 W/cm^2) or a 4-mm spot (398 W/cm^2), substantially changing the laser effect. The power density changes with the square of the spot size (see Figure 15-4). The surgeon must acquire the experience to achieve the spectrum of incision, ablation, or coagulation.

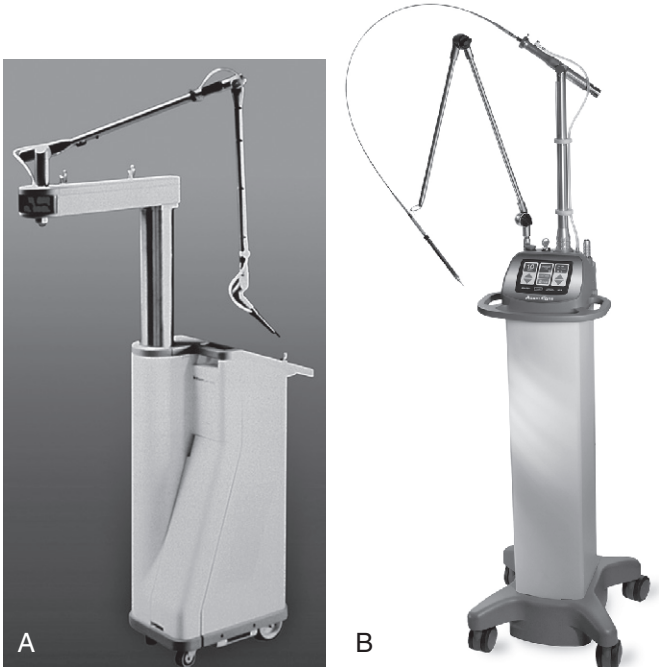

Figure 15-9. A, Typical higher-powered CO_2 laser delivered through an articulated arm with a lens-focusing handpiece. **B,** Typical CO_2 laser delivered through a flexible waveguide and handpiece with variable aperture tips. (**B,** Courtesy of Aesculight, LLC, Woodinville, WA.)

Hemostasis during CO_2 laser surgery is significant but not as profound as with lasers that penetrate tissue more deeply, even though lack of penetration is one advantage of using this laser. Hemorrhage from vessels 0.5 mm or less in diameter and lymphatic drainage are largely eliminated[14,16]; larger vessels or visible lumens should be ligated.

There is too much water in a quartz fiber to transmit CO_2 laser energy, so CO_2 lasers transmit the energy by reflection from mirrors through an articulating arm with a handpiece and lens to focus the beam (see Figure 15-9, *A*). Some models deliver the laser beam through a flexible waveguide with a handpiece, and spot size is varied by using interchangeable tips instead of a lens (Figure 15-9, *B*). CO_2 lasers are often equipped with pulsed modes (described previously), thus making incisional surgery similar to that of a steel scalpel possible.

Devices that attach to articulated arms of CO_2 lasers can deliver the beam laparoscopically, bronchoscopically, or arthroscopically. However, none of the instruments are flexible, and a gas medium is always required. The arthroscopic instrument has been applied to horses,[17] and it may be possible to adapt the other instruments for equine applications.

Some CO_2 lasers can be fitted with semiflexible waveguides to access deeper surgical sites. Waveguides are actually tubes and are not as flexible as quartz fibers. Many waveguides can be passed through the biopsy channel of some endoscopes, but they are fragile. Excessive bending will reduce the laser energy

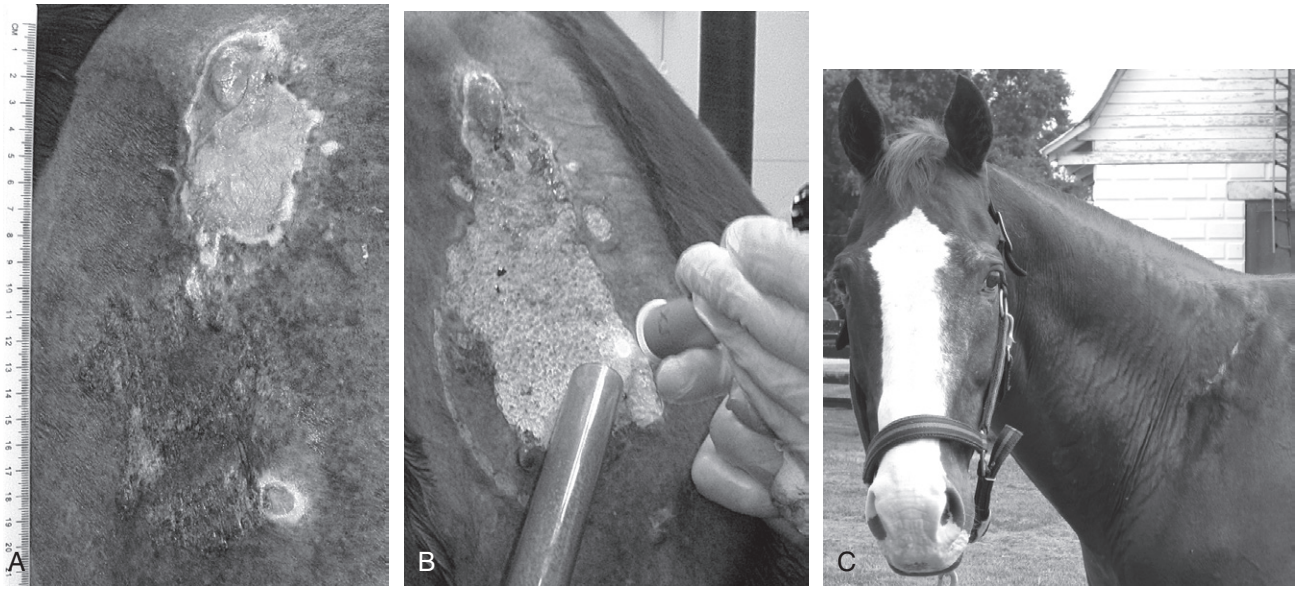

Figure 15-10. A, Preoperative image of large mixed sarcoid covering the scapular region of a horse. **B,** Computerized scanner attached to a CO_2 laser performing a partial (skin) thickness ablation of the sarcoid shown in **A.** The surface is even and there is no char formation. The entire lesion will be treated. Leaving the dermis intact facilitates healing and minimizes chance of recurrence. Topical fluorouracil was also used. **C,** End result of lesion shown in **A** and **B.**

or damage the waveguide, leading to a burnout; these should be kept relatively straight. One waveguide can function in a flexible endoscope, but it must be purchased with its specific laser (Omniguide).

Computerized pattern scanners are accessories that manipulate the focused laser beam across a preset scan size at a constant velocity to ablate tissue. Without a scanner, a slightly defocused beam is used to create a manual crosshatch pattern to vaporize a surface lesion, but char must be removed with a gauze sponge. The manual technique is workable but generates more heat and is less uniform than with the scanner (Figure 15-10). The difference between manual delivery and computer scanning is that scanners deliver focused laser energy, which ablates tissue completely. The beam moves away before collateral heating occurs and returns before the tissue cools sufficiently for char to form; less heating of deeper tissue occurs. The surgeon must acquire the "feel" of the scanner and keep it moving appropriately or it removes excessive tissue. The power settings should be kept low until the proper technique is acquired. Because this is focused laser energy, reducing the power simply slows the rate of surgery and produces no detrimental effect.

Equine general surgery holds many applications for the clean, efficient, and safe CO_2 laser.[18-20] Proper CO_2 laser surgery produces much less thermal injury than electrosurgery,[21] and tissue generally swells less than with conventional surgery. Surgical dead space tends to fill less with serum after laser dissection than with conventional surgical dissection.[22] In a gas medium, CO_2 laser energy can be used arthroscopically for specific applications.[17,23] Procedures requiring incision/excision of tissue or ablation of masses are all candidates.

Neodymium Yttrium Aluminum Garnet (Nd:YAG) and Gallium Aluminum Arsenide (GAL) Diode Lasers

The 1064-nm Nd:YAG and 980-mn GAL diode laser wavelengths behave almost identically in tissue, so the following discussion applies to both wavelengths. Many Nd:YAG lasers have been replaced by the less-expensive diode units. Nd:YAG lasers are generally sold with outputs up to 100 W, and GAL diode lasers are most often in the 15 to 50 W range. Higher power output is a reason some continue to use Nd:YAG lasers.

The immediately apparent difference between the two is the size of the "box" that produces the laser energy. The Nd:YAG laser is large and on wheels, whereas the GAL diode laser is on a table top or cart. The capability of the laser energy to be generated within very small semiconductor diodes versus a generating chamber results in this difference. The relatively few moving parts involved in the diode "box" cause it to be a very reliable instrument. Because lasers are manufactured with several semiconductors in the diodes, the more-specific term, *gallium aluminum arsenide (GAL) diode*, is used for this surgical laser.

In their purely optical forms, these lasers are absorbed by dark pigment (such as melanin and hemoglobin) and poorly absorbed by water (see Figure 15-2). When the tissue is not obviously dark, the laser energy will convert to heat more slowly as it encounters sufficient pigment or protein deep to the surface, which may take several seconds. That distance could be a few millimeters in pale skin or mucous membrane or a few centimeters in an eye if only cornea and clear aqueous or vitreous humor is encountered. As tissue blackens, more laser energy is absorbed until black char accumulates and limits penetration. To continue, the char must be physically removed or time for tissue to slough must be allowed.

Coagulation results in physical contraction of tissue, which will slough during the ensuing several days if the blood supply has been occluded. Vascular stasis occurs when melanin-rich tissues absorb the laser energy and conduct heat to the vascular endothelium, where the coagulation cascade is activated. In tissues with low melanin concentrations, hemostasis occurs when hemoglobin absorbs the laser energy and conducts thermal energy to plasma protein.[24,25]

The 980-nm diode laser is absorbed by water three times more than the Nd:YAG laser and lower-wavelength diode lasers.

The practical effect is a much more efficient contact incision in tissue in the upper airway with the 980-nm diode laser.

Deeply scattered laser energy can damage subsurface tissues, such as nerves or vessels, or coagulate darkly pigmented skin on the ear after passing through white cartilage of the pinna. Misdirected Nd:YAG laser energy in the pharynx can leave a horse dysphagic from damage to the pharyngeal branch of the vagus nerve, which lies deep to the dorsolateral pharyngeal wall. When deeper tissues are at risk, the beam should be directed tangentially across the surface or a contact technique should be used, and the integrity of the sculpted fiber or sapphire tip should be ensured (see later).

Diode and Nd:YAG lasers are the instruments of choice for equine endoscopic surgery because the energy is delivered through flexible quartz fibers, which can be inserted through the biopsy channels of video endoscopes. Two types of quartz fibers are in general use.

The *"bare" fiber* is covered with a plastic coating similar to insulation on an electrical wire. That plastic is stripped from the tip for use because it will melt and burn. After stripping, the end is cleaved by scoring the quartz and fracturing the fiber or cutting with scissors to yield a symmetric circle from the aiming beam. A uniformly circular shape of the aiming beam indicates the coherence of the light emitting from the fiber, which is important for uniform delivery of laser energy in a noncontact fashion. With normal use, bare fibers gradually crystallize and burn out, requiring cleaving back to a new area of the fiber, a continuous process until they are too short to use. Activating the laser only when the fiber is in contact with tissue prolongs the fiber life because tissue dissipates the heat. Bare quartz fibers are commonly available in diameters of 600 to 1000 µm.

Bare quartz fibers (Figure 15-11, *A*) used in contact fashion may be "sculpted" to a point to maximize the power density for incisive surgery. The sculpted tip burns away rapidly, leaving a fiber that is the same diameter as the entire fiber. The free beam (noncontact) effect of the fiber returns when the tip wears out. Adequate power density for cutting is generally provided with a 600-µm fiber at an output of 20 W. Larger-diameter fibers require more laser output or sculpting to maintain effective power density for incision and may emit excess laser energy into the deeper tissues at higher power settings. Sculpted 1000-µm fibers cut very well, and the sculpting will last for approximately one procedure. They are stiff enough to have a real tissue feel but may have difficulty bending to reach tissue during endoscopic surgery. Blackening the tip of a bare fiber by firing it on a tongue depressor or, more conveniently, with a black permanent marker causes the energy to be absorbed at the fiber tip so it cuts efficiently (Figure 15-11, *B*).

Noncontact application of laser energy requires relatively high power settings and high power densities for an adequate tissue effect. Smaller fibers transmitting 20 to 25 W can vaporize small areas but burn out very rapidly. With higher outputs, such as 50 W, more tissue effect is accomplished, but bare fibers still tend to overheat at these levels. A fiber burning out inside an endoscope can badly damage the scope.

Gas-cooled coaxial fibers contain a 600-µm quartz fiber passed through a plastic tube that conveys cooling gas or liquid (Figure 15-11, *C*). A metal tip joins the two at the end of the fiber, enabling the fiber to be used to deliver noncontact laser energy, or it can be fitted with a sapphire tip for contact lasing. Compared to the bare quartz fiber, higher powers can be transmitted without burning out the fiber. Care must be taken not to touch tissue with the cooling port because clogging will cause the fiber to burn out. If the fiber tip burns out, a new tip must be refitted or the tip must be replaced.[26] If the metal tip flares during burnout, it should be cut off from the fiber before withdrawing the fiber from the endoscope or the metal edges could lacerate the biopsy channel in the endoscope. Because they are generally higher powered machines, Nd:YAG lasers are equipped with mechanisms for gas or liquid cooling of coaxial quartz fibers. This capability can be added to diode lasers.

Holmium:YAG (Ho:YAG) Laser

The near-infrared (2100 nm) Ho:YAG laser is a pulsed laser that has been used in orthopedics, but more recently it has been applied in urology. The wavelength is substantially absorbed by water, an advantage for endoscopic ablation of soft tissue while protecting deeper structures.[27] The effect is enhanced in a water medium, which concentrates the energy within an air bubble formed where the laser contacts tissue. In an air medium, the delivery is noisy, and tissue is displaced slightly with each pulse. In the upper airway, the noise can be distracting for horses, and the delivery is not as precise as with the continuous Nd:YAG or diode lasers. This laser will ablate or "drill" cortical bone. I experimentally attempted distal tarsal articular cartilage débridement, but the fiber would not follow the contour of the joint and drilled into the depths of the distal tarsal bones.

The Ho:YAG laser has been used in equine arthroscopic surgery to remove palmar and plantar chip fractures of the proximal phalanx with good results.[28] The laser tip facilitated separation of the chip from the underlying bone and removal of hypertrophied synovial villi without bleeding. Additionally the fibrous tags could be vaporized to leave a smooth surface. However, this type of surgery was more time-consuming than the conventional chip removal with comparable results, which led to the cessation of Ho:YAG laser application.

The Ho:YAG laser is used for human[29,30] and small animal lithotripsy.[31] The Ho:YAG laser effect on uroliths has been described as primarily photothermal drilling of the stone[32,33] or surface ablation[34] compared to the broader effect described for the pulsed dye laser. The overall performance has been inefficient in horses,[35,36] but some smaller, less-compact equine uroliths have been successfully addressed.[37]

Pulsed Dye Laser

The laser-generating medium is an organic dye that is activated by a flash lamp or another laser, resulting in a visible 400 to 700 nm wavelength absorbed by hemoglobin and urinary calculi.[33] Some machines allow variation (tuning) of the wavelength.[33] Lithotripsy is performed in a water medium, producing a combination plasma formation and the photoacoustic effect derived from the pulsed delivery. Plasma formation is the result of focal accumulation of charged gas and ion particles resulting from the true optical laser-tissue interaction; mineral disappears in a manner similar to smoke in an air medium. Subsequently, expansion of minute cavities of steam along with the photomechanical energy from the pulse cause the stone to fragment.[33] Quartz fibers, which are generally smaller (200 to 320 µm) than for other lasers, are used for lithotripsy. Fiber rigidity is not necessary because it is strictly a contact "end-on" procedure. However, the small-diameter fibers are fragile and expensive.

The pulsed dye laser has proven useful for equine laser lithotripsy.[38,39] Calcium carbonate uroliths can be removed from

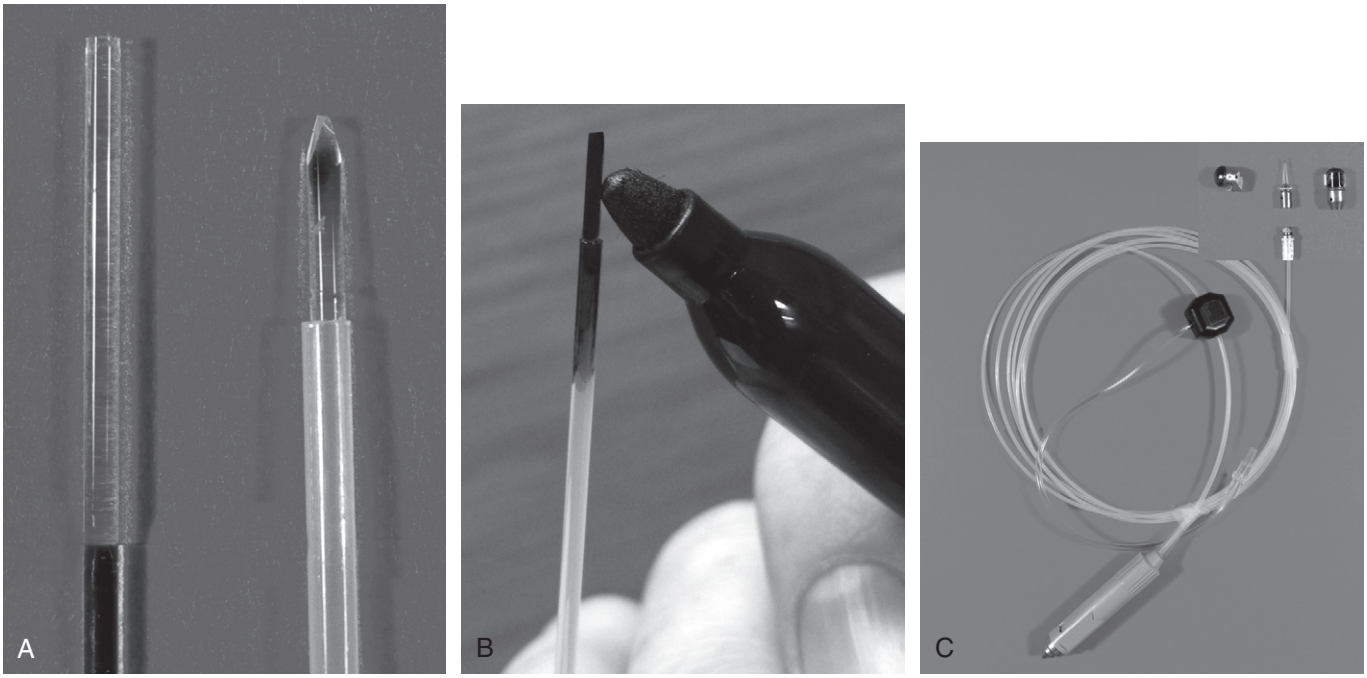

Figure 15-11. A, Bare quartz fibers (1000-μm) for use with Nd:YAG or diode lasers. The fiber on the *left* is a plain cylindric tip for free beam (noncontact) transmission of laser energy. The fiber on the *right* has been sculpted into a chisel point to increase power density for contact laser surgery. Both ends eventually burn out, requiring stripping back the plastic coating and cleaving the quartz in a fresh site. Although it is possible to manually resculpt the tip, it is tedious and not as accurate as replacing the fiber. **B,** A bare quartz fiber is being blackened with a permanent marker. The black pigment absorbs the laser energy for an immediate effect on tissue and limits deeper penetration of laser energy. As the marker pigment burns off, the heat itself and tissue char blackens the fiber for continuing until the tip must be cleaved again. **C,** Gas-cooled fiber for use with the Nd:YAG laser. The quartz fiber inside the plastic tubing can transmit 50-100 W of energy without burning out because the gas circulating in the tubing cools it. The ports in the tips *(inset)* must remain clean for cooling to continue. The fiber can be used in noncontact fashion with the bare tip only, or sapphire tips of various types can be screwed onto the tip. Illustrated in the *inset, left to right,* are right angle, conical, and end-on sapphire tips. The conical tips are used for incisions, and the others are used for contact ablation of tissue.

geldings by access with a videoendoscope through the penile urethra (see later). The Ho:YAG laser has largely replaced this instrument for human lithotripsy because of its applicability to multiple procedures and reduced maintenance requirements.

GENERAL SURGERY

The CO_2 laser is the workhorse of general surgery. As a scalpel or scissors replacement, it makes incisions or dissections that are clean, dry and efficient. Because the laser beam has no mass to separate tissue, as a blade does, tension on the tissue is absolutely necessary to achieve the separation and minimize collateral heating of adjacent tissue for optimal wound healing. For a conventional incision, thumb and forefinger suffice; tissue forceps or towel clamps provide extra purchase for more extensive dissections. Tissue will separate along planes with the proper combination of laser energy and traction. The power density (see earlier) can be varied with distance of the handpiece from the tissue to facilitate seamless transition from precise incision to coagulation or ablation of tissue. Small vessels will be sealed; blood flow must be stemmed with pressure to coagulate walls of larger vessels. Larger vessels can be included in the deep coagulation, but ligation is advisable for visible lumens. Subtle superficial coagulation of surfaces of dead spaces will minimize seroma formation, but dead spaces should also be minimized during closure by using conventional techniques. Some surgeons "paint" the wound surfaces with defocused laser energy to vaporize remaining tumor cells after resection. The CO_2 laser effect is significantly less than a millimeter; intentional heating of the tissue may result in delayed necrosis and slough of tissue. The laser should be considered the same as a scalpel in this respect; that is, margins for tumor resection should be adequate. Local chemotherapy widens the margins.

The Nd:YAG and diode lasers with handpieces can also be used for incisive procedures, but the skin margins will never be as precise as with a properly created CO_2 laser incision. Good results can be obtained in this manner, but primary closures should have sutures set back 2 to 3 mm from the margins. Mass excisions with wounds to be left to heal by second intention will heal normally, but smaller fiber diameters will require frequent cleaving, so a sculpted 1000-μm fiber or a sapphire tip will be more efficient. Hemostasis can be accomplished by using the contact tip to compress the vessel to stem the flow and applying *low* energy (3 to 5 W). Higher power densities will simply transect the vessel.

One advantage of laser over conventional surgery is the capability to ablate (vaporize) tissue, particularly masses. The CO_2 laser ablates all tissues efficiently because most tissues have high water content. Any handpiece can be used to ablate tissue by backing away from the tissue to reduce power density slightly from the incisive mode and increase the spot size for more efficiency. The effective distance for the specific handpiece can be quickly determined by observing of the effect; deeper tissues

are safe because of the limited absorption length of the CO_2 wavelength. The handpiece is patterned across the tissue until the desired depth is obtained. Occasional interruptions limit overheating of deeper tissues. On thinner structures, such as ears, holding an ice pack on the opposite side will minimize overheating of tissue.

A computerized scanner (see earlier) is a significant advantage of ablating with a CO_2 laser. The result is a very clean wound bed with healthy tissue remaining (see Figure 15-10). Partial-thickness ablation of thin cutaneous equine sarcoids, ablation of corneal cell carcinoma, or reduction of granulation tissue are examples of procedures that commonly use the scanner.

Noncontact ablation of tissue with the Nd:YAG and diode lasers is possible as well. Unpigmented or pale tissues respond only after a few seconds of lasing has caused the tissue to darken. The surgeon must remember that transmission of this wavelength of laser energy can be several millimeters, so deeper structures must be respected.[40] Darkly pigmented skin or melanomas begin to vaporize readily. As the process continues, superficial (black) char will accumulate and completely absorb the laser energy, necessitating its removal before proceeding.

Noncontact ablation requires a high power density to span the distance to the tissue and vaporize. To be effective with a plain fiber, a higher wattage laser with a larger diameter fiber is needed. Gas-cooled fibers can transmit much higher power and are much more efficient for vaporizing tissue; the gas ports in the tips must be kept clean for proper cooling.

ENDOSCOPIC SURGERY

Endoscopic laser technique has expanded the breadth and depth of upper airway and urogenital tract surgery as well as in laparoscopy. New applications appear as needs arise. Endoscopic surgery is much different from endoscopic examination and requires instruction and practice to avoid injury to the patient or damage to the equipment. Video endoscopy vastly improves execution of procedures. For some procedures, a dual channel endoscope improves efficiency because one channel can be used for suction to evacuate blood or fluid without taking time to remove the laser fiber from the other channel. Efficient completion of the surgical procedure is very important because hemorrhage and swelling (which hinder visualization and laser delivery) generally increase with time. A consistent operating space tremendously improves efficiency and outcomes (Figure 15-12).

Because quartz fibers pass readily through endoscopic biopsy channels, Nd:YAG and diode lasers dominate this area of veterinary surgery. In general, contact incision is more precise and safer for deeper tissues than noncontact ablation, particularly in the upper airway. However, if the target tissue mass is known to conceal no critical structures (such as an ethmoid hematoma), noncontact ablation may be the best choice. In contact incision, the laser energy is applied at the tip of the laser fiber, which must contact the tissue to be effective. Take care to not place the side of the fiber on the target tissue (like a scalpel would be placed) because the tip of the laser could burn tissue beyond the field of view. The laser should never be activated if the target tissue cannot be visualized. Movement of the tip of the fiber or sapphire tip across tissue is a practiced feel; it should be moved slowly to allow the laser to cut while traction is held on the tissue. If insufficient or no laser activity is occurring, immediate cessation of the procedure and inspection of the fiber is critical. A malfunctioning fiber may be damaging the endoscope. The fiber should be allowed to cool a few seconds after lasing stops before retracting into the biopsy channel to prevent damage to the endoscope.

Figure 15-12. Operating facility for standing endoscopic surgery. The video endoscopic monitor faces the surgeon, making all the movements in the patient mimic those on the screen. The floor around the surgeon and assistants is free of cables or other debris.

Accessory instruments are necessary to provide traction and retrieve tissue, such as a long tissue-grasping forceps. The preferred forceps is a heavy-duty 75-cm forceps with Oschner-type jaws (Figure 15-13). The vertical action of the jaws is universally effective, but instruments with horizontal action or rotating jaws are on the market. Other instruments may be adapted or built for specific needs for various procedures.

All standing upper airway laser procedures require profound sedation for the patient to tolerate an endoscope in one nostril and accessory instruments in the other. Access to the caudoventral pharynx and larynx is facilitated by complete extension of the head; support of the rostral mandible or suspension of the halter is helpful (see Figure 30-2). Topical mepivacaine sprayed over the target tissue and normograde through the opposite nostril via the endoscope reduces reactivity. Persistent swallowing is addressed more effectively by increasing sedation whereas pharyngeal spasm requires additional topical analgesia.

Horses undergoing upper airway laser surgery are treated with local and systemic anti-inflammatory medications (Table 15-2). Excessive granulation tissue can be a serious postoperative complication if medications are not administered.

Laser Thermoplasty of the Soft Palate

Dorsal displacement of the soft palate (DDSP) is a poorly understood condition affecting racehorses. The rationale for laser thermoplasty is that the ensuing scar tissue formation will stiffen the soft palate, making it more difficult to displace from beneath the epiglottis.[41] Immediately before the standing laser thermoplasty, perform a standing bilateral sternothyroideus tenectomy to facilitate rostral movement of the larynx into the caudal margin of the soft palate where possible.

Horses with significant pharyngeal inflammation from many causes may present with secondary DDSP, which is not the same

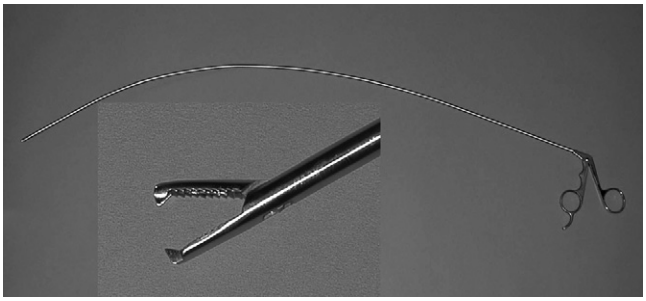

Figure 15-13. Esophageal grasping forceps (75-cm) used to provide traction for endoscopic laser surgery in the upper airway. (1404-881MT, Sontec Instruments, Centennial, CO.)

TABLE 15-2. Medications for Upper Airway Laser Surgery

Drug	Dosage
Dexamethasone*	20 mg IV
Prednisolone	1 mg/kg PO sid × 5 days 0.5 mg/kg PO sid × 5 days 0.5 mg/kg PO sid every other day 5 times
Phenylbutazone	2.2 mg/kg PO bid × 14 days
Oral antibiotic	PO 14 days
Throat spray†	20 mL bid via nasal tube × 21 days

*Given preoperatively.
†Formula: 225 mL glycerine, 50 mL dimethyl sulfoxide (DMSO), 50 mg dexamethasone, qs to 500 mL with saline.

situation as described previously. Correcting the underlying problem often corrects the DDSP, and laser thermoplasty in the presence of inflammation would be contraindicated.

Unless the soft palate will remain displaced (rare), the epiglottis must be elevated for access to the caudal margin of the soft palate, but the epiglottic cartilage should not be grasped or abraded. Use a diode laser set at 20 W with a 600-µm fiber applied in a "pin fire" contact fashion. The laser fiber is lifted from the tissue just as the pedal is released to prevent its sticking to the tissue; keeping the laser activated too long causes the fiber to burn out rapidly. A rhythm is soon acquired. Particular attention should be given to the caudal margin of the soft palate without injuring the subepiglottic mucosa; the entire treated area is slightly wider and longer than the epiglottis in its normal position, which requires approximately 1500 J. Care is taken to make pinpoint contacts and not linear incisions in the soft palate (Figure 15-14). Postoperatively, horses are treated with prednisolone, phenylbutazone, and an oral antibiotic (see Table 15-2). Training can be resumed approximately 10 days postoperatively, provided that the soft palate has healed completely and pale scar tissue has formed. More time is required if the surface remains ulcerated or hyperemic because fibrosis is incomplete.

Laser Ventriculocordectomy (LVC)

The ventricle and/or vocal cord (also called vocal fold) have been variably removed with or without prosthetic laryngoplasty (PL) for many years. Bilateral conventional ventriculocordectomy without laryngoplasty reduces the noise associated with

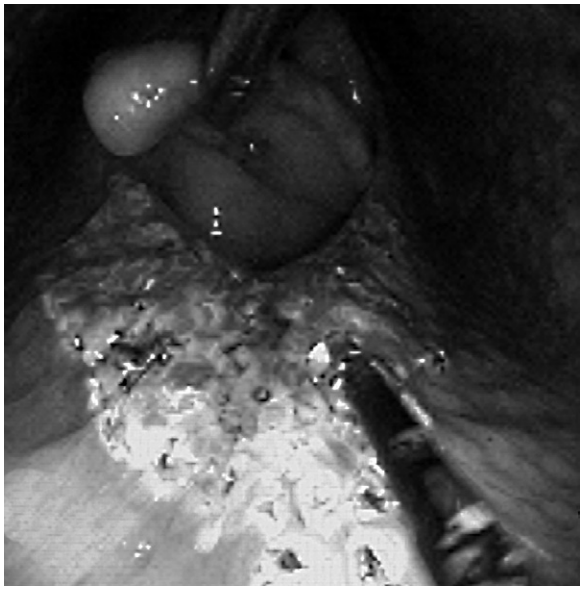

Figure 15-14. Laser thermoplasty of the soft palate. The epiglottis is elevated for access to the soft palate. A 600-µm quartz fiber is being applied to cover slightly more than the area of the epiglottis. Particular attention is given to the caudal margin.

left laryngeal hemiplegia (LLH),[42] and left LVC using a laser reduces airway noise and restores airway pressures to normal in experimental horses with LLH.[43] Those horses were shown to have complete removal of the ventricle and a solid arytenoid-thyroid cartilage fibrous adhesion to prevent arytenoid collapse during exercise.[44] From these data, LVC using the laser would seem to stabilize the arytenoid position after PL, and PL (when performed) would seem to facilitate the position of the adhesion following LVC. Although all horses with LLH can return to normal work with a left LVC, occasional failure of that scar has been observed in racehorses presumably as a result of repetitive and prolonged negative upper airway pressures. Perform LVC in all horses with LLH,[45] but racehorses and other high-level athletes also have a prosthetic laryngoplasty (see Chapter 45).

Noncontact laser mucosal ablation to obliterate the ventricular space has been reported.[46] Contact excision of the ventricle and vocal fold is preferred to obtain a tight arytenoid-thyroid adhesion[44]; no ventricular mucoceles have been reported using this technique. The dissection can be performed using a single grasping forceps for everting the ventricle and dissection of the vocal fold.[47] A transnasal sacculectomy burr that everts the ventricle and provides traction and tension on the vocal fold facilitates the procedure (Figure 15-15, *A* and *B*).[43,48]

Perhaps the single most important factor in performing LVC is efficient execution of the surgery because delay allows hemorrhage, which obscures vision and absorbs laser energy. The most consistent significant vessel encountered is located at the ventral aspect of the vocal fold, so it helps to save this area until traction can be applied to minimize the hemorrhage. Using the burr, dissect ventrad-to-dorsad while rotating the burr clockwise to elevate and retract the tissue. Repulsion and traction with the burr or manipulation with long grasping forceps may be required to complete the procedure; each case is slightly different. Care should be taken not to apply direct thermal energy to the arytenoid cartilage. Leaving a small amount of soft tissue on

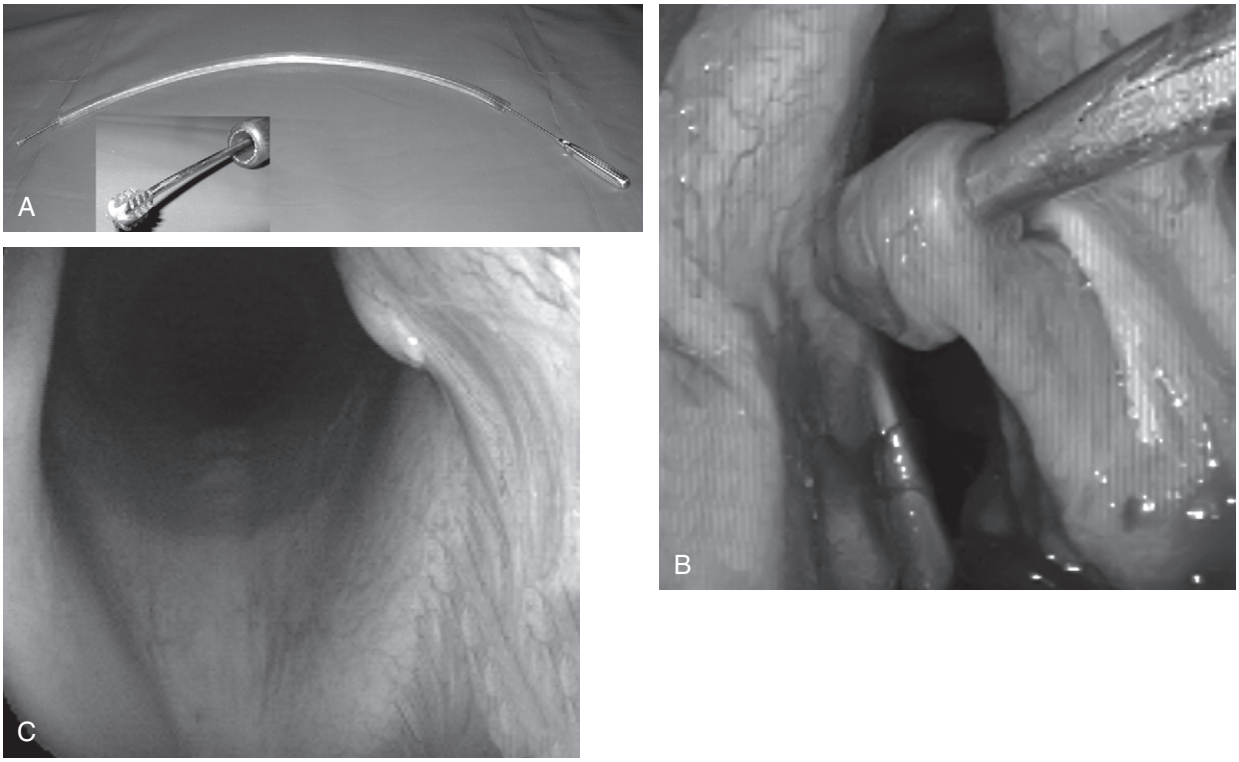

Figure 15-15. A, Transnasal sacculectomy burr shown in a segment of stomach tube used to safely conduct the burr to and from the larynx. (Virginia Roaring Burr, 1271-283 engages clockwise for the left ventricle. 1271-284 engages counterclockwise for the right ventricle. Sontec Instruments, Centennial, CO.) **B,** The transnasal sacculectomy burr has everted the ventricle. The clockwise torsion of the tissue provides traction, facilitating laser dissection of the tissue, and minimizes hemorrhage. **C,** Laser ventriculocordectomy site after healing.

the vocal process of the arytenoid cartilage facilitates faster healing. Approximately 10,000 J is required to efficiently complete the procedure using a 600-µm quartz fiber with the diode laser set to 20 W.

Postoperative care consists of 30 days of stall rest with unlimited hand walking and 30 days of light turnout in a small paddock. Training can resume during the third month but speed should be reserved for the fourth month to allow complete maturation of the adhesion (Figure 15-15, *C*). Early airway turbulence (including continuous screaming) or failure to properly medicate may cause excessive granulation tissue to develop; small to moderate granulation tissue masses usually shrink without treatment. Medications include the entire list in Table 15-2.

Epiglottic Entrapment

Epiglottic entrapment (EE) refers to the dorsal displacement of the aryepiglottic fold to stretch over and envelop the epiglottic cartilage (see Chapter 44). The goal of surgery to correct EE is to transect the restricting aryepiglottic fold effectively, lengthening it so it can no longer maintain the position; the procedure effectively converts the entrapment to a subepiglottic ulcer. The advantage of endoscopic laser transection is that neither general anesthesia nor laryngotomy is required.

Contact[49] and noncontact[50] laser techniques have been described. Both consist of sequential rostrad-to-caudad removal of tissue covering the epiglottis until it divides. The contact technique requires a wedge-shaped sapphire tip to separate the tissue without a separate traction instrument. With more substantial long grasping forceps available, the entrapment can be grasped and elevated from the epiglottic cartilage and transected in a vertical caudad-to-rostrad manner, which is more efficient and keeps the laser fiber away from the epiglottic cartilage (Figure 15-16). For thicker entrapments, a small dorsal incision may be created for the forceps to grasp. A diode laser set at 20 W using 600-µm quartz fiber can accomplish the transection in 5 minutes or less.

Some entrapping membranes are attached just ventral to the tip of the epiglottis and will not fall away when transected; care must be taken not to damage the cartilage before this is known. The membrane can be repositioned ventral to the epiglottis with the grasping forceps as the incision nears the epiglottis to evaluate how much should be transected. If the aryepiglottic fold remains tight enough to partially entrap when no more tissue is available to transect at the tip, releasing incisions in the aryepiglottic folds can be created along the sides of the fold. When transection is completed, the horse should be stimulated to swallow several times. Extremely thick entrapments may not disappear below the soft palate. If the epiglottis has been released, remaining tissue will contract with rest and medication. Further dissection of thick soft tissue from the ventral surfaces of the epiglottis is not advisable because increasing scar tissue may limit mobility of the epiglottis.

Postoperative care consists of topical and systemic medications and airway rest. All the medications listed in Table 15-2 are administered. The time required to heal depends upon the ulcer created by the transection. Thin entrapping membranes heal rapidly and horses can return to training relatively quickly,[49] but chronically thickened entrapments already have a granulating mass that must regress. If the subepiglottic mass fails to

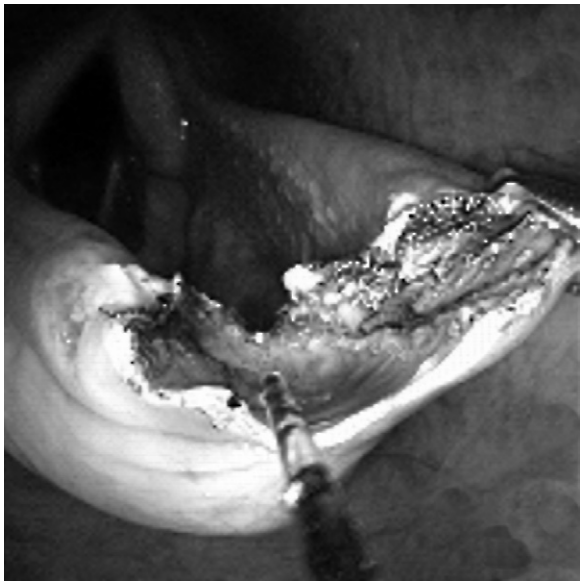

Figure 15-16. Transection of a moderately thickened epiglottic entrapment. The grasping forceps elevates and stabilizes the entrapping membrane while the laser fiber transects the membrane dorsally to ventrally. The membrane should be reduced below the epiglottis periodically to be sure the laser doesn't contact the cartilage.

regress or enlarges, it may physically displace the soft palate from its normal position ventral to the epiglottis. Monitoring of the healing process requires elevation of the epiglottis to examine the healing ulcer. Ideally, mucosal healing will be complete before training resumes, but a few horses will retain a small, mature-looking ulcer that will not seem to cover with mucosa or be a problem for training.

Occasionally a healing subepiglottic lesion granulates seemingly out of control; airway stertor at rest may appear. It should be checked endoscopically, but the throat spray should be given four to six times daily until it subsides. Further surgery on an inflamed area is not advisable.

When the entrapment has been relieved, the tip of the epiglottis may appear swollen and hyperemic from the pressure that has been there. Some of these epiglottic cartilages shorten in time in apparent atrophy from this inflammation, which may predispose to future DDSP. If that occurs, procedures to correct DDSP may be needed (see Chapter 44).

Although most horses with EE are having performance problems, one endoscopic survey of horses in training found EE as an incidental finding.[51] Additionally, some horses with EE and an obviously deformed epiglottis perform at least acceptably. If these horses are taken from training for surgery and then must regain their fitness with a shortened epiglottis, they may never regain their previous level of performance. The entire situation should be considered before deciding when to remove a horse from training to correct EE.

Guttural Pouch Tympany

Inflation of the guttural pouch(es) (GP) is a rare condition of young foals caused by the salpingopharyngeal orifice(s) functioning as a one-way valve. Unilateral or bilateral outward distention gives the foal a chipmunk-like appearance (see Chapter 46). The majority of cases are unilaterally affected, and treatment consists of perforating the median septum separating the two guttural pouches, thus allowing air to escape through the normally functioning side.[52] Another option is the creation of a new salpingopharyngeal opening on the affected side.[53] Bilaterally affected foals must have the median septum perforated and at least one salpingopharyngeal communication established. Alternatively, two salpingopharyngeal communications can be created without perforating the median septum.[54]

The procedure(s) can most often be performed in the standing foal. If general anesthesia is contemplated, pneumonia may be a factor. Laser division of the median septum is accomplished by driving the endoscope axially immediately after entering the GP. The membrane lies rostral to the vertical longus capitus muscle and may be slightly thicker than normal in affected foals. Entering the unaffected GP in unilateral cases usually reveals the septum displaced convexly into the unaffected GP, facilitating the initial contact. Cranial nerves IX through XII lie along the caudal margin and axial floor of the medial compartment, making the more-controlled contact laser perforation the preferred technique.

A 600-µm quartz fiber makes the axial bend easily; a diode laser set at 20 W is sufficient for the incision. Using an endoscope with the biopsy channel at 6 o'clock in the field of view, it is helpful to rotate the endoscope 90 degrees so the fiber presses easily against the membrane. The fiber is positioned well into the dorsal half of the septum at the caudal margin in preparation for a caudad-to-rostrad incision toward the endoscope. When the membrane is perforated, the fiber must be advanced appropriately to maintain contact of the tip of the fiber with the tissue. The hot fiber tip should not come too close to the endoscope. The incision will open a respectable hole in the membrane that is certainly adequate for airflow. However, many such perforations will heal closed[52]; removing a segment of the membrane is more apt to create a permanent opening (Figure 15-17, *A*). A section of membrane is removed by creating two parallel horizontal incisions followed by vertical transection of the rostral attachment. The dangling tissue is subsequently grasped with a long forceps inserted into the opposite GP opening to retract the tissue while the caudal attachment is severed. If the surgeon elects not to resect a piece of the membrane, the perforation should be monitored for a few weeks (Figure 15-17, *B*). If it begins to close, a Foley catheter can be placed for 3 weeks.

The simplest salpingopharyngeal orifice creation is ablation of the pharyngeal mucosa just caudad to the cartilage flap (Figure 15-18).[53] A Chambers catheter is inserted into the GP flap and rotated so the round tip presses axially, creating a bulge in the mucosa caudad to the external cartilage. The perforation must be created caudad enough to prevent the inner cartilage from obstructing the new opening. A noncontact Nd:YAG or diode laser can be used to vaporize the tissue overlying the catheter tip or a contact fiber can be rotated over the area until the fenestration is complete. A Foley catheter must be placed in the new opening for 7 to 10 days or until the tendency to close has passed. Alternatively, the pharyngeal cartilage flap of the GP opening may be dissected away using a contact technique and long grasping forceps for traction on the tissue.

Rarely, pharyngeal collapse caused by guttural pouch distention occurs in an adult horse. Although the affected guttural pouch will be asymmetrically distended and confirmed by radiography, the external distention typical of foals is not present. Treatment for adults is the same as for foals.

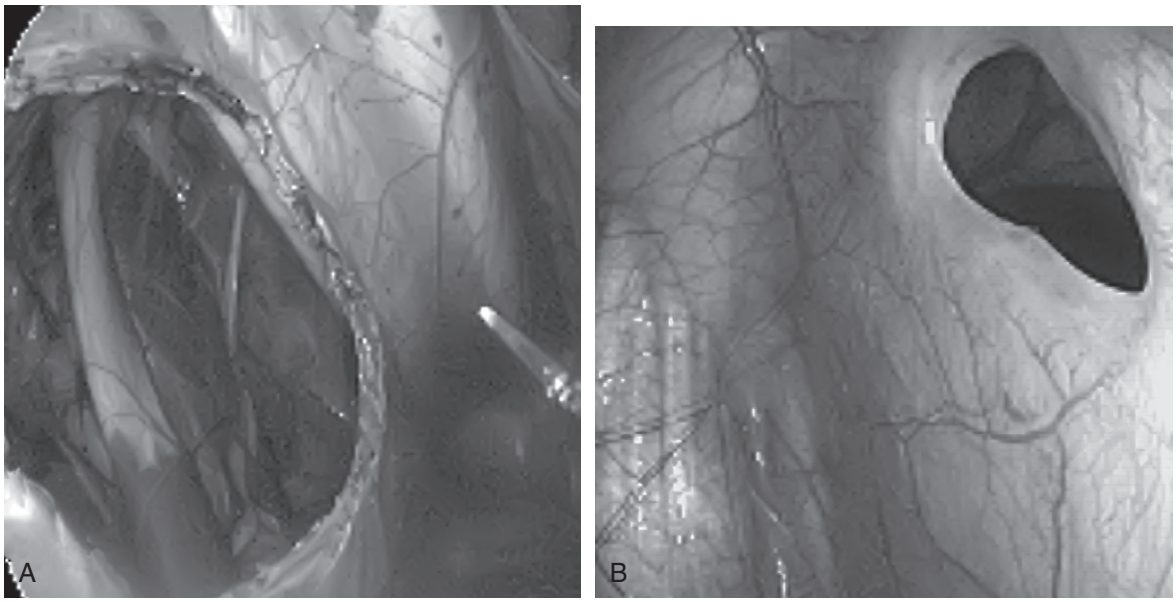

Figure 15-17. A, A segment of the guttural pouch septum has been resected, creating a large defect. This should be monitored for a few weeks to be sure it does not heal closed. **B,** The healed defect in the guttural pouch septum is usually much smaller than it was immediately postoperatively and often closes completely.

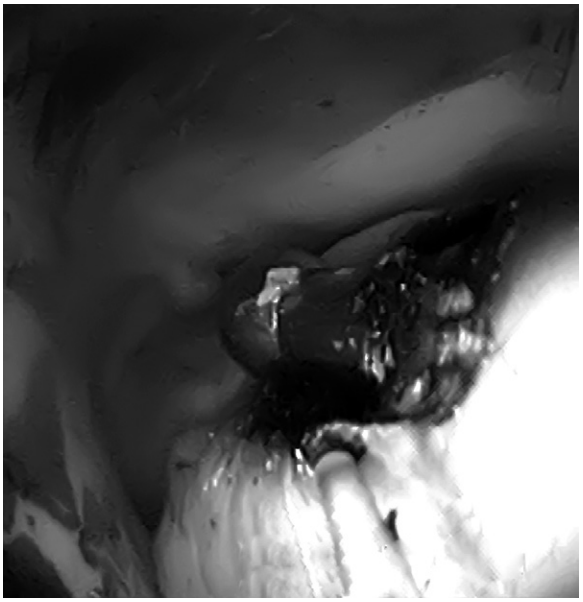

Figure 15-18. A salpingopharyngeal opening has just been created caudad to the left guttural pouch to correct tympany. The Chambers catheter *(arrow)* is protruding through the new opening; a Foley catheter will be placed through the guttural pouch flap opening and out this new opening to hold it open while it heals. The dorsal pharyngeal recess is visible in the upper left of the photo.

Progressive Ethmoid Hematoma (PEH)

Although usually progressive, in that these lesions enlarge over time, PEH is not always located on the ethmoturbinates and is not a simple hematoma. The laser is of limited value for lesions requiring frontonasal bone flaps for access because they must be conventionally debulked. Lesions visible by nasopharyngeal endoscopy may or may not be confined to the nasopharynx. Particularly lesions located against the lateral wall above the ethmoid shelf may also involve the frontal sinus. The laser is useful for lesions confined to the nasopharynx or ethmoid region. Radiographs should be taken to rule out sinus involvement, although these may be interpreted more easily after removal of obvious nasopharyngeal masses.

If the lesion is small, noncontact ablation with a higher-power Nd:YAG laser transmitted through a gas-cooled quartz fiber is feasible. For larger masses, it is important to treat the base of the PEH definitively; how the (usually) more rostral portion of the mass is removed matters little. Choices for debulking the larger masses include: formalin injection; snaring and amputation with obstetrical wire looped through a stomach tube; or simply grasping the lesion with a long instrument, such as a long sponge forceps, and pulling it out. If removal is incomplete, a combination of these methods is possible.

When the base of the lesion is visible, vaporization with the Nd:YAG laser is effective. Packing the ethmoid shelf with gauze sponges soaked in formalin is an effective ancillary treatment. The sponges are tethered to a long heavy suture that is stapled to the skin outside the external nares for overnight at least. The packing can be recharged with formalin through an endoscope or an endoscopically guided artificial insemination pipette if desired; injecting with the instrument held tightly against the packing soaks better and minimizes formalin dripping. The area should be endoscopically monitored for recurrence periodically for the first year.

Subepiglottic Cysts

Subepiglottic cysts (SCs) occur in foals and adults (see Chapter 45). These lesions may be associated with upper airway noise, respiratory disease, dysphagia, or poor performance[55,56] and may be asymptomatic at rest. Small SCs can be found in epiglottic entrapments and may provide inertia for the displacement. The SC must be removed completely to prevent recurrence.

SCs have been resected using contact laser decompression,[55] contact laser excision,[55,56] obstetrical (OB) wire amputation,[55,56] and electrosurgical loop amputation.[56,57] Decompression alone

led to recurrence. Although results were generally satisfactory, laser dissection and OB wire amputation were associated with a case of dorsal displacement of the soft palate; subepiglottic scar formation was responsible for one. The thermal effect of laser dissection on the ventral epiglottis was expressed as a concern.[56]

Some prefer electrosurgical amputation of SCs,[56,57] which may be performed standing or *per os* in the anesthetized horse. The unit is set to "pure cutting" and 100 W. The least amount of tissue possible should be in the snare when the amputation is performed. Small lesions that can be snared and retracted completely from the surrounding epiglottic cartilage and soft palate can be amputated with the horse standing. The typical SC in an adult horse is accessed more safely *per os* with the horse in dorsal recumbency.

The soft palate is left in its normal position ventral to the epiglottis to prevent the apex of the epiglottis from curling "up" (ventrally) and obstructing access to the SC. The electrosurgical loop is placed over the SC just "south of its equator" and tightened (Figure 15-19, *A*). The snare will gather mucosa and stretch it dorsally against the ventral surface of the epiglottis thus minimizing the amount of mucosa removed. Traction is placed on the snare, which is activated to amputate the SC in its entirety (Figure 15-19, *B*).

Postoperatively the horse is rested from airway turbulence approximately 2 weeks or until the subepiglottic ulcer has healed. Prednisolone, phenylbutazone, and an oral antibiotic are administered (see Table 15-2).

Neonates are treated the same but some additional caution may be needed. If respiratory disease is present, general anesthesia may be delayed; foals with large SCs may require tracheotomy as the respiratory disease improves. In one young foal with a large SC, for example, there was possible airway obstruction or difficulty intubating after general anesthesia was induced. With the foal sedated, the SC was endoscopically decompressed using an endoscopic injection needle. When the foal was intubated and in dorsal recumbency, the SC was reinflated with saline and amputated using electrosurgery.

Endometrial Cysts

Endometrial cyst ablation has been reported using the Nd:YAG laser in contact or noncontact fashion. With reasonable caution, there is little chance of full-thickness damage to the uterine wall.[40] Uterine distention for visibility is much better with the mare out of estrus.

For the noncontact technique, at 50 W or less, the gas-cooled fiber is positioned approximately 1 cm from the cyst wall, which is "painted" to visibly coagulate the visible tissue; the serous cyst fluid is heated in the process, further coagulating the cyst's lining. When all of the visible tissue has been blanched, the laser fiber set at 75 to 100 W is used to puncture the cyst and ablate all visible cyst tissue.[40] A similar contact technique is effective with a bare quartz fiber at lower power settings.[58]

Contact laser energy has been used to initially puncture the cyst so the fluid will heat and coagulate the cyst wall. The cyst wall will blanch and contract around the fiber; lasing is continued until it is only a small pale mass on the endometrial surface.

Complete electrosurgical removal of the structure with the fluid cyst intact is preferred.[59] The process is more efficient, and there is no potential for recurrence. However, extremely large cysts still require laser ablation. The electrosurgical loop/snare is tightened around the base of the cyst, distending the fluid contents. With the electrosurgical unit set at "pure cutting" and 100 W, the intact cyst is amputated and removed with suction through the endoscope or snaring with the electrosurgical loop. Postoperatively, the mare is short-cycled and examined again by the theriogeniologist.

Lithotripsy

Cystic or urethral uroliths can be removed completely endoscopically with laser energy transmitted through a quartz fiber inserted through the biopsy channel of the endoscope. The

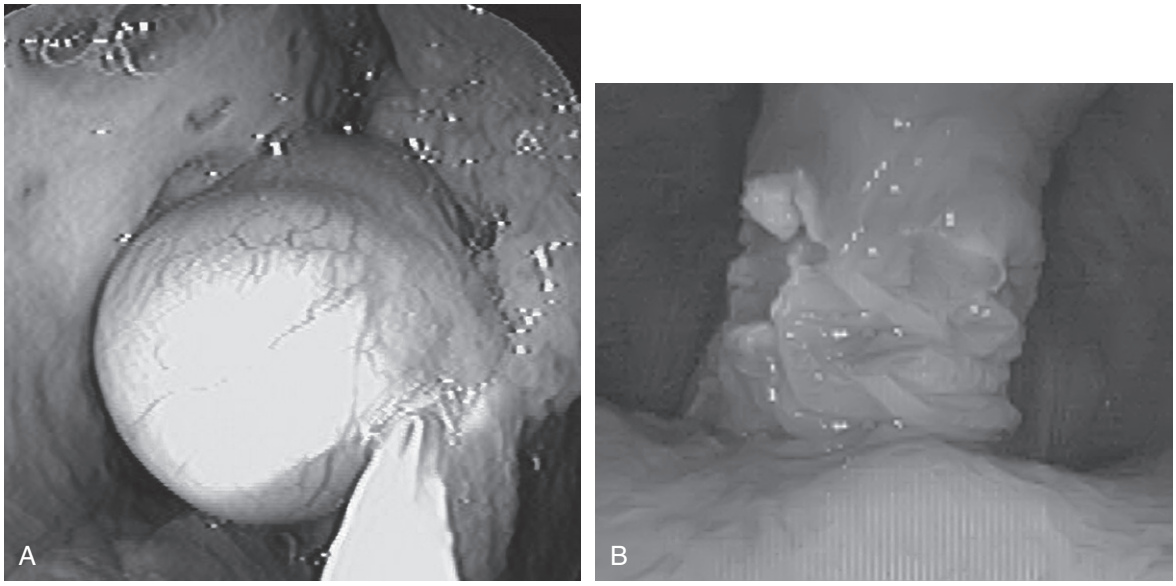

Figure 15-19. A, The electrosurgical snare is tight around the base of the subepiglottic cyst, ready to amputate. **B,** The site of amputation of the subepiglottic cyst is minimal if the mucosa is stretched tightly around the cyst. Excessive mucosal removal or damage may lead to excessive scar tissue immobilizing the epiglottis.

pulsed dye[38,60] and Ho:YAG[36-38] lasers have been used. Although the Ho:YAG laser is widely used in human and small animal urology, the pulsed dye laser is more efficient with large equine uroliths. The Ho:YAG laser essentially pulverizes the mineral, which is a lengthy process in dense stones; however, more porous smaller stones have been effectively addressed.[37] The pulsed-dye laser vaporizes the mineral into a plasma, and the pulsed hydraulic pressure fragments the stone.

Preoperative urine culture is advisable so treatment can be started in advance. If sabulous cystitis is present, preoperative lavage as needed with 0.5% acetic acid will dissolve the debris and sanitize the bladder. Cases with extreme sabulous cystitis should be evaluated carefully before surgery; for example, one urolith has been observed that was tightly adhered to the cystic wall, which perforated when it was separated endoscopically. No laser surgery had been performed.

Although it is possible to perform the procedure through the penile urethra with the horse standing, surgery is much more efficient with the horse in dorsal or lateral recumbency. The procedure is performed under water within the bladder (or urethra) with the fiber passed through the biopsy channel. Tubing from an arthroscopic fluid pump is attached to the biopsy channel and perforated with a needle to accommodate the very small quartz fiber. Fibers are expensive, so efficiency is helpful. When the bladder is distended with fluid, the urolith is located and the fiber is placed directly against the stone. When the laser is activated, the plasma will float away in the fluid medium and fragments will fall away (Figure 15-20). When the accumulation of fragments hinders access to the stone, the endoscope is replaced with sterile plastic tubing with the largest internal diameter the horse can accommodate. Place a hand on the bladder *per rectum*, which is inflated using the arthroscopic pump. When inflated, the tip of the tube is retracted to the neck of the bladder to funnel debris as the bladder is allowed to rapidly decompress; the bladder is "bounced" with the hand to keep the debris suspended. The process is repeated until the bladder is completely empty.

Figure 15-20. Endoscopic laser lithrotripsy of a cystic urolith. The pulsed-dye laser is turning the substance of the stone to a "plasma," which is floating away. Fragments that have fallen from the stone litter the foreground.

In most cases, the uroliths can be completely fragmented, but complete removal of the debris may be completed with the horse standing if anesthesia becomes prolonged. It is important to remove all the mineral debris; manual removal of small fragments may require the use of endoscopic biopsy forceps if they won't wash out.

Depending upon the horse, standing laser lithotripsy through a perineal urethrostomy may be preferable. General anesthesia may not be advisable; standing surgery for horses with extremely large stones is recommended. A sterile access tube can be placed in the bladder through the urethrostomy, significantly expediting the procedure.

Laser Treatment of Distal Tarsal Joints

Horses that have chronic distal tarsal disease (bone spavin) and that are refractory to medical therapy and corrective shoeing become candidates for surgery. Among the several approaches that have been reported is Nd:YAG or diode laser treatment of the tarsometatarsal and distal intertarsal joints.[61] Radiographic fusion of the joints does not occur, so the term *arthrodesis* does not apply, and fusion is apparently not required to resolve the lameness. The mechanism is more likely desensitization of the capsular sensory nerves. Compared to surgical drilling and sodium monoiodoacetate injection, laser-treated horses were more comfortable.[62] For additional information, review Chapters 81 and 97.

The procedure consists of fluoroscopically guided insertion of needles into the tarsometatarsal and distal intertarsal joints. Laser energy must not be applied to the proximal intertarsal joint, which communicates with the tarsocrural joint. Each joint must have ingress and egress to evacuate steam if the pressure is sufficient so superheating is prevented; the joint fluid reaches 100° C during the procedure. Because the joints are abnormal, they will typically not allow lavage across the entire joint, requiring two medial and two lateral needles in most cases. Laser energy is applied only if there is fluoroscopic confirmation of correct needle placement, and sterile saline will flush out at least one egress (may not be the adjacent needle). If one side of a joint cannot meet both criteria, that side is not treated. Lack of treating a part of a joint has not affected the outcomes to date.

Surgery is performed with the horse in dorsal recumbency, so the fluoroscope can be moved conveniently between limbs. I use a diode laser set at 20 W with a sterile 600-µm fiber. Sixteen-gauge needles are required to accommodate the laser fiber, but 18- or 20-gauge needles may be used for egress. Total energy per joint approximates 1500 J divided between the medial and lateral aspects of the joint, if needed. Gentle pressure is kept on the laser fiber as lasing progresses; some of the fiber will dissipate in the process (Figure 15-21). Removal of the needle and the fiber at once prevents the fiber from breaking inside the needle. Amikacin (250 mg) is placed in each joint after laser treatment. The needles become hot enough to cause skin necrosis, so sterile iced saline–soaked sponges are held over the needles and skin as lasing is performed. This is difficult on the down side of a horse in lateral recumbency, so dorsal recumbency is recommended.

Postoperatively, the horse is monitored for lameness and local swelling. Perioperative antibiotic and postoperative

Figure 15-21. Laser treatment of the distal tarsal joints. The horse is in dorsal recumbency and the needles are in the medial aspect of the tarsometatarsal and distal intertarsal joints. Laser energy is being applied to the more proximal joint and a plume is escaping from the vent needle.

NSAIDs are administered. If lameness or swelling appears, systemic DMSO (1 L 10% DMSO IV bid) is added and the affected areas are iced; topical NSAID is also helpful. Handwalking for 5 days followed by another 5 days of shed row or light turnout followed by return to training is prescribed.

LASER SAFETY

The authority for laser safety in the United States is American National Standard (ANSI) for Safe Use of Lasers in Health Care Facilities Z136.3. All surgical lasers are secured with a key lock and a separate interlock that is required to operate the machine. A designated laser safety officer responsible for lock security, warning signs during surgery, and other required safety measures is advisable.

Appropriate eye protection is required for all surgical laser wavelengths. Clear glass with protection from all angles is adequate for the CO_2 laser, but optical density recommendations are specific for the near-infrared and other wavelengths and should be followed for the wavelength of the laser. The patient's eyes must be considered as well. Because surgical lasers discussed here are not in the visible spectrum, a low-energy helium neon laser aiming beam is used. However, prolonged direct exposure, particularly to the eye, can cause damage.

All smoke generated from tissue should be evacuated using a filtered laser smoke evacuator. In spite of reports that insignificant concentrations of bacteria become aerosolized[63] and that horses are not adversely affected by routine upper airway laser surgery,[64] there is sufficient evidence that infectious, carcinogenic, and irritant material is present in laser smoke.[65] The vaporized debris and potentially viable cells or pathogens should not be inhaled by humans or the patient. Surgical suction is inadequate for this task because it is less efficient and the suction lines will eventually foul.

The surgical field should be protected by barriers when possible. Towels or lap sponges soaked with sterile water limit CO_2 laser energy from burning tissue off the field or drapes. Wet sponges should be held behind tissue when the laser might penetrate completely.

Accelerants should be avoided. Saline should be used instead of alcohol for surgical prep. Heliox (oxygen diluted with helium) can be substituted for pure oxygen when operating close to the airway with the horse under general anesthesia. If these few simple rules are followed, laser surgery is as safe as any other surgery.

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CHAPTER 16

Suture Materials and Patterns

Jan M. Kümmerle

The history of surgery is intrinsically tied to the development of suture materials. The use of silk and catgut was already described in AD 150 by Galen of Pergamon, who used these materials to suture wounds sustained in gladiator fights, a popular entertainment in the Roman Empire.[1] Since that time, tremendous advances have been made in the development of biomaterials, and today the veterinary surgeon can choose from a variety of suture materials. However, the one ideal suture material for every indication does not exist, and the surgeon needs to know the specific properties of each material to make an appropriate choice for each application.

SUTURE CLASSIFICATION

Sutures can be classified by several criteria[2]:

- Degradation behavior: absorbable versus nonabsorbable
- Composition: natural versus synthetic
- Structure: monofilament versus multifilament.

Absorbable sutures undergo degradation and loss of tensile strength within 60 days.[3] Degradation is mediated by hydrolysis, enzymatic digestion, or phagocytosis. Natural materials are degraded by proteolytic enzymes, whereas the new synthetic absorbable sutures are degraded by nonenzymatic hydrolysis of ester bonds that occurs independent of inflammation. After hydrolysis separates the ester bonds and depending on particle size, phagocytosis may take place.[4] Nonabsorbable suture materials are not significantly degraded after implantation and are indicated where extended wound support or implant function is required.

Sutures are made from naturally occurring substances, synthetic polymers, or metallic fibers. Natural materials tend to invoke a significant inflammatory reaction and currently have been replaced by synthetic materials.

Multifilament suture materials are composed of several filaments twisted or braided together. Generally, this leads to good handling and knot-tying properties and offers superior knot security. On the other hand, their braided structure increases capillarity, facilitates penetration of bacteria, and increases drag resistance while being pulled through tissue. Multifilament sutures may be coated to reduce tissue drag and capillarity. However, coating can reduce knot security, and the coating layer may be damaged during the suturing process, thus leading to recurrence of the aforementioned disadvantages of multifilament sutures.[5] A monofilament structure results in lower tissue drag, less risk of infection, reduced tissue reaction, and less tendency of pretied loops to collapse. On the other hand, the higher bending stiffness and greater memory of monofilament sutures as well as their lower coefficient of friction result in poorer handling properties and less knot security. In addition, their stiff cut ends can cause tissue irritation and mucosal ulceration.

SUTURE CHARACTERISTICS

Suture Size

The United States Pharmacopoeia (USP) standard for suture size (i.e., cross-sectional diameter) is still commonly used. This system uses 0 as the baseline average suture size. As suture diameter decreases, 0s are added or numbers followed by a 0 (2-0, 3-0, etc.; e.g., 000 and 3-0 are the same size). As suture diameter increases above 0, increasing numbers are assigned (1, 2, etc.). Another system is the European Pharmocopoeia. It was established in 1973 and uses a metric system. Suture size is expressed as a number (4, 5, etc.) that corresponds to 1/10 of the suture diameter in mm (Table 16-1).

TABLE 16-1. The USP and ESP Classification System for Suture Sizes of Synthetic Suture Materials

US Pharmacopoeia	European Pharmocopoeia	Suture Diameter
USP SIZE	METRIC SIZE	mm RANGE
11-0	0.1	0.010-0.019
10-0	0.2	0.020-0.029
9-0	0.3	0.030-0.039
8-0	0.4	0.040-0.049
7-0	0.5	0.050-0.069
6-0	0.7	0.070-0.099
5-0	1	0.100-0.149
4-0	1.5	0.150-0.199
3-0	2	0.200-0.249
2-0	2.5	0.250-0.299
2-0	3	0.300-0.349
0	3.5	0.350-0.399
1	4	0.400-0.499
2	5	0.500-0.599
3; 4	6	0.600-0.699
5	7	0.700-0.799
6	8	0.800-0.899
7	9	0.900-0.999
8	10.0	1.000-1.099

Flexibility

The torsional stiffness and diameter of a suture determine its flexibility.[6] Flexible sutures are required to ligate vessels or to perform a continuous suture pattern.

Elasticity

Elasticity is the capability of a material to undergo elastic deformation under tension, returning to its original length after stretching. High elasticity will allow the suture to stretch with wound edema but return to its original length when swelling has subsided.

Surface Characteristics and Coating

The surface characteristics of a suture determine the tissue drag (i.e., the resistance and subsequent trauma when pulled through tissue) and the coefficient of friction.[6] Rough sutures cause more injury than sutures with a smooth surface. In delicate tissues, such as the eye or a thin-walled viscus, low tissue drag is particularly important. However, sutures with a smooth surface and low tissue drag require greater tension to achieve good apposition of tissues and they have lower knot security. Multifilament sutures have more tissue drag than monofilament sutures. The coefficient of friction is a measure of the slipperiness of a suture that affects the tendency of the knot to loosen after it has been tied: multifilament sutures have higher frictional values[7] and thus knot security.[8] Coating provides a smoother surface, reducing tissue drag and the coefficient of friction.

Capillarity

Capillarity is the process by which bacteria and fluid are carried into the interstices of a multifilament suture material. Cells of the body's immune defense system are too large to enter these interstices, and therefore a persistent infection can result, particularly if a nonabsorbable suture is used. Coating can reduce the capillarity. Suture materials with significant capillarity should not be used in contaminated or infected surgical sites.

Memory

Memory refers to the capability of a suture to return to its original shape after deformation by tying. Sutures with a high degree

of memory, particularly monofilament sutures, are stiff and difficult to handle.

Tensile Strength

The suture material's tensile strength (TS) is the force that the untied suture strand can withstand before it breaks when the force is applied in the direction of its length.

Knot Holding Capacity

The knot holding capacity (KHC) is the maximum load to failure when tension is applied to the knotted suture material.

Relative Knot Security

Relative knot security (RKS) has been recommended as a standardized way to describe the knot-holding capacity. It is the knot-holding capacity expressed as a percentage of the unknotted suture's tensile strength by the formula RKS (%) = (KHC/TS) × 100.

SELECTION OF SUTURE MATERIALS

To select an appropriate suture out of the wide range of suture materials, its specific composition and structure as well as biological and biomechanical behavior as they relate to the requirements needed should be considered. The details on characteristics of relevant suture materials are summarized in Tables 16-2 and 16-3.

Selection of the Biomechanically Appropriate Suture Size and Material

Certain biomechanical principles should be taken into account when selecting a suture material and its size:

- The selected suture should be as strong as the normal tissue through which it is placed.
- Tensile strength reduction over time of the chosen suture material should correspond to the healing characteristics and gain in wound strength of the sutured tissue.
- A suture is not needed after a wound has healed.
- The strength of a wound is more dependent on the involved tissue's ability to hold a suture than the strength of the suture material itself.
- Elastic suture materials are indicated for skin closure to adapt to wound edema; suture materials with high stiffness are required to serve as a prosthesis and for abdominal closure, herniorrhaphy, or joint imbrications.
- The use of an oversized suture material may weaken the repaired wound by causing excessive tissue reaction.
- For a wound under tension, increasing the number of sutures applied (and/or the use of tension sutures) is preferable to increasing the suture size.[9]

SURGICAL NEEDLES

Surgical needles are manufactured from surgical steel and come in various shapes: straight, half-curved, or curved with 1/4-, 3/8-, 1/2- or 5/8-circle shapes (Figure 16-1). Easily accessible tissues, such as the skin, may be sutured by hand with straight needles, but curved needles are generally preferred because they are easier to use with instruments. There is limited indication for 1/4-circle needles, except for ophthalmologic surgery. For suturing in confined and deep locations, 5/8-circle needles are useful. In most instances, 3/8- or 1/2-circle needles are preferred because they do not require extensive rotational movement of the hand to penetrate tissue and allow precise wound adaptation.

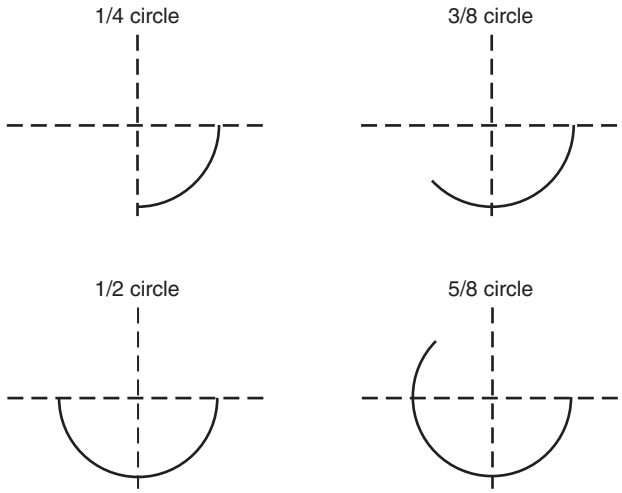

Figure 16-1. Various shapes of curved needles.

The three basic components of a surgical needle are the suture attachment end (i.e., swaged or eyed), the body, and the point[6] (Figure 16-2). In eyed needles, the suture must be threaded through the eye and a double strand of suture pulled through the tissue. Eyed needles are reusable and thus less expensive. However, they can become dull with reuse and this can exacerbate tissue trauma. Swaged needles have the suture attached to their ends. They are easier to handle, and tissue penetration results in less trauma than that caused by eyed needles because only a single strand of suture material is pulled through the tissues. Currently, the hole to introduce the suture material into the back of the needle is prepared with lasers. This process is more precise and has led to downsizing of the needles, which results in decreased trauma during suturing.

Needle length should be considered when choosing a suture-needle combination. The needle should be long enough to allow penetration of both wound margins. Chord length and needle radius become important factors in laparoscopic surgery when the needle needs to be inserted through a laparoscopic cannula.[10]

The shape of the point and body of the needle (Figure 16-3) are main determinants of its behavior in the patient's tissue. Taper point needles should be used wherever possible because they are least traumatic to adjacent tissue and minimize inadvertent damage to vessels and nerves. Indications include suturing muscle, subcutaneous tissue, or viscera. Cutting needles provide sharp edges that cut through dense connective tissue thus rendering them suitable for closing skin, tendon, and fascia.[11] Both the regular cutting needle and the reverse cutting needle have a triangular cross-sectional area. The regular cutting needle possesses a sharp edge on the inner curvature of the needle point and shaft. This may promote "cut out" of

Text continued on p. 188

TABLE 16-2. Absorbable Suture Materials

Suture Type	Trade Name	Composition	Structure	Absorption	Tensile Strength	Advantages	Disadvantages
Surgical gut	Catgut, Plain Gut, Chromic Gut, Catgut Chrom	Collagen obtained from bovine intestinal serosa or ovine intestinal submucosa; chromic gut is treated with a chromic salt solution	Multifilament	Evokes a moderate inflammatory reaction in tissue as it is broken down through a combination of enzymatic degradation and phagocytosis; rate of absorption is increased in the presence of infection and in tissues with high levels of proteolytic enzymes	Has less tensile strength than most synthetic absorbable sutures	Inexpensive; adhesion promotion can be desirable in some indications; good handling characteristics	Production and use of catgut was prohibited in the European Union in 2001 as the entire bovine intestine is classified as specific TSE risk material; chromic coating reduces soft tissue reaction and rate of absorption; chromic gut is difficult to handle and has poor knot security when wet
Polyglactin 910	Vicryl	Copolymer of glycolide and L-lactide; coating: polyglactin and calcium stearate	Braided multifilament; coated	Resorption time: 56-70 days	Tensile strength reduction by 25% at day 14, 50% at day 21, and by 100% at day 35	Good size-to-strength ratio; greater initial breaking strength and stiffness than polydioxanone; minimal tissue reaction; excellent handling properties	May cut through friable tissue (especially if not coated)
Polyglycolic acid	Dexon, Dexon II, Safil	Polymer of glycolic acid; Dexon II is coated with polycaprolate	Braided multifilament; can be coated	Resorption time: 60-90 days	Tensile strength reduction by 35% at day 14 and by 65% at day 21	Good handling characteristics	Very rapid absorption in the oral cavity; tends to drag through tissues; less knot-breaking strength than polyglactin 910
Braided lactomer	Polysorb	Copolymer of glycolide and lactide; coating: mixture of a caprolactone/ glycolide copolymer and calcium stearoyl lactylate	Braided multifilament; coated	Resorption time: 56-70 days	Tensile strength is 140% of minimum knot strength requirements of the European/ United States Pharmacopoeia initially, 80% at day 14 and 30% at day 21; biomechanically superior to polyglactin 910	High initial tensile strength; good knot security; excellent handling properties	

Glycomer 631	Biosyn	Combined polymer of glycolide, dioxanone and trimethylene carbonate	Monofilament	Resorption time: 90-110 days	Tensile strength is 75% of minimum knot strength requirements of the European/ United States Pharmacopoeia at day 14 and 40% at day 21.	Monofilament suture with only minimal memory and excellent handling properties; minimal tissue reaction	
Polyglytone 6211	Caprosyn	Copolymer of glycolide, caprolactone, trimethylen carbonate, and lactide	Monofilament	Resorption complete within 56 days	Loses almost all tensile strength within 21 days	Provides short-term tensile strength combined with very rapid absorption	
Polydioxanone	PDS II	Polymer of poly-*p*-dioxanone	Monofilament	Resorption time: 180 days	Tensile strength reduction by 25% at day 14, 30% at day 28, 50% at day 42	Absorbable suture material that maintains tensile strength over a prolonged period of time; less memory effect than polyglyconate	Moderate knot security, moderate handling characteristics
Polyglyconate	Maxon	Copolymer of glycolide and trimethylene carbonate	Monofilament	Resorption time: 180 days	Tensile strength reduction by 25% at day 14, 50% at day 28, 75% at day 42	Slow resorption and loss of tensile strength; three times stronger than polyglactin 910 at day 21 of wound healing; good knot security	Much memory effect, limited pliability and moderate handling properties
Polyglyconate	Monosyn	Copolymer of glycolide, trimethylene carbonate, caprolactone	Monofilament	Resorption time: 60-90 days	Tensile strength reduction by 30% at day 7, 50% at day 14 and 80% at 21 days	Very good handling properties and good knot security	
Poliglecaprone	Monocryl	Copolymer of glycolide and caprolactone	Monofilament	Resorption time: 90-120 days	Tensile strength reduction by 50% at day 7 and 80% at day 14; complete loss of tensile strength within 21 days	Very low tissue drag owing to smooth surface; only minimal memory effect and high pliability; provides high initial tensile strength and rapid absorption; minimal tissue reaction	

TABLE 16-3. Nonabsorbable Suture Materials

Suture Type	Trade Name	Composition	Structure	Tensile Strength	Advantages	Disadvantages
Silk	Sofsilk, Silkam	Raw silk, spun by silkworm	Braided multifilament; coated or uncoated		Excellent handling characteristics; useful for ligatures	Does not maintain tensile strength more than 6 months; may potentiate infection—should be avoided in contaminated sites; has significant capillarity; incites some inflammatory reaction
Surgical steel	Steelex	Alloy of iron	Monofilament or as a multifilament twisted wire	Greatest tensile strength of all sutures	Greatest knot security of all sutures; no inflammatory reaction	Tissue movements against the inflexible ends may cause inflammation and necrosis; poor handling properties; cannot withstand repeated bending without breaking; multifilament wire can fragment and migrate, leading to sinus tract formation
Nylon	Dafilon	Polymer of polyamide	Monofilament or multifilament	Intermediate tensile strength; monofilament nylon loses about 30% of its original tensile strength by 2 years because of chemical degradation; multifilament nylon retains no tensile strength after 6 months	Suitable for use in contaminated wounds; degradation products act as antibacterial agents	Poor handling characteristics and poor knot security; not recommended for use within serous or synovial cavities because buried sharp ends may cause frictional irritation
Polycaprolactam	Supramid	Polymerized caprolactam (=polyamide 6)	Multifilament with a polyamide coating	Better tensile strength than nylon	Excellent handling properties, high knot security	Intermediate tissue reactivity; has a tendency to form sinuses on implantation in tissues and is therefore best suited for use in the skin

Polyester	Mersilene, Synthofil, Dagrofil, Ethibond, Ticron	Polyethylene terephtalate	Monofilament or multifilament; uncoated or coated with polybutilate or silicone or polyethylene/vinyl acetate	Very high and sustained tensile strength		Noncoated polyester fibers have a high coefficient of friction; knot security is poor and is further reduced by coating; causes marked tissue reaction and fibrous encapsulation; should not be used in contaminated wounds
Ultra-high molecular weight polyethylene	FiberWire	Ultra-high molecular weight polyethylene	Multifilament with a polyethylene/ polyester coating	Superior strength; greater tensile strength and less elongation under load than polyester sutures	High abrasion resistance; good knot security; less tissue drag than polyester sutures	
Polypropylene	Premilene, Prolene, Surgipro	Polyolefin plastic	Monofilament	Moderate tensile strength	Greatest knot security of all synthetic monofilament sutures; least thrombogenic suture material makes it suitable for vascular surgery; minimal tissue reactivity and least likely to potentiate infection; high elasticity	Slippery handling and tying characteristics
Polybutester	Novafil	Copolymer of butylene terephthalate and polytetramethylene ether glycol	Monofilament		Good handling characteristics and knot security; more flexible than polypropylene or nylon; elongates elastically under load or tension when wound edema occurs and returns to its original form when edema subsides; minimal tissue reaction	

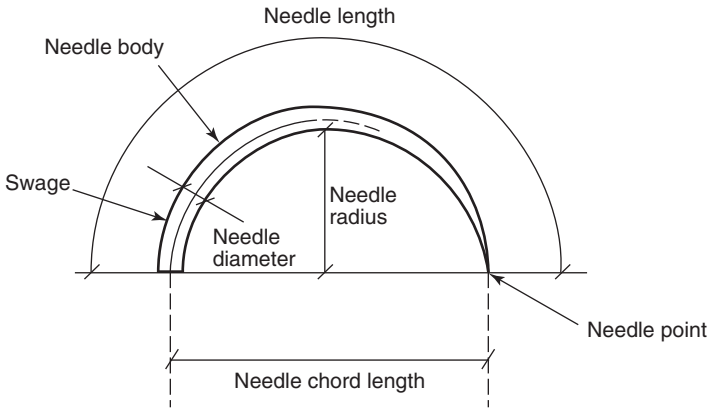

Figure 16-2. Anatomy of a surgical needle.

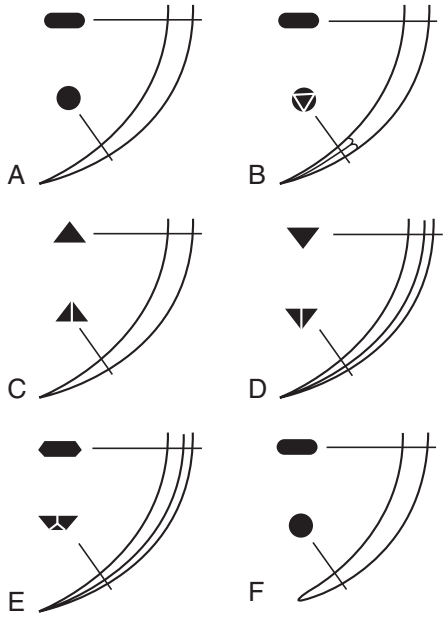

Figure 16-3. Various points and shaft designs of surgical needles. **A,** Taperpoint; **B,** tapercut; **C,** regular cutting; **D,** reverse cutting; **E,** spatula point; **F,** blunt point.

tissue because it cuts toward the edges of the wound or incision. Reverse cutting needles have the cutting edge located on the convex, outer curvature of the needle. This makes them stronger than similarly sized conventional cutting needles and reduces the risk of tissue cut out.[6] A tapercut needle combines the reverse cutting point that readily penetrates through tissue with a round shaft that does not cut through or enlarge the needle hole when passed.[11] These needles can be used to suture a delicate tissue to a denser one (e.g., suturing epithelium/mucosa to the skin as in urethrostomy, colostomy, or tracheostomy) or for dense but delicate tissues (e.g., periosteum). Spatula needles are flat on the top and bottom and have a side cutting action. They are indicated for certain procedures in ophthalmologic surgery.[12] Blunt-point needles have a rounded, blunt point that can penetrate friable tissue without cutting. They can be used to suture soft, parenchymal organs, such as the liver or kidney.[6]

The Deschamps needle (Figure 16-4) is a long thin instrument with a palm-held handle and a thin needle-like extension

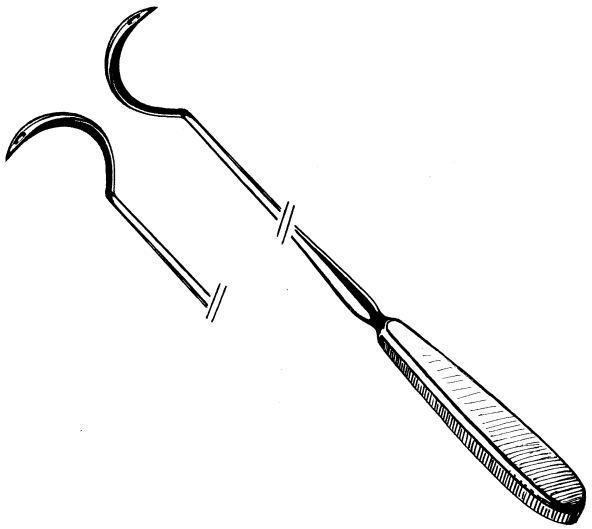

Figure 16-4. Deschamps needles, showing the left- and right-handed configuration, respectively. The threaded eye near the pointed tip allows easy retrieval of the suture without the need for complete penetration by the needle. When the suture is grasped at the tip of the needle, the instrument is rotated backward out of the tissue and can be rethreaded for the next bite.

that bends laterally at right angles at its tip and then continues as a semicircle in the same plane. The tip has a needle eye and a pointed but not sharp end. It is designed to place ligatures around vessels in poorly accessible sites and can be used for suturing in deep, confined areas.

SUTURE CONFIGURATIONS

Knots and Ligatures

Knot tying is an essential part of almost any surgical procedure. However, even a perfectly tied knot is the weakest part of a suture.[6,13] Therefore, it is of tremendous importance to perform knot tying correctly to prevent unnecessary weakening of this critical part of the suture, which could potentially leading to subsequent dehiscence.

Knot Tying Techniques

A knot is constructed by laying at least two throws on top of each other and tightening them. If the direction of the throws is reversed, a square knot results (proper); otherwise a granny knot is obtained (improper). During knot tying, opposing suture ends should be pulled perpendicular to the long axis of the incision except if sutures are placed deep in the tissues. In the latter situation, the suture ends are pulled parallel to the direction of the suture line and in doing so the tissues positioned above the knot are not pulled apart. Reversal of throw direction combined with pulling mainly on one end of the suture results in a half-hitch; if tension applied by the pulling hand is directed away from the incision by lifting this hand, a sliding half-hitch is formed (Figure 16-5). Granny and half-hitch knots are prone to slip.[13] However, this feature can be beneficial if the knot needs to be slid into a deep and confined space.

Generally, a superimposition of square knots is considered the most reliable knot configuration.[6] When the first throw of a square knot does not hold the wound margins in apposition,

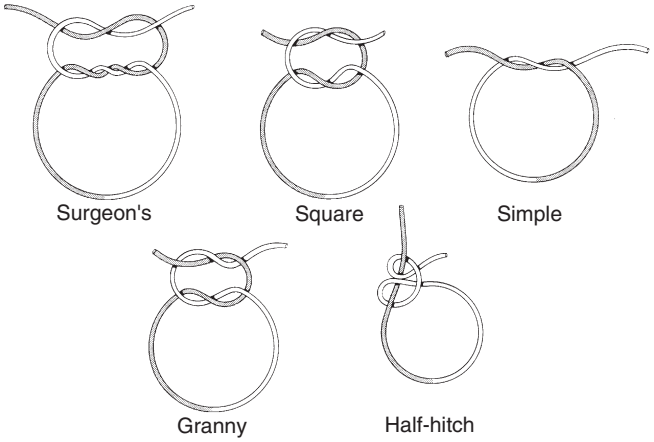

Figure 16-5. Surgical knots.

a surgeon's knot may be tied. However, the surgeon's knot should be avoided when not needed because it places more suture material into the wound and can decrease structural stiffness of a knot with some suture materials.[14] Clamping the first throw of a square knot to maintain tissue apposition after the first throw does not negatively affect mechanical properties of common multifilament suture materials; however, clamping can reduce breaking strength of monofilament sutures by 10%.[13,14] A square knot but not a surgeon's knot should be used to ligate vessels.[6] Knots can be tied using instruments or by hand. In veterinary surgery, instrument ties are more commonly used because there is less waste of suture material. If a square knot is formed at the end of a continuous suture line and a needle holder is used to tie the knot, it is important to grasp exactly at the center of the looped end to avoid asymmetric loads placed on either end of the loop. By applying tension to the suture loop with an open needle holder, the tension along the loop equalizes on its own. Hand ties are particularly useful in confined areas, when sutures have been pre-placed or to precisely adjust tension on the suture. Hand ties require that the suture ends be left longer than for an instrument tie. A one-handed or two-handed technique can be applied.

The knots of subcutaneous and intradermal suture patterns should be buried to reduce irritation caused by knots rubbing against more superficial tissue and to prevent suture extrusion.

Knot Efficiency

Loop security and knot security are ways of measuring a knot's effectiveness.[8] Loop security is the capability to maintain a tight suture loop as a knot is tied. Inadequate loop security results in loss of tissue apposition during knot tying.[15] Knot security is defined as the effectiveness of the knot at resisting slippage when load is applied. Knot security depends on the structural configuration of the knot and the type of suture material.[16] The characteristics of a suture material mainly affecting knot security are memory and coefficient of friction. Remember that body fluids come in contact with the suture material during surgery, which affects frictional behavior and thus the knot security of a suture.[17]

In addition to suture material and knot configuration, the number of throws and suture end length also influence knot security. A suture end length of at least 3 mm is recommended to optimize knot integrity.[18,19] The minimal number of throws needed (including the first) for a secure square knot using No. 2-0 USP suture materials is three for polyglycolic acid, polyglactin 910, and polypropylene and four for nylon and polydioxanone.[20,21] For larger-diameter suture materials, sufficient knot security is achieved with five throws. This was demonstrated for polyglactin 910 No. 2 USP, polyglactin 910 No. 3 USP, and polydioxanone No. 2 USP.[13] Knots at the end of a continuous suture line are constructed using one looped and one free end. These knots require two or three more throws to ensure knot security than do knots constructed from two single suture strands.[22] The Aberdeen knot represents a special configuration to end a continuous suture line and is recommended in human surgery when monofilament suture material is used (the configuration of the knot can be studied in the cited publication).[23] A recent *in vitro* study demonstrated superior relative knot security and reduced knot volume of Aberdeen knots compared to square knots to end a continuous suture pattern of polydioxanone.[21]

Another factor to consider is the wound environment. A fatty wound environment can increase the number of throws needed to achieve a secure knot. This was confirmed by the finding that fat-coated No. 2-0 USP polydioxanone requires one additional throw to form a secure square knot at the beginning of a continuous pattern compared to plasma-coated No. 2-0 USP polydioxanone.[21] Asymmetric knots like sliding half-hitch or asymmetric granny knots usually need two additional throws to achieve knot security. This was demonstrated in one study for polyglactin 910.[24] However, the superior knot security of braided lactomer (Polysorb) provided sufficient knot security even without additional throws.[24] In the clinical situation, the number of throws should be adequate to ensure knot security but not excessive to limit the amount of bulky foreign material in the tissues.

Finally, the suture diameter is also a determinant of knot security. Knot security decreases with increasing suture diameter.[13,16]

Loop Sutures

As an alternative to knots, the use of a loop suture to apply a simple continuous pattern has also been described in equine surgery.[25] The use of loop sutures reduces the number of knots and the double suture strand provides a larger surface area as the suture passes through the tissue; however, they result in an increased total amount of suture material remaining in the wound and the placement of a bulky four-stranded knot at the end of the suture line. In an *in vitro* experiment, USP No. 2 braided lactomer loop sutures applied in a simple-continuous fashion provided sufficient security for closure of the equine linea alba based on single-cycle to failure testing, with fascial failure being the main failure mode and without occurrence of suture or knot failure.[25]

Knot-Tying Techniques for Minimally Invasive Surgery

Minimally invasive surgical techniques require modifications in knot-tying techniques. In equine surgery, laparoscopy is increasing in importance, and extracorporeal knotting requires safe and efficient sliding-knot techniques. The first reliable sliding knot described for human laparoscopy was the Roeder knot.[26] Knot security was further improved by Sharp et al.[27] by

developing the 4S-modified Roeder knot (Figure 16-6). *In vitro* studies revealed that the 4S-modified Roeder knot outperformed several other slipknot ligatures in terms of knot security.[28,29] Monofilament suture materials are suitable for laparoscopic surgery because they perform well for knot rundown, have low tissue drag, and, unlike multifilament sutures, do not loose loop characteristics when wet. With regard to suture material and size used for the 4S-modified Roeder knot, polydioxanone and polyglyconate are biomechanically superior to polyglactin 910 and polyglycolic acid and sizes USP No. 1 or 2 are superior to smaller suture sizes.[28,29]

Suture Tension

Suture tension can be classified as intrinsic or extrinsic. Intrinsic tension refers to the tension on the tissue constricted within the suture loop. Excessive intrinsic tension can cause ischemic necrosis. Extrinsic tension represents the pulling tension from outside the suture loop. It depends on wound size, location, relationship to skin lines, and the amount of surrounding loose tissue.[30]

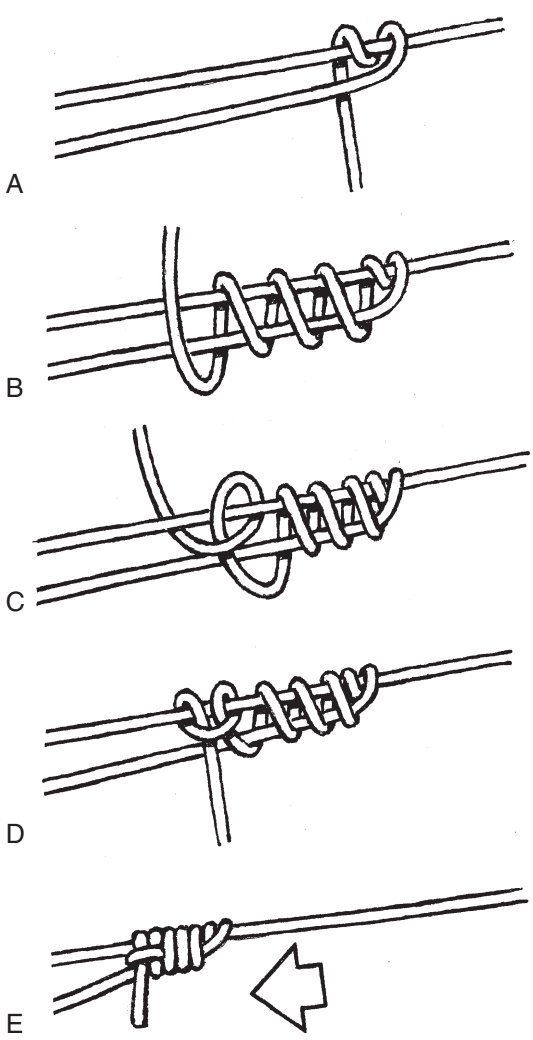

Figure 16-6. The 4S-modified Roeder knot is tied by: **A,** starting the knot with a single throw; **B,** wrapping the tail of the suture three times around both strands of the loop entering the abdomen; **C,** completing the knot with a half hitch knot around the standing part of the suture; **D,** completed knot is tightened by alternately pulling on the standing part and strand of the abdominal loop that exits from the cannula; **E,** the tightened knot is then slid into the abdominal cavity using a knot pusher.

Suture Patterns

Suture patterns can be classified as interrupted or continuous. Interrupted suture patterns have the following advantages over continuous patterns: increased security because failure of one suture does not jeopardize the entire suture line, precise reconstruction of irregular wound margins, precise control of tension at each point of the wound margin, less interference with blood supply of the wound margins, and no purse-string–like effect when tightening the suture applied in hollow viscera. Additionally, a part of the suture line can be re-opened in the postoperative period if drainage should be necessary.

On the other hand, the advantages of continuous patterns include: a smaller volume of suture material (in the form of knots) in the tissues, decreased surgery time, more even distribution of tension, better holding power against stress, and a tighter seal of skin and hollow viscera.

Suture patterns can be further characterized by the way they appose tissue: appositional sutures bring the tissue in direct approximation of the two cutting surfaces, everting sutures turn the tissue edges outward, and inverting sutures turn tissue inward. Appositional sutures are useful for anatomically precise closure. Inverting suture patterns are indicated to close hollow viscera or, in the form of the Lembert pattern, for fascial imbrication. Everting sutures eliminate dead space and counteract the tendency of wound edges to invert during healing. Most tension sutures have everting characteristics. Tension sutures redistribute the tension across the wound edges, thus drawing the wound edges together and minimizing marginal strangulation and necrosis. The capability of a suture pattern to withstand tensile forces is related to the number of segments that are parallel to the line of tension.[31] In horses, tension sutures are frequently used to close traumatic lacerations and surgical wounds over bone plates. Tension sutures are pre-placed well away from the wound margins, the skin edges can be apposed with the aid of towel clamps followed by tying of the tension sutures. Finally, the wound is closed with an appositional suture pattern.[9] Gauze, rubber tubing, or buttons can be incorporated into the tension sutures to reduce the risk of cutting out of sutures. This technique is termed "quilled" or "stented" suture. To approximate severed ends of a tendon or to secure one end of a tendon to bone or muscle, special tension suture configurations are indicated.

Tables 16-4 through 16-6 summarize the most common suture patterns. These patterns are illustrated in Figures 16-7 through 16-9.

Sutures for Specific Tissues

Skin

Monofilament suture materials are indicated for skin closure to reduce capillary transport of bacteria into deeper tissues. Nonabsorbable materials like nylon, polypropylene, and polybutester are preferred for skin sutures. Polybutester combines good handling characteristics with adequate elasticity to adapt to wound edema. A simple interrupted pattern is commonly used. Slight eversion is desirable to counteract the tendency of the

TABLE 16-4. Appositional and Everting Suture Patterns

Suture Pattern	Tissue Apposition	Characteristics
Simple interrupted (SI) (Figure 16-7, *A*)	Appositional; excessive tension may cause inversion	Easy and quick to place; precise anatomic closure and tension adjustment possible; knot should be offset so it does not rest on the incision
Interrupted intradermal/ subcuticular (Figure 16-7, *B*)	Appositional	Upside down SI suture placed in dermis/subcutis
Cruciate (Figure 16-7, *C*)	Appositional; excessive tension causes inversion	Stronger closure than SI; resists tension and prevents eversion; gains more space per suture than SI pattern
Gambee (Figure 16-7, *D*)	Appositional	Reduces mucosal eversion compared to SI pattern; may reduce wicking of bowel contents from the intestinal lumen to the exterior
Interrupted vertical mattress (IVM) (Figure 16-7, *E*)	Appositional to slightly everting	Precise apposition of wound edges; minimal interference with vascular supply; can be used for concurrent closure of skin and subcutis; places more suture material into the wound than SI
Allgöwer corium vertical mattress (Figure 16-7, *F*)	Appositional	Minimally traumatic suture pattern that provides good skin apposition and excellent cosmetic outcome; less holding strength than IVM
Interrupted horizontal mattress (Figure 16-7, *G*)	Everting	Degree of eversion depends on suture tension and distance to the wound margin; more everting than IVM; can also be applied in a continuous pattern
Simple continuous (SC) (Figure 16-7, *H*)	Appositional	Provides maximal tissue apposition; time and material saving; provides a relatively airtight and fluidtight closure; if used for skin closure: excessive tension can cause strangulation of the skin; anatomically less precise adaptation than SI pattern
Continuous intradermal (Figure 16-7, *I*)	Appositional	Bites are placed intradermally and parallel to the long axis of the incision; knots must be buried; superior cosmetic outcome; no need for suture removal; provides less strength than percutaneous skin closure
Ford interlocking (Figure 16-7, *J*)	Appositional	Synonym: Reverdin pattern; provides precise adaptation and offers greater security in the event of a partial failure; may be difficult to remove; may cause pressure necrosis and become buried when placed under tension

skin edges to invert during healing, and it results in the most cosmetic outcome. As mentioned earlier, wounds of traumatic origin or skin closure over implants may require the application of tension sutures.

To close a surgical incision, the needle enters the skin approximately 3 to 5 mm lateral to the incision line. Collagenase activity remains high within 5 mm of a skin incision, and sutures placed too close to the incision may be at greater risk of cutting through tissue.[9] Wounds of traumatic origin may manifest with traumatized or inflamed tissue margins that may require partial resection of the skin edges and larger needle bites. Suture spacing depends on skin thickness and the direction and magnitude of tension lines. Wounds along tension lines are pulled into better apposition and require fewer sutures than those oriented perpendicular to a tension line.[30] Placing interrupted sutures too closely together can result in excessive tissue reaction and unwarranted interference with cutaneous blood supply. Generally, it is recommended to place interrupted sutures 5 mm apart.[9]

As an alternative to percutaneous skin sutures, a continuous intradermal suture pattern using absorbable synthetic materials can be applied. Advantages of an intradermal suture are no need for suture removal, lack of skin irritation, lack of suture track infection, and excellent cosmetic outcome. Disadvantages include increased time for placement and less security than percutaneous skin patterns.[30]

Subcutis

Subcutaneous sutures are placed to eliminate dead space and decrease tension across the wound margin before placement of skin sutures. If drainage might become necessary, they can be placed in a simple interrupted pattern, otherwise a simple continuous pattern with the bites made perpendicular to the long axis of the incision is generally used.[6] Intermittent incorporation of the underlying soft tissue can reduce dead space. Synthetic absorbable suture materials are usually used.

Fascia

Fascia is considered a slowly healing tissue. Therefore, nonabsorbable or slowly absorbable synthetic suture materials are indicated for its closure.[32]

Concerning the equine linea alba, an experimental study on tissue strength after ventral midline celiotomy and closure of the linea alba using braided lactomer USP No. 2 in an interrupted cruciate pattern found a return to baseline tensile

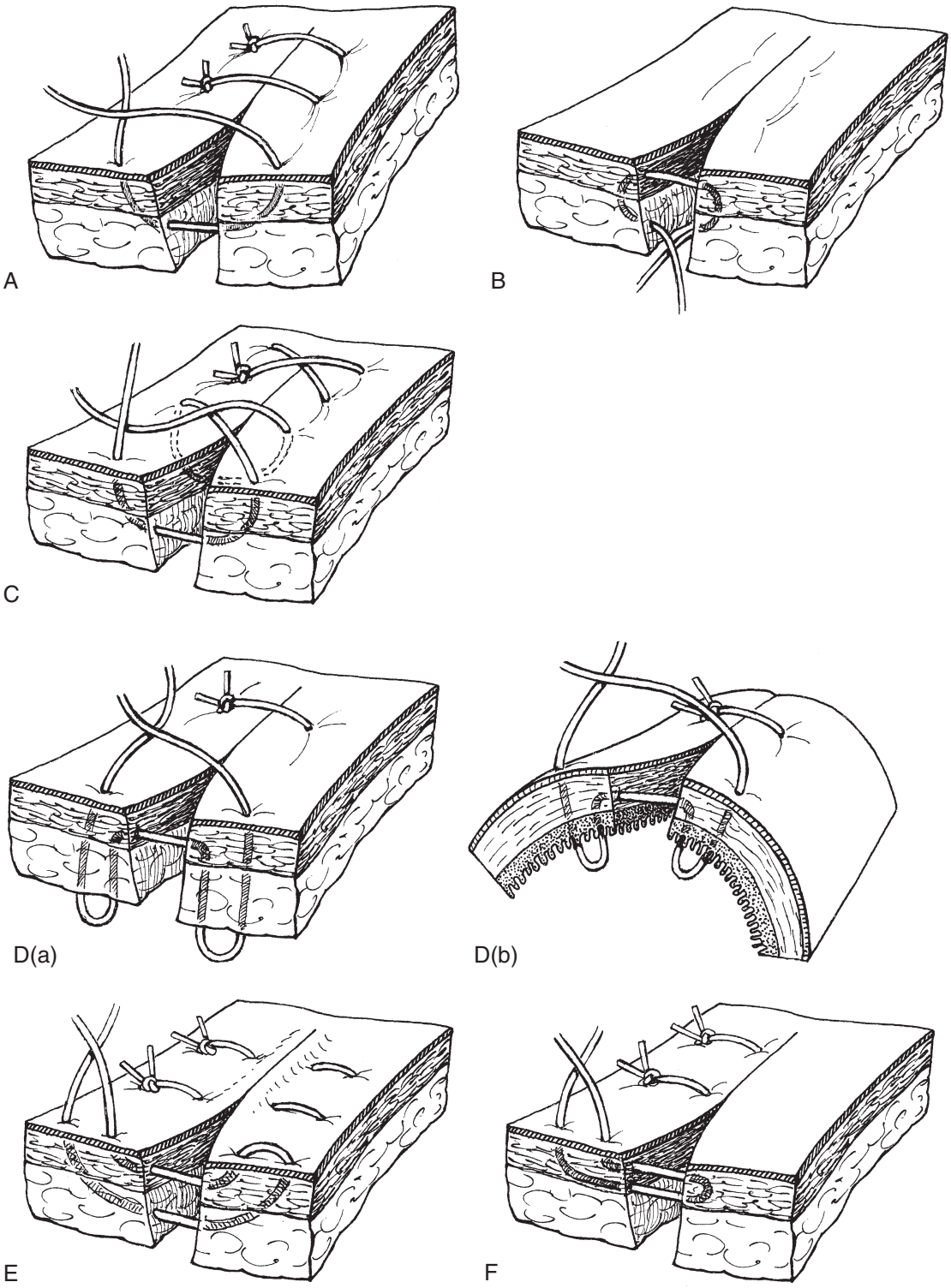

Figure 16-7. Appositional and everting suture patterns: **A,** Simple interrupted; **B,** interrupted intradermal/subcuticular; **C,** cruciate; **D,** Gambee; this pattern can be used as an appositional suture pattern for skin *(a)* or intestine *(b);* **E,** interrupted vertical mattress; **F,** Allgöwer corium vertical mattress.

strength values at 8 weeks postoperatively.[33] Furthermore, suture sinus formation has been reported following the use of polypropylene sutures for closure of the equine linea alba.[34] For this reason, synthetic absorbable suture materials like braided lactomer,[25,35] polyglactin 910, or polydioxanone are recommended. Suture size for closure of the linea alba in adult horses ranges from USP No. 2 to USP No. 7.[25,35] A simple continuous pattern sustains higher loads to failure than interrupted patterns.[36] Tissue bite size should be 15 mm[37] and the interval between the suture bites should be 15 mm as well.[25] This results in a ratio of suture length to wound length of 4:1 or more. This ratio is considered optimal for providing sufficient reserve suture material to accommodate incisional lengthening during episodes of abdominal distention.[25]

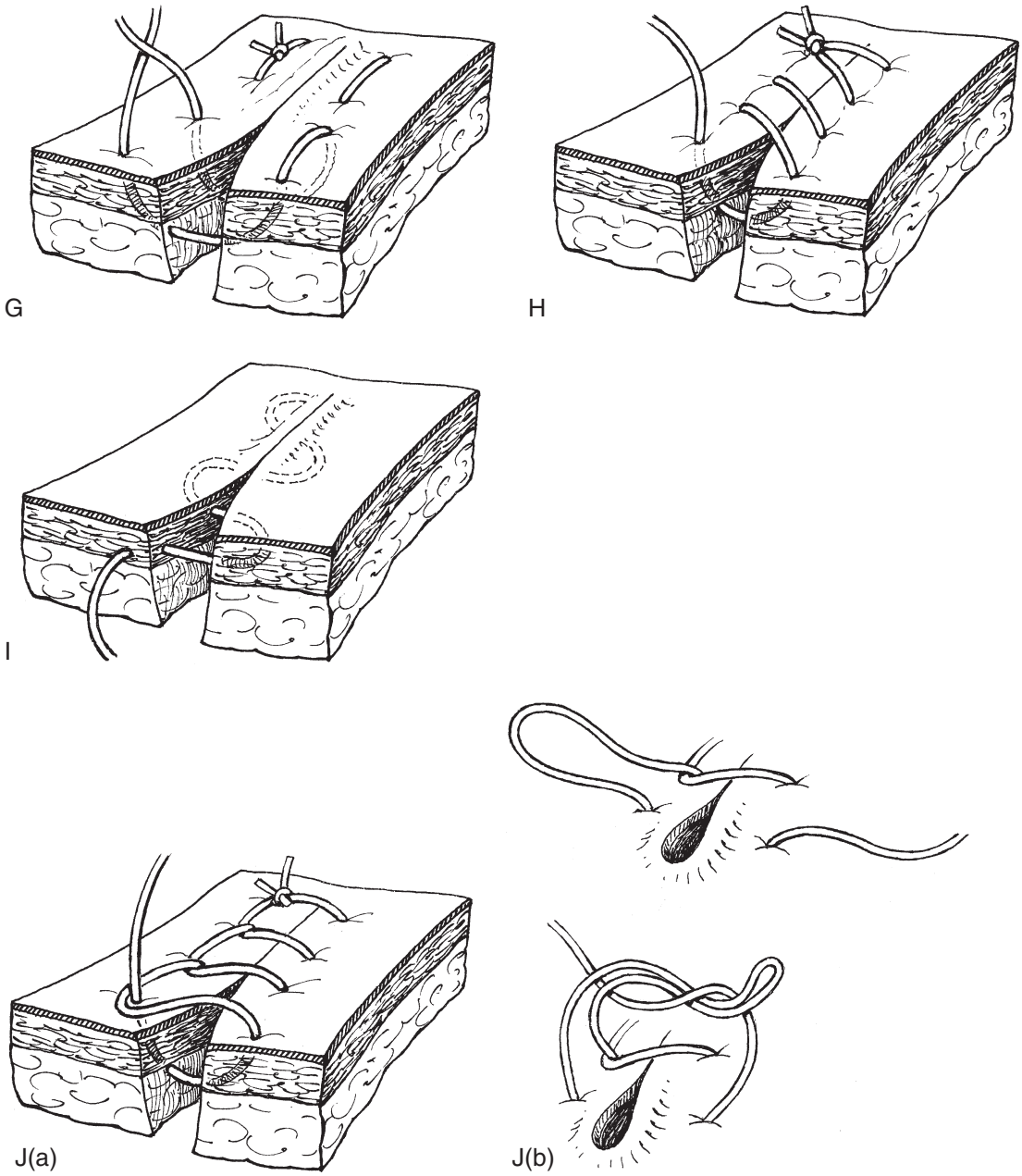

Figure 16-7, cont'd. G, Interrupted horizontal mattress; **H,** simple continuous; **I,** continuous intradermal; **J,** Ford interlocking *(a);* to terminate this pattern, the needle is introduced in the opposite direction from that used previously, and the end is held on that side; the loop of the suture formed on the opposite side is tied to the single end *(b).*

TABLE 16-5. Inverting Suture Patterns

Suture Pattern	Characteristics
Cushing (CU) (Figure 16-8, *A*)	Penetrates the submucosa but not the lumen of hollow viscera; results in a watertight seal, adequate inversion but less luminal reduction than the LE pattern
Connell (Figure 16-8, *B*)	Similar to CU pattern but penetrates all layers of the bowel; subject to wicking of visceral contents
Lembert (LE) (Figure 16-8, *C*)	Penetrates the submucosa but not the lumen of hollow viscera; results in considerable inversion; can also be used for imbrication procedures; can be used as interrupted or continuous pattern
Parker-Kerr (Figure 16-8, *D*)	Indicated to close hollow visceral stumps: a combination of a CU suture sewn over a clamp and pulled tight as the clamp is removed, oversewn by a continuous LE pattern
Pursestring (Figure 16-8, *E*)	Can be used to close the preputial cavity or anus temporarily; if used to close visceral stumps, the stump must be held inverted as the suture is tightened

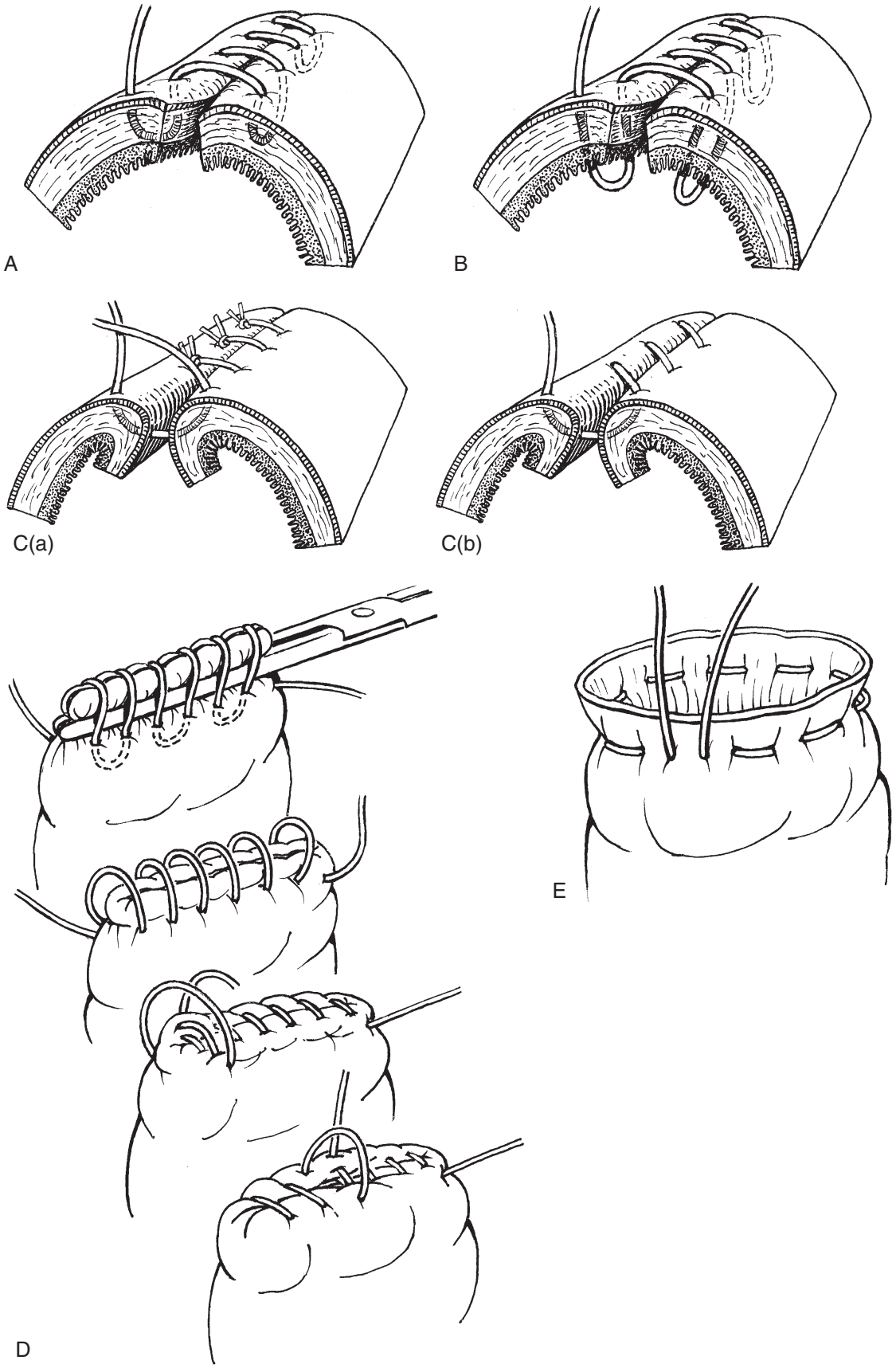

Figure 16-8. Inverting suture patterns: **A,** Cushing; **B,** Connell; **C,** Lembert; this pattern can be applied as an interrupted *(a)* or continuous pattern *(b);* **D,** Parker-Kerr; **E,** pursestring.

TABLE 16-6. Tension Suture Patterns

Suture Pattern	Characteristics
Interrupted vertical mattress (IVM) (Figure 16-9, *A*)	Appositional to everting; stronger under tension and less interference with vascular supply than IHM; stents of soft rubber tubing can be placed under the suture to prevent suture cut-through and impairment of skin circulation
Interrupted horizontal mattress (IHM) (Figure 16-9, *B*)	Degree of eversion depends on suture tension and distance to the wound margin; more everting than IVM; distributes tension over a wider area but is weaker under tension than IVM; higher potential for tissue strangulation and interference with blood supply than IVM; can also be applied in a continuous pattern
Quilled/stented (Figure 16-9, *C*)	Variation of IVM or IHM that loops over a stent/button/plastic tube on either side of the wound to reduce suture cut-through
Near and far (Figure 16-9, *D*)	Can be applied as near-far-far-near or far-near-near-far pattern; provides tension relief (far component) and apposition (near component); high resistance to tension because all suture passes are in the same vertical plane; places more suture material in the wound than other patterns do
Walking suture (Figure 16-9, *E*)	A buried tension suture that moves skin progressively toward the center of a wound; can be placed in rows no closer than 2-3 cm apart; walking sutures evenly distribute tension and obliterate dead space; can potentially damage cutaneous blood supply; large number of walking sutures can increase tissue reaction and foreign body response
Locking loop or modified Kessler (Figure 16-9, *F*)	Strong tension suture for tendon repair; maintains gliding function of the tendon owing to limited amount of suture material on the tissue surface; two locking loop sutures can be combined to form a double locking loop
Three-loop pulley (Figure 16-9, *G*)	Very strong tension suture for tendon repair with increased resistance to gap formation; may compromise gliding function because of a large quantity of suture material on the tendon surface

Infected or Contaminated Wounds

Sutures should be avoided in highly contaminated or infected wounds because even the least reactive suture can exacerbate infection. Multifilament nonabsorbable sutures should not be used in infected tissue because they potentiate infection and may lead to fistulation.[6] If a suture is required in a contaminated or infected wound, absorbable and ideally monofilament suture material is indicated. If implantation of a nonabsorbable suture is unavoidable, monofilament nylon and polypropylene are least likely to elicit infection in contaminated tissues.

Muscle

Muscle is difficult to suture because it has poor holding power. Sutures placed parallel to the muscle fibers are prone to pull out; therefore, sutures should be placed perpendicular to muscle bundles when possible. Whenever achievable, the fascial layer should be incorporated to improve holding capacity. Synthetic absorbable or nonabsorbable sutures may be used to suture muscle layers.

Gastrointestinal Tract

Gastrointestinal incisions demonstrate rapid postoperative healing. Physical strength is dependent on suture or staple strength during the lag phase (i.e., the first 4 days postoperatively) of wound healing. During the proliferation phase (3 to 14 days postoperatively), wound strength increases rapidly and the maturation phase has little clinical relevance.[38] Absorbable synthetic sutures are indicated for gastrointestinal sutures. Prolonged retention of tensile strength is not necessary. Low tissue reactivity is desirable to prevent further luminal reduction and adhesion formation. Polyglycolic acid, polyglactin 910, and polydioxanone can be used. The monofilament suture material glycomer 631 has the advantage of combining reduced capillarity and tissue drag with an appropriate resorption profile. Although the use of a simple interrupted or the Gambee pattern has been described for equine intestinal anastomoses,[39,40] a Lembert pattern or a simple continuous pattern oversewn with a Cushing pattern are more commonly used.[41,42]

Urinary Tract

Compared with healing of the gastrointestinal tract, the urinary bladder has a more rapid healing rate and gain in tensile strength.[43] Sutured cystotomy wounds need to withstand voiding pressures of 90 cm H_2O.[44] Suture materials used in cystotomy closure should provide adequate strength during the lag phase of wound healing, followed by rapid absorption to avoid lithogenesis in case of mucosal penetration. In addition, low tissue reactivity is needed to further reduce the risk of calculus formation.[45] Exposure to alkaline urine—as found in herbivores—results in accelerated hydrolysis of absorbable suture materials.[46,47] Urinary tract infections with pathogens like *Proteus mirabilis* can further accelerate loss of tensile strength if suture materials are exposed to urine.[47] Nonabsorbable sutures and metallic staples may be calculogenic and should be avoided.[48] Absorbable synthetic sutures are recommended, and monofilament sutures have the additional benefit of reduced capillarity and tissue drag. The use of quickly absorbable suture materials like poliglecaprone has not been evaluated in horses but seems a possible choice, given the rapid healing capacity of the urinary bladder. The suture pattern should be continuous to provide a tight seal and should be inverting. Penetration of the transitional epithelium should

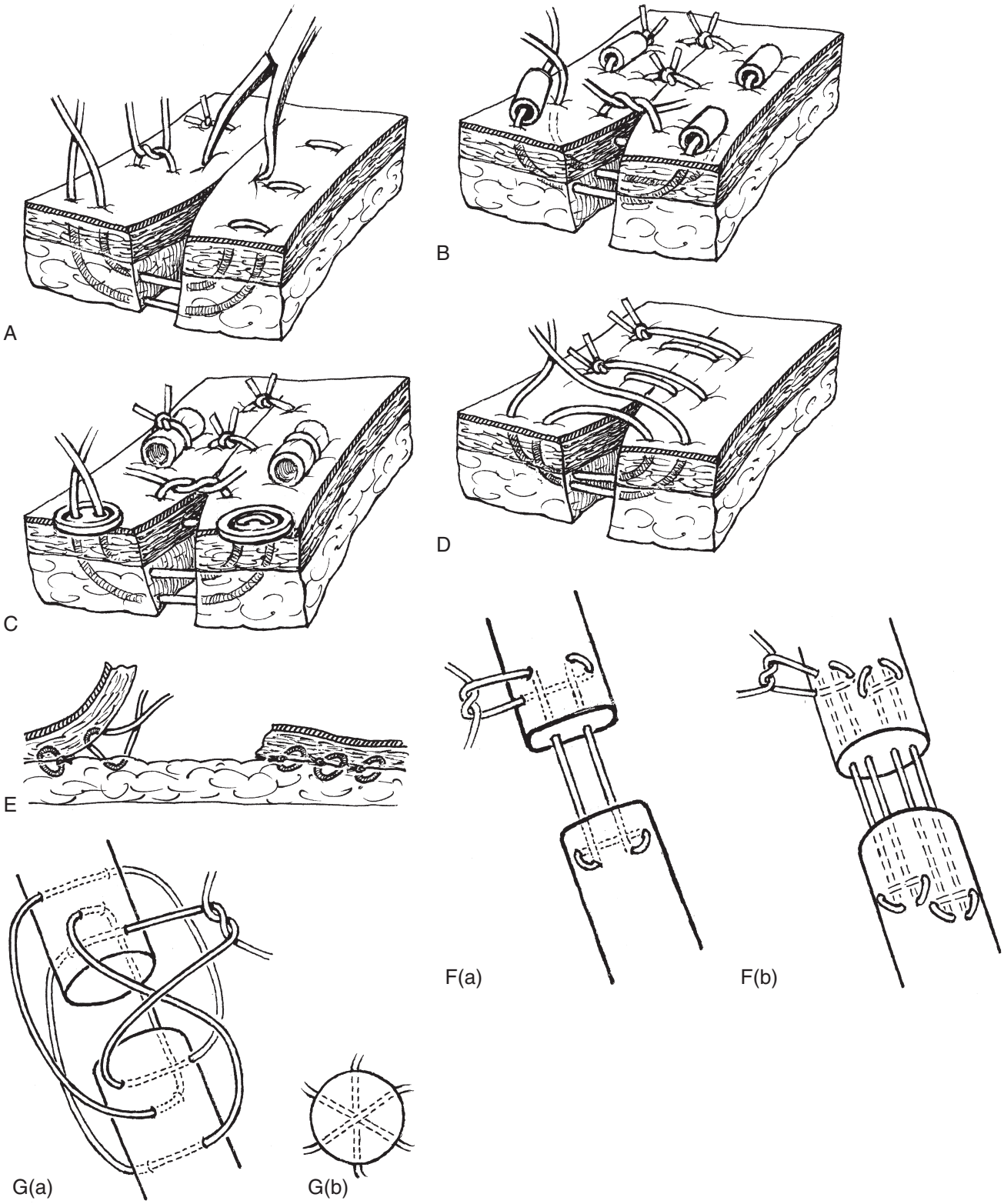

Figure 16-9. Tension suture patterns: **A,** Interrupted vertical mattress pattern used as a tension suture; **B,** interrupted horizontal mattress pattern placed as a tension suture with stents to reduce focal pressure on the skin, followed by a simple interrupted suture pattern to achieve wound closure; **C,** quilled/stented; **D,** far-near-near-far; **E,** walking suture; **F,** locking loop *(a)* and double locking loop *(b);* for the locking loop patterns, bites perpendicular to the tendon fibers are superficial relative to bites that are aligned parallel to the fibers; **G,** 3-loop pulley pattern *(a)* with a cross-sectional view *(b)* of this pattern demonstrating that each loop is oriented 120° relative to the others.

be avoided. For urinary tract procedures that result in exposure of the suture material to urine, polyglyconate or polydioxanone are recommended to avoid premature loss of tensile strength.[16,46]

Tendon

The most common suture patterns for tendon repair are the locking loop and the three-loop pulley suture (see Figure 16-9). The three-loop pulley suture pattern is more resistant to gap formation under tensile loading.[49] Appropriate suture materials include strong, nonabsorbable sutures or slowly absorbable materials with high tensile strength retention, like polydioxanone or polyglyconate. However, none of these sutures can maintain flexor tendon apposition under normal loading conditions in an adult horse[50] and additional external coaptation is required if tenorrhaphy is attempted. Application of bioresorbable tendon plates has resulted in superior immediate failure strength compared to 3-loop pulley sutures but has only been evaluated biomechanically in a cadaveric study.[51]

Blood Vessels

Vessels should be ligated with absorbable suture material. Vascular repair or anastomosis is performed with monofilament nonabsorbable suture material. Polypropylene is the material of choice because it is the least thrombogenic suture.[6]

Nerves

Nonabsorbable sutures with low tissue reactivity, like polypropylene or nylon, are recommended for nerve repair.[32]

Implant Prostheses

Strong nonabsorbable suture materials can be implanted to serve as a permanent prosthesis (e.g., for laryngoplasty, tie-forward or joint stabilization). Polyester sutures can be used for these purposes. However, the newer ultra-high molecular weight polyethylene sutures are stronger,[52] have less tissue drag, and provide better knot security.[53]

ANTIMICROBIAL-COATED SUTURE MATERIAL

Surgical site infections (SSIs) remain an important problem in the surgical community. There is some evidence that the suture knot may play a role as a repository for bacterial colonization and replication that can ultimately result in an SSI (see Chapter 7).[54] To achieve active inhibition of bacteria at the surgical site, antimicrobial-coated suture materials were developed. The agent most commonly used for this purpose is triclosan, chemical name 5-chloro-2-(2.4-dichlorophenoxy)-phenol.[55] Triclosan has antiseptic properties and good biocompatibility.[56] Experimental studies confirmed the inhibitory effects of triclosan-coated polyglactin 910,[57] polydioxanone,[58] and poliglecaprone[59] suture material on bacterial colonization. Most clinical studies in human medicine report reduced wound infection rates with the use of triclosan-coated suture materials,[54,60] although these results were questioned in one study.[61] Up to now, there is only one study published on clinical application of triclosan-coated suture material in the horse. This study could not find a beneficial effect on incisional complication rates when triclosan-coated suture material was used for subcutaneous closure following exploratory celiotomy.[62] However, the serious complications experienced with SSI in horses would make these materials attractive for further evaluation.

SUTURE ANCHORS

Suture anchors serve to attach soft tissues to bone or to fix a suture as a prosthetic implant. These devices are commercially available in a variety of configurations. Typically, they have a metal end configured as either a screw or a toggle bar and an "eye" for suture attachment (Figure 16-10).[63] Suture anchors have been used in equine patients for surgical repair of collateral ligament instability of the carpal and metacarpophalangeal joint in two foals[64] and for a prosthetic capsule technique in a pony with coxofemoral luxation.[65]

SURGICAL STAPLERS

Surgical stapling devices are commonly used in equine surgery, especially for intestinal resections, anastomoses, ligation of blood vessels, and skin closure. Potential benefits of stapling include reduced surgery time, less tissue trauma, less intraoperative contamination, preservation of blood supply, and utility in areas of difficult accessibility.[3,66]

Stapling Devices

Thoracoabdominal Stapler

Thoracoabdominal (TA) staplers (Figure 16-11) are loaded with a cartridge (also called *single-use loading unit*) and fire one double-staggered row of B-shaped staples to seal tissues and vessels with preservation of microcirculation. Titanium staples are commonly used but absorbable lactomer staples are available as well. Cartridge sizes for reusable stainless steel TA stapler devices are 30, 55, or 90 mm in length. Cartridges for disposable re-loadable staplers are available in 30, 45, 60, and 90 mm lengths.[66] Staple cartridges are color coded to indicate staple

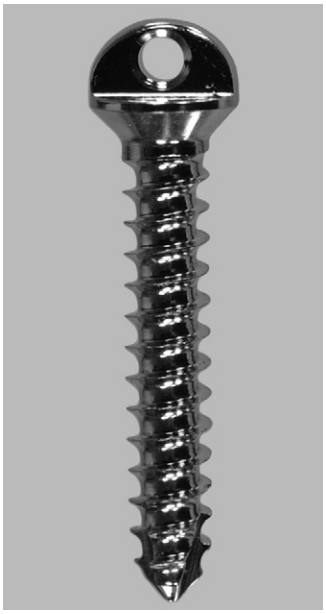

Figure 16-10. Example of a suture anchor: a self-tapping 3.5 mm diameter cortex screw with an eyed head.

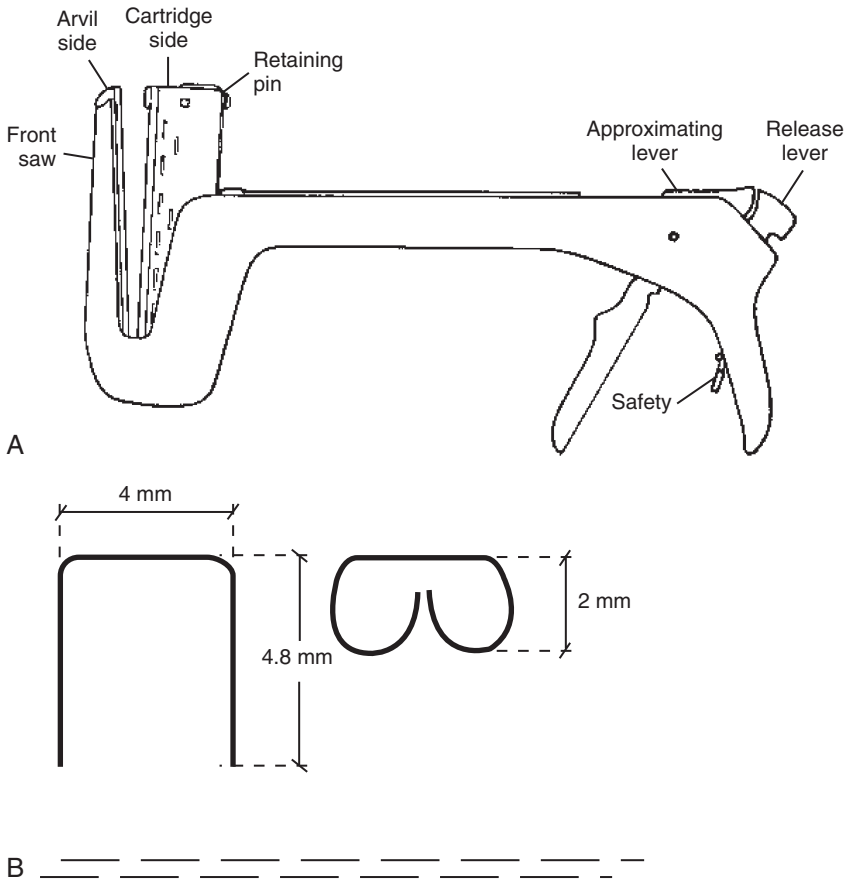

Figure 16-11. A, TA-90 Premium stapler with a disposable cartridge, schematic labeled view; **B,** the TA-90 Premium 4.8 loading stapler fires 33 staples arranged in a double-staggered row 91.5 mm long, schematic labeled view. The staples of the green cartridge have a crown width of 4 mm, a leg length of 4.8 mm, and a closed height of 2 mm.

size. Green cartridges contain staples that have a leg length of 4.8 mm, crown width of 4.0 mm, and closed height of 2.0 mm. The staples in the blue cartridge have a leg length of 3.5 mm, a crown width of 4.0 mm, and closed height of 1.5 mm (see Figure 16-11). TA staplers have a U-shaped opening through which the tissues are inserted. Tissues are secured within the device by a retaining pin. Activating the approximating lever closes the cartridge. After releasing the safety device, squeezing of the handle forces the staples out of the cartridge against the anvil. After firing, the instrument head can be used as a guide for tissue transection. The TA stapler is released by retracting the release lever and loosening the approximating lever.

In equine surgery, the 4.8-mm staples are commonly used because of the longer staple leg. The TA-90 is useful for colon resection,[67] jejunocecostomy,[68] jejunocolostomy,[69] ovariohysterectomy,[70] partial lung lobe resection,[71] rectal tear repair in postparturient mares,[72] and partial splenectomy.[73] It can also be beneficial to achieve hemostasis in areas that are difficult to access, such as bleeding from the testicular or ovarian artery after neutering.[74]

Gastrointestinal Staplers

Gastrointestinal anastomosis (GIA) and intestinal linear anastomosis (ILA) staplers are linear stapling instruments with two interlocking halves (Figure 16-12). Like the TA staplers, they are loaded with cartridges (single-use loading units). Cartridge sizes for reusable stainless steel GIA instruments are 50 or 90 mm in length.[66] Disposable reloadable GIA staplers are available in 60, 80, and 100 mm lengths. The reusable ILA stainless steel stapler is available in 52 and 100 mm lengths.[66]

Gastrointestinal staplers apply four staggered rows of staples; cartridges contain cutting blades that divide tissues between the second and third row of staples. The instrument separates into two halves so that each fork of the instrument can be placed into a bowel lumen or on either side of a hollow viscus (Figure 16-13). After closure, the push bar handle of the device is slid forward to fire the staples and the blade. The incision cut by the knife blade is 8 mm short of the last staple at the distal end.[66] Staples are made of stainless steel or titanium and, as with TA staplers, are B-shaped when closed. The B configuration of the closed staple permits blood flow through the tissue enclosed by the staple.[3] Color coding of cartridge size is the same as for TA staplers. Staples in green cartridges have a 4.8 mm leg length that compresses to a final height of 2.0 mm, whereas staples in blue cartridges have a 3.8 mm leg length that compresses to a final height of 1.5 mm. Staples in both cartridges are 4.0 mm wide. Reusable GIA instruments are only available with 3.8 mm staples.[66] When used for side-to-side or functional end-to-end anastomosis, the result is a stoma with two rows of staples on either side. The instrument insertion site remains open and must be closed by suturing or by applying of a TA stapler. When used for viscus resection, two rows of staples provide an everted seal along the cut margin of the healthy organ; the resected portion of the viscus is also sealed with two rows of staples, reducing intraoperative contamination.[66]

Common indications for use of GIA or ILA staplers in equine surgery include jejunojejunostomy,[75] jejunocecostomy,[68] and jejunocolostomy.[69]

Endoscopic versions of gastrointestinal staplers are also available in variable sizes and have been used for laparoscopic

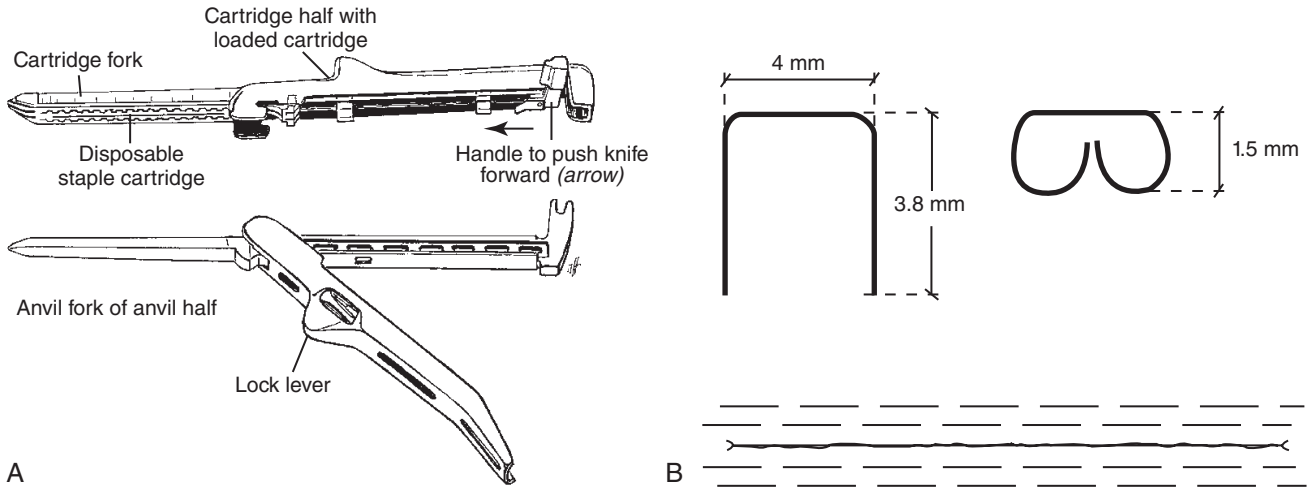

Figure 16-12. A, GIA-90 Premium stapler with disposable cartridge, schematic labeled view; **B,** The GIA stapler fires two double, staggered rows of staples. Staples of the blue cartridge for reusable GIA instruments have a crown width of 4 mm, a leg length of 3.8 mm, and a closed height of 1.5 mm. The instrument's knife blade cuts between the two sets of staple lines, ending approximately 5 mm short of the last staple in the distal end.

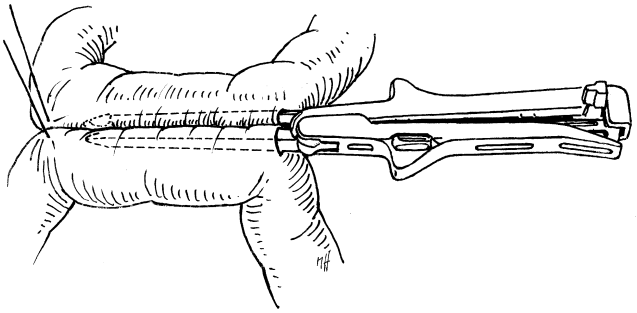

Figure 16-13. Each fork of the GIA instrument is placed into the bowel lumen; after closure, the push bar handle of the device is slid forward to fire the staples and the blade.

ovariectomy[76] and laparoscopic small intestinal biopsy in horses[77] as well as for laparoscopic sterilization of male donkeys.[78]

Ligating Dividing Stapler

The ligating dividing stapler (LDS) is a pistol-shaped instrument that places two vascular staples simultaneously while a cutting blade divides the blood vessel–containing tissue between them (Figure 16-14). In the horse, this instrument is mainly used for rapid ligation of mesenteric vessels during colic surgery. Metal staples are commonly used with this device and are made of surgical steel or titanium.

The U-shaped staples come in two sizes: regular, which is 5.8 mm wide × 5.2 mm tall, with a final closure width of 5.3 mm and a distance between staples of 6.35 mm; and wide, which is 8.0 mm wide × 7.2 mm tall, with a final closure width of 7.3 mm and a distance between staples of 9.53 mm.

The closed staple forms a thin crescent shape with the ends of the staples meeting at the center of its outer rim. Vessels that need double ligation require placement of a ligature or a single vascular clip before LDS application. The LDS should not be used on tissues that cannot be compressed to 0.75 mm.[66] In an experimental study in horses evaluating jejunal artery occlusion, mean arterial bursting pressure achieved with the LDS was significantly lower than after LigaSure application or 2-0 PDS

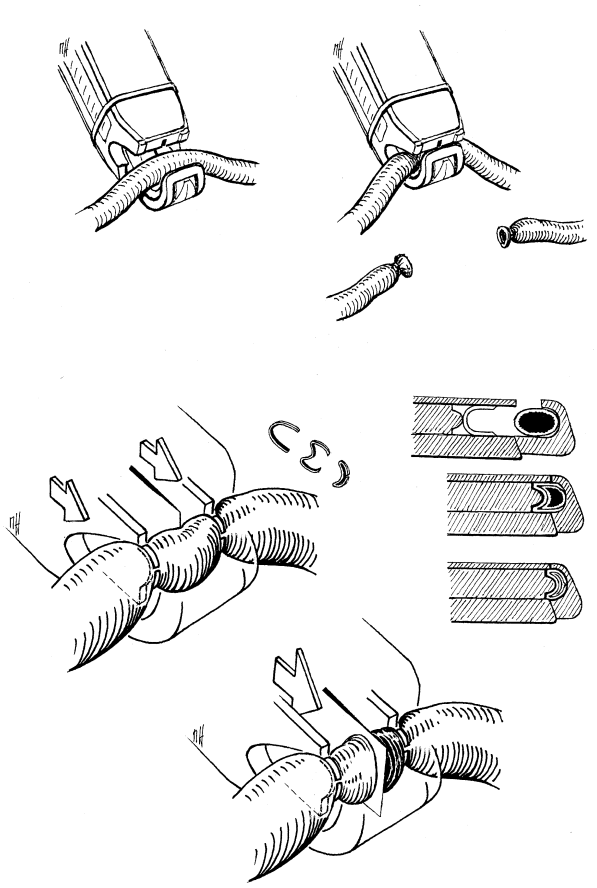

Figure 16-14. The ligating dividing stapler is a pistol-shaped instrument that places two vascular staples simultaneously while a cutting blade divides the blood vessel–containing tissue between them. The closed staple forms a thin crescent shape with the ends of the staples meeting at the center of its outer rim.

ligation but still far above systolic pressure values.[79] However, in the clinical patient, hemorrhagic strangulating obstruction is commonly associated with congested vessels and hemorrhagic changes of the associated mesentery. The subsequent increase in tissue thickness makes application of the LDS less reliable, and an additional suture ligation may be required.

Ligating Clips

Ligating clips can be useful to achieve hemostasis. Metal clips are commonly used but synthetic absorbable clips are available as well. The advantages of ligating clips include ease of application in poorly accessible areas, structural stability, and reduction of surgery time. To provide safe hemostasis, the diameter of the vessel should be one third to two thirds the size of the clip, the vessel should be dissected free of surrounding tissue before the clip is applied, and 2 to 3 mm of vessel should extend beyond the clip to prevent slippage.[6]

Manufacturer recommendations should be reviewed regarding clip size selection for specific vessel diameters. Potential disadvantages of ligating clips include the relative instability of the clip in the applicator, insecurity of an inadequately applied clip, potential slippage, and permanence of metallic clips in the tissue.[3] Caution should be used when manipulating tissues after placement of vascular clips because they are more easily dislodged than suture ligations.[66]

Skin Staples

Surgical skin staples are fabricated from surgical stainless steel. Before application, the skin staple is U-shaped. During application, the cross member is bent over an anvil, crimping it at two sites and bringing the legs together. This results in a rectangular shape of the closed staple, which is narrower than the original staple.[66] Staple removal is performed by a staple extractor, which compresses the cross member of the staple and straightens the legs, permitting easy extraction (Figure 16-15). Skin staples are suitable for rapid closure of surgical incisions that are not subjected to appreciable tensile forces. They provide excellent wound edge eversion without strangulation of tissue[80] and incite only minimal tissue reaction.[30] They are commonly used in equine surgery with excellent functional and cosmetic results. However, an experimental study in pigs demonstrated some inflammatory responses after skin staple application.[81] A recent meta-analysis found a significantly higher risk of developing a wound infection after orthopedic surgery in humans when the surgical wound was closed with staples rather than sutures.[82] Similarly, a large case series of horses undergoing exploratory celiotomy identified the use of staples for skin closure as a significant risk factor for development of an SSI.[83]

A novel form of skin closure that uses absorbable lactomer subcuticular staples is available. They are inserted into the subcuticular tissue with the help of a staple applicator and forceps. Subcuticular staples produced less inflammatory response and a superior cosmetic outcome than metal skin staples in human surgery[84] and in a porcine experimental model.[81] Application of absorbable subcuticular staples in equine surgery has not been described yet.

TOPICAL TISSUE ADHESIVES

2-Octylcyanoacrylate

Tissue adhesives based on 2-octylcyanoacrylate are available as a dermal suture replacement. Their advantages include faster closure, reduced cost, ease of application, and no need for suture removal. In human medicine, they are considered equivalent to other methods of skin closure in terms of cosmetic outcome, infection rate, and dehiscence rate.[80] Tissue adhesives should not be applied to tissues within wounds; instead, they should be applied to intact skin at the wound edges to hold the injured surfaces together. Adhesives are particularly useful in superficial wounds or wounds in which the deep dermal layers have been closed with sutures. Furthermore, 2-octylcyanoacrylate can be used to attach intravenous or nasolacrimal catheters, skin grafts, or wound dressings.[63]

Currently, the use of topical tissue adhesives in the equine patient is limited because they should not be used for wounds in mucous membranes, contaminated wounds, large or deep wounds, and wounds under tension.[80]

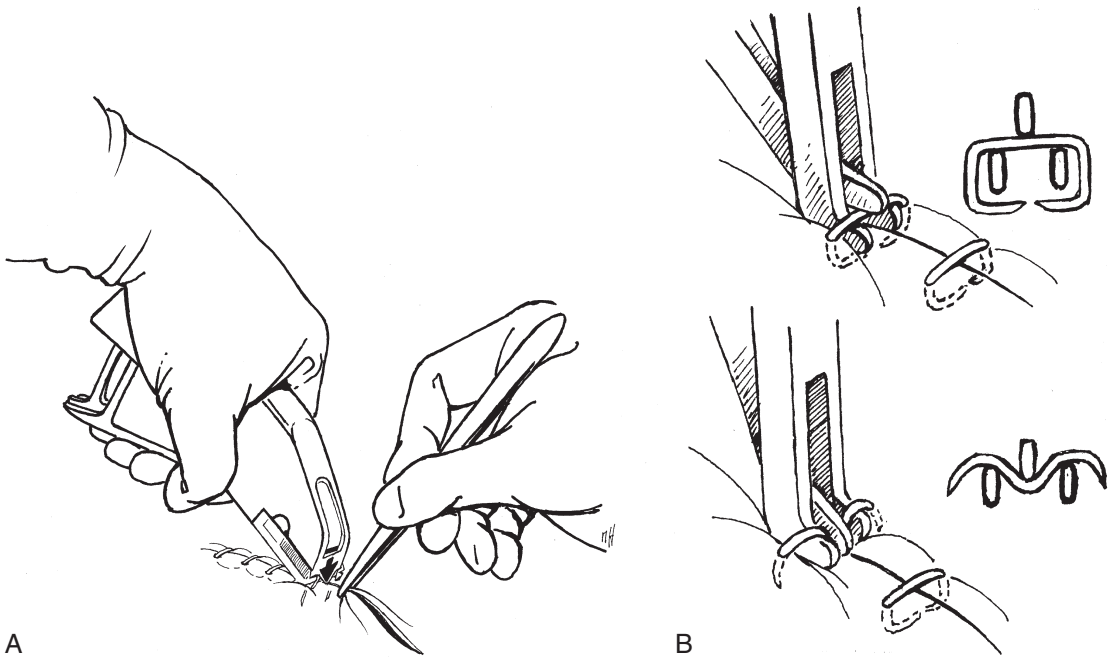

Figure 16-15. **A,** Skin staplers are applied with the help of a forceps to achieve slight eversion of the skin. **B,** Staple removal is performed by a staple extractor, which compresses the cross member of the staple and straightens the legs, permitting easy extraction.

Fibrin Glues

Fibrin glues are mainly composed of concentrated fibrinogen, thrombin, and calcium chloride, thus duplicating the final stage of the coagulation cascade. Fibrin acts as a hemostatic barrier, adheres to surrounding tissue, and serves as a scaffold for migrating fibroblasts.[85] Fibrin glues are used as a tissue adhesive for a variety of surgical procedures in human and small animal medicine (e.g., control of hemorrhage from parenchymal tissues, as a supplementary sealant in intestinal, parotid duct or vascular anastomoses, as a carrier or adhesive agent in bone regeneration–enhancing procedures, and for augmentation of skin closure).[86-89] Fibrin glues can also be applied in minimally invasive surgery. An experimental study in pigs demonstrated superior results achieved with the application of fibrin glue for laparoscopic closure of a ureterotomy compared to laparoscopic suturing or laser welding.[90] The main advantages of fibrin glues are tissue compatibility, biodegradability, and efficacy when applied to wet surfaces.[86] Few studies have evaluated the application of fibrin glues in equine surgery. One study showed no difference in graft acceptance between split-thickness skin grafts applied with cyanoacrylate alone or with a combination of cyanoacrylate and fibrin glue.[91] Another group used fibrin glue to fix a periosteal autograft over an osteochondral defect.[92] Use of fibrin glue as a carrier matrix for mesenchymal stem cells or bone marrow mononucleated cells for treatment of tendinitis represents a more promising application in the equine patient.[93] Further potential applications include laparoscopic and endoscopic procedures and its use as a sealant in wound closure in combination with other techniques.

Tapes: Steri-Strips

Modern cutaneous tapes play an important role in wound closure in human surgery. Closure with microporous tape produces more resistance to infection than other closure techniques.[80] Tapes maintain the integrity of the epidermis and thus result in less tension to the wound. They are indicated for linear wounds in areas with little tension. Tapes do not adhere to mobile areas under tension or to moist areas. These tapes can also be used over sutures to provide a partially closed environment and improve cosmesis. Wound edge approximation is less precise with tape alone than with sutures. Wound edema can lead to blistering at the tape margins and to eversion of taped wound edges.[80] Because of these disadvantages, tapes are not routinely used in equine surgery but may be used for certain specific indications.

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CHAPTER 17

Drains, Bandages, and External Coaptation

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The application of drains, bandages, and external coaptation is an important step in state-of-the-art wound management. The different dressings used in association with bandages are discussed in Chapter 26.

DRAINS AND DRAINAGE

History

Hippocrates, in the 4th century BC, was the first to use drains in the form of hollow tubes, to treat empyemas. In the 2nd century AD, Celsus and Galen used conical tubes of brass and lead to drain ascites, and these devices were used for 1500 years. In 1719, Heisler introduced capillary drainage via a gauze wick inside a metal tube.[1,2] In 1859, Penrose used soft rubber tubing as a drain, known today as the Penrose drain. Kehrer modified this technique in 1882 by placing gauze inside the Penrose drain to facilitate drainage, thus creating the "cigarette drain." Today's version of the cigarette drain consists of semirigid vinyl or polyvinyl tubing inserted into a Penrose drain to prevent soft tissue obstruction and increase capillary action.[3,4] All of these drains were applied in a passive system, allowing gravity, capillary action, natural pressure gradients, or overflow to control fluid and gas emanations. Negative pressure was subsequently applied to the semirigid tubes to provide an active system, and finally Raffle developed the technique of continuous suction in 1952.[5]

Purposes

Drains are implants designed to channel unwanted fluids (such as wound secretions, purulent material, bile, urine, blood, or gases) out of the body.[6] Proper use of drains generally speeds up healing time, whereas inappropriate use usually delays healing, occasionally even increasing morbidity and mortality. There are three reasons to place a drain: (1) to facilitate elimination of dead space, (2) to evacuate existing fluid and gas accumulations, and (3) to prevent anticipated formation of fluid collections.[7] Understanding the principles of drain selection, placement, and management minimizes the risks associated with these implants.

Materials

The ideal drain is inert, soft, nonreactive, and radiopaque. Table 17-1 lists common drain types and materials. Soft latex is frequently used in drains; it allows excellent passive drainage of wound fluids. Because it is pliant and does not maintain a rigid lumen, it fits comfortably within the wound. Polyvinylchloride (PVC) drain tubes provide excellent wound fluid evacuation, especially from body cavities and deep surgical wounds. They are less flexible than latex and have a rigid lumen, allowing them to be used for passive or active systems. Frequently, PVC drains are multifenestrated to permit fluids to exit the wound or body. Other drains are manufactured out of silicone, an organic compound in which all or part of the carbon has been replaced by silicon (a nonmetallic element occurring in nature as silica).[8] Silastic is the trade name for polymeric silicone substances having the properties of rubber; it is biologically inert and frequently used in applications other than drains.[8] It is softer than PVC, but at some diameters it maintains a rigid lumen. Therefore, Silastic can be used for active or passive drainage systems. The compliance of the material increases the animal's comfort and makes this type of drain ideal for placement in sensitive areas, next to bone, and within small spaces.[9,10]

Placement

The basic principles of wound management, such as clipping of the hair, aseptic preparation of the implantation site, and possibly local anesthesia, are considerations when placing a drain. In sterile wounds, the drain should be applied under aseptic conditions. Additionally, this sterile environment should be maintained as long as possible by covering the wound and by making frequent bandage changes. Passive drains should exit ventral to the most dependent aspect of the wound or dead space.

The drains should be placed into the space requiring the most drainage. Occasionally, several drains are needed to evacuate a large area or several different tissue layers. The shortest and most direct avenue for evacuation of secretions should be selected. Drains cause some mechanical irritation and therefore should not be placed in the immediate vicinity of blood vessels, nerves, and suture lines. To reduce the risk of suture dehiscence, drains should exit through separate incisions, and not through the suture line (Figure 17-1, *A*). It is important to secure the drains with individual sutures to prevent their loss into or out of the wound. A suture is placed from the skin into the wound, through the drain, and back through the skin, where it is tied (see Figure 17-1, *B*). The suture used for securing the drain should be easily distinguishable from the skin sutures to avoid inadvertent premature removal of an incisional suture. If a drain is placed into a wound that is to be closed, care should be taken to avoid inadvertent incorporation of the drain into the suture line, because drains are usually removed before incisional sutures (see later). The drain end should be long enough to prevent its disappearance into the wound when the patient moves and to evacuate drainage fluids. It is also important to protect the drains from attempts by the patient to remove them. Large openings provide better and longer-lasting drainage. Small exit incisions frequently become blocked, even with a drain in place, preventing effective evacuation of drainage material.

TABLE 17-1. Drains

	Material	Mechanism of Action; Function	Advantages	Disadvantages
PASSIVE DRAINS				
Gauze drains*	Fine mesh gauze	Gravity Capillary action	Economical	Adherence of fibrin clots to gauze
Penrose drain†	Soft, pliable latex available in various sizes Hollow tube	Gravity Capillary action Mostly drainage around periphery	Economical Many applications	May easily kink Not applicable in body cavities No suction possible May facilitate ascending infection
Silicone Penrose drain‡,§	Soft, pliable, nonreactive silicone	As Penrose drain	Less irritating Use in latex-sensitive patients Contains radiodense marker	Not applicable in body cavities No suction possible
Rubber tube drains	Red rubber Smooth surface	Gravity Capillary action	Because of relative stiffness, rarely compressed or occluded Suction may be applied	Increased foreign body reaction
Well drain¶ (German for "waved drain")	Waved sheet of red rubber, stiff, can be cut to size	Gravity Capillary action	Because of relative stiffness, rarely compressed or occluded	Increased foreign body reaction
Flexi-Drain§	12 silicone tubes, 3 mm in diameter, joined together parallel to each other	Gravity Capillary action Good drainage along the tubes where they join	Can be split longitudinally to adjust size of drain Suction may be applied	Main drainage externally
ACTIVE DRAINS				
Redon drain¶	Round, multifenestrated PVC tube with nonfenestrated extension	Closed or open drainage system	Can be used as closed or open drainage system Excellent for evacuation of fluids from body cavities	Depending on location, attaching the container may be difficult Tube cannot be used universally
Jackson-Pratt drain¶	Flat Silastic, multifenestrated drain with nonfenestrated extension	Closed or open drainage system	Can be used as closed or open drainage system Excellent for evacuation of fluids from body cavities Less reactive	Depending on location, attaching the container may be difficult
Blake drain*	Round pliable Silastic drains with slits at the end	Closed or open drainage system	Multifaceted slits reduce the risk of clogging up Minimal tissue irritation	Reactively voluminous Suction function possible only when skin suture is tight
Trocar catheter#	Round, multifenestrated tube Inserted with blunt trocar into the chest	Drainage of thoracic cavity	Minimal reaction and irritation Effective fluid drainage from thorax	Relatively easily disloged, interrupting effective drainage

*Johnson & Johnson, New Brunswick, NJ: Triclosan-Gauze IVF Hartmann, Neuhausen, Switzerland.
†Sherwood Medical, St. Louis, MO.
‡Easy-Flow drain, Degania Silicon LTD, Degania Bet, Israel.
§Cook Veterinary Products, Eight Mile Plains, Queensland, Australia.
¶Nelaton, Ruesch, Belp, Switzerland.
¶Zimmer, Inc. Dover, OH.
#Mallinckrodt Medical, Athlone, Ireland.

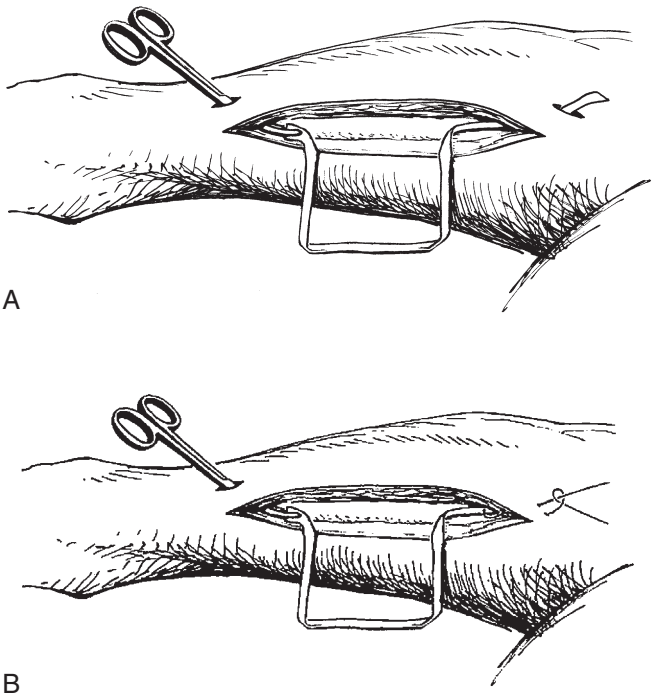

Figure 17-1. Proper placement of a Penrose drain for passive postoperative drainage. **A,** The two exit portals for the drain are placed distant from the primary incision. The ends of the drain are secured with a suture each (using sutures of a different type from the skin sutures for easy recognition). **B,** Only the distal exit portal is made, and the proximal-most aspect of the drain is secured within the wound with a suture that enters proximal to the wound through the skin, passes through the drain, and exits the skin again, where it is tied. The wound is subsequently closed.

Management

The amount of drainage and its consistency dictate the frequency with which bandages need to be changed or vacuum containers emptied. The exit site should be cleaned with antiseptic solutions at every bandage change. If a passive drain is used, it is advisable to protect the adjacent skin from irritation by covering it with a thin layer of Vaseline. Passive drains should seldom if ever be back-flushed, and active drains should not be back-flushed unless obstructed because of the risk of transporting microorganisms into the wound. Additionally, healing may be interrupted by the mechanical disturbance of flushing.

Re-establishing drainage in an obstructed drain exit wound should be performed carefully. If the drain exit site is obstructed, it should be reopened. First the site is prepared for aseptic surgery, followed by inserting a sterile hemostatic forceps into the opening and gently spreading its jaws to separate the wound ends. If needed, some surrounding tissues are removed with scissors or a small scalpel. Because round wounds heal much more slowly than triangular or square/rectangular wounds, it is advisable to create a round drainage opening to ensure an exit portal that will be open for a longer period of time.

Removal

As a general rule, drains should be removed as quickly as possible. An average time for maintaining drains is 2 to 4 days, the duration of the débridement period of wound healing. However, there are exceptions to this rule:

- When evacuating blood from small cavities, the drain may be removed after approximately 24 hours.
- When treating bacterial infections, the drain should be maintained for 48 to 72 hours.
- If large dead spaces remain, for example, after tumor removal, the presence of a drain may be necessary for as long as 2 weeks.

The best indicator for drain removal is an abrupt decrease in the drainage volume and a change in its characteristics to a serous, non-odiferous, slightly turbid fluid. Because drains are foreign material, they induce the production of secretions. At the time of drain removal, exit sites are prepared for aseptic surgery. While the proximal end is held in place, the distal securing suture is removed, followed by application of slight tension to the distal end of the drain, before it is cut off at skin level. This ensures that the contaminated external part of the drain is not pulled through the wound bed, possibly recontaminating it. The proximal suture is removed, and the rest of the drain is pulled out of the wound bed through the distal portal. The two incisions are left to heal by secondary intention. In cases where only one exit portal exists, the securing suture(s) is (are) cut and the drain is removed through the distal portal.

If gauze packs are used as tamponade in a bleeding or actively secreting wound bed, they are removed in stages, with a portion withdrawn and cut off daily, each time leaving a protruding stump to facilitate removal of the next portion.

Complications

Foreign body response and ascending infection are the most common complications associated with drain use. Because drains are foreign bodies, a certain adverse response to the drain cannot be avoided. If a portion of the drain is accidentally left in the wound, wound drainage will persist until it is removed. Therefore, the removed drain should be carefully examined to verify that it is intact. Ascending infection may aggravate an already existing infection, and the microorganisms in the wound may be resistant to previously used antibiotics. Cultures should be obtained from the drain if the character of the wound fluid changes or the volume increases while a drain is in place.

Loss of function may be encountered, especially if the distal exiting portal is too small. It is therefore advisable to initially remove a triangle of skin and subcutaneous tissue at the exiting portal. Cutting a round hole ensures longer persitance of a patent drainage hole. Another cause of loss of function is the kinking of a tube drain, effectively obliterating the drain lumen. Repositioning of the drain and gentle traction may restore function.

Suture dehiscence is an occasional complication that may be attributed to the placement of a drain. Also, vessels and nerves may be damaged during drain placement through stab incisions and blind implantation. Rigid drain tubes may cause pain if they are located near osseous protuberances.

Types of Drains

Drain selection depends on the wound and on expected activity level of the patient. Additionally, the preference of the surgeon,

based on experience, plays an important role in the drain selection.

Passive Drains

GAUZE DRAINS

Gauze drains are prepared from gauze rolls or gauze sponges. They may be soaked with an antibiotic or even with a mild or diluted antiseptic. The antibiotic may be added at the time of drain placement, or the gauze may come commercially prepared (Figure 17-2, and see Table 17-1). If a large amount of gauze is used to pack a cavity, several rolls are tied together securely to ensure that eventually all of them are completely removed. Drainage occurs by gravity and capillary action.

Gauze drains are applied as packing in profusely bleeding cavities (e.g., after nasal septum removal) or in abscesses that cannot be drained at the lowest point. They can be used to evacuate a hematoma (after closed castration). In Europe, gauze drains are frequently attached to the stump of the spermatic cord after castration to facilitate drainage and prevent fluid accumulation in the periscrotal tissues. The advantages of gauze drains include cost effectiveness and ease of removal in stages. The adherence of fibrin clots to the gauze is a disadvantage because it may result in bleeding after removal. Conversely, the aderance of fibrin to the drain may also support débridement of the cavity.

PENROSE DRAINS

Penrose drains are the most commonly used drains because they are soft, pliable, easily sterilized, readily available, and economical, and they cause little foreign body reaction (see Table 17-1).[4] They are available in lengths from 30 to 45 cm (12 to 18 inches) and in widths from 6 to 25 mm (¼ to 1 inch) (see Figure 17-2). Most drainage occurs extraluminally and is driven by gravity and capillary action. To facilitate intraluminal drainage, the drain may be installed inside the body at its most proximal aspect or fenestrated. However, despite providing access of drainage to the inside of the drain, fenestrations reduce the surface area, which decreases the drain's efficacy.[4] Also, the fenestrations weaken the drain and may result in breakage when traction is applied to remove it. The risk of subsequent incomplete removal if adhesions between the drain and the soft tissues develop obviates any advantage that fenestration might provide.

Penrose drains can be successfully used in wounds that cannot be completely débrided and in the presence of residual foreign material, massively contaminated tissue, questionably viable tissue, and fluid-filled dead spaces.[3,5,7] Additionally, these drains have been applied with favorable results underneath skin grafts, in open wounds left to heal by secondary intention, and even in septic joints and tendon sheaths left open for lavage.[11-14] Penrose drains are not suitable for use with suction (because they collapse under a vacuum), in the abdominal cavity (because they are walled off within a short time in the abdomen), or in the thoracic cavity (because they allow air to pass into the thorax).[7]

SHEET DRAINS

Frequently, large wounds over muscular areas have to be drained. In these instances, several drains are needed to effectively drain the entire wound. The sheet drain represents an alternative in these situations (see Figure 17-2 and Table 17-1). The drain is manufactured of red rubber and has a cross-section shaped like a sine wave. The sheet can be trimmed to the desired size and width. To facilitate additional space in the field to be drained, the sheet can be folded or rolled over. Because of its inherent stiffness, there is a gap between any two layers of drain when folded or rolled, which resists obstruction. Because red rubber generally induces a significant foreign body reaction, these drains are left in place for only a couple of days, but they work efficiently during that time.

TUBE DRAINS

Tube drains differ in form and material. They can be relatively stiff, single tubes of red rubber; contain a cross-sectional wave pattern; be of soft, pliable, ribbed, flat Silastic; or be tubular silicone drains that consist of 12 single tubes joined together, each with a diameter of 3 mm (Figure 17-3, and see Table 17-1). These drains function by extraluminal and intraluminal flow and have been successfully applied for draining fluids from wounds as well as from the abdomen and thorax. The more rigid tube drains have a tendency to induce a greater tissue irritation than Penrose-type drains. Simple tube drains provide only weak capillary action but they are effective for gravity drainage.[4,7] The outer and inner surfaces of the tubes should exhibit a low coefficient of friction to facilitate evacuation of blood clots as well as the drain's removal. Some of the drains can be connected to a suction apparatus to evacuate fluids without lumen collapse and to allow irrigation. These drains are inexpensive and readily available, and they cause less interference with tissue healing than Penrose drains.[4]

One disadvantage of tube drains in a passive system is that they are easily obstructed by debris, so that they become ineffective until they are back-flushed to render them patent again, and this may have to be repeated frequently. Therefore, the use of these drains is limited to grossly contaminated areas where bacterial contamination by back-flushing is not too worrisome. Some materials (such as red rubber) induce greater inflammatory reactions than others (such as PVC or Silastic). Polyethylene contains certain impurities that support bacterial growth.[3,7] When used intra-abdominally, omentum can easily obstruct tube drains.

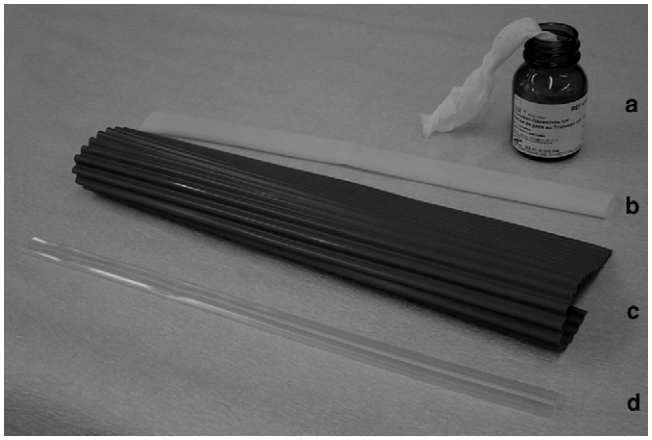

Figure 17-2. Materials frequently used as drains. *a,* Gauze drain soaked with Triclosan (antibiotic) (IVF Hartmann, Neuhausen, Switzerland). *b,* Latex Penrose drains (Sherwood Medical; St. Louis, MO). *c,* Sheet drain of waved red rubber (Ruesch, Belp, Switzerland). The sheet is folded over. *d,* Easy-Flow silicone drain (Degania Silicone LTD, Degania Bet, Israel).

Figure 17-3. A Flexi-drain (Cook Veterinary Products, Eight Mile Plains, Queensland, Australia) folded up in a plastic cup. The 12 single tubes joined to a single drain system is shown.

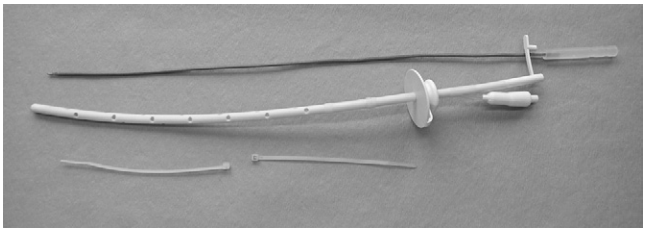

Figure 17-4. A Lepage drain (Cook Veterinary Products, Eight Mile Plains, Queensland, Australia) shown with its insert to provide rigidty during insertion. Two plastic arm bands are shown that are used to attach the negative suction device at the bandage.

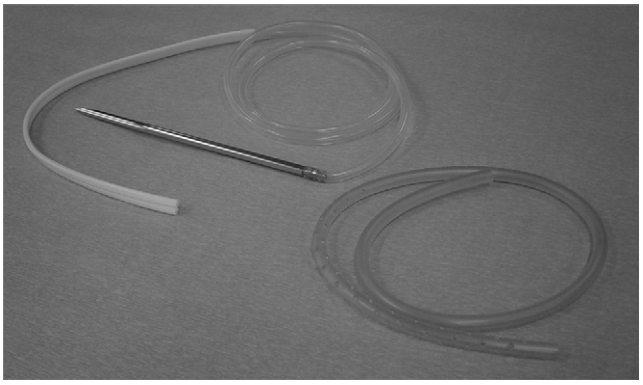

Figure 17-5. A Blake drain (Johnson & Johnson, New Brunswick, NJ) and a multifenestrated Snyder-type tube drain (Zimmer, Inc, Dover, OH). The insert represents the cross-section of the Blake drain. The trocar at the other end is used to place the drain through the skin.

Active Drains

CLOSED SUCTION SYSTEMS

In equine practice, simple tube drains attached to a suction apparatus providing either intermittent or continuous suction are frequently used in infected joints and in large, deep wounds to evacuate the pleural space and under full-thickness skin grafts. Fenestrated tube and Blake drains are often used in these situations (Figures 17-4 and 17-5; see Table 17-1), and occasionally Snyder Hemovac drains are used (Figure 17-6; see Table 17-1). Either the end of the drain is multifenestrated or the cross-section consists of a modified cloverleaf pattern with four slits and protected spaces (i.e., the Blake drain). The external end is made of smooth tubing and is connected through a three-way stopcock, most frequently to a syringe; the plunger of the syringe is withdrawn and held in that position by introducing a large needle or a small pin across a hole prepared across the plunger and resting it on the syringe end to achieve the desired persistent negative pressure (see Figure 17-6). This provides the most economical suction apparatus. The three-way stopcock allows interruption of the suction action prior to removing the syringe for emptying. This is also an effective means to fight against ascending infection. A study comparing Penrose drains to closed suction drains showed that at 24 hours, 34% of the Penrose drains were contaminated compared with none of the closed suction drains.[2,4,7] If suction is applied in a continuous manner, soft tissues can rapidly occlude the drain. High negative pressure may cause injury to tissues, and if the system is suddenly disrupted, reflux of evacuated fluid may occur, increasing the risk of infection. Adding a Heimlich valve to a suction system can prevent reflux of fluid (Figure 17-7).

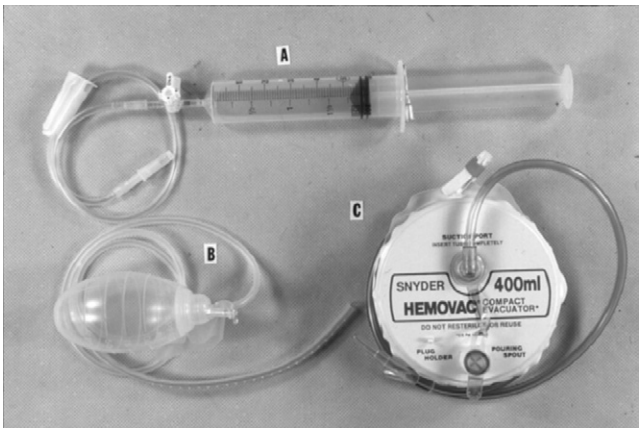

Figure 17-6. Devices used as active drainage systems. *a,* Syringe-adapted closed-suction device made from a 60-mL syringe by drilling a hole in the shaft, near the plunger. A three-way stopcock and extension set is attached to the syringe and fixed to the drain. The syringe is held open by a 14-gauge needle whose tip is ground flat, placed across the syringe shaft. *b,* Snyder Hemovac–100 mL (Zimmer, Inc, Dover, OH) with a flat silicone fenestrated drain. *c,* Snyder Hemovac–400 mL (Zimmer, Inc).

A special closed suction system has been used in humans to promote granulation tissue production in large open wounds,

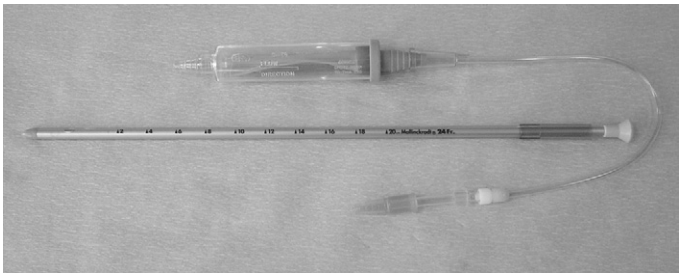

Figure 17-7. *Top:* A spontaneous pneumothorax aspiration system (Heimlich valve) (Cook Veterinary Products, Eight Mile Plains, Queensland, Australia) used to prevent access of ascending air and microorganisms into the cavity to be drained. *Bottom:* A PVC thorax drain (Trocar Catheter, Mallinckrodt Medical, Athlone, Ireland).

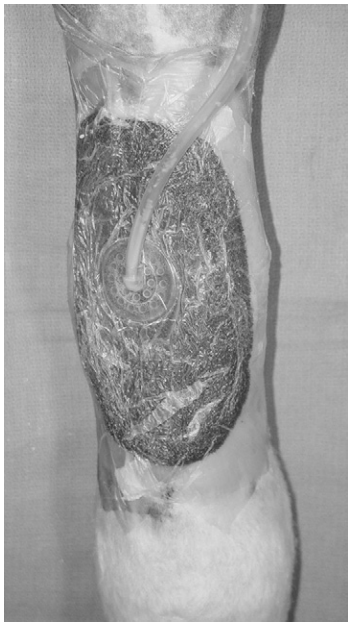

Figure 17-8. The wound in Figure 17-9 was covered with a suction device and sealed under a plastic bandage.

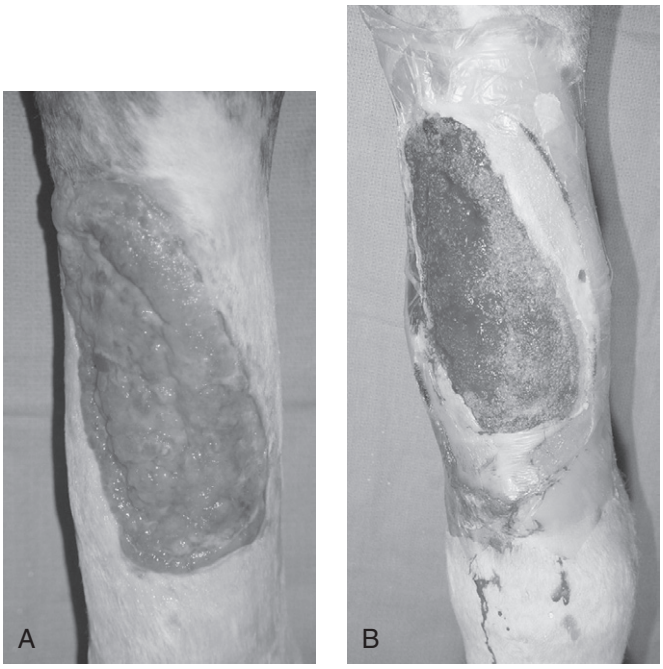

Figure 17-9. A, An old, infected, nonhealing wound over the dorsomedial aspect of third metatarsal bone. The granulation tissue is unhealthy looking and nonresponsive to treatment. **B,** The same wound 4 days later, after removal of the suction device. Healthy granulation tissue covers the wound. The size of the wound is significantly reduced.

especially when there is bone involvement. This device has been successfully applied in horses. The wound to be treated by suction is prepared for aseptic surgery and the wound edges are clipped and trimmed. A sponge is cut to slightly overlap the wound size. The continuous suction device is installed into the sponge. The entire sponge and the suction device are covered by a special adhesive tape, which provides an airtight seal between the wound and the normal skin (Figure 17-8). A bandage is applied to protect the device and maintain external pressure. When suction is applied, the evacuated fluid accumulates in a container. Movements of the horse must be restricted to ensure continuous suction. This method of wound treatment can change an infected, odiferous wound into one covered with healthy granulation tissue within 4 days (Figure 17-9).

OPEN SUCTION SYSTEMS

Open suction is rarely applied in equine surgery. One system involves a sump drain, consisting of a large drain tube with a second, smaller tube in the wall or within the lumen of the larger tube. This "vented" suction apparatus allows air to enter the wound through the narrow lumen tube while debris and fluid are evacuated through the larger tube. Suction may be applied in continuous or intermittent form.[4,7] The airflow improves drainage and decreases the risk of occlusion. However, sump drains do not adapt well to many veterinary hospital situations. Large, portable, or built-in wall units are needed. Also, the large quantities of air needed to keep the suction end open may increase the risk of infection and tissue irritation. Bacterial filters over the air inlets have been shown to effectively reduce infection rates.[2]

The application of a Heimlich valve provides an effective barrier to ascending infection in open drainage of body cavities. This device prevents inflow of air but facilitates drainage of fluid and debris (see Figure 17-6).

Drainage of Body Spaces

Drains in Synovial Spaces

Removal of purulent debris from synovial spaces is facilitated by drain placement. Passive or active drainage systems can be employed for this purpose, but the passive Penrose drains are best. It is important that they be placed in the distal dependent aspect of the synovial space and maintained beneath a sterile bandage. Conversely, active drainage systems can be uncomfortable and abrasive to articular cartilage and tendons because of the rigidity of the material. However, Jackson-Pratt drains, made from Silastic, are multifenestrated and can be placed in these small spaces to provide efficient active drainage.[8]

Drains in Body Cavities

ABDOMEN

Passive drainage of the abdominal cavity requires dependent placement of a rigid-lumen drain tube.[7] PVC and Silastic drains can be used effectively for this purpose. (Penrose drains are not functional for this purpose and should not be used.) Intraoperative placement of multifenestrated drains should be considered after abdominal lavage or when large volumes of exudate or transudate are expected. The drain is placed in a dependent position away from the abdominal incision and sutured to the skin to prevent dislodgement. A sharp trocar with a threaded end is provided to facilitate entrance of the drain at the desired location (see Figure 17-4). It is important to use the trocar to prepare the drain exit portal so that it is just large enough to allow drainage to occur through the drain lumen but not around it. An exit wound that is too large may allow eventration of omentum through it. Bandage placement over abdominal drains is impractical because of the drainage volume obtained. If used, the drains should be removed as soon as drainage slows or ceases. Protecting the drain end is important to prevent ascending infection. A simple method to reduce this risk is to cut off the end of a latex condom, or a finger from a surgical glove, and to place it over the drain, where it acts as a one-way valve. Such valves are commercially available under the name of Heimlich valves (see Figure 17-7).

Thoracic trocars made from PVC and Silastic can be placed percutaneously for drainage of air, urine, exudates, or lavage fluid from the abdominal cavity. Functional time may be limited by the number of fenestrations in the commercial products, so it is helpful to provide additional fenestrations. Square holes in the drain may provide better drainage than round holes.[4] To place the drain, a dependent position is identified. If a long-standing peritonitis is present, or if there has been previous surgery, ultrasonographic guidance may be indicated to identify bowel adhered to or near the body wall. The site is prepared for aseptic surgery, and local anesthetic is infiltrated. A 1-cm incision is made through the skin and the external rectus sheath. An appropriate-diameter thoracic trocar is selected (16-30 Fr) and carefully inserted through the rectus abdominis muscle, internal rectus sheath, and peritoneum. When the abdominal cavity is penetrated, the obturator is removed, minimizing the risk of inadvertent bowel puncture. The drain is subsequently positioned properly and secured. Drains can be sutured to the skin in a variety of patterns. Two useful patterns are the *Chinese finger trap* suture and the *double clove hitch* pattern (Figure 17-10).[9] If the drain is left in place, its end is protected, as previously described, or a Heimlich valve may be added.

In cases that benefit from open peritoneal drainage, polypropylene mesh can be used to provide drainage over several days.[7] After correction of the primary problem, the mesh is secured into the abdominal closure with sutures, leaving a gap for fluids to escape. The mesh is left in place until drainage subsides, and it is removed during a second surgical procedure (Figure 17-11).

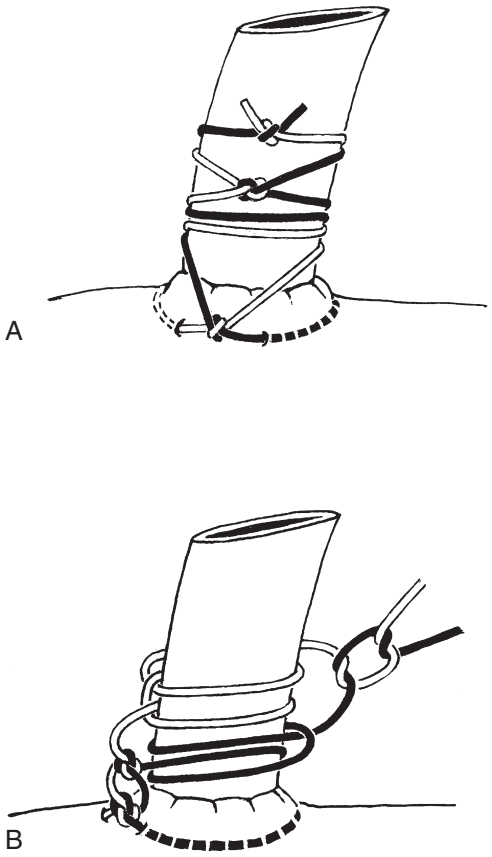

Figure 17-10. Suture patterns used to secure a drain to the body wall. **A,** The "Chinese finger trap" suture pattern. **B,** The "double clove hitch" pattern.

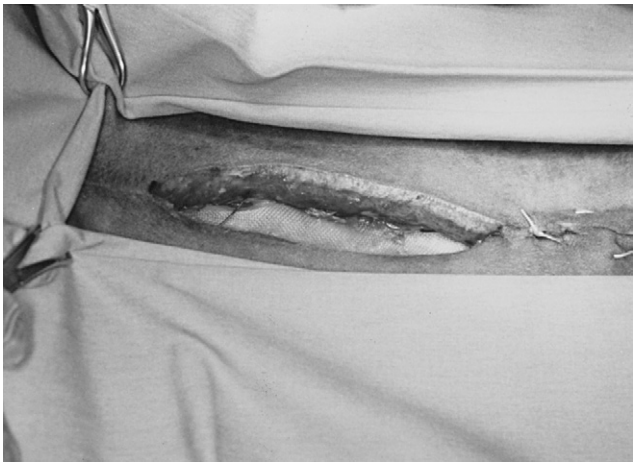

Figure 17-11. Polypropylene mesh used for open peritoneal drainage. The mesh, seen interposed between the wound edges, is ready for removal.

THORAX

Thoracic drainage presents special problems because negative pressure needs to be maintained in the chest despite the frequent presence of air. The use of a rigid drain tube is necessary. Removal of air can be achieved through active or passive mechanisms. To place a drain for removal of air, a dorsal site is selected and prepared for aseptic surgery. Local anesthetic is infiltrated prior to establishing a 1-cm stab incision through the skin. A thoracic trocar is inserted and tunneled cranially for one or two rib spaces, followed by insertion into the thorax along the cranial edge of the rib, avoiding the intercostal vessels located on the caudal border of the ribs (Figure 17-12). When the thorax has been penetrated, a Heimlich valve is placed on the drain end. The Heimlich valve has a rubber liner, which allows air to exit during expiration, and it collapses on inspiration,

restricting backflow of air (see Figure 17-7). If a large volume of air is present, suction can be applied to the open end of the Heimlich valve, rapidly removing air and reestablishing negative pressure. The drain is secured by one of the means previously described. If the primary problem is corrected, the drain can usually be removed within 24 hours if an active drainage system was used initially to drain fluid from the lower thorax. Multifenestrated PVC drains surgically placed or thoracic trocars percutaneously placed are suitable for this purpose. A closed suction device is applied to the catheter and is maintained until drainage subsides. It is important that the closed suction device not become dislodged from the drain because this would cause a rapid loss of negative pressure and introduce environmental contaminants into the thorax (see Figure 17-12).

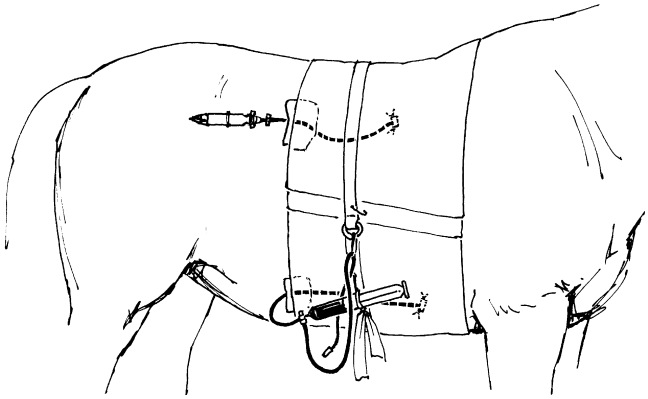

Figure 17-12. Proper placement of thoracic drains. A drain in the dorsal thorax is placed with a Heimlich valve (Heimlich chest drain valve, Bard Parker, Becton Dickinson, Inc., Lincoln Park, NJ) to prevent the backflow of air. A ventral drain uses a syringe-adapted closed-suction device to provide safe removal of fluid accumulating in the ventral thoracic cavity.

BANDAGES

Bandages are applied to cover wounds protected by dressings, to prevent edema formation after injuries of the limb, and to support the limb in conjunction with an added splint in the case of a ligament injury or fractured bone.[15,16] The type of bandage is chosen on the basis of the location and the nature of the injury.

Foot Bandage

Foot bandages are applied to manage a variety of problems. Part of a roll of cotton is placed over a primary wound dressing (Figure 17-13). The padding is secured with gauze, and it can be held in place with either cohesive or adhesive bandaging tape. Duct tape placed over the bottom of the bandage will render the bandage more durable and less permeable to urine and water (see Figure 17-13).[15] Moisture can be kept from entering the bandage by placing plastic over the foot. An empty 5-L fluid-bag can be opened with a pair of scissors and placed over

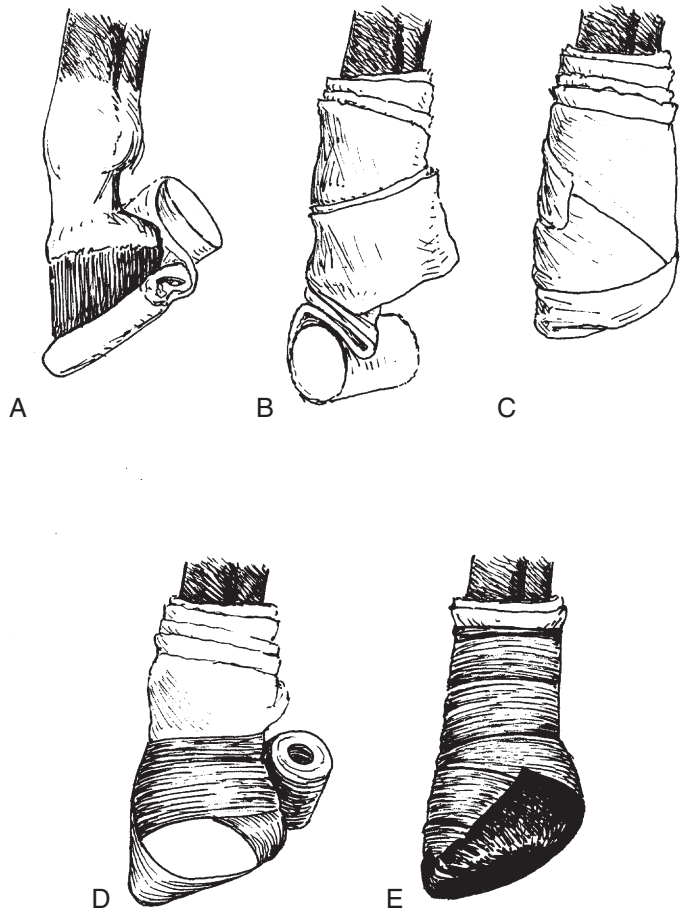

Figure 17-13. A, Several layers of folded-up cotton are placed over the sole of the foot. **B** and **C,** The roll of cotton is subsequently applied in several layers over the foot and fetlock area. **D,** A roll of elastic adhesive tape, tightly applied, finishes the bandage. **E,** Some protection may be applied to the sole to prevent wearing of the bandage. If deemed necessary, the bandage may also be covered with several layers of casting tape to further reduce motion in this region and to stabilize an injury of the bulbs of the heel.

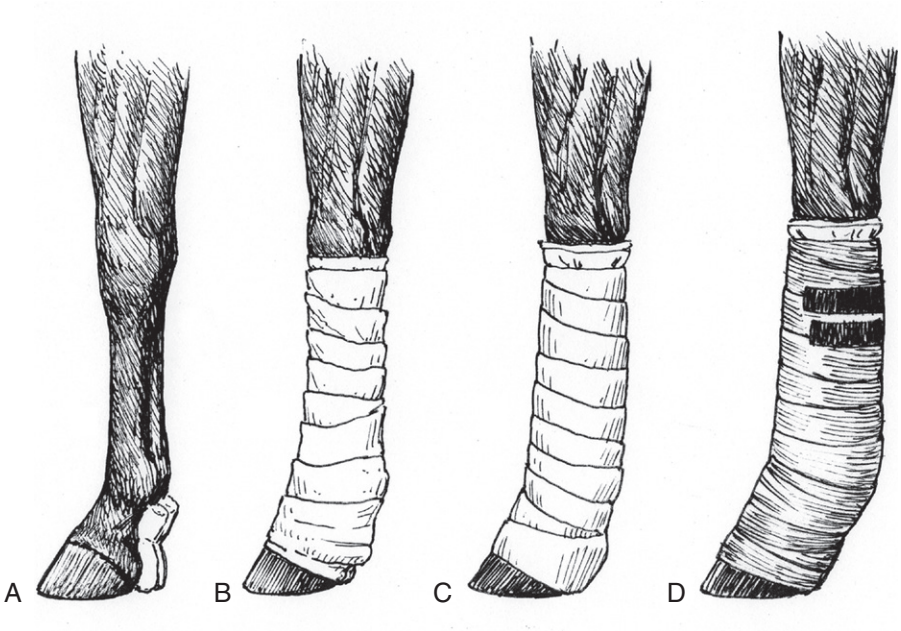

Figure 17-14. A, The heel area is first padded with some cotton. **B,** The first layer of the lower limb bandage is placed on the hind limb. **C,** After tightening the first layer with gauze, a second layer is applied. **D,** The bandage is covered with elastic adhesive tape and secured with two pieces of duct tape. To finish the bandage, adhesive tape is applied to its top and bottom to prevent bedding and dirt from gaining access to the wound (not shown).

the hoof capsule and fastened with adhesive tape, attaching it effectively to the foot. This type of bandage is useful if it is desirable to exclude water from the wound environment, when a poultice or soak is applied to the foot, or when preparing a foot or pastern for any type of aseptic surgery.

Lower Limb Bandage

A lower limb bandage is applied from the bulbs of the heel up to just below the carpus or tarsus. It usually consists of a roll of cotton or sheet, applied in the standard clockwise fashion (pulling the tendons to the inside) (Figure 17-14). The underlying medical problem dictates the thickness, or number of layers, of the bandage. Each layer is secured with conforming roll gauze, wrapped snugly in a spiral pattern overlapping half the tape width, to prevent the padding from slipping or bunching. The gauze is overlaid with either adhesive or cohesive bandaging tape to secure the bandage in position. A single wrap of adhesive tape around the bottom of the hoof and the top of the bandage prevents bedding materials from gaining access to the underlying skin or wound, respectively. Care should be taken to extend the bandage to the level of the carpometacarpal or tarsometatarsal joint and to prevent inadvertent tendon damage if a considerable amount of tension is applied to the elastic bandage tape. At the level of those joints, the tendons are lodged between the vestigial metacarpal bones, which provide protection. Additionally, the coronary band should be included in the bandage so that tape can be applied directly to the hoof capsule.

Full Limb Bandage

Forelimb

A full limb bandage is applied from the bulbs of the heel up to the elbow region (Figure 17-15). When applying a full limb bandage, movement of the carpus requires that special attention be given to this area to prevent decubitus ulcers. The bandage is usually *stacked,* beginnning with a lower limb bandage followed by proximal limb bandages, to prevent slippage and subsequent irritation over bony prominences. Padding materials are the same as for the lower limb bandage and therefore require placement in two stages. The distal bandage is initially applied as previously described. The proximal part is subsequently added on top of the lower limb bandage, overlapping it for 5 to 10 cm. Applying a doughnut-shaped cotton ring or incising the gauze over the accessory carpal bone helps prevent skin irritation over that area and potential development of skin ulcers. Tightening of the bandage in layers provides more stability and increases the support. If the bandage becomes displaced distally, it is imperative that it be changed at once to prevent skin ulcers from developing over bony prominences.

Hindlimb

Motion of the tarsus requires special attention when applying a bandage to that region. Primary wound dressings are held in place using gauze applied in a figure-of-eight pattern (Figure 17-16). The crossing of the "8" occurs over the dorsal aspect of the tarsus, with the loops applied around the proximal metatarsus and the distal tibia of the limb, leaving the point of the hock open. Caution should be used in applying tension over the gastrocnemius tendon. The bandage is also applied in two steps, as described for the forelimb. The proximal part of the bandage overlaps the distal bandage. Applying soft cotton patches medially and laterally between the tibia and the gastrocnemius tendon provides support and reduces the pressure of the latter, thereby serving as protection against tendon damage that could result from excessive tension. Each layer of padding material is first secured with gauze, applied at a right angle to the limb, as opposed to the figure-of-eight pattern for the primary dressing. Application of cohesive or adhesive tape completes the bandage (see Figure 17-16). The bandage is finished by applying elastic adhesive tape around the hoof capsule below and to the skin on top of the proximal end of the

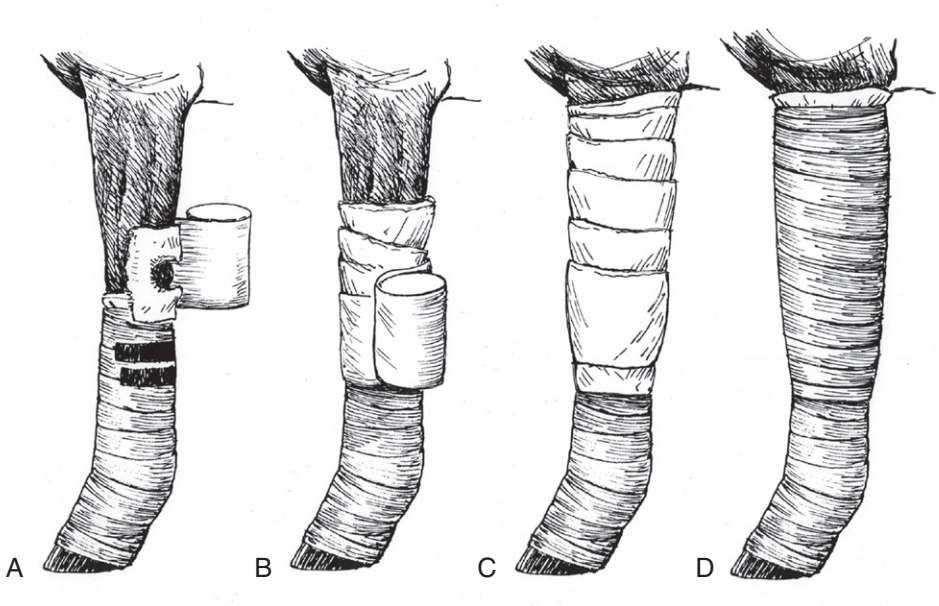

Figure 17-15. A full limb bandage applied to the hind limb. **A,** The distal part of the bandage (shown in Figure 17-14) is first applied. Cotton arranged in a doughnut shape or a piece of felt with a central hole is placed over the accessory carpal bone before roll cotton is applied to the proximal aspect of the limb. **B,** The carpal area is covered with roll cotton in figure-of-eight fashion. **C,** The proximal limb is evenly covered with cotton layers, each separately tightened with heavy gauze. **D,** The bandage is covered with tightly applied elastic adhesive tape, and the top and bottom are sealed to prevent access of bedding and dirt.

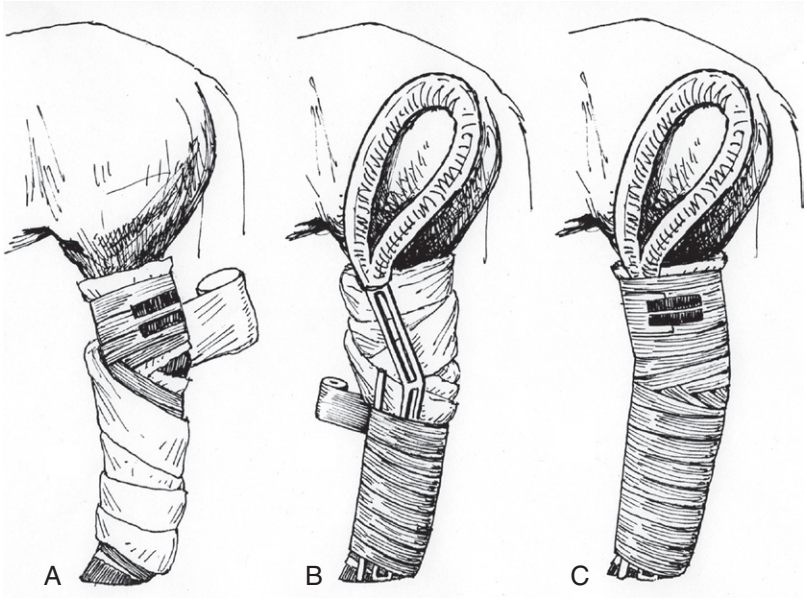

Figure 17-16. A Robert Jones bandage with a lateral splint applied to immobilize a distal tibial fracture prior to surgery. **A,** First, a multilayered full limb bandage is applied to the limb using a technique similar to that described in Figure 17-15. The tarsus is covered with a figure-of-eight bandage. **B,** The proximally padded commercial metallic splint is applied to the lateral aspect of the limb and attached to the bandage with broad nonelastic tape. **C,** The bandage is tightly applied up to the stifle. The padded loop in the hip area provides counter-pressure and resists the development of a valgus deformity during weight bearing. It is prudent to surgically prepare the skin and use a sterile dressing in the first layer in the event the fracture becomes open after the bandage is applied or during transport.

bandage, thus preventing access of bedding to the skin underneath the bandage.

The application of a full limb bandage to the hind limb decreases the movement of all joints in the limb because of the reciprocal apparatus. Some horses have more problems coping with this situation, especially when rising from recumbency. Therefore, the patient will need to be observed for a while after such a bandage is applied.

Splints

A special type of full-limb bandage is the Robert Jones dressing (RJD), for which several layers of cotton are evenly applied over the entire limb, each layer tightened separately with elastic nonadhesive tape. The final cover of the RJD consists of a layer of tightly applied elastic adhesive tape. The size of the RJD should be approximately double the size of the limb and produce a dampened "ping" when snapped with the finger on the outside. This type of bandage provides good support to a severely injured or fractured extremity, because it adds rigidity, especially if a splint of some kind is incorporated into the bandage. An RJD with an incorporated splint allows weight bearing on a fractured limb.

Splints must be applied carefully to prevent decubitus ulcers. Splint materials commonly used include: wood, PVC pipe, or metal, or they can be assembled from cast material incorporated into the bandage. Wood splints are not ideal because they lack strength in small conforming widths, and larger boards do not conform well to the limb. This limitation is overcome by incorporating several small-width slats into the bandage. The sum

of the slats used increases the bandage rigidity and achieves the desired result. With adequate padding in place, 1 × 4-inch (2.5 × 10 cm) boards can be incorporated into a bandage and arranged in at least two right-angle planes. Board splints should extend from the hoof to the joint proximal to the affected area in at least one plane. If the radius or tibia is to be immobilized, a padded lateral splint extending beyond the top of the bandage should be incorporated to prevent adduction of the limb (see Figure 17-16).

Excellent rigidity can be achieved by using PVC pipe as splints. The diameter of the schedule 40 PVC pipe selected depends on the size and location of the limb to which it is applied. The material should be split longitudinally in half. The splint may be modified by removing half-moon–shaped portions at strategic locations to allow access to regions with a wider diameter, such as the carpus. A good compromise has to be found between the PVC pipe diameter and the diameter of the widest part of the limb to be incorporated into the splint.

Neither PVC pipe nor wood conforms well to the limb, however. By applying a hot air gun to strategic locations of the PVC pipe some adaptations to anatomic locations are possible. Casting tape, on the other hand, conforms well to the bandaged limb, but it does not provide the bandage rigidity that can be achieved with wood or PVC pipe. Splints may be made from casting tape rolls, or they can be purchased in that configuration as a longuette. The addition of a casting tape splint reduces the amount of padding needed and provides suitable immobilization in most circumstances. Casting tape splints cannot be applied to extend to the shoulder or hip to prevent adduction of the limb for immobilization of the antebrachium or crus, respectively.

Stainless steel splints are commercially available for temporary immobilization of the distal limb, including the metacarpus in the forelimb. These splints are used as emergency fixation for breakdown injuries of the suspensory apparatus, for flexor tendon injuries, for fractures of the metacarpal condyle, and for phalangeal fractures when a strut of bone remains to support the limb. They are especially useful for transport of horses with such injuries (see Chapter 73).

Boots

A variety of commerical equine boots are manufactured from different materials and in different sizes and styles, ranging from low to high profiles and for the front and rear limbs. Low flexible boots are used to replace conventional horse shoes. Some boots reach the pastern region and contain a thick, rugged sole (Easyboot, EasyCare, Inc., Tucson, AZ). Silicone pads are available that can be trimed to fit the sole of the foot to be placed into the boot (Figure 17-17). This boot is well suited to raise the opposite foot of a horse placed in a cast (see later in this chapter).

Other boots are available to apply to an injured horse on an emergency basis (Figure 17-18). Indications for these boots are breakdown injuries and phalangeal fractures. A tight bandage is applied to the distal limb to ensure a tight fit in the boot. These boots can also be applied following internal fixation of phalangeal and distal metacarpal or metatarsal fractures. When the horse has recovered from anesthesia, the boot is usually removed. However, in selected cases it can be maintained for a longer period of time. The same control measures have to be applied similar to casts (see later in this chapter).

Figure 17-17. A high profile Easyboot (EasyCare, Inc., Tucson, AZ) with a thick sole and a silicone pad for the support of the sole. Additional pads can be added to elevate the foot in cases where a cast was applied to the opposite foot.

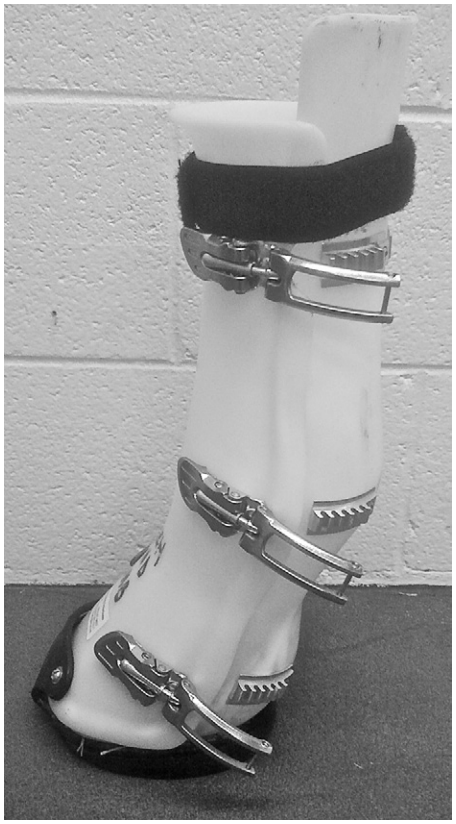

Figure 17-18. A distal limb boot (Equine Bracing Solutions, Trumansburg, NY) used for emergency treatment of distal limb fractures and to protect an internal fixation of such fractures during recovery from anesthesia. (Courtesy L. Bramlage, Rood & Riddle Equine Hospital, Lexington, KY.)

EXTERNAL COAPTATION (CASTS)

Cast Application

Materials

Historically, plaster of Paris casts have been popular for external coaptation. Plaster is still a viable casting material because it is easy to apply, has good molding capability, and is inexpensive. Unfortunately, plaster casts also are heavy, disintegrate when wet, and do not allow tissues to be exposed to air, which makes this type of cast uncomfortable when worn for a prolonged period.[17] Furthermore, plaster is not as strong as fiberglass and thus requires more material to prevent breakage. This results in a heavier cast.

The shortcomings of early fiberglass casts were corrected and they are now manufactured from materials of superior quality; they are lightweight, strong, and radiolucent, and they have excellent molding capability. Additionally, the porosity of the material allows air to reach the skin. Although these types of casts are more expensive than plaster casts, they are more durable and require less material for adequate strength. A variety of fiberglass casting materials are currently available on the market. In 1983, the mechanical properties of several of these materials were compared, and the differences were recorded.[18] However, since then, major improvements in handling capability and strength have been implemented. For practical purposes today, there are no significant differences between the various fiberglass products on the market.

The strength of a cast is determined mainly through bonding between the tape layers, so swift cast application to avoid lamination is necessary to produce a strong cast. Fiberglass casts are about 20 times stronger and 4 times lighter than plaster casts. All cast materials exhibit an exothermic reaction (they release heat) during setting; the more layers applied, the greater the reaction. Immersion in water hotter than 27° C (80.6° F) immediately before application also results in heat production. However, warm water reduces the curing time considerably. Therefore, veterinarians inexperienced in the application of casts should use cool water, which permits a longer application time but ensures that all tape layers will bond together as the cast hardens. Unlike in the procedure for application of plaster casts, water should not be expressed from the fiberglass material before application, because the cooling effect of the water is lost. Also, freshly applied casts should not be covered with bandage materials before they have set, as is frequently done to facilitate intertape-bonding and ensure good rigidity of the cast. The casts usually set within 4 to 5 minutes and allow weight bearing within 20 to 30 minutes.

Indications

External coaptation by casting is indicated in selected fractures of the phalanges, as adjunct treatment to internal fixation of fractures, for immobilization after tendon repair, and to stabilize wounds that are healing in regions of continuous motion, such as heel lacerations (see Chapter 90). Casts are also applied to protect a limb during recovery from anesthesia—for example, after repair of a condylar fracture of the distal third metacarpal or metarsal (MCIII/MTIII). Tube casts may be applied to foals with incomplete ossification of the cuboidal carpal and tarsal bones to facilitate ossification while weight is distributed evenly across the joints (see Chapter 86).

Technique

Most casts are applied with the horse under general anesthesia. This prevents the animal from moving during the application and setting of the cast, which may weaken the cast or cause pressure points, with subsequent development of decubitus ulcers. However, with adequate sedation, casts can also be successfully applied in standing horses.

Before starting, all materials should be laid out for efficient and swift cast application. The entire portion of the limb to be covered with a cast should be cleaned and dried. It is not advisable to clip the hair unless that is required for a surgical procedure. Special attention should be given to the hoof. It should be trimmed and all excessive sole and frog material removed. It is advisable to paint the sole and frog with a solution containing iodine. Any lacerations or wounds should be débrided, sutured if necessary, and covered with a sterile nonadhering dressing. This dressing should be secured by a gauze or elastic bandage. It is advisable to apply boric acid to the portion of the limb that will be covered with a cast. Boric acid is a drying agent with antibacterial properties. Applying zinc-containing soft gauze is an alternative method to provide protective properties to the skin. These measures are especially important if the cast is to be left in place for an extended period of time. A piece of stockinette somewhat longer than twice the proposed length of the cast is prepared by rolling it from each end toward its center. One side is rolled outward and the other side inward (Figure 17-19). For foals, a 5-cm (2-inch) diameter stockinette should be selected. A 7.5- or 10-cm (3- to 4-inch) stockinette is adequate for adult horses. The stockinette should be neither too loose nor too tight.

The stockinette is applied to the limb with the portion previously rolled up in the outward direction (viewed from the stockinette). The rolled up portion is now unrolled, and in doing so the stockinette is applied to the limb (see Figure 17-19). When the first layer of stockinette is applied, it should be pulled distally for about 2 cm (¾ inch) to ensure normal alignment of the hair along the limb. The other half of the stockinette is then twisted at the sole region and unrolled like the first half (the previously inwardly rolled part can now be rolled outwardly). At this stage, the stockinette should extend about 5 to 10 cm (2 to 4 inches) past the proximal end of the cast to be applied. Generally, a ring of orthopedic felt, about 7 cm (3 inches) wide, is applied to the most proximal aspect of

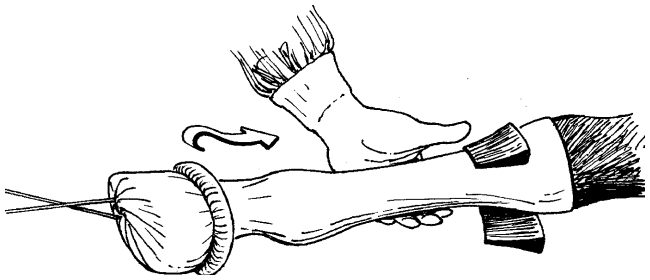

Figure 17-19. The limb is placed in traction and the inner layer of stockinette is rolled up along the limb. The outer layer of stockinette, which was initially rolled up in an inward direction, is twisted axially 360 degrees at the bottom of the foot and rolled up along the limb as well. At the proximal aspect of the cast, a wedge-shaped piece of thick felt is fitted to the limb, secured with tape, and covered with the outer layer of stockinette.

the cast between the two layers of the stockinette (see Figure 17-19). This ring of felt should not be overlapped but should be adjusted to the correct length to perfectly appose both ends. The ends are held in place temporarily by nonelastic adhesive tape.

For plaster casts, a cotton stockinette usually is used, whereas synthetic stockinettes are preferred for fiberglass casts. Because synthetic stockinette is manufactured from acrylic fiber that has little capacity to hold moisture, moisture is transferred away from the body. Also, synthetic stockinette maintains greater bulk, adding to the padding. Some clinicians prefer to add a thin layer of synthetic cast padding between the two layers of stockinette. Additional attention should be given to potential pressure points, such as over the accessory carpal bone, ergot, or calcaneus regions. Extra padding, consisting of a silicone doughnut or orthopedic felt with an elliptic hole, should be applied to these areas. After the stockinette and padding have been applied, the limb should be positioned for application of the cast.

In most cases, the limb should be extended with the metacarpal and phalangeal regions in the same frontal plane. In special cases, it may be preferable to cast the limb in a normal weight-bearing position. For this purpose, the carpus is flexed and slight pressure is applied either to the dorsum at the fetlock region or to the sole in a dorsal direction (Figure 17-20). An assistant must hold the limb in the desired position. The palm of the hand, not the fingertips, should be used to apply pressure to a specific region and thereby help prevent pressure point development. It must be kept in mind that attempts to cast a limb in its normal angulation fails in most cases when the cast is applied with the horse in a non–weight-bearing position (i.e., on the surgery table). Casting a limb in an "almost weight-bearing" position renders it more vulnerable to the development of pressure sores than if the limb is cast with the metacarpal/metatarsal and phalangeal regions aligned in the same plane. Therefore applying the cast in the standing, weight-bearing horse that is properly sedated is the method of choice for a normal weight-bearing position.

The polyurethane, resin-impregnated foam (3M Custom Support Foam) introduced in the early 1990s is an efficient means to reduce cast sores.[15,19] This material is immersed in warm water for about 1 minute. After minimal squeezing, the soft foam is applied evenly over the stockinette. Care is taken to overlap each turn half of the width, with the result that a double layer of foam is applied evenly over the part of the limb being covered with a cast. Minimal tension is applied. Wearing gloves during application of the foam is strongly encouraged.

To facilitate cast removal under practice conditions, one Gigli wire attached to a long felt strip (Figure 17-21, *A*) may be placed medially and laterally over the padded limb (see Figure 71-21,

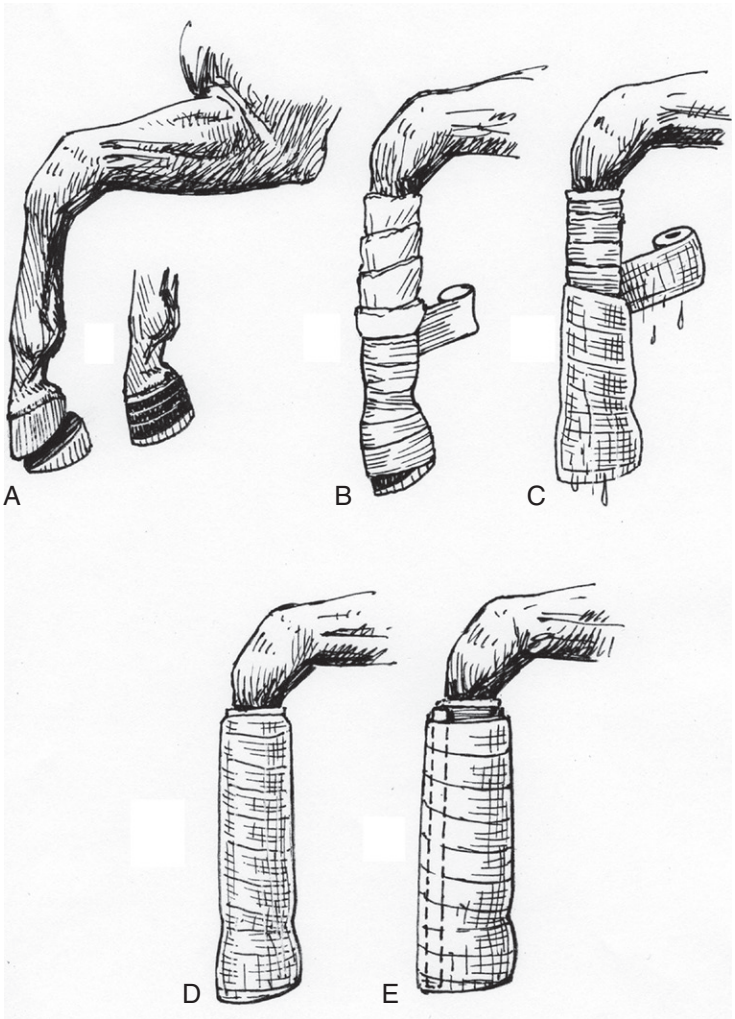

Figure 17-20. A short limb cast applied to the forelimb. The phalanges and third metacarpal are aligned in the same plane. **A,** A wooden wedge is applied to the foot with adhesive tape. **B** through **D,** The cast material is evenly applied over the synthetic foam in several layers. **E,** If deemed necessary, a straight dorsal splint may be incorporated into the cast.

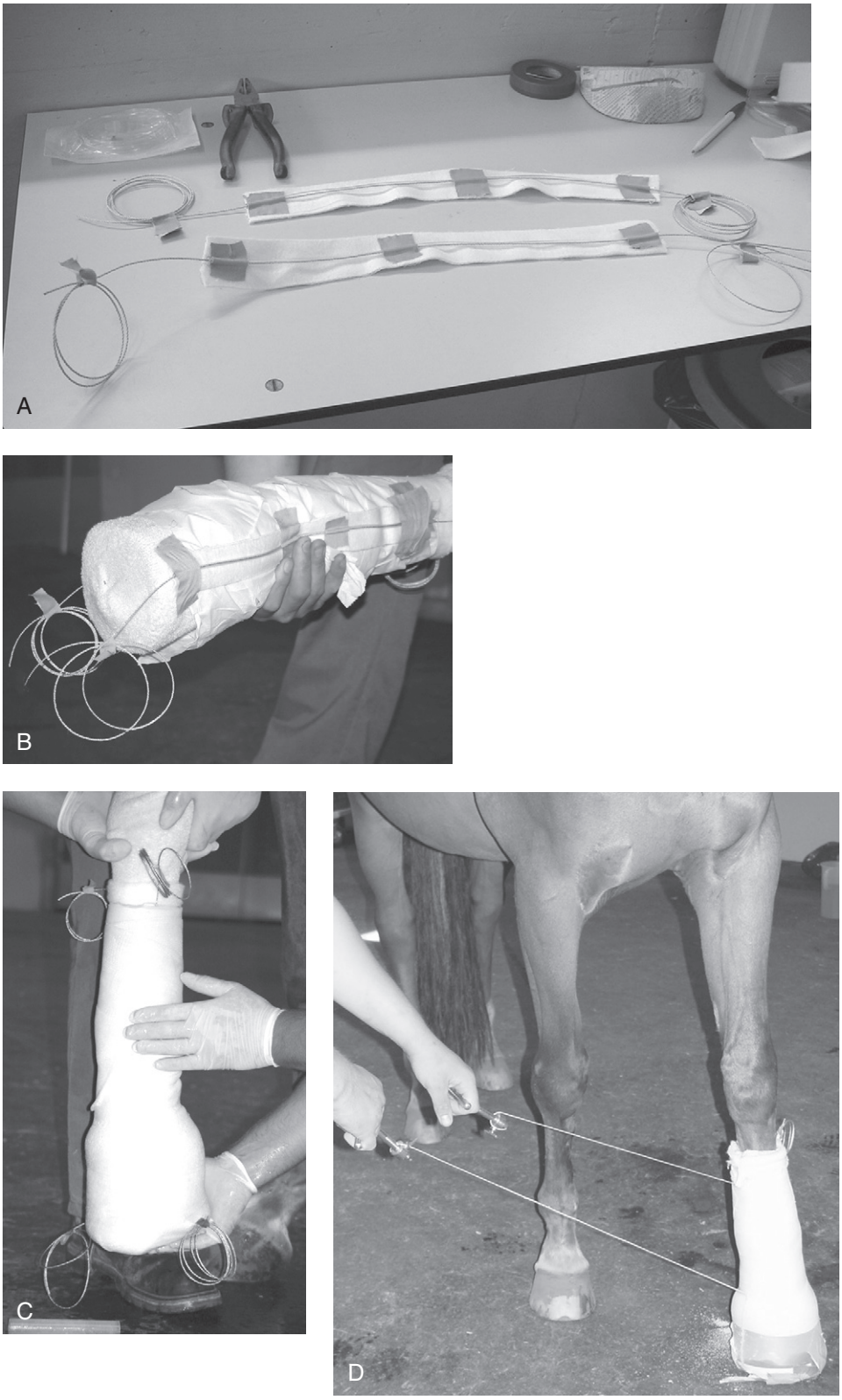

Figure 17-21. A, Two pieces of Gigli wire are attached to felt strips. The two ends are rolled up. **B,** The felt strips with the wire are applied medially and laterally to the padded limb. **C,** The cast is applied in routine fashion. The rolled-up ends of the wire are covered with tape at the end of cast application. **D,** At the time of cast removal, the ends of the wire are attached to the handles, and by slow sawing movements, the medial and lateral sides of the cast are severed apart. Finally, the two shells still connected at the sole are split dorsally and palmarly, allowing the limb to be removed from the cast. (Courtesy C. Lischer, Zurich, Switzerland.)

B).[20] The wire should be long enough that some excess wire protrudes proximally and distally on either side of the leg. When the cast is finished, the excess wire is rolled up and placed underneath the elastic tape applied to this region (see Figure 17-21, *C*).

Latex gloves must be worn when applying a fiberglass cast. The airtight packages of the fiberglass tape are opened immediately before application. The fiberglass tape is held with both hands, separating the free end from the rest of the material, before submerging it in the water.[21] The fiberglass tape is held in water at about 21° to 27° C (70° to 80° F) for 5 seconds. During this time, the tape is squeezed four or five times to encourage complete penetration by water. The fiberglass tape is removed from the water dripping wet and immediately applied to the limb. Cast application is started at the foot and progresses in a proximal direction, overlapping at least half the width of the roll until the most proximal aspect is reached. After applying two layers at the top, the cast material is directed distally and applied evenly over the limb. As a rule, the cast bandages are applied in progression by continuing with the next bandage where the previous one ended. Changing direction during cast application is done by folding the cast material at one place and smoothing out the fold with the flattened hand. The newer materials adapt so well that in most cases directional changes can be carried out without folding the material over. Care should be taken to follow the contours of the limb and not to apply too much tension to the tape, which could interfere with circulation.

After the first few layers of cast material are applied, the extra stockinette extending on top of the cast is folded distally and covered by the following layers of cast tape (see Figure 17-20). Fiberglass cast material is applied until the cast reaches a thickness of 7 to 8 mm (¼ to ½ inch) throughout the total length of the cast. This requires four to six rolls of 12.7-cm (5-inch) fiberglass casting tape for a half-length cast in an adult horse and 10 to 12 rolls for a full-length cast.

If deemed necessary, a straight splint, which could be an old hoof rasp or any similar type of material, may be incorporated into the cast on its dorsal aspect (see Figure 17-20, *E*). This splint should be covered with cast material to prevent accidental trauma to another limb. Such a splint would reduce the amount of cast material needed for a weight-bearing cast and is proposed only for preoperative support of phalangeal fractures.

When sufficient cast material is applied, the cast is molded over its total length and the surface is smoothed out. It is important not to flex the joints under the cast from the time cast application begins until the cast has set. Most casts harden within 5 to 7 minutes after the final roll is applied.

If the limb is cast in an extended position, a wedge should be incorporated in the cast under the heel (see Figure 17-20). The wedge permits weight to be applied over a greater surface than just at the toe. It is advisable to protect the bottom of the cast with a layer of hoof acrylic, a piece of old inner tube, or the bottom of a gallon plastic bottle taped to the bottom of the cast with nonelastic tape.

To prevent foreign material, such as wood shavings or straw, from entering at the top of the cast and causing irritation, a collar of adhesive elastic tape should be loosely placed around the top of the cast and continued about 6 cm (2¼ inches) proximally up the limb.

Casts applied with the limb in extension result in a longer limb than the ipsilateral counterpart. Therefore, the cast limb is usually held in an extended or non–weight-bearing position. This may lead to continuous overload of the good limb, which increases the risk of foundering. It is advisable to tape a rubber pad to the ipsilateral foot and in so doing to lengthen it as well, preferably to the same extent as the cast limb. This comforts the patient and facilitates even weight bearing.

Generally, hindlimb casts are applied in the same way as casts for the forelimb. The most likely areas for pressure sore development are the Achilles tendon and the dorsal aspect of the tarsus. It is advisable to attach a wedge to the sole of the foot to facilitate weight bearing and prevent upward fixation of the patella. The tarsal region presents an additional problem in a full-leg hind limb cast because of the reciprocal apparatus. Attempts of the horse to flex the hind limb in a full limb cast may cause the peroneus tertius tendon to avulse from its attachment or rupture in the tarsal region, allowing flexion of the stifle without flexion of the tarsus. Treatment of this problem is discussed in Chapter 97.

Exercise should be limited for a horse with a cast. It is preferable to keep the horse in a cool environment to prevent excessive sweating under the cast. In this respect, fiberglass casts are superior to plaster casts because fiberglass casts are porous and dissipate heat from the body.

It is advisable to palpate the cast every day, especially over possible pressure points. A localized area of increased heat, palpable through the cast, is an early sign of a developing skin ulcer. Sudden decreased use of the limb under a cast or increased lameness of the affected limb are signs of irritation under the cast. Another sign of such a problem is cast abuse through chewing, stomping, or rubbing. Swelling above the cast and/or a fetid odor usually signify a far more serious problem under the cast. Should any of these signs be noted, the cast should be changed or removed to alleviate the problem. Repairing a cast or making adjustments is rarely successful and is therefore not recommended.

Cast Removal

Removal of the cast with the horse under general anesthesia is usually uneventful. Removal of the cast while the horse is standing may be more complicated. In most cases, some degree of chemical or physical restraint is necessary to permit safe removal of the cast. If Gigli wires were incorporated into the cast, the ends can be freed up and connected to their handles, and with slow sawing motions the cast can be split in half (see Figure 17-21, *D*). If cast cutters are used, the cast should be grooved medially and laterally along its entire length to ensure the correct location of the cut. Then, the proximal aspect of the cast is cut completely through, down to the foam or orthopedic felt. This allows assessment of the thickness of the cast and gives the person removing the cast an indication of how deep to cut. Using excessive force may result in perforation of the underlying skin. When the proximal area of the cast is cut through, the rest of the cast should be cut by maintaining the blade at the same location until the cast is cut through completely before moving distally. Cutting through the entire thickness of the cast is appreciated by a little faster progression of the cast cutters, which should be anticipated by the person using the cast cutters and immediately reacted to by retracting the machine and reapplying it somewhat further distally. Dragging the cast cutter parallel to the limb when it rests on the

skin will promote skin lacerations. After the entire thickness of the cast has been cut through, the two portions of the cast tend to separate somewhat.

The cast covering the foot should be split carefully, because the density of the hoof is similar to that of the cast, and it is often difficult to differentiate between them, resulting in inadvertent penetration of the hoof wall by the saw blade. Although a standing horse may object to such treatment, there will be no reaction to this in the anesthetized animal.

When the cast is split into two half shells, a cast spreader is applied to widen the gap and allow transection of the adhering cast padding with scissors. The cast is removed and the limb is washed thoroughly. If radiographs are taken after cast removal, it is advisable not to wash the limb with soap containing iodine. After cast removal, the limb should be covered with a pressure bandage for some time to allow gradual relief of external pressure. Any sores that developed under the cast should be treated immediately using routine wound management.

In selected cases, a bivalve cast is applied to the limb. This can be made on the standing and sedated horse or with the horse under general anesthesia. In either case, the padding of the cast is made somewhat thicker and usually consists of a thin bandage, which can later be changed at regular intervals. The cast is subsequently applied using routine technique. It is advisable to let it set for about a day before splitting it into two half-shells. After the bandage is changed, the two shells are reapplied and maintained in apposition by tightly wrapping the two half-shells with nonadhesive tape.

Complications

Cast complications may develop from an overly tight application, resulting in dermal pressure necrosis (which will damage deeper structures if undetected) or in an overly loose application. If the cast is too loose, the limb can shift in the cast, which may result in the development of skin pressure in areas not anticipated. Cast loosening may result from a decrease in the limb swelling, from muscle atrophy, or from compacting of cast padding materials. Application of too-short a half-cast may result in severe tendon injury, because the limb may be partially flexed, causing the top end of the cast to apply a considerable amount of linear pressure on the unprotected tendons. In a properly applied cast, the tendons are protected by the proximal ends of the vestigial metacarpal or metatarsal bones. Wear on the bottom of the cast will also cause the limb to shift within the cast, resulting in serious dermal pressure necrosis.

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CHAPTER 18

Balanced Inhalation Anesthesia

Regula Bettschart-Wolfensberger

The concept of balanced general anesthesia is based on the theory that administration of a mixture of small amounts of several neuronal depressants summates the advantages but not the disadvantages of the individual components of the mixture. Therefore, with a combination of different drugs, desired effects are achieved and untoward side effects are minimized. In horses, longer procedures (more than 2 hours) are usually performed under inhalation anesthesia. All currently used inhalation anesthetics depress cardiopulmonary function in a dose-dependent manner.[1,2] Hence, a minimal dose (just enough to induce unconsciousness) should be used, and analgesia and muscle relaxation should be provided by adding other drugs to the anesthetic protocol. In horses, maintenance of good intraoperative cardiopulmonary function followed by calm and coordinated anesthetic recovery is crucial. Therefore, balanced anesthetic techniques for horses should be directed at these two goals.

This chapter provides an overview of the use of modern inhalation anesthetics in combination with sedatives, analgesics, and/or muscle relaxants. Table 18-1 lists the recommended dose regimens and the respective dose rates.

ANESTHETIC RISK

The fatality rate in horses undergoing general anesthesia is much higher than in companion animals or humans and varies between 1% and 0.1% depending on the study design.[3-7] Horses undergoing colic or emergency surgery and horses undergoing fracture repair carry a several-fold increased risk compared to horses undergoing elective surgical procedures.

Only a few risk factors can be influenced by the choice of anesthetic agent. Nevertheless, it is generally accepted that most equine fatalities are related to either poor cardiopulmonary performance during anesthesia or to fatal injuries during violent, poor-quality anesthetic recovery.

Further studies investigating factors that might influence morbidity and mortality are necessary to determine drugs or anesthetic techniques that will improve outcome.

MODERN INHALATION ANESTHETICS

Inhalation anesthetics currently used in equine anesthesia include isoflurane (IsoFlo ad us. vet.), sevoflurane (Sevorane), and desflurane (Suprane). These drugs are usually used for maintenance of anesthesia. Their use for induction of anesthesia in foals is not recommended, because they are associated with an increased fatality rate compared to intravenous drug induction.[5]

Modern inhalation anesthetics are less potent and less soluble than older agents, such as halothane (Halothane B.P.). Drug potency is represented by the minimum alveolar concentration (MAC), defined as the alveolar concentration of inhalation anesthetic that prevents movement in 50% of subjects in response to a noxious stimulus. Thus, the MAC of novel drugs is higher than that of the older drugs (halothane: 0.88%,[8] isoflurane: 1.31%,[8] sevoflurane: 2.31%,[9] desflurane: 7.6%[10]). That means that with modern inhalation anesthetics, higher concentrations are needed to keep the horse anesthetized.

The lower blood solubility of the modern inhalation anesthetics means that changes in anesthetic plane can be achieved more readily and onset or disappearance of clinical effects is faster. The quicker disappearance of clinical effects might influence recovery characteristics, which led to several studies comparing recovery following different inhalation anesthetics (see Chapter 21). Biodegradation of modern inhalation anesthetics is very low (isoflurane, sevoflurane, and desflurane are metabolized by the liver at a rate of 0.2%, 3% to 5%, and 0.02%, respectively)[11] and probably does not influence recovery.

In general, cardiovascular depression from inhalant anesthetics is dose dependent. Dose-dependent decreases in cardiac output, stroke volume, and blood pressure as well as respiration in spontaneously breathing horses are common. While cardiovascular variables between isoflurane and halothane MAC multiples were similar in one study, they were less depressed by isoflurane and sevoflurane than halothane in another.[1,12] Under controlled ventilation, isoflurane causes less depression of cardiac output and stroke volume than halothane, and it causes similar changes in blood pressure. Vascular peripheral resistance decreases with isoflurane more than with halothane.[12]

Isoflurane, sevoflurane, and desflurane lower blood pressure as a result of decreased peripheral resistance and tend to cause less depression of cardiac output and contractility than does halothane. The effects of isoflurane and sevoflurane on cardiac output are very similar,[1,13] and desflurane does not depress cardiac output at 1 MAC.[2] Based on these findings, isoflurane, sevoflurane, and probably desflurane provide better tissue blood flow and therefore may be safer, especially in the critically ill patient.

Halothane-anesthetized horses breathe at a faster rate than horses on isoflurane. The respiratory rate also decreases progressively with increasing doses of isoflurane or sevoflurane but less

TABLE 18-1. Drugs Recommended for Reducing Minimum Alveolar Concentration in Combination with Inhalation Anesthesia, to be Administered Following Anesthesia Induction

Drug Name	Dose Rates	Comments
Lidocaine	Bolus: 0.65-1.2-(2.5) mg/kg (over 15 min) followed by 25-50-(100) µg/kg/min	Dose carefully in cardiovascularly compromised patients Toxic effects masked by anesthesia Prolonged use (more than 2 hrs) might result in ataxia during recovery; to reduce this, switch off 30 minutes before the end of anesthesia
α_2-Adrenoceptor agonist	Bolus for sedation before inducing anesthesia	Increased urinary production, urinary catheter mandatory Smoother recoveries than with lidocaine or ketamine
Medetomidine Romifidine	3.5 (-5) µg/kg/hr 0.3 µg/kg/min	CRI can be used for several hours without accumulation
Ketamine	0.5-1 (-2) mg/kg/hr	Sympathetic stimulation
S(+)-Ketamine	0.5-1 mg/kg/hr	Rough recoveries following prolonged use (more than 1.5 hrs), less with S(+)-ketamine; switch off infusion 15-20 min before end of surgery and sedate the horse during recovery to reduce this

CRI, Constant-rate infusion.

with halothane.[1,12] Despite these differences, $PaCO_2$ tends to be similar with all inhalant anesthetics, indicating similar minute-ventilation with all of them. This is most probably the result of an increased tidal volume in horses receiving isoflurane or sevoflurane, to compensate for the slower respiratory rate and a smaller tidal volume in horses receiving halothane.

DRUGS USED FOR BALANCED ANESTHESIA

Lidocaine

Lidocaine (Lidocain HCL 5%) has gained widespread popularity in equine inhalation anesthesia during the last decade to reduce the requirements for the volatile agents, in addition to its use perioperatively to improve gut motility or to provide analgesia.

Lidocaine is highly metabolized by the liver and has a very short half-life.[14] It has to be administered by constant-rate infusion (CRI). To achieve constant plasma levels within an acceptable time, an initial bolus has to be injected followed by CRI. A study conducted by Doherty and Frazier,[15] who administered a bolus of lidocaine (2.5 mg/kg/min) to six experimental ponies followed by either saline or two doses of lidocaine, 50 µg/kg/min or 100 µg/kg/min for 1 hr, revealed that lidocaine reduces MAC of halothane in a dose-dependent fashion. The same authors[15] also stated that with these dose rates no steady state was achieved and that lidocaine plasma levels were very variable between 1 and 4 µg/mL with the lower infusion rate and between 3 and 7 µg/mL with the higher rate. Plasma levels less than 2 µg/mL had a minor effect on MAC (maximal effect 20% MAC reduction) whereas levels more than 5 µg/mL reduced MAC by 50% to 70%. In another clinical study that compared isoflurane-lidocaine anesthesia to pure isoflurane anesthesia, lidocaine was administered as a bolus dose of 2.5 mg/kg (given over 10 min) followed by 50 µg/kg/min in combination with isoflurane anesthesia and administered for 75 min. This resulted in an average MAC reduction of 25%. Plasma levels of 0.03 to 4.23 µg/mL were recorded without causing any untoward side effects.[16] The horses in the lidocaine group recovered with less excitement from anesthesia.[16] In contrast to this, following a similar anesthetic period, horses had significantly worse recoveries with lidocaine (125-minute infusion) in comparison to balanced anesthesia with medetomidine.[17] Another clinical study that investigated the influence of lidocaine CRI on recovery from isoflurane or sevoflurane anesthesia showed that horses receiving lidocaine until the end of surgery had a significantly higher degree of ataxia and a tendency toward a lower quality of recovery.[18] Therefore, this study recommended the discontinuation of lidocaine CRI 30 minutes before the end of surgery to reduce ataxia during the recovery period.

In healthy awake horses, it has been shown that signs of lidocaine intoxication such as muscle tremors and ataxia occur at plasma levels as low as 1.85 to 4.53 µg/mL.[19] Feary et al.[20] showed in clinical cases undergoing routine arthroscopy that anesthesia with sevoflurane has a profound effect on lidocaine disposition. Lidocaine plasma levels were considerably higher during anesthesia than in awake horses. These authors recommended lower dosage rates in anesthetized horses than generally advocated because general anesthesia might mask neurologic manifestations of toxicosis. In another study that investigated the effects of lidocaine on small intestinal function and recovery after colic surgery, considerably lower dosage rates were used than in previous studies (0.65 mg/kg loading dose followed by 25 µg/kg/min).[21] Nevertheless these authors measured lidocaine plasma levels as high as 2.72 µg/mL in one horse and advocated prudent intraoperative dosing, especially in compromised patients. Contrary to this, Driessen reported in a retrospective clinical study the successful use of lidocaine in combination with isoflurane or sevoflurane in 25 horses undergoing colic surgery.[22] A bolus of 1.5 mg/kg lidocaine was administered immediately before surgery and the infusion of 30 µg/kg/min was stopped when the surgeon started to close the abdomen. In this comparison, horses with lidocaine did not show worse recoveries than those without, and no signs of toxicity were noted.

To summarize, a lidocaine bolus (0.65 to 2 mg/kg administered over 10 to 15 min) followed by CRI 25 to 50 µg/kg/min) can be used as part of a balanced anesthesia regimen in horses. It decreases MAC dose dependently. Higher dosage rates might induce toxicosis, especially in compromised patients with impaired cardiovascular function and thus reduced liver blood flow and metabolism. Toxicosis only becomes apparent after the effect of the inhalant anesthetic has vanished and might negatively influence recovery. Thus lidocaine should be administered with care and stopped 30 minutes prior to the end of

anesthesia, to reduce the occurrence of ataxia and uncontrolled recoveries.

α_2-Adrenoreceptor Agonists

α_2-Adrenoreceptor agonists are potent analgesics, and they reduce MAC of inhalation agents dose dependently.[23,24] Therefore, all available α_2-adrenoreceptor agonists are commonly used for balanced anesthesia in horses. Boluses of α_2-adrenoreceptor agonists impair cardiopulmonary function considerably for 20 to 120 minutes at clinically used dosage rates.[25,26] However, medetomindine is different in this regard. A bolus of medetomidine followed by a CRI results in a drop in heart rate and cardiac output for the first 10 minutes only.[27] Throughout a 2-hour medetomidine infusion, heart rate and cardiac output do not differ from pre-sedation values.[27] Among all available α_2-adrenoreceptor agonists being used for balanced anesthesia in horses, medetomidine has been investigated most intensively.[27-32]

Medetomidine's high clearance rate and short half-life necessitate its use as a CRI to achieve a persistent effect.[28] A CRI of medetomidine (3.5 μg/kg/hr) during experimental desflurane anesthesia in ponies decreased MAC by 28%.[29] In 40 clinical patients, the use of medetomidine CRI (3.5 μg/kg/hr) in combination with isoflurane resulted in significantly reduced isoflurane requirements compared to isoflurane anesthesia alone.[30] With medetomidine in this study, CRI adjustment of anesthetic depth was easier, requiring less additional drug to deepen anesthesia. Another clinical study applying balanced anesthesia in 69 cases that compared lidocaine/isoflurane (1.2 mg/kg bolus followed by 50 μg/kg/min) with medetomidine/isoflurane (7 μg/kg bolus for sedation prior to anesthesia induction followed by 3.5 μg/kg/hr throughout anesthesia) revealed that following a mean anesthesia time of 2 hours, recovery with medetomidine was longer but of better quality.[17] Maintenance of anesthesia was also easier with medetomidine, and less additional drug had to be administered to maintain a stable plane of anesthesia. The cardiac index was higher in horses anesthetized with lidocaine/isoflurane, but this was related to very high cardiac index values in some horses that were insufficiently anesthetized rather than to depressed cardiovascular function with medetomidine/isoflurane. Contrary to this, an experimental study that compared the use of either lidocaine CRI or lidocaine in combination with medetomidine CRI showed no differences in cardiopulmonary function but better-quality recoveries when medetomidine was added.[31]

A retrospective study that reported the use of medetomidine/isoflurane anesthesia in 300 clinical cases with a mean anesthesia duration of 146 minutes (range: 40 to 420 min) outlines the safety of this drug combination in horses, with only 1 poor recovery reported.[32] In comparison to other clinical studies, the incidence of hypotension or hypoxemia was similar or even lower. These authors emphasize that anesthesiologists need to be aware that judgment of depth of anesthesia is different from other inhalation anesthesia regimens.[32] Under medetomidine/isoflurane anesthesia, eye reflexes are brisker. Only the appearance of nystagmus may serve as an indicator of insufficient depth of anesthesia. Further, α_2-adrenoreceptor agonists and especially medetomidine increase urine production, and catheterization of the urinary bladder after induction of anesthesia is mandatory.

The use of romifidine (Sedivet ad us. vet.) for balanced anesthesia was tested in a clinical study in 20 horses.[33] All horses were premedicated with romifidine (80 μg/kg), and anesthesia was induced with ketamine (3 mg/kg) and diazepam (0.1 mg/kg). Ten horses were maintained under anesthesia with isoflurane only, and in the other 10 horses isoflurane was supplemented with 0.3 μg/kg/min of romifidine. Although horses with romifidine CRI needed less isoflurane, had sufficient spontaneous ventilation, and needed less dobutamine for maintenance of appropriate blood pressures, the results of this study should not be overinterpreted. There were two different anesthesiologists administering isoflurane to effect, and the duration of anesthesia was only 45 to 80 min. Furthermore, the impact of mechanical ventilation on cardiovascular function was completely neglected, even though ventilatory support was used only in some horses in the study group. A description of the recovery characteristics was also lacking in this study.

Detomidine (Equisedan ad us. vet.) CRI for balanced anesthesia in combination with halothane was used in an equine study that also investigated the effect of neurectomy on cardiopulmonary function.[34] Five horses were maintained on halothane in combination with detomidine, and four horses were maintained on halothane alone. Administration of detomidine began following anesthesia induction with a target-controlled infusion device that aimed at a plasma level of 25 ng/mL. Duration of detomidine administration was 1 hour 40 minutes to 2 hours 50 minutes and the average infusion rate was 0.18 μg/kg/min. With halothane only, horses had higher heart rates, but otherwise no other differences between the groups concerning cardiopulmonary function or recovery were noted.

In conclusion α_2-adrenoceptor agonists reduce MAC by about 30%. With medetomidine CRI at a dosage rate of 3.5 to 5 μg/kg/hr, cardiopulmonary function is relatively well maintained, and large trials showed that recovery after medetomidine/isoflurane anesthesia is better than after lidocaine/isoflurane anesthesia, and in comparison with other regimens it seems to be generally of better quality. Data of balanced anesthesia including other alpha$_2$-agonists is limited.

Ketamine

The currently licensed form of ketamine is a racemic mixture containing 50% S-ketamine and 50% R-ketamine. Ketamine (Narketan 10 ad us. vet.) is a dissociative agent, which in systemically healthy horses induces analgesia, amnesia, and immobility without depressing cardiovascular function. On the other hand, there is some sympathetic stimulation, which might help to maintain cardiovascular function in combination with inhalation anesthesia. Respiratory function is only minimally impaired by ketamine.

These properties make ketamine an ideal agent for balanced anesthesia in horses. During inhalant anesthesia, ketamine has been administered in incremental intravenous doses (0.1 to 0.2 mg/kg) or as a CRI. Muir and Sams[35] investigated ketamine's halothane-sparing effects by continuously administering the drug at several infusion rates while administering halothane in oxygen at different concentrations. The authors found halothane reduced MAC by up to 37%, and cardiopulmonary function was better in horses with ketamine-halothane in comparison to only halothane. Another study investigated ketamine in combination with isoflurane.[36] With a target controlled-infusion pump, the investigators aimed at an arterial concentration of S-ketamine (Keta-S ad us. vet.) of 1 μg/mL. The initial ketamine loading dose was approximately 0.3 to 0.4 mg/kg IV followed by a linearly decreasing infusion rate beginning at 9 mg/kg/hr and eventually reaching 5 mg/kg/hr. At these infusion rates,

ketamine was found to decrease nociception during isoflurane anesthesia in a more pronounced fashion than when the inhalant anesthetic was used alone.

Unfortunately, ketamine as well as its metabolites exhibit undesirable excitatory central nervous system effects. Following prolonged ketamine infusions (more than 1 to 2 hours) or repetitive IV boluses, horses might show those side effects. Ketamine can provoke emergence reactions during the anesthetic recovery period characterized by muscle tremor and rigidity, mydriasis, oculogyric movements, sweating, excitation, ataxia, and schizophrenia-like behavior that can turn into a fatal event in horses.[35,37] These phenomena are related to the plasma concentration of the drug, the length of drug infusion, and the concurrent formation of S-norketamine.[38] To minimize such reactions, ketamine infusions can be reduced progressively and/or be stopped 15 to 20 minutes before the end of the procedure, and patients should receive additional post-anesthetic sedation with alpha$_2$-adrenoceptor agonists.[39] Another option to reduce such unwanted reactions is to use S-ketamine, which has been used mostly under clinical circumstances instead of the racemic ketamine. S-ketamine has been tested in a study in horses undergoing elective arthroscopy.[40] Following xylazine (Xylazin Streuli ad us. vet.) injection (1.1 mg/kg), S-ketamine was administered (1.1 mg/kg IV) and anesthesia was subsequently maintained with a CRI of S-ketamine (0.5 mg/kg/hr) in conjunction with isoflurane in oxygen. This balanced anesthesia regimen resulted in better quality of anesthetic recovery than when horses received twice the dosage of racemic ketamine, especially when the anesthetic episodes lasted for more than 2 hours. Similarly, Filzek et al. found that guaifenesin (Myolaxin 15% ad us. vet.)–S-ketamine–xylazine combinations provided better recovery qualities than guaifenesin–racemic ketamine–xylazine combinations in horses undergoing castration.[41] In a clinical study in 50 horses, balanced anesthesia with S-ketamine and isoflurane resulted in better cardiovascular function than with medetomidine and isoflurane but worse recovery scores.[42]

Dissociative anesthetics preserve some reflexes usually used to evaluate anesthetic depth, such as swallowing or eye blinking, and thus horses undergoing balanced anesthesia with ketamine may not seem to be at an adequate surgical plane.[37] Therefore, special attention should be given while evaluating these patients to avoid drug overdosing. Also, sympathomimetic effects of ketamine may impair judgment of anesthetic depth. Increases in heart rate and/or arterial blood pressures should be considered indicators of an inadequate plane of anesthesia only if they are associated with surgical stimulation. Thus, if ketamine-based balanced anesthesia protocols are chosen, one may better use other parameters such as respiratory rate in spontaneously breathing horses, muscle relaxation, and absence of nystagmus to evaluate adequacy of depth of anesthesia for surgical procedures. The presence of reflex activity can be disturbing when performing surgery in the upper airway or ophthalmologic procedures. It has therefore been suggested to avoid ketamine for such procedures.[43]

In conclusion, low-dose IV infusions or repetitive boluses of racemic ketamine or S-ketamine might be beneficial when administered in conjunction with other anesthetic agents. This applies in particular to horses in need of additional analgesia and/or improved hemodynamic function. When racemic ketamine is used, the additional boluses should not exceed 2 mg/kg, and a CRI (1 mg/kg/hr) should not be used for anesthesia exceeding 90 to 120 minutes, to avoid violent recoveries. CRI should be discontinued 15 to 20 minutes prior to transferring the patient to the recovery stall. Administration of an α_2-adrenoceptor agonist before emergence from anesthesia is highly recommended.

Opioids

The intraoperative use of opioids as part of a balanced anesthesia regimen has not yet gained widespread popularity in horses, contrary to other species and humans. Their effect is debated by many authors. Several experimental and clinical studies have tried to determine the influence of opioids on MAC.[44-49] Morphine (Morphin HCl sintetica 10 mg), butorphanol (Alvegesic 1% forte ad us. vet.), or alfentanil (Rapifen) did not consistently reduce MAC.[44-46] Individual horses within each study showed either an increase in MAC, a decrease, or no change at all. Individual horses recovered violently from anesthesia, showing signs of central nervous excitement, especially when high doses of opioid agonists were used.[46]

The use of *naloxone* (Narcan) did not prevent this excitement during recovery.[46] A clinical study tested the use of a bolus of morphine (0.15 mg/kg) followed by infusion of the drug (0.1 mg/kg/hr) in comparison to halothane anesthesia alone.[47] No significant differences between the groups were identified. The same authors also tried to show a beneficial effect of morphine on recovery from anesthesia but were unable to do so.[48] Morphine's influence on MAC of halothane was also tested when administered concurrently with xylazine.[49] The results of this study indicate that xylazine reduces inhalant anesthetic MAC but morphine does not enhance this effect any further.

In one study, the isoflurane MAC-sparing effects of fentanyl, dosed based on previously determined pharmacokinetic data in individual horses, were tested.[50] It was concluded that there may be a therapeutic dosage range of fentanyl that consistently decreases MAC, even though with the different plasma levels that were tested some horses showed an increase in MAC and others showed a decrease or no change at all. Furthermore, in the same study,[50] two horses needed active cooling with ethanol to maintain their body temperature below 38.6° C and one of eight horses showed a violent recovery during which it frantically attempted to circle in both directions, falling over several times. This observation is in agreement with studies in awake horses.

A recently published retrospective study showed that butorphanol deepened anesthesia when administered in conjunction with isoflurane and that sympathetic stimulation caused by surgery was blunted when butorphanol was used.[51] The fact that opioids when used for balanced anesthesia reduce propulsive gastrointestinal motility[52] and may slow respiration needs to be considered.[46]

In conclusion, only very few studies support the regular pre- or intraoperative use of opioids for MAC reduction in horses. Nevertheless, it is advisable to administer 0.1 mg/kg morphine at the end of every anesthesia to provide some additional analgesia. This results in a smoother recovery with no untoward effects.

Centrally Acting Muscle Relaxants

Guaifenesin

Formerly known as glyceryl guaiacolate, guaifenesin is used as an adjunct to balanced anesthesia in horses to induce muscle

relaxation. Guaifenesin has a wide margin of safety and sedative properties that can potentiate other sedative drugs.[53] It provides good relaxation of laryngeal and pharyngeal muscles, allowing easier intubation, and also produces relaxation of skeletal muscles. Clinical dosages (in the range of 100 to 150 mg/kg) do not affect diaphragmatic function, preserve respiratory function, and exert no significant effect on cardiac output and arterial blood pressure.[54] Whether guaifenesin has mild analgesic properties in horses is still under debate. For this reason, the use of guaifenesin as the only adjuvant to inhalation anesthetics is not advised.

Spadavecchia et al. combined guaifenesin (1 to 0.3 mg/kg/min) with ketamine (39 to 13 μg/kg/min) to reduce the required halothane dosage in horses that were presented for a variety of surgical procedures, including emergencies.[39] The combination of these two drugs resulted in a more stable surgical anesthesia compared with halothane alone, with fewer episodes of patients moving in response to surgery. The quality of recovery was acceptable and similar to horses receiving halothane alone. Similarly, infusions of ketamine/guaifenesin or ketamine/guaifenesin/romifidine facilitated a reduction in isoflurane dosages in horses undergoing various surgical procedures and resulted also in more stable and better cardiovascular performance than when isoflurane was used alone.[55] The authors attributed these observations to the anesthetic-sparing effects and the analgesic properties of the drug combination. Infusion of guaifenesin, ketamine, and medetomidine to horses anesthetized with sevoflurane resulted in better transition and maintenance phases while improving the cardiovascular function and reducing the attempts needed to stand up during the recovery phase, compared with inhalation of sevoflurane alone.[56]

Thrombophlebitis can occur especially with solutions containing 10% guaifenesin, and hemolysis has been reported after administering IV solutions containing a concentration greater than 10% of guaifenesin.[57,58]

In conclusion, although the effect of guaifenesin alone on MAC has never been quantified, and neither have its analgesic properties, this drug can be added to balanced anesthesia protocols because it improves muscle relaxation. Administration to horses at risk of thrombophlebitis is not recommended.

Benzodiazepines

Traditionally, benzodiazepines (such as midazolam [Dormicum]) have been used in equine anesthesia to reduce the muscle contraction produced by ketamine, especially during the induction phase of anesthesia. Water-soluble benzodiazepines have been incorporated in balanced anesthesia protocols in an attempt to potentiate muscle relaxation and to reduce the dose of volatile agents required to maintain a surgical plane of anesthesia.[59] Controversy exists with regard to their analgesic effects. Although literature on the pharmacologic properties of benzodiazepines did not consider them as being analgesics, more recent studies provide some evidence to suggest that they might enhance the analgesic properties of co-administered drugs.[60]

Kushiro et al. administered CRI of ketamine, medetomidine, and midazolam to six horses undergoing a 4-hour surgery twice at an interval of 1 month.[60] The horses were mechanically ventilated and received sevoflurane in oxygen. With this drug combination, cardiovascular function was well preserved and sevoflurane delivery could be reduced to an end-tidal concentration of 1.7%, which is lower than the MAC value (2.3%) reported in horses.[61] In these horses, recovery from anesthesia was uneventful, although ataxia was recorded for 15 to 20 minutes after standing, and an assisted recovery technique with use of head and tail ropes was advised. To minimize the postanesthetic ataxia induced by benzodiazepines, it has been suggested to antagonize their effects by administration of specific benzodiazepine antagonists, such as sarmazenil (Sarmasol).[37]

In conclusion, water-soluble benzodiazepines can be administered together with α_2-adrenoceptor agonists or ketamine to enhance muscle relaxation. Their role as analgesic co-adjuvants remains to be determined. Antagonization with a specific antagonist at the end of the anesthesia is advised to reduce the risk of postoperative ataxia.

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CHAPTER
19

Modern Injection Anesthesia for Horses

Regula Bettschart-Wolfensberger

The use of inhalation anesthesia is generally limited to larger clinics because of equipment costs, the requirement for an oxygen source, and the need for a scavenging system for waste gases. There is also strong evidence that inhalant techniques are associated with a higher mortality rate in horses (see Chapter 18). Therefore, the use of safe intravenous anesthesia techniques in practice is both desirable and advantageous. The most important features of intravenous protocols are a smooth, excitement-free induction phase with a slow lowering of the body into sternal and lateral recumbency, minimal

cardiopulmonary depression, no reactions to surgical stimuli, and a calm recovery with a single attempt to stand and minimal ataxia. Other factors include good muscle relaxation and analgesia, as well as the possibility to assess depth of anesthesia and to modify depth and duration of anesthesia in a quick and predictable manner.

Injectable anesthetic combinations are currently used for anesthesia induction and for short, minor surgical procedures up to 30 minutes. Longer surgeries are performed with intravenous anesthesia induction followed by inhalation, or less commonly by total intravenous anesthesia (TIVA). Features of short-duration injection anesthesia are discussed separately from long-duration (anesthetic duration more than 30 minutes) TIVA. Anesthetics discussed in this chapter include ketamine and propofol and useful combinations of these two drugs. Unfortunately there are only very few recent reports that further investigated these drugs or new combinations. The only new drug in equine intravenous anesthesia is alphaxalone (Alfaxan 10 mg/mL). Its first use in horses has been reported in anesthesia conferences.[1,2] Further clinical studies are necessary to determine whether this drug has advantageous properties compared to the currently used injectable anesthetics. Older agents, such as barbiturates, chloral hydrate, and drugs suitable for the anesthesia of wild Equidae, such as Immobilon (etorphine-acepromazine), will not be discussed.

SHORT-DURATION INJECTION ANESTHESIA

For anesthesia induction, ketamine (Narketan 10 ad us. vet.), tiletamine/zolazepam (Zoletil ad us. vet.), or propofol (Propofol 1% MCT Fresenius) can be used. Adult horses must be adequately sedated before anesthesia induction with a calculated dosage of the selected drug. Only in very young foals and in recumbent, severely compromised horses can anesthesia be induced with administration of an anesthetic to effect. For compromised horses, a mixture of equal volumes of diazepam (Valium 5mg/mL) and ketamine (100 mg/mL) represents a safe method for anesthesia induction and usually requires 1 mL/25 kg of the mixture (0.1 mg/kg diazepam plus 2 mg/kg ketamine). This protocol avoids the cardiovascular-compromising side effects of α_2-agonists. In foals, the administration of propofol to effect is a good alternative (see Chapter 20).

Ketamine

Ketamine is the most widely used drug for anesthesia induction in horses. It provides good, mainly somatic analgesia without inducing hypnosis. However, it is not suitable as a sole agent because it may cause seizure-like activity and muscle rigidity. Appropriate sedation with α_2-agonists and/or acepromazine (Prequillan), eventually in combination with opioids, prior to anesthesia induction is very important.[3-16] In a stressed, not well-sedated horse, ketamine does not result in a satisfactory quality of anesthesia. The addition of such drugs as guaifenesin (Myolaxin 15% ad us. vet.) or benzodiazepines (diazepam or midazolam [Dormicum]) will further improve muscle relaxation.[17-23] Guaifenesin is a safe drug with minimal side effects at clinical dosages. It should be used as a 5% (50 mg/mL) solution, because higher concentrations are associated with significant irritation of the veins and intravenous hemolysis.[24]

Guaifenesin is administered to the sedated horse to effect (preferably under pressure, because effective dosages are large: 50 mg/kg, or 500 mL for a 500-kg horse). When the horse begins to buckle at the knees, the induction drug, most commonly ketamine (2 mg/kg), should be administered.

Benzodiazepines can be used instead of guaifenesin and can be given together with ketamine without causing irritation of the vein. Depending on the dosage used, these drugs may increase the respiratory depression caused by ketamine. To reduce the risk of apnea (under field conditions where respiratory support is not available) and ataxia during recovery, low dosages of benzodiazepines (0.02 to 0.04 mg/kg diazepam or midazolam IV) are advocated. For anesthesia induction followed by inhalation anesthesia, higher dosages (up to 0.2 mg/kg IV) can be used and will facilitate intubation. Mechanical ventilation will counteract respiratory depression. To guarantee adequate muscle relaxation in the field where low dosages of benzodiazepines should be used, very deep sedation with relatively high dosages of α_2-adrenoceptor agonists, such as xylazine (Xylazin Streuli ad us. vet.), detomidine (Equisedan ad us. vet.), or romifidine (Sedivet ad us. vet.), is recommended.

A recent study investigated the effect of a bolus of 3 mg/kg lidocaine in ponies anesthetized with xylazine/ketamine for castration.[25] The use of lidocaine did not reduce the need for top ups with xylazine/ketamine for maintenance of unconsciousness and recumbency during castration. Only time-to-standing was prolonged by lidocaine (Lidocain HCl 2%), indicating an analgetic effect of this drug. The significance of this for clinical practice remains to be tested.

Ketamine is a safe anesthetic for horses. Properly sedated, undisturbed horses will slowly sink into sternal and then lateral recumbency. Recovery at the end of anesthesia is usually quick and coordinated.[3-16] Respiratory depression is minimal. Ketamine's sympathomimetic action is ideal to counteract the bradycardia and hypotensive effects of the drugs used as sedatives.[26] For example, xylazine has been shown to have only minimal influence on cardiovascular function in combination with ketamine in the horse.[9] Because the eyes remain open and the reflexes are only minimally depressed, the assessment of anesthetic depth is difficult and requires some familiarization time. Movements as a result of awakening may occur very suddenly and may be of a strong nature. Preferably, administer ketamine in healthy horses according to a fixed time scheme rather than to effect (Table 19-1).

S(+)–Ketamine

The currently licensed form of ketamine is a 50 : 50% racemic mixture of the S(+) and the R(−) enantiomers. The S(+)-ketamine (Keta-S ad us. vet.) is the active compound, and several studies have tested its use in horses.[27,28] At a dosage rate of 50% to 66% of the racemic ketamine the effects were similar to the racemic ketamine. Recoveries were smoother and of better quality when S(+)-ketamine was used in combination with xylazine[28] administered as boluses (xylazine 0.5 mg/kg, S[+]-ketamine 0.5 mg/kg) every 10 minutes for 50 minutes to perform castration. S(+)-ketamine given as a constant rate infusion (0.5 mg/kg/hr) in combination with inhalation anesthesia also resulted in better recoveries than the racemic ketamine at double the dosage rate.[27] Other effects were identical.

TABLE 19-1. Protocols for Use of Ketamine for Short Surgical Procedures

Sedation		Anesthesia Induction		Duration of Anesthesia	Prolongation
α_2-AGONIST		**MUSCLE RELAXANT AND ANESTHETIC**			
Administer slowly to effect; wait 5 (xylazine)-10 (others) min	Xylazine 0.5)-1 mg/kg IV	*Either* guaifenesin 25-50 mg/kg IV Administer under pressure before ketamine to effect (until horse starts to become wobbly)	Ketamine 2 g/kg IV	10-25 min Duration of anesthesia tends to be longer with detomidine or romifidine sedation	Xylazine-ketamine: half the initial dose of each drug every 10 min
Combine with butrophanol (0.02 mg/kg) *or* methadone (0.1 mg/kg to increase sedation	*or* detomindine 20-(40) mcg/kg IV *or* romifidine (80)-100 mcg/kg IV	*or* benzodiazepine 0.02-(0.2) mg/kg IV (diazepam, midazolam, climazolam) administer together with ketamine	Ketamine 2 mg/kg IV	Shorter duration in stressed, nervous animals	Ketamine: 0.5-1 mg/kg every 10 min following detomidine or romifidine sedation
Administer (IM) detomindine *or* romifidine to uncooperative horses					Triple drip (see Table 19-2)

Tiletamine

Tiletamine is a drug similar to ketamine, and both are classified as dissociative agents. Tiletamine is commercially available as a powder in a fixed (1:1) combination with zolazepam, a benzodiazepine, and it is reconstituted with sterile water immediately before use. It has been used in horses and other Equidae at dosage ranges of 1.1 to 1.65 mg/kg IV after sedation with α_2-adrenoceptor agonists.[29-33] Depending on the dosage selected, the recumbency time was considerably longer than with ketamine combinations, and respiratory depression was more pronounced. Ataxia during recovery was more accentuated than with ketamine, with horses making several attempts to stand up. These features make this combination less desirable than xylazine-ketamine, especially under field conditions.

Propofol

Propofol is a short-acting anesthetic that provides very good hypnosis and muscle relaxation but poor analgesia. It is not suitable as a sole agent in adult horses because anesthesia inductions are unpredictable, and the large volumes required make it impractical and prohibitively expensive.[34] Propofol has been used mainly after sedation with α_2-adrenoceptor agonists under experimental conditions.[35-38] Only a few investigations have looked at propofol in clinical patients.[39-41] Some authors were happy with propofol as an induction agent.[39] Others reported that the quality of anesthesia induction in individual horses was "not ideal" or "unacceptable."[40,41] It was hoped that slow administration of propofol would smooth anesthesia induction, but other factors, such as respiratory depression and lack of inherent analgesia in addition to cost, have prevented widespread use of propofol for anesthesia induction and its maintenance during short surgical procedures.[40]

In the neonatal foal, the use of propofol administered to effect for anesthesia induction (approximate dosage, 2 mg/kg IV) is, however, a good alternative to ketamine; the duration of action of propofol is short and not dependent on liver function. Relaxation is better and thus endotracheal intubation is easier than with ketamine. Propofol is contraindicated in hypotensive foals, because it will further compromise these patients. For additional information on foal anesthesia, refer to Chapter 20.

TOTAL INTRAVENOUS ANESTHESIA OF GREATER THAN 30 MINUTES' DURATION

Table 19-2 summarizes drugs and dosages suggested for maintenance of anesthesia with TIVA in horses.

In modern equine practice, TIVA of longer duration is commonly maintained with constant infusions of the so-called triple-drip (ketamine–guaifenesin–α_2-agonist), ketamine-climazolam (Climasol ad us. vet.), or propofol in combination with various drugs.

Ketamine combinations can be safely used for up to 1.5 hours to perform minor surgeries under clinical practice conditions. A recumbent horse breathing air will, however, become hypoxic. To prevent complications associated with hypoxia during procedures of 30 minutes or longer, inspired air should be supplemented with oxygen (15 L/min, via a nasal or endotracheal tube). If the duration of anesthesia exceeds 2 hours, ketamine should not be used, because it produces active metabolites that are eliminated very slowly and thus influence the recovery period negatively.[42,43]

TABLE 19-2. Dosage Regimens for Maintenance of Ketamine-Based TIVA in Horses

Technique	Drugs	Approximate Dosage	Comments
Triple drip	500 mL guaifenesin 10% + 1 g ketamine + 10 mg detomidine	1 mL/kg/hr	Even higher dosage during initial 10-15 min; try to reduce dosage rate after 1 hr of infusion
	500 mL guaifenesin 5% + 650 mg ketamine + 325 mg xylazine	2 mL/kg/hr	
	500 mL guaifenesin 5% + 500 mg ketamine + 250 mg xylazine	2.75 mL/kg/hr	Better relaxation than with preceding mixes (because guaifenesin dose is higher); more ataxia during recovery
Ketamine-climazolam	Ketamine + climazolam	6 mg/kg/h + 0.4 mg/kg/hr	20 minutes after the end of infusion, sarmazenil 0.04 mg/kg must be given IV to prevent ataxia
Medetomidine-ketamine-midazolam	0.1 mg/mL medetomidine + 40 mg/mL ketamine + 0.8 mg/mL midazolam	0.1 mL/kg/hr	Tested for anesthesias up to 60 mins; sarmazenil 0.04 mg/kg can be given IV to prevent ataxia in recovery

Ketamine–Guaifenesin–α_2-Adrenoceptor Agonist (Triple-Drip)

The triple-drip drug combinations are the most widely used for TIVA. Cardiovascular and respiratory depressions are minimal, and these combinations have been successfully used to perform major surgical procedures as well as to prolong field anesthesia in a relatively controlled manner.[44-47] The use of triple-drips should be restricted to procedures of up to 2 hours in duration, not only because of the cumulative effects of metabolites of ketamine but also because large doses of guaifenesin may result in severe ataxia during recovery. Anesthetic induction performed prior to triple-drip anesthesia should not include guaifenesin, so that the total amount of guaifenesin given to the individual is as low as possible. A suitable induction technique is xylazine followed by ketamine. Another successful protocol tested by Mama et al. applies xylazine, guaifenesin, and ketamine and maintains anesthesia with only xylazine and ketamine at various dosage rates.[48] The horses went through a good to excellent recovery phase but took relatively long (mean times to standing, 46 to 69 minutes) following anesthesia durations of 66 to 73 minutes.

During painful procedures, analgesia should be provided with local anesthesia or the inclusion of opioids, such as butorphanol (Alvegesic 1% forte ad us. vet.) or morphine (Morphin HCl sintetica 10 mg).

Ketamine–Midazolam–α_2-Adrenoceptor Agonist

Because guaifenesin is not available in some countries, some authors have tried to replace the guaifenesin with midazolam (Dormicum). A protocol using medetomidine (Dorbene) for sedation and ketamine (2.5 mg/kg IV) and midazolam (0.04 mg/kg IV) for anesthesia induction was followed by a mixture containing 0.8 mg/mL midazolam, 40 mg/mL ketamine, and 0.1 mg/kg medetomidine at a rate of about 0.09 mL/kg/hr for maintenance of anesthesia was used satisfactorily for castrations lasting 38 ± 8 minutes.[49] Whether this protocol is useful for longer-duration surgeries remains to be tested.

Ketamine-Climazolam

The infusion of climazolam (a long-acting benzodiazepine) together with ketamine causes less cardiovascular depression than triple-drip techniques.[19,50] Because climazolam causes severe ataxia, its action has to be reversed with sarmazenil (Sarmasol) for recovery. Analgesia is sufficient to perform superficial surgeries causing somatic pain. To provide adequate analgesia for visceral procedures, local anesthesia or additional analgesia (opioids, α_2-agonsits) must be administered.

Propofol

In contrast to ketamine, propofol is an ideal anesthetic for prolonged TIVA. It possesses a short context-sensitive half-life, permitting rapid recovery.[42] It has been used in combination with different α_2-adrenoceptor agonists, guaifenesin, ketamine, and opioids.[40,41,51-54] In combination with medetomidine, anesthesia was maintained for up to 4 hours, and major surgical interventions could be completed successfully. Although recovery was uneventful and relatively quick in all reports, problems, mainly of a respiratory nature (apnea and severe respiratory acidosis) and the relatively high cost of propofol have prevented widespread use of propofol in clinical practice. It offers no major advantages over commonly used inhalation anesthetic protocols, which represents an additional reason for its limited use.

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CHAPTER 20

Anesthesia and Analgesia for Foals

Bernd Driessen

Foals are born after a gestation period of approximately 11 months (335 to 342 days)[1] and birth takes place quickly, consistent with the status of a horse as a prey and flight animal. Unlike many other species, the foal is developmentally much more mature at the time of birth, reaching the status of a juvenile physiologically within 6 to 8 weeks of life.[2] Based on physiological parameters, anesthesiologists may classify foals from birth to 1 month of age as neonates and as pediatric and then juvenile animals when they are 1 to 3 and 3 to 4 months old, respectively. They may be treated anesthesiologically like young adults when they have acquired mature cardiopulmonary function and metabolic pathways at 4 to 5 months of age.[2,3]

Foals may require deep sedation; local, regional, or general anesthesia; and analgesia care for a variety of reasons, most commonly for abdominal, urogenital, traumatic, orthopedic, endoscopic, and diagnostic imaging procedures. In 1995, the overall perioperative mortality rate for equine patients under 1 year of age was reported as high as 1.9%, which was higher than the rate reported for the general horse population.[4] However, recent data indicate that the perianesthetic mortality rate can be reduced to 0.2% or less, similar to that reported in adult horses,[5] provided anesthetic techniques and analgesic regimens applied are tailored to the developmental stage and the specific needs of the individual foal.

PHYSIOLOGICAL AND PHARMACOLOGICAL CONSIDERATIONS AS THEY RELATE TO ANESTHESIA IN THE NEONATAL AND MATURING FOAL

In the first days and weeks of life, the newborn foal undergoes major physiological changes that will affect almost all organ systems and functions, including circulation, respiration, oxygen (O_2) and nutrient delivery and consumption, central and peripheral neuronal activity, cell and organ metabolism, thermoregulation, and immune system activity. Administering safe anesthesia in the foal requires a thorough understanding of those changes, which are summarized in Table 20-1.

Cardiovascular System

Transition from Fetal to Neonatal Circulation

In mammals, the most dramatic change in cardiovascular function occurs at birth with the transition from fetal to neonatal circulation.[6] The primary function of the circulatory system of both the fetus and newborn is to deliver O_2 and nutrients to metabolizing organs and return deoxygenated blood to the gas exchange organ to replenish the O_2 and eliminate waste products including carbon dioxide (CO_2). In the fetus, the gas exchange organ is the placenta, and its vascular connections are

TABLE 20-1. Most Relevant Aspects of Foal Physiology that Affect Anesthetic Management

System	Neonate (1 Month or Younger)	Pediatric/Juvenile Foal (1-4 Months)
Cardiovascular	Transition from fetal to neonatal circulation Risk of return to fetal circulation HR-, not SV-dependent cardiac output Low systemic vascular resistance	More SV-, less HR-dependent cardiac output Increasing systemic vascular resistance
Respiratory	Maturation of pulmonary microanatomy, neuromuscular control, compliance, surfactant production High RR-dependent V_{min}, low V_T High O_2 consumption but low PaO_2	Respiratory function Higher V_{min} and RR with normal V_T Close to normal PaO_2
Nervous	Immature central, autonomic, and peripheral nervous system function Higher BBB permeability	Matured central, autonomic, and peripheral nervous system function Close to adult BBB permeability
Metabolism and tissue composition	High ECF compartment, CBV, CPV Low glycogen reserves No fiber intake High body surface area (heat loss)	Higher ECF but close to adult CBV and CPV Larger glycogen reserves Increasingly more fiber intake
Hepatic	Maturing liver function in first 3-4 wks	Overall close to mature
Renal	Immature Reduced concentrating ability	Overall mature
Hematology and biochemistry	Physiologic anemia Gradual increase in WBC Elevated serum enzyme activities	Normalizing PCV Adult WBC Elevated serum enzyme activities

BBB, Blood-brain barrier; *CBV*, circulating blood volume; *CPV*, circulating plasma volume; *ECF*, extracellular fluid volume; *HR*, heart rate; *PaO_2*, arterial oxygen tension; *PCV*, packed cell volume; *RR*, respiratory rate; *SV*, stroke volume; *V_{min}*, minute ventilation volume; *V_T*, tidal volume; *WBC*, total white blood cell count.

in a parallel arrangement with the other systemic organs, remote from the pulmonary circulation. To supply deoxygenated blood to the placenta and return oxygenated blood to systemic organs, a series of extracardiac shunts (ductus venosus, patent ductus arteriosus) and an intracardiac communication (foramen ovale) are necessary. At birth, the function of gas exchange is transferred from the placenta to the lungs, and therefore from the systemic circulation to the pulmonary circulation. The venous and arterial circulations are now separated, and not only are the fetal shunts unnecessary, but their persistence may compromise circulatory functions. Therefore, transition from fetal to neonatal circulation includes elimination of the placental circulation; lung expansion and increase in pulmonary blood flow; and closure of the foramen ovale, ductus arteriosus, and ductus venosus. Closing of those pathways does not occur immediately at birth and thus right-to-left shunting may continue and murmurs consistent with a patent ductus arteriosus may be auscultated in normal foals during the first 3 to 5 days of life,[7] with partial reopening possible up to the moment of complete fibrosis of those pathways, which occurs within 2 to 3 weeks.[8] As part of the transition from fetal to neonatal circulation, the left ventricular wall increases in thickness in parallel with a rise in systemic vascular resistance, reflecting the shift from the physiological right ventricular hypertrophy during fetal life to the physiological left ventricular hypertrophy in postnatal life.[9] An understanding of fetal hemodynamics and the acute and chronic changes that occur with transition to the newborn circulation are important for the care of normal newborns and are crucial to the recognition and management of a newborn with significant congenital heart disease or transient hemodynamic changes that may occur during general anesthesia and trigger a reversal to conditions of fetal circulation.

Hemodynamic Function

Cardiac output (CO) is defined as the amount of blood ejected by the heart per minute and is calculated as the product of heart rate (beats per minute) and stroke volume (mL). It is the most appropriate index of overall cardiovascular function and, when normalized to body weight, is referred to as cardiac index (CI, mL/min/kg). Fullfilling the needs of metabolically highly active organs and tissues during the early postnatal life, CI in resting foals up to 2 to 3 months of age is markedly higher when compared to adults and primarily rate-dependent (Table 20-2).[10-14] If CO is adjusted for metabolic size (0.75/kg),[11] the average CI in foals is approximately twice that of adults but the average stroke volume index 30% less.[7,15] Therefore, the normal heart rate of a resting equine neonate is significantly higher to maintain higher CO (see Table 20-2).[16,17] It is in this early period of life that any drug with heart rate–decreasing properties like α_2-adrenoceptor agonists may compromise hemodynamic function to an extent that the neonate cannot tolerate.[3,7] From 4 months of age onward, heart rates reach close to adult values and remain relatively stable throughout the remainder of the first year.[18]

Mean systemic arterial blood pressure is substantially lower in the early days of life but pulse pressure amplitude is higher in the neonate compared to the adult owing to a lower vasomotor tone and hence systemic vascular resistance (see Table 20-2).[7,10,11,13,14] By 1 month of age, foals tend to have a lower CI and heart rate (see Table 20-2) but a larger stroke volume, and their mean arterial pressure increases during this period because of a marked increase in vascular resistance indicative of the maturing sympathetic branch of the autonomic nervous system.[7]

Respiratory System

It is pertinent for every anesthetist to appreciate that any impairment of respiratory function, whether caused by sedative, analgesic or anesthetic drugs, recumbency and positioning, or surgical/diagnostic interventions, may severely compromise vital functions of the newborn. At birth, neither neuromuscular control of ventilation nor the lung itself is fully developed in foals.[7,15,19-21] Pony lungs are microanatomically more mature at birth than horses' lungs,[21] but still sufficient surfactant production is lacking and gas exchange occurs across terminal air spaces and more primitive alveoli.[8] Compliance of the chest wall is large in the neonate but lung elasticity is decreased.[8] Therefore, functional residual capacity (FRC), which is the gas

TABLE 20-2. Hemodynamic, Respiratory, and Acid-Base Parameters in Normal Awake Foals Compared to Adults*

	Age				
Parameter	**1-3 days**	**1 week**	**2 weeks**	**4-6 weeks**	**Adult**
HR (beats/min)	118 ± 10	110 ± 30	103 ± 21	84 ± 11	39 ± 4
SAP (mm Hg)	137 ± 31	–	–	–	142 ± 12
DAP (mm Hg)	62 ± 7	–	–	–	99 ± 11
MAP (mm Hg)	87 ± 10	100 ± 20	100 ± 11	115 ± 14	114 ± 11
CI (mL/kg/min)	271 ± 3	225 ± 56	229 ± 74	167 ± 40	69 ± 17
RR (breaths/min)	44 ± 19	42 ± 11	38 ± 11	36 ± 9	16 ± 6
V_T (mL/kg)	6 ± 0.5	8 ± 1.2	14 ± 2	13 ± 2	14 ± 2
V_{min} (mL/kg/min)	848 ± 231	744 ± 169	523 ± 126	436 ± 116	162 ± 45
PaO_2 (mm Hg)	75 ± 8	87 ± 5	–	89 ± 6	94 ± 3
$PaCO_2$ (mm Hg)	46 ± 3	47 ± 3	–	42 ± 2	40 ± 3
pH	7.4 ± 0.05	7.37 ± 0.03	–	7.4 ± 0.01	7.4 ± 0.03
BE (mEq/L)	1.7 ± 7.4	1.4 ± 2	–	0.8 ± 2.4	6 ± 3

BE, Base excess; *CI*, cardiac index; *DAP*, diastolic arterial pressure; *HR*, heart rate; *MAP*, mean arterial pressure; *$PaCO_2$*, arterial carbon dioxide tension; *PaO_2*, arterial oxygen tension; *RR*, respiratory rate; *SAP*, systolic arterial pressure; *V_{min}*, minute ventilation volume; *V_T*, tidal volume.
*Data from references 7, 15, 16, 20, 45, 62, 84, 97.

volume left in the lung after a normal expiration, and tidal volumes are markedly smaller than in the adult (see Table 20-2). Thus, in the immediate postnatal period foals are hypoxemic, with PaO_2 values being significantly lower than during adult life, whereas $PaCO_2$ values being similar. Still, because O_2 needs of the rapidly developing organism are much higher than in the adult, especially in the first week postpartum, O_2 consumption (6-8 mL/kg/min) exceeds that of the adult horse by two- to threefold,[15] requiring increased respiratory minute ventilation. To compensate for the smaller FRC and tidal volume, newborn foals typically breathe up to 60 to 80 times per minute, which in the fourth to sixth week declines to 30 to 40 breaths per minute for the remainder of the first 3 months of life before gradually approaching adult values. In addition, neonates close the upper airway during end expiration and therefore do not allow the lung to collapse easily; however, this protective mechanism (often referred to as "auto-PEEP") is often lost during anesthesia. This in conjunction with a lower sensitivity of the respiratory center to changes in PaO_2 and $PaCO_2$, most prominent after sedation with α_2-adenergic drugs, which particularly predisposes neonatal foals to hypoxemia and hypercarbia.

Nervous System Development

At the time of birth, the nervous system of a newborn still has substantial development to complete, as behavioral, anatomic, and physiologic evidence suggests. Studies in various species, including horses, indicate that the basic anatomical structures of the brain and spinal cord are present at birth as are specific cellular groups, synapses, and neurochemical markers.[22,23] Unlike in other species, neurogenesis of the cerebellar cortex is fairly complete in the newborn foal, indicating the horse's brain is maturing quite rapidly.[22] Because the cerebellum is responsible for coordinated movements and ambulation, this also explains why the foal can rise about 30 minutes after birth and start running minutes later. Although most of the large neurons differentiate early during fetal development, small neurons and neuroglia differentiate later, and myelination of nerve fibers is incomplete at many levels of the nervous system at the time of birth. As a result, transmission of nerve impulses from the periphery to the central nervous system is slower than in the adult and the ability to localize stimuli may be relatively poor. Therefore, when the neonatal foal is traumatized, it may or may not reply quickly enough with target-oriented nocifensive reflex responses. This phenomenon, however, does not preclude functioning nociceptive pathways or pain sensation in the newborn, and it thus calls for appropriate analgesic treatment, local and regional anesthesia, or even general anesthesia whenever a foal is exposed to noxious stimulation.

There is substantial laboratory animal evidence that the blood–brain barrier to proteins and other macromolecules, principally a property of tight junctions between the cells, is well formed early in brain development, whereas postnatal modifications in tight junction structure is in part responsible for the decline in blood-brain and blood–cerebrospinal fluid (CSF) permeability to smaller molecular compounds, such as many endogenous substances, nutrients, and drugs.[24] In addition, during the early postnatal period, the open tubulocisternal endoplasmic reticulum components of cerebral endothelial and chorioid plexus epithelial cells close, thereby restricting more and more diffusion of endogenous substances and drugs from the blood into the central nervous tissue.[25] Those changes, immaturity of the central nervous neuronal function as a whole, and different pharmacokinetic behavior of drugs in neonates may account for differences in responses to agents administered to a foal for purposes of chemical restraint, sedation, analgesia, and anesthesia.

There are numerous age-dependent changes in the autonomic nervous system responsiveness of myocardial contractile and conducting tissue and vasomotor tone reported in laboratory animal species,[26] but little is known in the horse. Those studies suggest that at birth parasympathetic nervous activity dominates while sympathetic innervation of heart and vasculature is still immature, which may in part explain the low systemic vascular resistance and mean systemic blood pressure as well as higher rate of bradyarrhythmias observed in the newborn foal subject to hypoxemia and/or hypothermia.

Early Postnatal Behavior

The significantly advanced development of the nervous system in the equine compared to other species provides the newborn foal with the ability to quickly escape from predators. Immediately postpartum, the equine neonate can maintain sternal recumbency and within 1 hour after birth will stand up and nurse, and soon thereafter can trot and canter.[17] Most can gallop by the next day. The suckle reflex is present within 30 minutes, and the average foal is nursing the mare within 2 hours postpartum. Any deviation from those normal behaviors, similarly to deviation from normal cardiovascular and respiratory parameters in the early phase of a foal's life, should alert the anesthetist to existing or impending problems.

Body Metabolism, Biotransformation, and Excretion

Body Water Content and Body Tissue Composition

Unlike in neonates of most other species, in the newborn foal total body water content is around 72%[27] or 74.4% ± 2.4%[28] of total body mass and hence is relatively low compared to puppies and kittens; and it does not change much over the first 5 months of life.[29] It also is close to the 67% of body weight measured in the adult horse.[30] The extracellular fluid (ECF) compartment is on a per kilograms of body weight basis about one third larger in foals than in adults, as are the blood and plasma compartments (Table 20-3),[27,31,32] which must be accounted for during perianesthetic fluid therapy and intravascular volume substitution. The higher ECF volume implies a larger apparent volume of distribution for many drugs, which must be taken into account for appropriate drug dosing and for predicting of drug uptake and distribution in the body.[25] Furthermore, because of the presumably higher capillary permeability in the neonate yet increasing systemic arterial blood pressures postpartum, intravascular water rapidly redistributes into the interstitial space, where it accumulates.[33] As a result, no sustained increase in intravascular volume occurs, which in the adult animal triggers diuresis by modulating release of vasopressin, renin, and atrial natriuretic peptide.[33] Consequently, neonates, especially ill neonates, retain administered fluid over a much longer time and thus do not handle large fluid loads well. At the same time, the expanded interstitial space in the neonate serves as a reservoir for fluid and can be rapidly mobilized in situations of acute hemorrhage or hypovolemia, restoring total blood volume

TABLE 20-3. Hematologic and Biochemical Parameters in Normal Awake Foals Compared to Adults*

	Age						
Parameter	**1-3 Days**	**1 Week**	**2 Weeks**	**4-6 Weeks**	**2 Months**	**4-6 Months**	**Adult**
Body weight (kg)	54 ± 5	–	–	98 ± 14	133 ± 15	200 ± 14	400-650
CBV (mL/kg)	152 ± 33	–	99 ± 16	93 ± 10	93 ± 10	78 ± 11	72 ± 11
CPV (mL/kg)	95 ± 9	–	70 ± 9	62 ± 6	60 ± 9	50 ± 8	48 ± 6
ECF volume (mL/kg)	394 ± 29	–	364 ± 53	348 ± 45	301 ± 24	287 ± 32	234 ± 22
PCV (%)	40 ± 4	35 ± 3	–	33 ± 4	34 ± 5	38 ± 3	32-52
Hb (g/dL)	8.1 ± 0.8	13.3 ± 1.2	–	13 ± 1	8.7 ± 0.5	8.4 ± 0.6	11-19
WBC ($\times 10^3$ cells/μL)	7.4 ± 2.3	6.3-13.6	–	12.1 ± 2.5	12.8 ± 2.8	12.8 ± 2.8	5.5-12.5
Neutr ($\times 10^3$ cells/μL)	5.4 ± 0.6	4.4-10.6	–	6 ± 1	7.3 ± 1.3	7.8 ± 1.5	2.7-6.7
Lymph ($\times 10^3$ cells/μL)	1.9 ± 0.6	1.4-2.3	–	3.3 ± 1	4.7 ± 1.3	6.5 ± 1.4	1.5-5.5
TP (g/dL)	5.4 ± 0.8	6.4 ± 0.6	–	6.1 ± 0.5	5.2 ± 0.3	5.4 ± 0.3	4.6-6.9
Albumin (d/dL)	3 ± 0.3	2.7 ± 0.7	–	2.4 ± 0.2	3 ± 0.2	3.3 ± 0.2	2.5-4.2
Glucose (mg/dL)	140 ± 20	162 ± 18	–	162 ± 22	119 ± 9	112 ± 11	75-115
BUN (mg/dL)	13 ± 7	4-20	–	8 ± 2	9 ± 2	16 ± 3	10-24
Creatinine (mg/dL)	1.4 ± 0.3	1.3 ± 0.2	–	1.5 ± 0.2	–	1.6 ± 0.3	0.4-1.8
$BILI_{tot}$ (mmol/L)	47 ± 16	32-56	–	25 ± 6	22 ± 6	19 ± 5	7.1-34.2
$BILI_{conj}$ (mmol/L)	2.6 ± 0.7	2-7	–	2.5 ± 0.5	2.2 ± 0.4	1.8 ± 0.8	0-6.8
CK (IU/L)	40-909	52-143	–	81-585	–	97-396	2.4-23.4
AST (IU/L)	137 ± 49	237-620	–	160 ± 19	165 ± 21	158 ± 14	226-366
SDH (IU/L)	9.4 ± 5.5	5.1 ± 5.1	–	2.6 ± 1.9	–	–	1.9-5.8
LDH (IU/L)	799 ± 264	–	–	751 ± 417	703 ± 192	539 ± 142	162-412
γ-GT (IU/L)	29 ± 27	16-98	–	18 ± 8	14 ± 3	13 ± 3	4.3-13.4
AP (IU/L)	1787 ± 893	137-1169	–	983 ± 245	871 ± 180	782 ± 118	143-395

AP, Alkaline phosphatase; *AST*, aspartate amino transferase; $BILI_{conj}$, conjugated bilirubin; $BILI_{tot}$, total bilirubin; *BUN*, blood urea nitrogen; *CBV*, circulating blood volume; *CK*, creatine kinase; *CPV*, circulating plasma volume; *ECF*, extracellular fluid volume; *γ-GT*, gamma glutamyl transpeptidase; *Hb*, hemoglobin; *LDH*, lactate dehydrogenase; *Lymph*, lymphocyte count; *Neutr*, neutrophil cell count; *PCV*, packed cell volume; *SDH*, sorbitol dehydrogenase; *TP*, total protein; *WBC*, total white blood cell count.
*Data from 7, 31, 35, 36, 43, 45 and clinical biochemistry data in adults from reference 98.

much faster than in an adult.[33] As a result, the neonate can tolerate a greater blood loss before any significant decrease in blood pressure and tissue hypoperfusion is noted.

During the first weeks of life, the mare's milk provides the primary nutrional source the growing foal needs for sustenance. Daily fluid intake (milk plus water) of foals is high, with animals aged 11 to 18 days drinking 246 g/kg and those aged 30 to 44 days consuming 202 g/kg.[34] Maintenance fluid rates in neonatal foals are variable but higher than in adults, and as much as 120 mL/kg per day is required in foals up to 1 month of age.[33]

Glycogen reserves in liver and muscle are smaller in the newborn foal than in neonates of other species and last only for a few hours, making the foal more susceptible to hypoglycemia and energy deficits if the foal does not nurse.[27] However, from 2 to 4 weeks of age onward the foal's diet changes gradually to solid food with high-quality grains and forage increasingly covering the foal's dietary requirements, and by 4 months of age the mare's milk is no longer a significant source of nutrients. Therefore, foals are weaned by 3 to 5 months of age. In parallel, the foal's body mass as a whole (especially skeletal musculature and fatty tissue) increases rapidly over the first 12 to 16 weeks, providing the animal with increasingly larger glycogen stores and rendering it less susceptible to hypoglycemia and loss of energy reserves when food is withheld or the sick animal stops eating. In addition, uptake, distribution, and elimination kinetics of anesthetic drugs change rapidly as the foal's body mass increases. Growth rates of horses are not as well defined as those of other farm animals.[35,36] Studies in Thoroughbreds revealed that in this breed body weight at birth (day 0) ranges from 39 to 67 kg and increases rapidly over the following 6 months to 237 ± 19 kg, which is approximately half that of an adult animal (see Table 20-3).[35,36] Consequently, in the maturing foal, uptake and distribution of anesthetic and other drugs are expected to approach patterns as described for the adult horse more rapidly than in many other species.

Thermoregulation

Rectal temperature of foals ranges from 37.2 to 38.6° C (99 to 101.5° F). The much higher ratio of body surface area to weight, thin skin, and scarce subcutaneous fat tissue (poor insulation) increase environmental heat loss in the neonate compared to the adult horse.[8,37] Conduction, convection, radiation, and evaporation all play a role and can expose the newborn to rapid heat loss. In addition, mature equine neonates have the ability to generate heat through shivering, but they can respond with non-shivering (cellular) thermogenesis and behavioral actions as well.[38] Anesthetic drugs and commonly used sedatives will interfere with thermoregulation and therefore promote extended periods of hypothermia.

Hepatic Function and Development

The liver is the principal site of drug metabolism. The microsomal cytochrome P450 enzyme system is primarily responsible for transformating lipophilic compounds to polar and pharmacologically less-active or inactive substances (phase I reactions), whereas glucuronidation and other conjugation processes (phase II reactions) render the metabolites more hydrophilic, facilitating renal elimination. Functional maturity

of the liver is incomplete at birth and thus the capacity to metabolize endogenous substances such as bilirubin or drugs is markedly lower in newborn foals than in the adult horse.[17,25] As a result, metabolism and half-lives of organic waste products (e.g., bilirubin) are expected to be prolonged causing higher plasma concentrations to persist in the newborn foal (see Table 20-3). Likewise drugs have longer plasma half-lives and may accumulate on repeated dosing, thereby extending effects and slowing elimination from the body.[39] As blood flow to the liver increases after birth, enzyme induction begins with exposure to various endogenous and exogenous substances. In the horse, metabolic pathways seem to mature more rapidly than in other species. In particular, microsomal enzyme activity increases rapidly during the first 3 to 4 weeks of life, while conjugation processes approach activity levels similar to those measured in the adult more gradually.[25,39] Nevertheless, by 6 to 12 weeks postpartum most hepatic metabolic pathways are completely functioning.

Renal Function and Development

In horses, renal development, in terms of glomerular number, is complete by 30 to 40 weeks of gestation, although the kidney volume continues to grow until 50 to 90 weeks of postnatal life.[21] As a result, on a per kilogram of body weight basis, glomerular filtration rate and effective renal plasma flow of the full-term newborn foal is already comparable with that of the adult.[40,41] Foals have a relatively greater renal tubular internal surface area available for reabsorption but reduced renal concentrating ability in the postpartum period as compared to adult animals. Normal urine output in neonatal foals is reported to be approximately 6 mL/kg/hr but then decreases gradually over the subsequent 12 weeks of life.[21,32] Reflecting a high water intake and urine excretion, normal urine specific gravity in newborn foals, after the first 24 hours of life, is usually hyposthenuric (1.008 or less) and is reported to range from 1.001 to 1.027.[32,42] When compared with values reported in adults, excretion, clearance, and fractional electrolyte excretions (FE) in 4-day-old foals are similar for sodium but somewhat higher for potassium, phosphorus, and calcium.[42] Blood urea nitrogen values of 2 mmol/L or less (6 mg/dL or less) are normal up to 3 months of age, whereas the mean adult value is 3.5 mmol/L or less (9.8 mg/dL or less).[42]

Hematology and Biochemistry

Blood volume in neonates is higher than in adults, approximately 13% to 15% of total body weight, and then decreases to near adult values (8% to 10%) by 12 weeks of age.[31] A detailed review on clinical pathology findings in the maturing foal can be found elswhere.[43] Key hematologic and clinical chemistry parameters recorded in foals within the first 6 months of life are listed in Table 20-3. Most noticeable, packed cell volume and hemoglobin values typically increase to maximum values at birth as a result of placental blood transfusion and then gradually decline. In horses and ponies (as in human infants) this physiologic anemia of the newborn develops during the first 2 weeks of life with PCV to remain just below or in the low adult reference range.[31,43] This decline in red blood cells (RBCs) may be further aggravated by neonatal isoerythrolysis, an alloimmune hemolytic anemia caused by antibodies in the mare's colostrum against the newborn's erythrocytes that is accompanied by neonatal icterus.[17,43] Thereafter values increase again, reaching levels of adult animals by about 1 to 3 months of age.

Although there is no fetal hemoglobin in the equine species, levels of 2,3-diphosphoglycerate (2,3-DPG) in fetal and therefore neonatal erythrocytes is slightly lower than in adults, thereby increasing the affinity of hemoglobin toward O_2 and thus facilitating O_2 loading of hemoglobin in the placenta during fetal life and in the lung of the newborn while impeding O_2 off-loading at the tissue sites.[43,44] The impact of this phenomenon, however, is soon reversed in the pediatric/juvenile foal when phosphate substrate becomes available through active bone turnover, and the short-living fetal erythrocytes are increasingly replaced by new RBCs of bone marrow origin.[31] The 2,3-DPG of RBCs of these foals increases and thus O_2 off-loading in the tissues increases. Therefore, fewer RBCs are needed to maintain adequate tissue oxygenation. Data in other species indicate that when rapid bone growth slows, this trend reverts back to normal adult values.

In contrast, total white blood cell count of a foal increases gradually after birth, attributable to an increase in neutrophils, but considerable inter-individual variability exists with regard to total neutrophil numbers.[43] Lymphocyte numbers decrease immediately after birth to resume adult levels by about 3 months of age.[17,43] It is important to recognize that lymphocyte counts per se are not diagnostic in the neonate as numbers may be normal or reduced in foals with immunodeficiency.[43]

At birth, total plasma protein content may vary widely and then increase following colostrum intake, but a wide range persists, although the albumin concentration remains relatively constant.[17,43,45] Marked hyperbilirubinemia in the first week of life is a common finding and can be attributed to an accelerated breakdown of neonatal erythrocytes and immature hepatic function.[43] Serum enzyme activities (including creatinine kinase, sorbitol dehydrogenase, γ-glutamyl transferase, lactate dehydrogenase, and aspartate aminotransferase) have been reported to be transiently elevated in the first few weeks after birth as a result of hepatocellular maturation (see Table 20-3).[17,45] Serum lactate concentrations are high immediately after birth (3 to 5 mmol/L), likely because of temporary tissue hypoperfusion and hypoxia, but then decrease soon to normal values (2 mmol/L or less).[43]

ANESTHETIC MANAGEMENT OF THE SYSTEMICALLY HEALTHY NEONATE AND MATURING FOAL

Since the 1990s the scope of anesthetic management in adult horses and foals has expanded to accommodate new developments in equine surgery (e.g., laser surgery in the airway, laparoscopy, thoracoscopy), medicine, and diagnostic imaging (e.g., computed tomography, magnetic resonance imaging [MRI]). Today protocols for continuous deep sedation, loco-regional anesthesia, and analgesia are techniques that in combination may replace or complement general anesthesia. Methods of general anesthesia in the foal have been described previously.[1,3,7,8,37,46] The rapid maturing of the foal postpartum with all its implications described earlier and both clinical and experimental observations suggest that sedation, anesthetic, and analgesic protocols must be tailored to the needs of the individual foal, such as its developmental stage and health status. For clinical purposes, it is meaningful to distinguish anesthetic management of the neonate foal (up to 1 month of age) from that of the pediatric or juvenile foal (1 to 4 months old).

Weanlings (more than 4 to 5 months old) usually have adult circulatory and respiratory reserves and mature metabolic pathways, and they do not require specific precautions. Therefore, they can be treated anesthetically as adult horses.

Preanesthetic Examination and Preparation

A thorough history and physical examination of the foal in the presence of the mare, involving assessment of mental status and temperament, cardiopulmonary functions (heart rate and rhythm, pulse pressure, capillary refill time, mucous membrane color and moisture, respiratory rate and rhythm), hydration status, and body temperature are essential before any suitable protocol for sedation, anesthesia, and/or analgesia can be formulated. The need for ancillary tests (e.g., chest radiographs, ultrasound, electrocardiogram) and laboratory analyses (e.g., complete blood cell count, clinical chemistry profile, blood gas analysis, urinalysis) is largely dependent on the physical status of the foal, the presenting complaint, and the intended surgical or diagnostic procedure and should take into account age-dependent differences in vital, hematologic, and biochemical parameters between foals and adult horses (see Tables 20-1 and 20-2). As a minimum, packed cell volume, white blood cell count and differential, total plasma protein content, and blood urea and glucose concentrations should be determined in any foal undergoing prolonged sedation or general anesthesia. If the foal is a newborn, the assessment should include a detailed history of the perinatal period and a test of the adequacy of passive antibody transfer; if the foal is more mature, a complete medical history may be all that is necessary.[17]

Nursing foals up to 2 months of age have little fiber intake and should not be muzzled prior to anesthesia but should have free access to their mother. Suckling helps maintain adequate blood glucose levels, liver glycogen reserves, and hydration status. Older foals with increased solid food intake may be muzzled and held off feed for 3 to 6 hours prior to anesthesia. These older foals, particularly when hypovolemic, may profit from antiulcer medication (ranitidine [Zantac] 1.5 mg/kg IV every 8 hr, famotidine [Pepcid AC] 0.3 mg/kg IV every 12 h, omeprazol [GastroGard] 2 to 4 mg/kg PO every 24 h).[47] In foals of any age, the mouth should be rinsed out with water close to the time of induction of anesthesia to prevent feed or bedding material that may be present in the pharynx from being pushed into the airway during the process of endotracheal intubation.

In preparation for long-term sedation or general anesthesia and to ensure safe fluid and/or drug administration, a 16-gauge (18-gauge in minihorse or small pony foals) jugular venous catheter should be placed in the equine neonate using aseptic technique. Catheter placement is facilitated by infiltration of the subcutaneous tissue with local anesthetic (e.g., 2% lidocaine [Lidocaine HCl USP]) at the site of skin and blood vessel puncture. In the healthy neonate, mild sedation (Table 20-4) may be nessary to facilitate aseptic placement of an IV catheter. If anesthesia is being induced using an inhalant anesthetic technique, IV catheter placement may be postponed to the moment following induction of anesthesia. If antibody titers indicate inadequacy of passive immune transfer, the neonate should receive either colostrum or plasma, as appropriate, and antibiotics because newborns are highly susceptible to serious infections when stressed by injury, metabolic disease, anesthesia, or surgery.

Sedation of the Mare

In most instances, it is desirable to have the mare present when handling an awake or mildly sedated foal because separation from the mother may trigger anxiety, excitement, and stress. To facilitate preparation of the foal for anesthesia, sedation of the mare is highly desirable because it prevents her from becoming agitated or even aggressive toward personnel handling the foal.

A physical examination of the mare should precede any administration of sedatives or tranquilizers. Ideally the mare should be tranquilized while still in the stall with her foal. Sedative agents or a combination of drugs with relatively long duration of action are preferred. Depending on the temperament of the mare and the anticipated length of separation of mother from foal, acepromazine alone (PromAce, 0.02 to 0.05 mg/kg IV/IM) or in combination with α_2-adrenoceptor agonists (xylazine [Rompun] 0.2 to 0.3 mg/kg IV, detomidine [Domosedan] 5 to 10 µg/kg IV/IM, or romifidine [Sedivet] 0.02 to 0.04 mg/kg IV/IM) will provide adequate and long-lasting sedation.[3,7,8]

Anesthetic Management of the Neonate (1-Month-Old or Younger)

The immaturity of its central nervous, cardiopulmonary, hepatic, renal, and metabolic systems described earlier in this chapter must be kept in mind when designing the anesthetic plan for a neonate so as not to expose the foal to an increased risk of perianesthetic complications. Sedation and anesthetic drug regimens that are the least likely to impair vital functions and to cause prolonged central nervous depression are preferred.

Sedation

Foals up to 14 to 21 days of age usually do not require any chemical restraint or tranquilization to be handled and instrumented prior to induction of general anesthesia or locoregional anesthesia for brief and less-invasive surgical or diagnostic procedures.[2,3,7,8,37] If, however, sedation is required or the animal is older than 2 to 3 weeks, a benzodiazepine derivative is the preferred choice because it has limited adverse cardiopulmonary effects.[3,7,37] All benzodiazepines listed in Table 20-4 provide sufficient sedation and muscle relaxation, thereby facilitating minor interventions such as radiographic or ultrasonographic examinations, cast application and changes, synovial or cerebrospinal fluid aspiration, rhinolaryngoscopy, intravenous catheterization, or short surgical procedures under local anesthesia or induction of general anesthesia.[2,3,7,8,37] If infusion or repeated drug dosing is anticipated to maintain sedation, midazolam (Versed) may be the better choice because the propylene glycol vehicle in other benzodiazepine preparations (diazepam [Valium], lorazepam [Ativan], climazolam [Climaxolam]) can cause metabolic acidosis and nephrotoxicity.[48] In the more mature neonate (older than 2 to 3 weeks), benzodiazepines may be supplemented with one of the opioids listed in Table 20-4 and/or a low dose of xylazine (0.05 to 0.1 mg/kg) to enhance sedation and provide some analgesia.[3,8,37] If desired, the benzodiazepine effect can be countered at the end of the procedure using flumazenil (Romazicon; 0.025 to 0.1 mg/kg IV) or sarmazenil (Sarmasol; 0.025 to 0.1 mg/kg IV). The opioid can be antagonized with with naloxone (10 to 15 µg/kg IV) or levallorphan (Lorfan; 22 µg/kg IV),[49,50] and

TABLE 20-4. Anesthetic Management of the Systemically Healthy Foal

	Neonate (1 Month or Younger)	Pediatric/Juvenile Foal (1-4 Months)
Sedation (IV)	None (≤2-3 wk) Benzodiazepines (≥2-3 wk) Midazolam 0.05-0.1 mg/kg Diazepam 0.1-0.25 mg/kg Lorazepam 0.02-0.05 mg/kg Climazolam 0.1-0.2 mg/kg α_2-Agonists (not preferred) Xylazine 0.2-0.5 mg/kg Supplementation with: Morphine 0.03-0.06 mg/kg L-Methadone 0.05 mg/kg Butorphanol 0.05-0.1 mg/kg	Benzodiazepines (4-8 wk) Midazolam 0.05-0.1 mg/kg Diazepam 0.1-0.25 mg/kg Lorazepam 0.02-0.05 mg/kg Climazolam 0.1-0.2 mg/kg α_2-Agonists (>8 wk) Xylazine 0.2-0.5 mg/kg Detomidine 0.005-0.01 mg/kg Romifidine 0.02-0.04 mg/kg Phenothiazines Acepromazine 0.03-0.05 mg/kg Supplementation with: Morphine 0.03-0.06 mg/kg L-Methadone 0.05 mg/kg Butorphanol 0.02-0.1 mg/kg
Induction of anesthesia	Pre-oxygenation (2.5-5 L/min) via mask or nasotracheal tube Inhalant anesthetic in O_2 Isoflurane Sevoflurane Desflurane Injectable anesthetics (after sedation) Ketamine 2-2.5 mg/kg Propofol 2-2.5 mg/kg	Injectable anesthetics (in combination with benzodiazepine listed above or guaifenesin 20-50 mg/kg IV) Ketamine 2-2.5 mg/kg Propofol 1-3 mg/kg Ketamine 1.5 mg/kg + propofol 0.5 mg/kg Thiopental 4-6 mg/kg
Maintenance of anesthesia	Inhalant anesthetic in O_2 Isoflurane Sevoflurane Desflurane Total intravenous anesthesia (TIVA) Propofol 0.2-0.4 mg/kg/min	Inhalant anesthetic in gas mixture (FiO_2 > 0.3) Isoflurane Sevoflurane Desflurane Supplementation* with Lidocaine CRI Ketamine + propofol CRI Dexmedetomidine CRI Total intravenous anesthesia (TIVA)* Triple drip CRI Propofol 0.1-0.3 mg/kg/min

CRI, Constant rate infusion; *FiO_2*, inspired fraction of oxygen.
*See text for more details.

xylazine can be reversed with yohimbine (Yocon; 0.1 to 0.2 mg/kg IM).

Induction and Maintenance of Anesthesia

Induction and maintenance of general anesthesia can be achieved with one of the currently approved volatile anesthetics (isoflurane [Isoflo], sevoflurane [Sevoflo], or desflurane [Suprane] in O_2) or an injectable agent such as ketamine (Ketaset) or propofol (Propoflo) (see Table 20-4). Use of only a volatile anesthetic offers several advantages in neonates: (1) rapid uptake and elimination of the anesthetic via the lungs aided by the usually high minute ventilation and CO; (2) easy and rapid adjustment of anesthetic depth if untoward cardiovascular or respiratory depression or arrhythmias occur; (3) elimination of the anesthetic independent of hepatic and renal function. While the previous multicenter study[4] indicated a 4.5 times higher risk of perioperative mortality in neonatal foals that had received an inhalant anesthetic (halothane) versus ketamine for induction of anesthesia, this finding does not coincide with a clinical investigation of the safety of two inhalant anesthetics (halothane and isoflurane) for induction and maintenance of anesthesia in foals.[51] Also personal experiences do not corroborate a higher risk associated with using inhalant anesthesia in foals. Of the 153 neonatal foals anesthetized over the past 10-year period approximately 43% received one of the inhalant anesthetics (predominantly isoflurane) for induction and 57% an injectable anesthetic, yet only one animal with a perforated esophagus in which anesthesia was induced with ketamine suffered a fatal outcome because of an airway obstruction in the recovery period.

Considering the high O_2 consumption and predisposition of neonates to develop hypoxemia when being deeply sedated or anesthetized, it is recommended to have them breathe O_2 or at least an O_2-enriched gas mixture (FiO_2 0.5 or more), independent of technique used. (It is important to administer O_2-enriched gas because the risk of rapid desaturation is fairly high and an FiO_2 more than 0.5 buys valuable time). Circle

rebreathing systems and anesthesia equipment designed for use in humans or small animals are well suited for equine neonates. Dependent on the age and hence size of the animal, 3- to 5-L rebreathing bags or bellows are sufficient. Preoxygenation is recommended in all animals being anesthetized. For this purpose, the foal is intubated using a lidocaine gel (Xylocaine Jelly 2%) lubricated 6- to 9-mm ID cuffed silicone rubber nasotracheal tube of sufficient length (30 to 55 cm [12 to 22 inches]) that is passed through the nostril and ventral nasal meatus into the trachea. Subsequently, with the cuff inflated or alternatively using a tight-fitting mask, O_2 or an O_2-enriched gas mixture is delivered at 40 to 60 mL/kg/min for 3 to 5 minutes before anesthesia is induced.

For induction of anesthesia the volatile agent, like O_2 alone during preoxygenation, can be delivered via the rebreathing circuit using the previously placed nasotracheal tube or face mask. Preanesthetic sedation in neonates is often not necessary. Following preoxygenation the vaporizer output is incrementally increased up to the maximum output setting. At a fresh gas flow rate of 2.5 to 5 L/min, the volatile anesthetic concentration rises rapidly in the breathing circuit with the onset of anesthesia typically occurring within 3 to 8 minutes.[51] Sevoflurane and desflurane are characterized by a 50% and 64% lower blood solubility than isoflurane, respectively, and therefore induction is somewhat faster with these agents than with isoflurane.[52] If anesthesia is induced by mask, the foal should be orotracheally intubated as soon as it has lost consciousness and the swallow reflex. For this purpose, the fresh gas flowmeter shall be momentarily turned off and a lubricated 8- to 14-mm ID cuffed canine endotracheal tube be used. When the foal is intubated, the delivered inhalant concentration can be decreased and carefully adjusted to the individual foal's requirements and the fresh gas flow rate can be reduced to a setting of 4 to 10 mL/kg/min to avoid unnecessary and costly waste of anesthetic gas.

Among the injectable anesthetics, ketamine is currently the most commonly used agent for induction of anesthesia in the equine neonate, typically following sedation with a benzodiazepine derivative alone or in combination with an opioid and/or low-dose xylazine. It will induce anesthesia lasting 10 to 20 minutes (see Table 20-4).[3,8,37] Alternatively, either with or without benzodiazepine sedation, propofol may be administered slowly (over 45 to 60 seconds) to effect (to avoid severe respiratory depression and apnea).[3,8,37,53,54] Induction of anesthesia with thiopental or other barbiturates should be avoided in neonates because of the prolonged recovery period.

In most neonates, anesthesia is maintained with one of the volatile agents to avoid drug accumulation and slow awakening from anesthesia if injectable agents (ketamine or barbiturates) are being infused or repeatedly administered. For isoflurane, an average anesthetic vaporizer concentration setting of 2.8 ± 0.1% has been reported,[51] which accords well with my own observations of an average dial setting of 2.2 ± 0.7% and end-tidal isoflurane concentration of 1.5 ± 0.4% recorded in 152 neonate anesthetics. In foals undergoing major trauma surgery, a balanced anesthesia regimen involving intermittent (every 1 to 2 hours) subcutaneous administration of a low dose of medetomidine (Dormitor, 1 to 2 µg/kg) or dexmedetomidine (Dexdormitor, 0.5 to 1 µg/kg) has advantages over maintaining anesthesia only with an inhalant anesthetic. While providing analgesia, these drugs reduce the need for high doses of volatile anesthetic that typically cause severe hypotension. Recovery (commonly assisted) to standing position usually occurs quickly, within 15 ± 1 minutes after 86 ± 4 minutes of isoflurane anesthesia[51] and within 27 ± 18 minutes after 133 ± 66 minutes of isoflurane anesthesia. If higher doses of a benzodiazepine or xylazine have been used for preanesthetic sedation or if anesthesia was relatively short, reversal of the premedication agents with appropriate antagonists (e.g., flumazenil, yohimbine) should be considered to speed up recovery.

Using propofol in the neonate allows maintenance of anesthesia without risk of untoward drug accumulation and prolonged recovery. It facilitates safe anesthesia administration over extended periods of time when inhalant anesthesia may not be a feasible option, for example, for MR imaging when compatible anesthesia equipment is not available. An anesthetic technique considered suitable under those circumstances in neonates (3 to 6 days of age) includes xylazine (0.5 mg/kg IV) premedication followed 5 minutes later by a bolus (2 to 2.5 mg/kg IV) and subsequent infusion of propofol (0.2 to 0.4 mg/kg/min).[53,54] However, the hemodynamic effects of α_2-agonists at such high doses cannot be ignored in this age group. A study of xylazine sedation in healthy 10- and 28-day-old foals indicated a decrease in heart rate by 20% to 30%, yet without causing second-degree atrioventricular block that is typically seen in adult horses.[55] In addition, a biphasic (initial increase followed by a decrease) change in blood pressure, similar to that in adult horses, occurred, but mean arterial pressure did not fall below 60 mm Hg.[55] Therefore, one should still exercise caution when using α_2-agonists in the very young or sick neonate and keep doses at a minimum. In one study, recovery time after constant rate infusion of propofol (0.30 ± 0.07 mg/kg/min) for 60 to 122 minutes ranged from 15 to 32 min (mean, 27 min), and foals suckled within 10 minutes of standing.[54]

Anesthetic Management of the Pediatric/Juvenile Foal (1 to 4 Months Old)

Beyond 1 month of age, the normally developing foal of common breeds (i.e., Thoroughbreds, Standardbreds, Arabians, Quarter Horses, Warmblood horses, and Paint horses) has arrived at a stage of maturation when anesthetic techniques used in the adult can be applied with some modifications. In parallel with the maturation process, the risk for fatal perianesthetic complications seems to decrease markedly.[4]

Sedation

Systemically healthy foals 4 to 8 weeks of age (more than 120 to 150 kg body weight) or older are more difficult to physically restrain and therefore frequently require adequate tranquilization for preanesthetic catheter placement or other minor procedures. In younger pediatric foals, sedation with one of the benzodiazepine derivatives listed in Table 20-4 again offers the advantage of little adverse cardiovascular and respiratory effects yet profound calming and immobilization.[3,7,37]

In fractious individuals or foals older than 2 months, benzodiazepine administration often causes inadequate sedation and muscle relaxation or even excitement similar to what is described in the adult horse.[49] In these foals α_2-adrenoceptor agonists such as xylazine (0.2 to 0.5 mg/kg IV, 0.5 to 1 mg/kg IM), which is the most widely used drug in this group, as well as detomidine or romifidine, provide more reliable sedation and muscle relaxation, and in addition

profound analgesia (see Table 20-4 for dosages).[2,3,7,8,37] Overall, hemodynamic and respiratory side effects observed after α_2-agonist administration in foals up to 2 to 3 months old are similar to those noted in adults with maybe the exception of atrioventricular blocks occuring rarely in younger foals.[3,7,37,55,56] Of note, xylazine has been shown to cause hypothermia in foals.[57] Unlike in adult horses, α_2-agonists do not seem to produce hypoinsulinemia and hyperglycemia in 4-week old foals, indicating differences in pancreatic responses to α_2-agonists in early life and further emphasizing the need to monitor blood glucose levels during prolonged sedation and anesthesia.[57] Lower dosages of xylazine (0.2 to 0.3 mg/kg IV) usually provide adequate sedation for 15 to 30 minutes and are associated with minimal cardiovascular and respiratory changes,[7] making this drug the agent of choice for use in foals of that age group. In contrast, detomidine and romifidine have a longer duration of action and also carry a higher risk for untoward effects, including arrhythmias.[3,56] When combined with one of the opioids listed in Table 20-4, either the foal will lie down or it can be placed into lateral recumbency, allowing performance of minor surgical (in combination with local and regional anesthesia) or diagnostic procedures of short duration.[2,7] If desired or necessary, the α_2-agonistic effects can be antagonized at the end of the procedure using atipamezol (Antisedan, 0.05 to 0.1 mg/kg IV/IM) or yohimbine (0.1 to 0.2 mg/kg IV/IM).[49]

Acepromazine in clinically common dosages produces overall mild but long-lasting sedation.[3,37] It may be used to enhance and prolong sedation with xylazine. Clinically relevant hypotension secondary to vasodilation is a rare observation in normovolemic foals and therefore is not a concern.[3]

Induction and Maintenance of Anesthesia

Liver and kidney functions are significantly more mature in foals older than 1 month of age, and physical restraint becomes increasingly more difficult as the foal matures. Therefore, an IV technique is often considered the preferred method of induction of anesthesia (see Table 20-4). Ketamine is currently the most commonly used agent for induction of anesthesia in pediatric and juvenile foals and, to obtain good muscle relaxation, it is commonly combined with a benzodiazepine, unless this type of drug had been already administered for purposes of sedation. Alternatively and preferably in foals older than 3 to 4 months of age, ketamine may be coadministered with the centrally acting muscle relaxant guaifenesin (5%; Gecolate), which is administered IV to effect (dropping of head, general muscle relaxation and calmness, fetlock knuckling) at a rate of 2 to 3 mL/kg/min.[3,7,37] To avoid inadvertent guaifenesin toxicity, the infusion container (bag, syringe, or bottle) should only contain up to the calculated maximum dosage for the individual foal, about 50 mg/kg. Following xylazine administration (0.25 mg/kg), ketamine in combination with diazepam produces anesthesia in 4- to 6-week-old foals typically of 10 minutes' duration.[7] Ketamine may be replaced by propofol (see Table 20-4) for induction of anesthesia, but respiratory depression is likely to occur even in the more mature foal, and anesthesia may last only 5 minutes.[3,8,37,54] Alternatively, ketamine and propofol may be combined for induction of anesthesia (see Table 20-4).[37] Thiopental in conjunction with a benzodiazepine or guaifenesin is suitable for induction of anesthesia in the more mature foal and under certain circumstances (e.g., foals with seizures or brain trauma) it is the preferred technique (see Table 20-4).[7,37]

As in neonates, general anesthesia is most commonly maintained with isoflurane (MAC in adults, 1.3% to 1.6%)[52] in O_2 or an O_2-enriched gas mixture in pediatric and juvenile foals[51] although sevoflurane (MAC in adults, 2.3% to 2.8%)[52] or desflurane (MAC in adults, 7.0% to 8.1%)[52] may be used as well. Circle rebreathing systems and anesthesia equipment designed for use in humans or small animals are not suitable for foals when they reach a body weight in excess of 125 to 150 kg. Hence, a large animal anesthesia machine equipped with a 3- to 10-L rebreathing bag (accommodating the threefold increase of an average tidal volume to 10 to 15 mL/kg) is required to administer inhalation anesthesia in older foals.[8,37,51] Following induction with injectable agents, the foal is intubated using a 14 to 18 (70 to 100 kg body weight), 18 to 22 (150 to 200 kg body weight), or 22 to 24 (250 to 400 kg body weight) mm ID cuffed orotracheal tube of sufficient length. Initially, a high fresh gas flow rate of 3 to 5 L/min and vaporizer dial setting of 3% to 5% for isoflurane, 4% to 6% for sevoflurane, and 9% to 10% for desflurane ensures rapid rise of the volatile anesthetic concentration in the breathing circuit and airways of the foal. Within 5 to 15 minutes the dial setting should be carefully adjusted to maintain the required depth of anesthesia and the fresh gas flow rate should be reduced to a lower setting of 4 to 6 mL/kg/min. The foal's body size, previously administered anesthetic drugs, and blood gas solubility of the volatile anesthetic agent used must be considered when adjusting the maintenance dose.

As in adult horses, balanced anesthesia protocols in which an inhalant anesthetic is combined with administration of short-acting analgesic agents and/or injectable anesthetics also have become popular in foals.[37,46] Infusions of lidocaine (50 µg/kg/min after an IV bolus of 1.2 to 1.5 mg/kg)[46] or ketamine (50 µg/kg/min for 45 minutes followed by 25 µg/kg/min) plus propofol (0.1 mg/kg/min)[37] have been used in an attempt to reduce the required dose of the volatile anesthetic and provide better analgesia. In the older foal, combination with an α_2-agonist, such as dexmedetomidine (0.75 to 1.5 µg/kg/hr or 1 to 3 µg/kg IM q 1 to 2 h), also may be considered for painful orthopedic or trauma surgery.

Total intravenous anesthesia techniques have been applied in foals, whereby anesthesia is maintained by constant rate infusion of injectable anesthetics to effect (see Table 20-4). Infusion of 5% guaifenesin solution containing xylazine (250 mg/L) and ketamine (1 g/L), often referred to as *triple drip solution*, is applied most frequently.[7,8,37] This drug combination may also be administered after sedation of the foal at a rate sufficient to rapidly induce anesthesia. For maintenance of anesthesia, the drip can be continued at a rate of 2 to 3 mL/kg/hr. As described for neonates, anesthesia may also be maintained with propofol.[7,8,37,54] Lacking any analgesic properties, propofol administration may be combined with techniques of local and regional anesthesia or infusions of lidocaine, ketamine, or dexmedetomidine as mentioned for use with inhalant anesthesia (see Table 20-4).

Monitoring during Anesthesia

Anesthetic emergencies are typically of short duration and can have dire consequences, but in most instances, warning signs precede fatal developments and if recognized and responded to

in a timely and appropriate manner, emergency situations can be avoided altogether. This applies particularly to the neonate and maturing foal with its limited physiological reserves. Therefore, with the exception of very minor procedures, invasive diagnostic or therapeutic procedures requiring anesthesia of longer duration should only be performed in foals in specialty practices and hospitals that are appropriately equipped and have personnel familiar with foal anesthesia.

Guidelines for anesthesia in horses have been published previously by the American College of Veterinary Anesthesiologists[58] and more recently revised[59] and commented on.[60] Guidelines also have been issued by the equine practioner's association in Germany and recently discussed by the Association of Veterinary Anaesthetists. These guidelines state that a veterinarian or veterinary support staff familiar with foal anesthesia shall monitor the animal at all times during the anesthetic and recovery period and be prepared to intervene, when indicated. All drugs administered (including time, route, and dosage) and monitored variables are to be recorded every 5 to 10 minutes in an anesthesia record. It is important that monitored variables such as anesthetic depth, cardiovascular and respiratory functions, body temperature, hydration, and metabolic and hematological parameters be interpreted as interdependent variables taking into account the developmental stage of the foal and related normal physiology, the disease status of the patient, and the procedure being performed, before decisions to intervene are made. Monitoring techniques used in foals are principally not different from those applied in adult horses. The reader may refer to texts providing a more comprehensive review of monitoring in the anesthetized horse[61,62] and critically ill neonate.[63,64]

Anesthetic Depth and Drug Concentration

Although it is not always obvious, anesthesia is not a static situation but a rather dynamic process. Inadequate depth of anesthesia may be associated with periods of awakening or subconscious responses to noxious, auditory, mechanical and other stimuli, resulting in excessive stress, movement with potential contamination of the surgical site, and injury to the animal.[62] Excessively deep anesthesia is associated with severe depression of the brain stem and hence dangerous impairment of many vital functions, including those of the cardiovascular and respiratory systems. It is therefore essential that the anesthetist repeatedly assess the depth of anesthesia and, if needed, adjust anesthetic drug administration.

Physical signs such as position of the globe, nystagmus, degree of depression of protective eye reflexes (palpebral and corneal), presence or loss of swallowing reflex, rate and depth of breathing, lacrimation, skeletal muscle shivering/trembling or tightening, anal sphincter reflex, and hemodynamic responses to noxious stimulation are commonly evaluated.[62] Multiparameter anesthesia monitors with built-in multigas analyzer modules have become increasingly more affordable and are enjoying great popularity. They allow measurement of the end-tidal concentration or partial pressure of an inhaled anesthetic, which serves as an index of the partial pressure of the inhalant anesthetic in the alveoli and brain. Knowing the concentration or partial pressure of the inhalant anesthetic agent at every moment, the anesthetist has an additional tool at hand to better predict anesthetic depth and adjust anesthetic drug administration.

Cardiovascular System

Routine monitoring of hemodynamic function must include assessment of rhythm and rate of heart beats and arterial pulses, peripheral blood perfusion, and arterial blood pressure. Simple techniques such as cardiac auscultation, digital pulse palpation, and evaluation of capillary refill time and mucous membrane color may be adequate for foals subject to sedation only or brief periods of anesthesia (30 minutes or less). For longer-duration anesthetic periods and those involving inhalation anesthesia; however, more sophisticated monitoring techniques should be applied. Those entail continuous recording and display of the electrocardiogram (ECG) and measurement of arterial blood pressures, using either a noninvasive [ultrasonic Doppler or an oscillometric (Dynamap or Cardell)] or invasive but more accurate method involving catheterization of a peripheral artery (e.g., facial, transverse facial, auricular, or metatarsal artery). For oscillometric measurements, a cuff that is placed around the base of the tail with a bladder width–to–tail girth ratio of 1 : 2 to 1 : 3 provides the best measurements.[65] For invasive blood pressure recordings, the intraarterial catheter is connected via heparinized-saline–filled tubing to a sphygmomanometer or, more commonly, a pressure transducer, which then transmits the signal to an amplifier unit and monitor for continuous display of the pulse waveform and readout of systolic, mean, and diastolic pressures. The use of pulse oximeters with plethysmographic trace display assists in the assessment of peripheral perfusion and hence overall hemodynamic status of the foal.

In the critically ill foal, additional hemodynamic monitoring, such as central venous pressure, urine output, and cardiac output recordings, may become necessary or at least offer advantages in assessing the seriousness of cardiovascular function impairment and providing guidance for volume replacement and inotrope and vasopressor treatment aimed at optimizing systemic organ perfusion.[63,64] Various minimally invasive techniques have been developed in recent years and found to be appropriate for assessing cardiac output in foals under clinical conditions. One of these is the lithium dilution (LidCO) technique,[66] a noninvasive cardiac output technique (NICO) based on the Fick principle and partial rebreathing of CO_2,[67] and another is an ultrasound velocity dilution technique (UDCO).[14]

Respiratory System

Considering the predisposition of the newborn foal to develop respiratory depression after anesthetic drug administration with subsequent hypoxemia and hypercarbia, routine monitoring of respiratory function under anesthesia is warranted and must include at least observations of respiratory rate and rhythm and mucous membrane color. For anesthestic periods in excess of 30 minutes, involving inhalant anesthetics, performed in a hospital setting, or in animals not breathing gas mixtures high in O_2 concentration, continous monitoring of arterial hemoglobin oxygen saturation (SpO_2) by pulse oximetry is highly recommended. This is a noninvasive technique and involves placing a clip-type probe on the ear, tongue, or nonpigmented skin or mucosa. Although generally of great clinical value, device-dependent technology and software algorithms, transducer type, placement site, and tissue perfusion at the site of recording markedly influence the accuracy of a pulse oximeter readout. Therefore, pulse oximetry should be supplemented by

intermittent arterial blood gas analysis whenever the adequacy of respiratory function is a concern.

Capnometry, which is the breath-to-breath measurement of the end-tidal CO_2 tension ($ETCO_2$) at the end of expiration, provides a simple, noninvasive method for assessing ventilation. The usefulness of $ETCO_2$ measurements in isoflurane-anesthetized foals has been documented.[68] The $PaCO_2$ changes proportionately with metabolic activity and hence production of CO_2 in the body and inversely with its elimination (i.e., alveolar ventilation). Under physiologic conditions $ETCO_2$ changes with alveolar PCO_2 ($PACO_2$) and therefore with $PaCO_2$, with some predictable inaccuracy. An $ETCO_2$ value in excess of 45 mm Hg (hypercapnia) indicates hypoventilation, a value below 35 mm Hg (hypocapnia) indicates hyperventilation. In addition, changes in $ETCO_2$, whether sudden or gradual, may reflect changes in circulatory function (CO) as blood transports CO_2 from the periphery to the lungs. Most modern capnometers are equipped with screens that continuously display the $ETCO_2$ concentration over time (capnography), which provides the anesthetist with a valuable tool to recognize anesthetic equipment malfunction and gas flow changes in the airways, such as leaks in the circuit system, kinked endotracheal tubes, airway obstruction, exhausted carbon dioxide absorbent, and incompetent one-way valves.

Arterial blood gas analysis is the most accurate technique for evaluation of pulmonary gas exchange and, depending on equipment, allows direct assessment of arterial O_2 saturation (measured SaO_2), acid-base, and electrolyte status. With the development of portable, cartridge-based analyzers (e.g., Ometech OPTI CCA-TS blood gas analyzer; IDEXX VetStat, i-STAT System) blood gas analysis has become affordable in private practice.

Blood Glucose

Limited glycogen reserves in the neonate make it susceptible to the development of hypoglycemia during prolonged anesthesia (more than 1 to 1.5 hours). Blood glucose levels below 40 mg/dL may produce deleterious central nervous effects such as seizure activity, cerebral depression, and even permanent neuronal damage,[46] all of which are difficult to detect under general anesthesia. Therefore, blood glucose concentrations should be determined on a regular basis throughout a long-term anesthesia by using either bedside glucometers in the operating room or multiparameter laboratory analyzers.

Body Temperature

Foals, more than adult animals, are susceptible to heat loss because of their high ratio of surface area to body weight, lack of subcutaneous fat depots, and compromised thermoregulation under anesthesia.[7,8,37] For this reason, pharyngeal, esophageal, or rectal temperature should be continuously monitored or intermittently measured using either standard thermometers or electronic temperature probes that are connected to a multiparameter monitor for continuous recording.

Fluid Management in the Perianesthetic Period

In foals, use of balanced electrolyte solutions with normal strong ion difference (e.g., lactated Ringer, Plasmalyte, Normosol-R) has been recommended to avoid the acidifying effect associated with fluids without strong ion difference such as physiologic saline solution or 5% dextrose in water.[33] In the systemically healthy, normovolemic foal undergoing anesthesia, an infusion rate of 7.5 to 10 mL/kg/hr has been reported as adequate to maintain an appropriate circulatory volume.[3,7,8] Relative hypovolemia caused by anesthetic drug–induced vasodilation, use of high fresh gas flows causing a greater than normal respiratory loss, evaporative losses, and intraoperative hemorrhage may temporarily justify further increasing the IV fluid rate by up to five times the maintenance rate of 1.5 to 2 mL/kg/hr reported for foals, to a total of 20 mL/kg/hr.[32,33] However, the described differences between the neonate and adult in the response to isotonic fluid loading warrant a judicious approach to prolonged high-volume infusion of crystalloids in the neonate.[33] In adults, 20% to 50% (depending, in part, on state of hypovolemia and dehydration) of an isotonic fluid load is retained in the intravascular space 30 to 60 minutes after infusion, but this is much lower in the neonate, where fluid rapidly accumulates in the interstitial space and escapes regulatory mechanisms of fluid homeostasis. As a result, neonates retain infused fluids for a long time and do not handle large fluid loads well.[33] The situation is further complicated by a decrease in urine output under anesthesia.[69]

Dehydration and absolute hypovolemia as a result of persistent diarrhea, sepsis, septic shock, or acute hemorrhage require immediate intravenous fluid administration.[32,33,70] The previously mentioned balanced electrolyte solutions are adequate fluids whenever volume deficiencies are caused by insensible and isotonic fluid losses and may be administered in volumes of up to 50 to 80 mL/kg, typically given one third at a time followed by reassessment of the foal's volume status.[32,70] They provide rapid extracellular (intravascular and interstitial) rehydration.

Colloids, including synthetic solutions (e.g., dextrans, hetastarch, pentastarch) and plasma, may be required if the total plasma protein or albumin concentrations are low and may be used to supplement crystalloid fluid therapy.[32,70] Hetastarch in doses of 3 mL/kg at a rate of 10 mL/kg/hr may supplement crystalloid fluid therapy under those circumstances for rapid volume support.[64] Alternatively, hetastarch may be administered slowly (0.5 to 1.0 mL/kg/hr) up to a dose of 10 mL/kg/day for treatment in hypooncotic animals.[64]

Oxygen Supplementation and Mechanical Ventilation

Independent of sedation or anesthetic protocol used, supplemental oxygen therapy is principally indicated whenever the SaO_2 or SPO_2 value in a foal decreases below 90% and the PaO_2 value below 60 mm Hg.[44] Oxygen may be delivered via face mask, nasal cannula, or a nasotracheal tube and at a rate of 5 to 10 L/min. Preferably, O_2 shall be humidified by means of a bubble humidifier to minimize nasal and tracheal mucosal irritation and avoidable water losses in the foal, especially if administered over several hours.

In the foal, multiple factors may contribute to severe respiratory depression and impairment of pulmonary gas exchange leading to poor arterial oxygenation and CO_2 retention: persistent pulmonary hypertension, drug-induced central respiratory center depression, reduced FRC, exhaustion of respiratory muscles from increased work of breathing, immature lung, lung disease, and airway obstruction.[44] Therefore, especially in young

foals, ventilatory support is commonly required during general anesthesia as soon as respiratory minute ventilation decreases below 150 to 200 mL/kg/min, significant hypercarbia starts to develop and arterial oxygenation decreases. In spontaneously breathing foals under anesthesia the $PaCO_2$ rises commonly to values in excess of 80 mm Hg. Ventilators designed for use in humans or small animals are suitable for foals up to a body weight of 120 to 150 kg; for larger foals, large-animal ventilators are needed. Among all different modes of ventilation, controlled mandatory ventilation (CMV) is the mode most commonly used during equine anesthesia in general, even in foals. With CMV mode, the ventilator delivers breaths at a preset interval, regardless of any ventilatory effort made by the animal.[44] Typical settings to begin mechanical ventilation in the neonatal foal are a tidal volume of 6 to 10 mL/kg; a rate of 20 to 30 per minute; a peak flow of 60 to 90 mL/min; I:E ratio of 1:2; and a peak inspiratory pressure of 8 to 12 cm H_2O.[44] The initial inspired FiO_2 value should be set based on the preventilation PaO_2 and maybe as low as 0.3 to 0.5. It is not necessary to ventilate foals with 100% O_2. All ventilator settings should be dynamically readjusted based on postventilation blood gas results and tailored to the situation of the individual foal.

Recovery

Foals should recover in a dry, warm and quiet environment, preferably on a soft mattress suitable for the size of the foal to prevent decubitus injuries.[3,7] When the animal resumes spontaneous breathing efforts, it may be positioned in lateral (most frequent) or sternal recumbency and remain intubated at least until it is able to maintain adequate arterial oxygenation (SpO_2 more than 90%) and protect its airways. Continuation of endotracheal or nasal O_2 insufflation is recommended until the foal has resumed a normal breathing pattern and can maintain adequate arterial oxygenation when breathing room air. When the animal makes strong attempts to get up, it may be moved into sternal recumbency and then be given assistance at the moment of rising into standing position. The mare should be reintroduced to the foal when the foal is stable and able to stand on its own.

Perioperative Pain Management

Pain management should be an integral component of the anesthetic plan and perioperative care. A detailed review of analgesic therapy in horses can be found in Chapter 23. Currently no specific approach to pain assessment in neonatal foals has been described. However, the immaturity of hepatic and renal mechanisms in drug metabolism and elimination during the first 1 to 2 months of life would principally favor the use of locoregional techniques of analgesia and anesthesia over systemic pain therapy. As in adult horses, systemic analgesia in foals relies predominantly on administration of nonsteroidal antiinflammatory drugs (NSAIDs), opioids (primarily butorphanol [Torbugesic, Fort Dodge Animal Health]), and lidocaine.[3,71] Table 20-5 lists drug doses.

The NSAIDs flunixin meglumine (Banamine),[72,73] phenylbutazone (Phenylbutazone USP),[74,75] ketoprofen (Ketofen),[76] and ibuprofen (Caldolor)[77] have been studied in neonatal foals. Data from those studies indicate that clearance of these drugs is significantly slower and volume of distribution larger in the neonate than older foals and adult horses, causing prolonged half-lives. As a result, NSAIDs often need to be administered differently in neonatal foals, compared with adults. Under similar clinical circumstances, flunixin meglumine doses administered in neonatal foals during the first 24 hours of their life may be increased by as much as 1.5 times to induce comparable therapeutic concentrations, but in general, dosage intervals should be increased to avoid drug toxicity, including gingival and gastrointestinal ulceration, hypoproteinemia,

TABLE 20-5. Systemic Analgesics for Perioperative Pain Management

	Neonate (1 Month or Younger)	Pediatric/Juvenile Foal (1-4 Months)
NSAIDs	Flunixin meglumine IV/IM q 24-36 hr 1.4 mg/kg (foal <24 hr) 0.5-1 mg/kg (foal 1-4 wk) Phenylbutazone 2.2 mg/kg IV/PO q 12-24 hr Meloxicam 0.5-0.6 mg/kg IV q 8-12 hr Ketoprofen 1-2 mg/kg IV q 24 hr Ibuprofen 10-20 mg/kg IV/PO q 8 hr	Flunixin meglumine 1.1 mg/kg IV/IM q 24-36 hr Phenylbutazone 2.2 mg/kg IV/PO q 12-24 hr Meloxicam 0.5-0.6 mg/kg IV q 8-12 hr Ketoprofen 1-2 mg/kg IV q 24 hr Ibuprofen 10-20 mg/kg IV/PO q 8 hr
Opioids	Butorphanol 0.01-0.04 mg/kg IV Butorphanol 0.02-0.08 mg/kg IM Morphine 0.1-0.2 mg/kg IV, IM L-Methadone 0.05-0.1 mg/kg IV, IM Transdermal fentanyl (100-µg/hr patch)	Butorphanol 0.01-0.4 mg/kg IV, IM Morphine 0.1-0.2 mg/kg IV, IM L-Methadone 0.05-0.1 mg/kg IV, IM Transdermal fentanyl (one or two 100-µg/hr patches)
α_2-Agonists	Xylazine 0.1-0.5 mg/kg IV (use sparingly in foal colic because of adverse effects on cardiovascular and respiratory systems and GI motility) Medetomidine 1-2 µg/kg SC Dexmedetomidine 0.5-1 µg/kg SC	Xylazine 0.1-0.5 mg/kg IV/IM Detomidine 2-5 µg/kg IV/IM Medetomidine 2-5 µg/kg IM/SC Dexmedetomidine 1-3 µg/kg IM/SC
Other	Lidocaine 50 µg/kg/min following 1.3-1.5 mg/kg IV loading dose	Lidocaine 50 µg/kg/min following 1.3-1.5 mg/kg IV loading dose

GI, Gastrointestinal; *NSAIDs*, non-steroidal antiinflammatory drugs.
See text for more details.

colitis, nephrotoxicity, and platelet dysfunction, especially in sick foals.

The opioid agonist-antagonist butorphanol (0.05 mg/kg IV/IM) has been tested in newborn foals.[78] In animals up to 3 weeks of age, the elimination half-life was 2.1 hours after IV injection (about twice as long) and bioavailability was 66% ± 12% (twice as high as in adults).[79] In neonates, butorphanol has minimal effects on vital signs but makes the animals more sedate and even mildly ataxic compared to older foals and adults, in which higher doses commonly cause excitement. Morphine or L-methadone (L-Polamivet) at doses similar to those used in adults have been used,[37] but there are no reports on the pharmacokinetics or pharmacodynamics of these drugs in foals. Application of fentanyl patches (Duragesic) has been tested in neonatal foals, but not for analgesic efficacy.[80] After placement of one 100-µg/hr fentanyl patch on the skin above the jugular vein, fentanyl was detected as early as 20 minutes after patch placement, and plasma concentrations peaked after 14 ± 8 hours and returned to baseline concentrations 12 hours after patch removal.[80] All foals satisfactorily tolerated the patch application and showed no significant adverse effects.

Foals undergoing soft tissue surgery and especially those with abdominal pain respond well to lidocaine infusion (50 µg/kg/min following a loading dose of 1.3 mg/kg) and seem to tolerate such an infusion as well as adult animals.[81,82]

Perianesthetic Complications Requiring Intervention

Impaired Cardiovascular Function

Multiple causes may contribute to hemodynamic instability during the perianesthetic period, including an underlying disease process, hypovolemia and dehydration, anemia, endotoxemia, and impaired pulmonary gas exchange causing hypoxemia. Other potential causes include tissue hypoxia, cardiac arrhythmias, congenital heart defects, disturbances in metabolic and electrolyte homeostasis, and anesthetic drug–induced changes. The risk of cardiovascular decompensation is particularly high in the neonate and sick foal and requires careful preanesthetic assessment of the foal, correction of hypovolemia prior to induction of anesthesia, and vigilant monitoring of hemodynamic parameters during the perianesthetic period.

Table 20-6 lists average hemodynamic parameters recorded prior to any intervention aimed at improving hemodynamic or respiratory function in 482 foals undergoing inhalation anesthesia for various reasons. Retrospective analysis of these cases revealed that perianesthetic complications are not necessarily common in this patient population. In only 4% of the foals an arrhythmia was detected during the anesthesia period including, most commonly, second- or third-degree atrioventricular (AV) blocks, occasional ventricular extrasystoles, hyperkalemia-associated changes in ECG trace, and hypoglycemia-, hypothermia-, and/or hypoxia-associated bradycardia. If atropine (Atropine SA, 20 to 40 µg/kg IV) does not restore atrioventricular conduction, the indirect sympathomimetic drug ephedrine (Ephedrine Sulfate Injection, USP, 25 to 50 µg/kg IV) exhibiting both α- and β-adreneric activity may. Ventricular tachyarrythmias are treated if they are associated with significant impact on hemodynamic function with lidocaine as an initial bolus of 1 mg/kg IV followed by subsequent IV doses of 0.5 to 0.75 mg/kg as required or by constant rate infusion (20 to 50 µg/kg/min).

TABLE 20-6. Hemodynamic and Respiratory Parameters Recorded in 482 Anesthetized Foals (1 Day to 6 Months Old)

Parameter	Minimum	Maximum	Average
HR (beats/min)	55 ± 15	80 ± 25	66 ± 18
SAP (mm Hg)	85 ± 13	124 ± 17	103 ± 14
DAP (mm Hg)	44 ± 9	74 ± 16	58 ± 12
MAP (mm Hg)	59 ± 9	95 ± 16	76 ± 12
RR (breaths/min)	6 ± 4	12 ± 5	9 ± 4

DAP, Diastolic arterial pressure; *HR*, heart rate; *MAP*, mean arterial pressure; *RR*, respiratory rate; *SAP*, systolic arterial pressure.
Data recorded prior to drug treatments or mechanical ventilation.

The most frequent hemodynamic complication during anesthesia is systemic arterial hypotension as a result of anesthetic drug-induced decrease in systemic vascular resistance and/or bradycardia. Based on the individual foal's situation, volume replacement therapy as described earlier and/or infusion of dobutamine (Dobutrex, primarily used as a positive chronotropic agent in the neonate; 2 to 8 µg kg^{-1} min^{-1}), phenylephrine (Phenylephrine HCl Injection, USP) 0.1 to 1 µg kg^{-1} min^{-1}), or norepinephrine (Norepinephrine Bitartrate Injection, USP, 0.1 to 1.0 µg kg^{-1} min^{-1}) titrated to effect or IV ephedrine injections at increments of 0.05 to 0.1 mg/kg are used to increase and maintain mean arterial blood pressures above 60 to 70 mm Hg.[12,63,64,83,84]

Impaired Respiratory Function

Most neonatal but also pediatric and juvenile foals hypoventilate or even become apneic after administration of induction agents and inhalant anesthetics. In addition, reduced FRC as a result of pharmacodynamic effects of the anesthetics used, recumbency, development of absorption and compression atelectases, increased respiratory workload imposed by relatively narrow endotracheal tubes and the anesthetic circuit, and ventilation-perfusion mismatching and intrapulmonary shunting all may compromise pulmonary gas exchange in neonates. As a result, arterial CO_2 retention and hypoxemia, possibly even tissue hypoxia, may occur. Retrospective analysis of the anesthesia records of the population of 482 foals undergoing inhalant anesthesia (FiO_2 more than 0.8) revealed that hypoventilation is common with 20% of the foals developing marked arterial hypercarbia ($PaCO_2$ more than 65 mm Hg), but intraoperative hypoxemia (SaO_2 less than 90%) is rather rare with only 1% of foals affected. Adjustment of anesthetic drug administration and mechanical ventilation as described previously are required to improve respiratory function.

Hypothermia

The younger the foal, the higher the risk of developing hypothermia, which promotes bradycardia, slows awakening from anesthesia, and predisposes the animal to a higher risk of infections.[2,3,7,37] To prevent heat loss during anesthesia and surgery, the animal should be placed on a circulating warm water mattress and be covered with forced warm air blankets (e.g., Bair Hugger). Heating of the surgery room, administration of warmed maintenance fluids during anesthesia, avoidance of

cold disinfectant solution for aseptic preparation of the surgical site, and use of body-warm irrigation solutions may further help reduce heat loss.

Return to Fetal Circulation

A return to fetal circulation should be suspected in newborn foals that present without cyanosis but unexpectedly desaturate under anesthesia (SaO_2 less than 80%) and become cyanotic (decreasing to 20 to 40 mm Hg) despite being mechanically ventilated and inhaling 100% O_2.[17,44] This will be accompanied by persistent pulmonary arterial hypertension and massive right-to-left shunting of blood flow through the foramen ovale or ductus arteriosus. It occurs more commonly in compromised animals with respiratory disease, prematurity, or septicemia. Increasing the anesthetic depth in an attempt to reduce pulmonary vascular resistance may successfully reverse this life-threatening situation; however, treatment with sildenafil citrate (slowly, 0.5 to 2.5 mg/kg IV), a type 5 phosphodiesterase inhibitor that produces selective pulmonary arterial vasodilatation and thus ameliorates clinical signs of pulmonary hypertension, appears to be more efficacious and reliable.[44] In addition, an echocardiographic exam should be performed to exclude any primary cardiac cause of cyanosis.[17]

Cardiac Arrest and Cardiocerebral-Pulmonary Resuscitation

Causes of cardiac arrest and the technique for effective cardiocerebral-pulmonary resuscitation (CCPR) in the foal have been reviewed in detail.[85] Pulseless electrical activity (PEA) and asystole are the cardiac rhythms most commonly associated with cardiac arrest in foals.[85] Cardiopulmonary failure in foals usually occurs secondary to systemic disease or anesthetic overdose that initially cause respiratory arrest, followed by development of a nonperfusing bradycardia, PEA, and, finally, asystole. Causes of secondary cardiopulmonary arrest encountered in foals include severe hypovolemia, low cardiac output, severe metabolic acidosis, hyperkalemia (e.g., ruptured bladder), vasovagal reflex, severe hypoglycemia, severe hypothermia, septic shock/endotoxemia, and finally pulmonary arterial hypertension with return to fetal circulation and right-to-left shunting of blood causing systemic tissue hypoxia. Less commonly, cardiac tamponade, tension pneumothorax, or trauma is encountered. Without treating the underlying cause of the arrest, CCPR may temporarily revive the patient but not result in rescue.

The approach to any cardiac arrest situation is guided by the A(airway), B(reathing), C(irculation or cardiac massage), D(rugs) protocol of CCPR.[86] The anesthetized foal is usually already intubated and attached to an anesthetic circuit. Immediate discontinuation of any anesthetic drug administration and flushing of the anesthetic circuit with O_2 to eliminate residual anesthetic gas is essential, as is manual ventilation with the rebreathing bag at a rate of 6 to 10 breaths per minute. Should the animal not yet be intubated, immediate orotracheal intubation with a 7- to 14-mm ID tube followed by manual ventilation (6 to 10 per minute) using 100% O_2 are essential. Of equal priority is the initiation of chest compressions at a rate of 100 per minute as soon as a nonperfusing cardiac rhythm is diagnosed. Cardiac compression can be done most successfully by placing the foal on a firm surface in right lateral recumbency. The resuscitator should place the palm of one hand with the fist closed over the foal's heart, while the other hand is placed on top of the first to reinforce the compressing hand. The elbows should remain straight, and the motion for compression should originate from the waist (with the upper body weight powering the compression for increased endurance). Chest compression results in no more than 25% to 30% of normal cardiac output. Delaying cardiac contractions or ventilation significantly decreases the chances of successful resuscitation. If not established yet, jugular venous access should be obtained as soon as possible to allow reliable drug administration. In addition, ECG leads should be attached to the patient using ECG gel to facilitate conductivity. The use of alcohol could increase the risk of a fire hazard if electrical defibrillation is needed. Also a capnometer or capnography sampling line connected to the adaptor of the endotracheal tube is very useful because the progress of CCPR efforts can best be monitored by recording ECG and $ETCO_2$ changes. In the absence of cardiac contractility and therefore cardiac output to the lungs, no CO_2 is exhaled (alveoli are ventilated but not perfused) but with effective chest compressions cardiac output and lung perfusion resume and thus $ETCO_2$ starts to climb to values of 12 to 18 mm Hg. Capnography provides the resuscitator with the immediate feedback needed to adjust the chest compression technique until an optimal CCPR technique is achieved.

Among all drugs tested in CCPR, only two exhibit significant efficacy in cardiac arrest situations: epinephrine and vasopressin.[85,86] Epinephrine possesses strong vasoconstrictive properties (via activation of α-adrenoceptors) and has been shown to improve coronary perfusion pressure during cardiac arrest, thereby increasing myocardial blood flow during cardiac compressions. This helps resolve myocardial hypoxia and contractile failure. Therefore, epinephrine administration is indicated in any form of cardiac arrest regardless of cause or rhythm. It is appropriate to initially administer a dose of 0.01 to 0.02 mg/kg of epinephrine intravenously or intraosseously every 3 minutes. If jugular venous access is not available, epinephrine may be given by the endotracheal route at a dose of 0.05 to 0.1 mg/kg diluted in 1 to 2 mL of saline. Complications of epinephrine administration include most frequently ventricular fibrillation (V-fib), pulseless ventricular tachycardia (V-tach), and an increase in systemic vascular resistance, all increasing myocardial O_2 demand and cardiac workload. Electrical defibrillation is indicated as soon as V-fib and pulseless V-tach are diagnosed. A paddle should be placed on each side of the chest after shaving the area, and the initial charge should be 2 J/kg, with subsequent defibrillations using a charge of 4 J/kg, delivered 30 to 60 seconds later if no conversion of the rhythm occurs. In between shocks, at least 2 minutes of chest compressions and ventilation with 100% O_2 should continue to ensure coronary perfusion.

Like epinephrine, vasopressin is a potent peripheral vasoconstrictor with particularly high pressor activity in situations of cardiopulmonary failure and sepsis, and thus it may help the return to spontaneous circulation in cardiac arrest. The dose of vasopressin for cardiac arrest is a total of 0.6 IU/kg given as a single dose or divided. Because the effect may last 10 to 20 minutes, repeat doses are not recommended.[85] Postresuscitation hypertension or arrhythmias are much less likely after vasopressin administration.

Lidocaine may be an appropriate antiarrhythmic agent for treatment of V-fib, V-tach, or ventricular extrasystoles after epinephrine administration or electrical defibrillation.[85,86] The

initial dosage is 1 mg/kg IV and additional IV dosages of 0.5 to 0.75 mg/kg up to a maximum dosage of 3 mg/kg may be administered at intervals of 5 to 10 minutes if the arrythmia persists. Alternatively, a constant-rate infusion of 20 to 50 μg/kg/min may follow the initial loading dose.

During the period of cardiac arrest per se, large-volume fluid administration is contraindicated. Even with optimal chest compressions, the cardiac output is only 25% to 30% of normal. If fluids are given rapidly, the venous pressure rises, impeding coronary perfusion and return of a normal cardiac rhythm, despite effective chest compressions and doses of epinephrine.[85] Thus, a low fluid rate should be used until spontaneous cardiac rhythm returns. Thereafter, increased fluid rates may be indicated to maintain adequate systemic circulation.

Provided CCPR measures were successful, continued monitoring of cardiopulmonary and metabolic functions for at least 24 to 36 hours and adjustments in supportive therapy are critical to keep the foal stable and ensure long-term success.

ANESTHETIC CONSIDERATIONS FOR THE CRITICALLY ILL NEONATE AND MATURING FOAL

Foal with Uroperitoneum

The clinical presentation as well as medical and surgical management of uroperitoneum in newborn foals (up to 3 weeks of age) has been summarized.[87-89] Reports in the past suggested a sex predilection for males and emphasized the presence of characteristic electrolyte abnormalities including hyponatremia followed by hyperkalemia and then hypochloremia, in conjunction with azotemia and metabolic acidosis. However, more recent retrospective analyses challenge this traditional view.[88,89] A sex predilection was not observed, nor were the classic electrolyte abnormalities present in more than half of the affected foals. The clinical symptomatology appeared to be different when uroperitoneum was not the primary presenting complaint but rather developed as a secondary complaint during hospitilization. Foals having received fluids for other reasons were more likely to be septic despite having normal serum electrolyte concentrations. However, serum creatinine concentrations were always greatly elevated in foals with uroperitoneum. Of importance for the anesthetist is the fact that most foals develop respiratory distress with increased respiratory rate and respiratory effort as a result of significant abdominal distention, and lung auscultation often reveals wheezes and harsh lung sounds. Arterial O_2 saturation and tensions are frequently reduced. The heart rate is commonly and greatly increased with an irregular rhythm because of abdominal pain, hypovolemia, and hypoxemia, and a grade II systolic murmur commonly can be auscultated.

Urinary tract defects, located in the bladder wall or the urachus, require surgical repair as the treatment of choice, and thus emphasis must be placed on preanesthetic stabilization of the patient. This should include O_2 supplementation via nasal insufflation or mask delivery, restoration of circulating blood volume, correction of electrolyte and acid-base abnormalities (primarily hyperkalemia and metabolic acidosis) and slow drainage of the peritoneal fluid by abdominocentesis or by peritoneal dialysis to prevent the development of hypovolemic shock. Continuous recording of heart rate and rhythm via ECG and noninvasive measurement of blood pressures aids in monitoring the progress achieved with treatments. After initial blood volume restoration, with physiological saline or isotonic crystalloid solutions low in K^+ content (5 mEq/L or less), hypertonic saline may be infused to correct the Na^+ (and Cl^-) deficit. At Na^+ concentrations less than 110 mEq/L, seizures commonly occur.[68] The Na^+ deficit in mEq can be calculated as: normal serum Na^+ in mEq/L: measured serum Na^+ in mEq/L × 0.4 × body weight in kg. The Cl^- deficit is of similar magnitude. If hyponatremia prevails over several days, the brain slowly adapts by altering the osmolality of its cells through loss of intracellular potassium and organic solutes. Therefore, hyponatremia should be corrected slowly (i.e., 0.5 mEq/kg/hr or less) to avoid central pontine myelinosis.[69] Serum K^+ can be effectively decreased to clinically acceptable levels by giving regular insulin at a dose of 0.1 to 0.2 IU/kg slowly IV in 2.5% to 5% dextrose over 30 to 45 minutes prior to induction of anesthesia. The management of metabolic acidosis (pH less than 7.2) may necesiitate administration of sodium bicarbonate ($Na^+HCO_3^-$). The required dose of $Na^+HCO_3^-$ (in mEq) to be administered can be determined based on the base deficit (-BE): -BE in mEq/L × 0.4 × body weight in kg. One half of the calculated bicarbonate dose should be administered first over 20 to 30 minutes and then a blood gas analysis repeated to assess the effect before the second half of the dose is administered.

Sedation is rarely needed in sick foals with uroperitoneum, but premedication with a low dose of a benzodiazepine may be considered in less-compromised animals. Use of α_2-agonists (e.g., xylazine) should be avoided because of their respiratory depressant and proarrythmogenic properties. Induction of anesthesia with any of the modern inhalant agents (isoflurane, sevoflurane, or desflurane in O_2) is rapid and very smooth followed by the use of those agents to maintain anesthesia. Alternatively, a combination of ketamine (2 mg/kg) and diazepam or midazolam (0.2 mg/kg) may be used for induction of anesthesia, especially in the older foal, followed by inhalant anesthesia for maintenance.

The most common life-threatening arrhythmia observed in foals with uremia and hyperkalemia is a third-degree AV-block, which may be precipitated by surgical stimulation. Discontinuation of surgical stimulation and administration of atropine (20 to 40 μg/kg IV) and/or ephedrine (25 to 50 μg/kg IV) may resolve the arrhythmia. If these are not effective, epinephrine (10 to 20 μg/kg IV) and closed chest massage must be initiated.

Foals Affected by Acute Intestinal Disease

Strangulated inguinal hernia, persistent meconium impaction, intestinal intussusception, and intestinal volvulus belong to the most frequently encountered abdominal diseases requiring an emergency laparotomy in young foals.[90] Dehydration and circulating volume depletion as well as bowel distention are the main causes for severe pain and compromised respiratory and cardiovascular functions, which in the neonate rapidly culminates in respiratory and metabolic acidosis, hemoconcentration, lactatemia, and eventually central depression with cardiopulmonary failure. Preanesthetic fluid therapy, in an attempt to restore an adequate circulating volume, O_2 supplementation via face mask or nasotracheal tube as needed, and judicious use of analgesics (preferably an opioid or lidocaine infusion instead of α_2-agonists in neonates) are essential to stabilize the foal prior to anesthesia and surgery. A rapid-sequence induction with ketamine and a benzodiazepine may

be the preferred induction technique in the older or struggling foal followed by maintenance of anesthesia with either one of the inhalant anesthetics. Mechanical ventilation aids in overcoming the problems of reduced FRC as a result of recumbency, abdominal distention, and the effects of anesthetic drugs. Continued fluid administration plus ephedrine or a combination of dobutamine and phenylephrine infusion may be necessary to support the circulatory system. Respiratory system compliance and hemodynamic status usually improve dramatically as soon as the abdominal cavity is opened and gas-filled intenstines are decompressed or exteriorized.

Foal with Thoracic Trauma

Thoracic trauma is frequently encountered in neonatal foals as previous studies indicate.[91-94] Rib fractures may account for as many as 37% of life-threatening fractures in foals younger than 6 months of age with thoracic trauma as the primary cause of death in this age group.[91] The fractures occur most commonly at the costochondral junction or the area immediately above it and are best detected with ultrasonography.[93] Typically three or more ribs are affected with those on the left side of the chest (3rd to 8th) most often involved.[91-93] When performing CCPR, post-resuscitation examination of the rib cage is warranted to exclude the possibility of iatrogenic rib fractures.

External trauma may cause soft tissue injury including severe lung contusions or muscle damage, blood vessel laceration, and rib fractures, with systemic consequences including pain and shock. Depending on the nature of the trauma (blunt or perforating) and dislocation of fractured ribs, internal thoracic structures may be severely affected, resulting in severe complications such as pneumo- or hemothorax, pulmonary laceration, acute parenchymal lung injury with atelectasis formation (ALI), and pleuritis.[92,94,95] Clinical signs in foals commonly include tachycardia; tachypnea or respiratory distress (because of pain and impaired pulmonary gas exchange); subcutaneous emphysema, anemia, and hypovolemia if blood loss was significant; and mental depression.[92,94,96]

Preanesthetic emergency care of the foal with compromising thoracic trauma should include nasal insufflation of O_2 (5 to 10 L/min), IV fluid therapy with isotonic polyionic crystalloid solutions for circulatory volume replacement plus fresh-frozen plasma or fresh whole blood for supplementation of red blood cells, coagulation factors and platelets when indicated. Thoracocentesis may be needed to evacuate the chest in case of pneumo- or hemothorax. Appropriate antiinflammatory, analgesic (e.g., flunixin meglumine, methadone, butorphanol) and antimicrobial treatment should be instituted. Any surgical intervention to repair fractured ribs should be postponed until the foal has been stabilized. If sedation is required, premedication with a benzodiazepine derivative (e.g., midazolam 0.2 mg/kg IV) and potentially another dose of butorphanol (0.2 to 0.3 mg/kg IV) is suitable, followed by induction with IV ketamine (1 to 2 mg/kg) and orotracheal intubation. Any of the inhalant anesthetics is appropriate for maintenance of anesthesia. A constant-rate lidocaine infusion (30 to 50 µg/kg/min following a loading dose of 1.0- to 1.5 mg/kg IV) may help reduce the dose of the inhalant anesthetic and maintain adequate spontaneous ventilation. Positive pressure ventilation with or without positive end-expiratory pressure (PEEP) may become necessary if the foal remains markedly hypercapneic ($PaCO_2$ more than 60 mm Hg) and hypoxemic (PaO_2 less than 55 to 60 mm Hg; SpO_2 less than 85%). Under these circumstances a smaller than usual tidal volume (i.e., 4 to 6 mL/kg) and the lowest PEEP necessary to improve pulmonary gas exchange and arterial oxygenation should be employed in order to avoid ventilation-induced lung injury in an already compromised pulmonary parenchyma. Fluid therapy, nasal O_2 insufflation, and antiinflammatory and analgesic drug treatment should be extended into the postoperative period until the foal can maintain a stable cardiopulmonary status and does not express significant pain.

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CHAPTER
21 Recovery from Anesthesia

Regula Bettschart-Wolfensberger

Recovery is one of the most critical phases of equine anesthesia. Recent results from a multicenter prospective study of equine anesthetic fatalities reported that 23% of all nonsurvivors sustained inoperable orthopedic lesions during recovery.[1] Because of the size and temperament of the horse, it is not possible to continue full monitoring, and mechanical ventilation during recovery and even fluid administration is discontinued in most cases. Significant problems, such as hypoxemia or hypotension, that may develop can be recognized only in severe cases. When the horse starts to awaken, intervention is dangerous for personnel and, depending on the size of the horse and its temperament, often simply impossible. This chapter describes all aspects of recovery from anesthesia in horses.

MANAGEMENT OF HORSES DURING RECOVERY

General Aspects

Although a recent study failed to demonstrate a beneficial effect of recovering horses in a darkened room,[2] anesthetists commonly agree that during recovery, external stimuli (e.g., noise, bright light, physical stimuli) should be minimized. The horse's head should be protected by a padded head cover. The recovery box should have a padded floor and walls. Ideally, the box would have an octagonal shape to prevent horses from being trapped in the corners. In some clinics, horses are placed on heavy foam pads for the recovery. Depending on the nature of the surgery, the premises, and the personal preferences of anesthetists and surgeons involved, assistance during recovery (ropes, slings, hoists) may be desirable.[3] Sling assistance is sometimes (6%) not well tolerated, and to prevent self-inflicted injury, the patients may have to be re-anesthetized.[3] Successful use of a sling requires trained personnel. Ideally a horse should be accustomed to the sling at least a day before anesthesia and surgery.[4] In an experimental study, desflurane (Suprane) inhalation anesthesia was prolonged with propofol (Propofol 1% MCT Fresenius) and xylazine (Xylazin Streuli ad us. vet.) to position horses into the Anderson sling suspension system for recovery.[5] The quality of recovery was characterized by a smooth and careful return to a standing position, but a very high number of horses showed apnea caused by propofol and had to be ventilated during recovery. Fracture patients are most successfully recovered in a hydro pool (see "Pool Recovery," later).

In the early stages of recovery, an anesthetist should stay with the horse. Thereafter, continuous observation of the horse will reveal potential problems at an early stage, allowing immediate intervention. Ideally, horses remain recumbent for at least 20 minutes and then roll into sternal recumbency for another 10 to 20 minutes. The attainment of sternal recumbency is associated with the restoration of more normal breathing patterns, and it reduces ventilation-perfusion mismatching in the lung, which should improve oxygenation.[6] Horses that rest in sternal recumbency prior to attempting to stand usually get up without complication. They recover best if they are allowed to exhale most of the volatile anesthetic agent before trying to stand and thus should not be stimulated to get up too soon. If a horse has not attempted to stand within 60 minutes after the end of anesthesia and is still in lateral recumbency, stimulation may be appropriate. If it is reluctant to get up, its status must be reevaluated and any problems treated. Orthopedic patients are frequently exhausted when they are anesthetized for fracture treatment because they had to sustain all their body weight for some time on three legs and had to endure a potentially long transport to a specialized clinic. As a result of this, they

frequently remain for a prolonged time in lateral recumbency following the procedure.

For any surgery longer than 1 hour, the use of a urinary catheter is recommended because it prevents overdistention of the bladder, which is uncomfortable for the horse. Without a urinary catheter, a horse often tries to get up before it is ready to do so. As soon as it is standing, despite being ataxic, it will try to urinate. This can represent a considerable risk because the floor can become slippery and the horse may lose its balance and injure itself.

Maintenance of Adequate Respiration During Recovery

Insufflation of oxygen (15 L/min per 500 kg), first via the endotracheal tube and after extubation via nasal tubes or directly into the nasal cavity, helps to reduce the development of hypoxemia.[7] If apnea is present, the use of a demand valve helps to ventilate the horse until spontaneous respiration resumes.[8] Short periods of apnea (3 to 5 minutes)—for example, during transport from the surgery suite to the recovery stall—are not harmful. One study demonstrated that mechanically ventilated, anesthetized horses maintain higher values of arterial oxygen partial pressures (PaO_2) postoperatively during a brief period of apnea than horses weaned from the ventilator prior to disconnection.[9]

Controversy exists as to the optimal point during recovery for removal of the endotracheal tube. Most anesthetists advocate the removal of the endotracheal tube when the horse is actively swallowing. The presence of an active swallowing reflex helps to ensure that fluid or gastric reflux is not aspirated and that the horse replaces its soft palate into the normal position. Others believe that the tube should be removed only when the horse is standing. Extubation while the horse is in lateral recumbency allows earlier identification of potential respiratory obstruction. The endotracheal tube is then easily reintroduced, and if necessary a tracheostomy can be performed, which would be very difficult in a standing horse with acute dyspnea.

Another reason to remove the endotracheal tube as soon as the horse is swallowing is that some horses object to this foreign body when they regain consciousness. This negatively influences recovery because the animal may try to get up too early. Therefore, I prefer to remove the endotracheal tube before the horse regains a standing position. An alternative approach is to remove the endotracheal tube when the swallowing reflex is regained and to place a small endotracheal tube via the nares for recovery. This technique avoids the inadvertent damage to the endotracheal tube from the teeth and still maintains a patent airway (see later). Preexisting hemiplegia,[10] trauma, or hyperextension of the neck[11]; prolonged duration of recumbency[12]; or surgeries in the upper airway region are all factors that might predispose the upper airway to obstruction. If these factors are present, the endotracheal tube should not be removed until the horse is standing.

Most recumbent horses develop nasal edema, and because horses are obligate nasal breathers, this swelling will impair breathing after removal of the endotracheal tube. In severe cases, significant airway obstruction may occur. Transient airway obstruction during recovery can also be caused by a variety of other problems, such as laryngospasm, laryngeal hemiplegia, dorsal displacement of the soft palate, kinking of the head in a corner of the recovery box, or obstructing foreign bodies in the upper airways.[11] Regardless of the cause, any airway obstruction may lead to pulmonary edema or pulmonary hemorrhage, resulting in cardiovascular collapse and death.[10-15]

Phenylephrine 0.15% (Phenylephrin HCl 1.5 mg/mL) can be instilled into the nares to decrease nasal congestion and help prevent obstruction after extubation.[16] An alternative is to insert nasal tubes approximately 20 cm (8 inches) long into one or both nasal cavities. It is necessary to have various diameters available to fit a wide range of horses. Fractious animals find this tube in their nares uncomfortable, and therefore the use of an anticongestant is preferred. To achieve the optimal effect, the drops are instilled 10 minutes before extubation. If, despite the use of nasal drops, labored breathing or snoring occurs after extubation, nasal tubes should be inserted and the head extended with the tongue pulled out. Should this not result in normal regular breathing, other causes of airway obstruction should be investigated. Orotracheal intubation or a temporary tracheostomy (see Chapter 47) may become inevitable in these cases.

Management of Cardiovascular Function

Administration of fluids, dobutamine (Dobutrex), or other vasopressive agents is usually discontinued before recovery. All inhalation anesthetics and total intravenous anesthesia regimens depress cardiovascular function in a dose-dependent manner.[17] At the end of anesthesia, modern anesthetics are relatively quickly exhaled or redistributed, and thus cardiovascular function improves rapidly during recovery, and it is usually not necessary to administer fluids during the recovery period. If horses suffer from severe blood loss or hypotension (possibly as a result of endotoxemia) during anesthesia, continuing fluid administration and inotropic support during recovery is advocated. Depending on the severity of the hypotension, the horses can continue to lose fluid from their circulating volume and thus have problems in maintaining adequate cardiovascular function despite waning of the depressive effect of anesthetic agents. Such horses, especially if they are large, have great difficulty in getting up.

FACTORS INFLUENCING RECOVERY

Severity of Surgery, Temperament of the Horse

Under clinical circumstances, the quality of recovery and ultimate outcome correlate inversely with duration of anesthesia and severity of surgery.[1,18] On the other hand, an experimental study on horses anesthetized with isoflurane (IsoFlo ad us. vet.) or halothane (Halothane USP) but without undergoing surgery, showed that the temperament of the individual was the major factor determining the quality of recovery.[19] Therefore, the temperament of the patient must be considered during recovery from surgery.

Inhalation Anesthetics

Different inhalation anesthetics possess different physical properties. Blood gas solubility is the major determinant of the speed of action of an anesthetic and thus duration of recovery. With halothane, the relatively high rate of metabolism (about 20%), in comparison to isoflurane (about 0.2%) or sevoflurane (Sevorane Sevoflurane) (2% to 3%), may have an influence on the quality of recovery in compromised patients. An early report

that investigated duration and quality of recovery after 2 hours of anesthesia showed that recovery after isoflurane anesthesia is quicker and of a better quality than after halothane anesthesia.[20] After 3 hours of anesthesia, no difference in duration or quality of recovery was detected between isoflurane and halothane.[19] In another report, the recoveries of horses with or without surgery with isoflurane or halothane were compared.[21] The authors found no difference in the quality of recovery but reported shorter recoveries following isoflurane. This is in contrast to clinical studies that showed that recoveries with halothane are of better quality.[22-24] Another experimental study comparing halothane, isoflurane, and sevoflurane revealed that recovery quality was better with sevoflurane and halothane, and duration was shorter with sevoflurane and isoflurane.[17] Comparison of recovery after just 90 minutes of isoflurane or sevoflurane anesthesia showed that horses recovered significantly quicker and better with sevoflurane than with isoflurane.[25] A clinical study that compared recovery in 100 horses following MRI with either isoflurane or sevoflurane anesthesia could not demonstrate a difference between the two.[26] Desflurane (the least soluble and therefore fastest-acting inhalant agent currently available) was reported to cause a very rapid recovery, but of unacceptable quality.[27] One study used six horses and each of them was anesthetized for 2 hours with each of the three anesthetics sevoflurane, isoflurane and desflurane.[28] The horses were sedated for the recovery with xylazine. No significant differences between the recovery times were found, but with each anesthetic time for horses (which received all three anesthetics), recovery quality became better.[28] These clinical studies were retrospective or not well standardized. The experimental studies included very low numbers and were not able to exclude the recovery "learning factor." This might explain the different results. Use either isoflurane or sevoflurane in combination with suitable drugs in a balanced manner to achieve best possible recoveries (as described in Chapter 18).

Sedation, Analgesia

Pain has a negative influence on recovery in horses. To prevent central and peripheral sensitization, analgesia should be addressed before and during the anesthetic period. To provide maximal analgesia for recovery, especially after long, major surgical procedures, some additional analgesia, preferably using a multimodal approach, should be provided (see Chapter 23). The balanced anesthesia regimen used intraoperatively will influence recovery also (see Chapter 18).

In a clinical study of 25 horses undergoing arthroscopy, the preoperative use of phenylbutazone (Butadion ad. us. vet.) did not alter recovery time but did slightly improve recovery score compared with patients not receiving this nonsteroidal anti-inflammatory drug (NSAID).[29] These results are somewhat in contrast to the previous statements, which advocated continuous administration of analgesics during anesthesia.

The use of opioids in horses is controversial because of the fear of excitement and the effect of the opioids on the gastrointestinal tract. Some authors have tried to show the benefit of using opioids for recovery but have failed to do so, probably because too many other factors also influence this period.[30,31] They were, however, able to show that opioids do not negatively influence and prolong recovery or result in an elevated $PaCO_2$ level compared to untreated horses. Every horse benefits from the administration of morphine (Morphin HCl sintetica 10 mg) (0.1 mg/kg IM) 10 minutes before disconnection from the inhalation anesthetic.

α_2-Adrenoceptor agonists, such as xylazine, slightly prolong the recovery period.[25,32] In clinical cases, this usually results in superior recoveries. The fact that α_2-adrenoceptor agonists act not only as sedatives but also as analgesics certainly adds positively to the calming effect. Most authors advocate the use of 0.1 to 0.2 mg/kg xylazine IV; others use low doses of detomidine (Equisedan ad us. vet.), romifidine (Sedivet ad us. vet.), or medetomidine (Dorbene).[33] Because α_2-adrenoceptor agonists negatively influence cardiovascular function, the use of low doses of the shorter-acting xylazine (especially in compromised patients) is probably safest, because its side effects are less severe and of shorter duration. The α_2-adrenoceptor agonists should be administered only when brisk palpebral reflexes or even nystagmus is present. This guarantees that levels of inhalation anesthetics are lower than during maintenance of anesthesia and that no overdose occurs. Slow administration of the sedative (the full dose over 1 minute or slower) reduces the risk of profound side effects and thus probably increases safety. Two recent experimental studies[34,35] intended to improve recovery quality by exchanging maintenance of inhalation anesthesia with TIVA at the end of anesthesia. A combination of xylazine and ketamine (Narketan 10 ad us. vet.) was used following 90 minutes of isoflurane anesthesia for 30 minutes[34] and a combination of xylazine and propofol was used following 4 hours of desflurane anesthesia.[35] The use of xylazine-ketamine resulted in longer recovery times, whereas with xylazine-propofol, the transition from lateral recumbency to standing was qualitatively better. Propofol caused respiratory depression that might be dangerous under clinical circumstances. The use of propofol in this manner is too dangerous for everyday clinical practice and offers no benefit. As recoveries following short xylazine-ketamine TIVA are usually very coordinated and of good quality, this regimen might offer some potential to improve recovery quality following inhalation anesthesia, but this remains to be tested in a larger number of horses.

RECOVERY SYSTEMS AND AIDS

Recovery Stall Design and Construction

Although there is no literature comparing the influence of different types of recovery stalls on fatality rates, most anesthetists agree on a number of important design features for recovery stalls. First, the recovery box should not be too large; this allows the horses to lean against the walls during their arousal or as soon as they are standing, because most horses show some degree of ataxia immediately after recovery. Ideally, the angles between walls are greater than 90 degrees (Figure 21-1) to prevent horses from getting trapped in a corner with their head in a kinked position. Padding of walls and floor is most important. Floors should provide secure footing with a surface that is not slippery, even when wet (by urine, blood, excessive sweating, obstetrical lubricant, etc.). On the other hand, the surface must be soft enough to prevent abrasion injuries if horses fall during recovery. Last, the padding should be easy to clean. Linatex is a commercially available surface providing these qualities. In one study, recoveries with the aid of a rapidly inflating and deflating air pillow was compared to recoveries in a padded recovery stall.[36] With the pillow, horses rested longer before they attempted to attain sternal recumbency and took fewer attempts to standing. Quality of their standing was better as well.

A recovery stall should also have metal rings (for attaching ropes), an oxygen supply, heating, air conditioning, lights that can be dimmed, a hoist, and easy access for recovery personnel in case of an emergency. Figure 21-2 shows a design for a recovery stall that allows intervention and help for the horse from outside the box, thereby minimizing the risk of injury to people. The ideal size of a recovery box depends on the horse's size. The best option is to have several of different sizes. The octagonal shape with a diameter of 4.4 m of the box in Figures 21-1 and 21-2 has proved optimal for horses ranging in size from 400 to 800 kg.

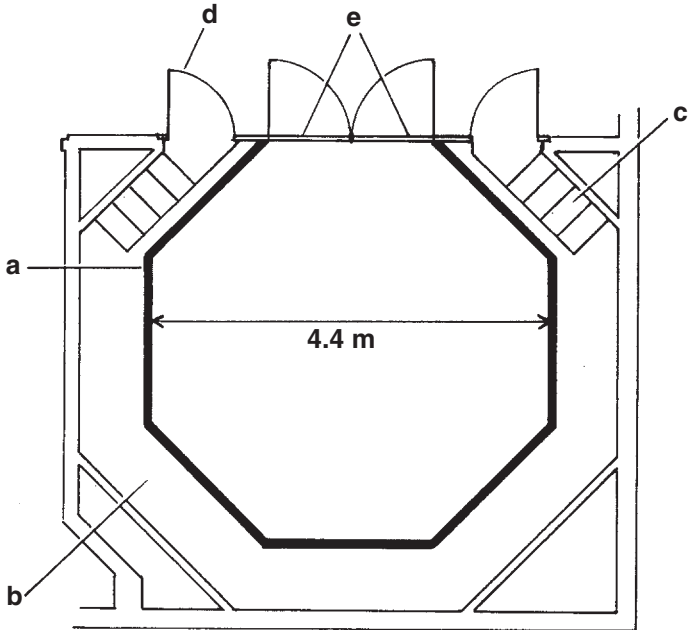

Figure 21-1. Bird's-eye view of a recovery box, including walkway around the box with access to the horse from above. *a,* Recovery stall wall; *b,* the walkway, the floor of which is elevated relative to the recovery box floor, provides the ideal, safe position to assist the horse's recovery; *c,* staircase to walkway; *d,* doors to walkway; *e,* doors to recovery box.

Thick Mattress

A thick mattress prevents the patient from making premature attempts to rise, because it takes a controlled and coordinated effort to "get out" of the mattress and attain sternal recumbency. Frequently, human assistance is necessary to roll the horse from the mattress onto the recovery box floor. Because the patient cannot leave the mattress too early, there is additional time for inhalant anesthetic to be exhaled, which eventually results in a smoother recovery. Soft mattresses may also prevent nerve damage in cases of prolonged recovery.

Rope-Assisted Recovery

As mentioned earlier it is possible to help horses during recovery with the aid of head and tail ropes. The aim should be to support the horse when it is getting up by itself, to stabilize it and not to pull it up. There are no studies comparing the outcome of recovery with or without rope assistance. It is likely that a trained team helps the horse with this support. It remains to be tested as to whether fatalities can be prevented. Care has to be taken not to disturb very heavy, difficult, or fractious horses while they are awakening. This could provoke attempts to stand up too early, resulting in a horse that is not yet able to stand but trying to do so because of the "help."

Pool Recovery

The major benefit of recovery in a hydro pool is that self-inflicted trauma is minimized because the partially submerged

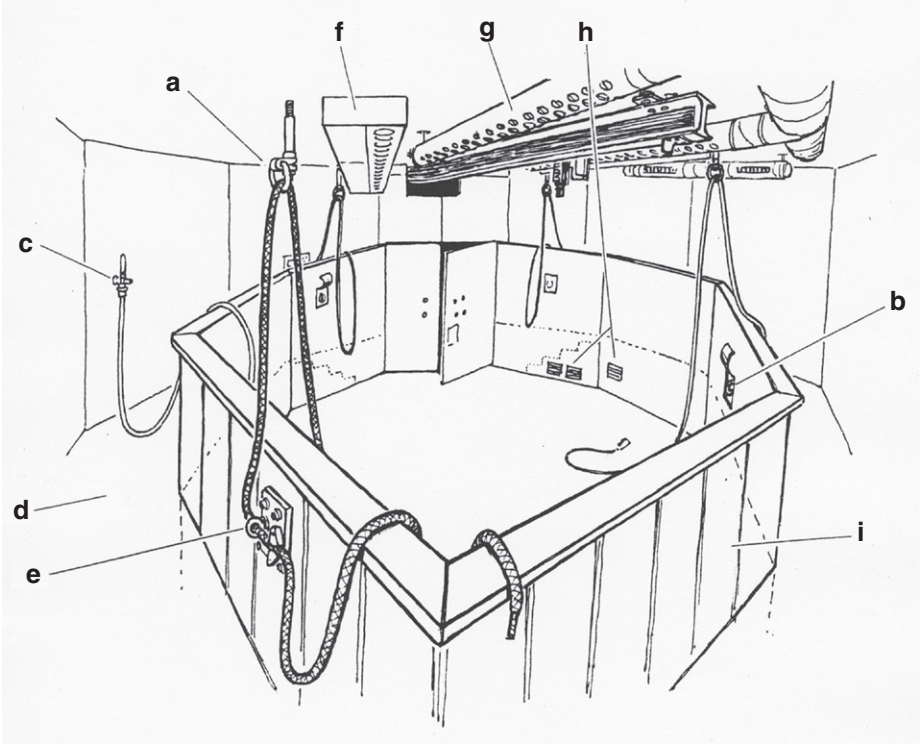

Figure 21-2. Lateral view from the walkway around the recovery box. *a,* Metal rings attached to the ceiling and *(b)* metal rings in the recovery box wall to engage ropes for assisted recovery; *c,* oxygen supply; *d,* walkway; *e,* quick-release devices for attaching ropes firmly; *f,* row of infrared lamps; *g,* air conditioning; *h,* fresh air supply *(left),* air removal–suction *(right); i,* recovery box wall.

Figure 21-3. A horse is lifted out of the pool with a Liftex-sling at the New Bolton Center. (Courtesy Larry Nann.)

horse struggles against the resistance of the water until it is fully capable of standing. For the personnel involved in recovery, there is minimal danger while the horse is inside the pool. The critical phase is the removal of the horse from the pool. There are two different systems. At the University of Pennsylvania's New Bolton Center, the horse is secured in a raft inside a round pool. The raft contains a sleeve for each leg and a flotation ring that has supports for the head. With this design, the horse has only minimal contact with the water. However, the stabilization of the horse inside such a pool can be difficult and requires several people well familiar with the procedure to remain with the horse during the entire recovery period. Removing the horses from this pool requires it to be lifted out of the raft by a sling attached to an overhead hoist (Figure 21-3). In this critical phase, if horse does not cooperate, injuries might occur. It may be necessary to implement short-term sedation or anesthesia until the horse is safely in a stall.

Another hydro pool system has been designed to facilitate the process of getting the horse out of the water.[37] The pool is a rectangle: 3.5 m long, 1.2 m wide, and 2.5 m deep. The water is heated to 38° C and continuously filtered to remove dirt. Additionally, bacterial contamination is minimized by passing the water through an ultraviolet filter. Because the horse is immersed in the water, it is impossible to totally prevent exposure of the surgical site to water, so application of a routine bandage for recovery is contraindicated. The wound is carefully closed with suture, followed by covering the incision site with cyanoacrylate (super glue) and some adhesives surrounding the incision site. Subsequently the limb region is covered with an Ioban bandage (Ioban 2). The Ioban sheet is further protected by an elastic adhesive tape.

The horse is placed inside the pool restrained by a sling or a rescue net designed for the helicopter rescue of horses (Figure 21-4). The scissor table installed within the pool is lowered all the way to the bottom of the tank. While the horse is unconscious, its head is supported by a floating mattress. The horse is maintained in a sternal position in the water. The head is secured in a vertical position by several ropes attached to metal bars on the sides of the pool (see Figure 21-4). When the horse starts to move, the table is elevated by a hydraulic system and the horse is allowed to "feel" the table underneath its feet. With time, the horse recognizes the presence of the table and with some positive encouragement eventually places its weight on all four feet. After the patient has stood securely on all four feet in the water for some time, the table is elevated, lifting the horse

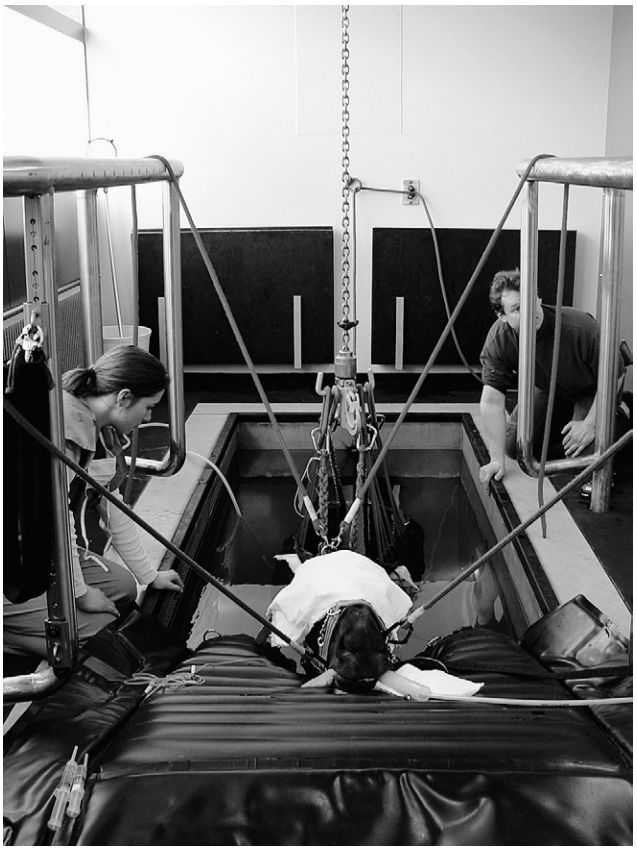

Figure 21-4. A horse recovering from anesthesia in the pool at the University of Zurich. The head is fixed with four ropes on the side bars, the head rests on an air mattress, and the body is secured in the water with a custom recovery net. The eyes are covered and ear-plugs are in place.

Figure 21-5. A patient quietly standing on the scissor table with the feet still in the water. The recovery net is still in place.

Figure 21-6. Horse during recovery from anesthesia with the help of an Anderson Sling Suspension System. The horse is still unconscious hanging in the sling in an upright position. (Courtesy L. Galuppo, Davis.)

up to the level of the room floor (Figure 21-5). The sling is subsequently disconnected, the bandage removed, the surgical site prepared aseptically, a bandage reapplied, and, when ready, the horse is walked into the recovery box, where it is allowed to dry under an infrared solarium.

This type of hydro pool was originally designed in California and has since been used regularly at Washington State University, Del Mar Race Track, and at the Vetsuisse Faculty University of Zurich, Switzerland. A report on the use of this type of recovery pool on 60 horses documented the following: mean anesthesia time, 182 minutes; mean time in the pool, 108 minutes (range, 20 to 270 minutes).[37] Furthermore, three horses showed severe lung edema (one of them died), two horses showed multiple skin abrasions from violent attempts to leave the pool during recovery, two horses showed incisional infections, and one horse developed septic arthritis following stifle joint surgery. These results demonstrate that hydro pool recovery is not risk free but that it is a relatively safe option when a difficult recovery is anticipated.

During hydro pool recovery, extrathoracic and pulmonary pressures are increased, which may contribute to an increased incidence of pulmonary edema during anesthetic recovery.[38] To minimize the likelihood of edema formation, all anesthetized horses in Zurich are treated with hetastarch (HAES-steril 10%) (4 L/500 kg IV, in addition to Ringer lactate) to maintain plasma oncotic pressure during hydro pool recovery. Furthermore, acepromazine (Prequillan) (0.03 mg/kg) is administered (IM) to produce peripheral vasodilatation and improve perfusion. The administration of acepromazine also provides some calming effect during recovery, without causing ataxia. Supplemental oxygen administration has decreased the incidence of edema at Washington State.[39] As with any surgery, it is very important to provide multimodal analgesia to optimize recovery quality before hydro pool recovery. For any orthopedic surgery, the following are advisable: the presurgical administration of phenylbutazone, α_2-agonists, and possibly opioids (e.g., morphine 0.1 to 0.2 mg/kg IM); the intraoperative use of a balanced anesthesia regimen (e.g., isoflurane in oxygen plus 3.5 µg/kg per hour medetomidine IV); plus additional analgesia for recovery (e.g., 2 µg/kg medetomidine IV plus 0.1 mg/kg morphine IM). The use of a α_2-agonist, such as medetomidine, for recovery is advantageous because it provides analgesia as well as sedation. Using this management protocol, lung edema has not occurred in 50 consecutive pool recoveries.[40]

Anderson Sling Recovery

The Anderson sling (Figure 21-6) is probably the best known sling device and has been tested by several authors.[4,5] It is relatively easy to apply and is tolerated well by most horses, especially when they are accustomed to it a day before surgery.[4] To optimize recovery in the Anderson sling, a recent paper looked at the use of propofol before recovery.[5] The quality of recovery was good and quiet. Whether this regimen improves safety under clinical circumstances remains to be tested because the incidence of apnea (as a consequence of the use of propofol) was high.

PROBLEMS ENCOUNTERED DURING RECOVERY

Reluctance or Inability to Stand

Most problems that become evident during recovery have developed during anesthesia. Hypotension (resulting in poor muscle perfusion), hypoxemia, and malpositioning on the surgery table are the major factors that result in the problems encountered during and immediately after recovery in horses.[41-48] Possible causes of prolonged recoveries include myopathy, neuropathy, and other problems.

Myopathies

Myopathies may affect single groups of muscles or they can occur in a generalized form. Myopathies occur most frequently in cases where hypotension (blood pressure less than 70 mm Hg) is present for more than 15 minutes or anesthesia time exceeds 3 hours.[49,50] Affected muscles are swollen to different degrees, are hard, and are very painful. Depending on the severity of the myopathy, horses may be unable to stand or place full weight on the repaired leg. Therapy of the acute phase is supportive and symptomatic and should aim at reducing pain, swelling,

and anxiety. NSAIDs, opioids, α_2-agonists, and, in severe cases, dimethyl sulfoxide (DMSO 20%), dantrolene sodium (Dantrolen IV "P & G"), and corticosteroids should be administered. A small dose of acepromazine (0.02 to 0.03 mg/kg IM) may help to calm the horse and to improve peripheral perfusion, but this should be administered only if no major cardiovascular disturbances are present. As long as the horse is recumbent, oxygen should be administered via nasal tube or directly into the nasal cavity. Ringer lactate solution or other balanced electrolyte solutions should be infused to help maintain cardiovascular function. In excited horses, the most important factor is to calm them, because repeated attempts to stand up exhaust the horse and increase the likelihood of a catastrophic injury. Human assistance with head and tail ropes helps the horse to lift its weight when getting up and provides some stability, because most affected horses show some degree of ataxia. Some horses resent the pull on the tail, and if this occurs this type of assistance should be abandoned.

When the horse is standing, the major life-threatening problem is myoglobinuria, resulting in kidney dysfunction. Appropriate fluid administration (to ensure maintenance of urine output) and monitoring of urine production are mandatory. Physiotherapy and infrared light may help the horse to relax its muscles and thus will reduce suffering.

Neuropathies

Neuropathies that impair recovery include femoral nerve paralysis, radial or brachial nerve paralysis, peroneal nerve paralysis, and hemorrhagic myelopathy. Neuropathies can also be accompanied by myopathies, and vice versa. Horses suffering from neuropathies of single nerves are not as high risk as horses suffering from severe myopathies, but the risk of fractures during recovery is relatively high in all horses with neurologic weakness. Horses are often unable to cope with paralyzed extremities: they get excited and injure themselves before intervention becomes possible. Therapy during recovery is symptomatic and supportive, as for myopathies.

Miscellaneous Problems

Other, less common causes of reluctance, difficulty, or inability to stand after general anesthesia are hyperkalemic periodic paralysis (HYPP), hypocalcemia, glycogen disease of draft horses, fractures or luxations, severe hypothermia or blood loss, generalized fatigue as a result of underlying disease, and pulmonary embolism.[51]

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CHAPTER 22

Chemical Restraint for Standing Surgery

Simone K. Ringer

Chemical restraint is used daily in equine practice because many diagnostic procedures as well as surgical or medical treatments are impossible without a proper restraint. Horses are not as tolerant to mechanical immobilization as are, for example, ruminants, and they may react violently to excessive restraint, pain, or fear. However, many procedures can be performed safely on standing horses using combinations of sedative and analgesic drugs, supplemented by appropriate local or regional anesthesia and physical immobilization.

An advantage of standing sedation over general anesthesia is the decreased risk of mortality. Compared to human anesthesia, mortality rate in equine anesthesia is still very high.[1,2] Further advantages include decreased costs as well as reduction in personnel needed during the procedure. Disadvantages compared to general anesthesia are less ideal surgical conditions because of movement as a result of ataxia or insufficient analgesia, which may lead to an increased risk to personnel. Also, depending on the localization of the surgical field, asepsis may be more difficult to maintain than in a recumbent horse.

The decision of whether to perform a surgical procedure standing or under general anesthesia should be based on type of procedure, temperament of the horse, anesthetic and surgical facilities, personal experience, and pre-existing medical conditions of the patient.[3]

The goal of chemical restraint for standing surgery is to keep the horse calm, sedated, and indifferent to environmental or noxious stimulation and physical manipulation. The horse should remain standing, and only mild ataxia is acceptable. Sedation must be reliable, and ideally the horse should not be arousable by noise, touch, handling, or movement.

Different drugs and protocols will be discussed in this chapter. Whenever painful procedures are performed, standing sedation should be combined with local or regional anesthesia (see Chapter 23).

GENERAL PRINCIPLES OF DRUG ADMINISTRATION

All sedatives work best when given to a quiet horse and in a quiet environment. Drug effects may be suboptimal if the horse is excited before or during drug administration. Also adequate time should be allowed for drugs to exert their maximal effect before starting any manipulation of the horse. Generally, if an intramuscular (IM) route of drug administration is used, longer onset times and higher doses are needed compared to an intravenous (IV) administration. Also, IM sedation will have less-predictable results.

Sedatives are most commonly administered as a bolus and re-dosing is frequently needed for procedures longer than 30 minutes. Therefore, for prolonged procedures, constant-rate infusion (CRI) of sedatives may be advantageous over repeated bolus administration because it is less cumbersome for the veterinarian, may cause less adverse effects, and provides a more constant level of sedation.[4] If CRI is used, a loading dose should be considered; otherwise the desired effects will take a protracted time because 4 to 5 half-lives are needed until steady state plasma concentrations and sedation are reached.

The catheter placed for CRI also serves as a quick access route for intravenous fluid administration needed during prolonged sedation.

DRUG SELECTION

Many drugs have been used and are available. The sedation protocol should be chosen based on health status and temperament of the horse, type and duration of the planned procedure, drug availability, and personal experience.

To achieve optimal sedation, often two and frequently even three drugs are administered simultaneously. The administration of multiple drugs may allow the use of reduced dosages and may be safer and more effective than administering larger doses of a single drug.

DRUGS AVAILABLE FOR CHEMICAL RESTRAINT

α_2-Adrenoreceptor Agonists

α_2-Adrenoreceptor agonists are probably the most commonly used drugs for standing sedation of horses. The most frequently used α_2-adrenoreceptor agonists in equine practice are xylazine (Xylazin Streuli ad us. vet.), detomidine (Equisedan ad us. vet.) and romifidine (Sedivet ad us. vet.) (Table 22-1). Medetomidine (Dorbene) and dexmedetomidine (Dexdomitor), even if not registered for horses, have been used successfully in this species.[4-7]

Mechanism of Action Leading to Sedation and Analgesia

α_2-Adrenoreceptor agonists provide reliable and profound sedation with analgesia and muscle relaxation. The sedative effect of α_2-adrenoreceptor agonists is the result of activation of α_2-adrenoreceptors located on the locus coeruleus in the pons of the brainstem. The exact mechanism for antinociception is currently not completely understood; both supraspinal and spinal sites of action seem to be involved.[8] The increased sensitivity to touch reported clinically with α_2-adrenoreceptor agonists[9] may be induced by modification of the activity of fast conducting non-nociceptive afferent Aβ fibers and seems to be strongest and longest with detomidine compared to romifidine and xylazine.[10]

Uses

For very short procedures a bolus of xylazine or medetomidine can be used, whereas for sedations between 15 and 30 minutes, the longer-acting romifidine or detomidine should be selected. Procedures taking longer than 30 minutes benefit from CRI of α_2-adrenoreceptor agonists.

Even if horses appear deeply sedated with α_2-adrenoreceptor agonists, they may suddenly respond to stimulation, especially to touch.[9] To avoid this, α_2-adrenoreceptor agonists are often combined with other drugs, mainly opioids, to improve quality of sedation (Table 22-2).

Administration, Doses, Routes

α_2-Adrenoreceptor agonists can be administered intravenously (IV) or intramuscularly (IM). Peak effects are reached within the first 3 to 10 minutes with the IV route. For IM administration, approximately twice the dose should be used to achieve the same effect. Time to peak effect may be as long as 30 to 60 minutes.[11] Unlike romifidine,[12] detomidine produces clinical sedation following sublingual administration.[13-15] However, the oral route is less predictable and may require up to 45 minutes for maximum drug effect.[13]

The IV route is the most predictable and should be the route of choice whenever possible. Compared to the IV route, after IM administration, the initial hypertensive response associated with α_2-adrenoreceptor agonists is less dramatic.[9] Therefore, if hypertension is undesirable (i.e., risk of bleeding associated

TABLE 22-1. Recommended Dosages for the Use of α_2-Adrenoreceptor Agonists in Horses

Drug	Dose (mg/kg) and Route (IV)	Comments	Reference
Xylazine	0. 55, 1.1	Stimulation by needle prick	21
Xylazine	2.2	After a period of maximal exercise Administration to effect recommended	33
Detomidine	0.005-0.02	Clinical study: – Radiology: <0.01 – Routine sedation: 0.01 – Difficult horses, prolonged sedation: 0.02	98
Detomidine	0.01-0.02	Normally effective if administered to calm and resting horses	Personal experience
Detomidine	0.01-0.04	Stimulation	99
Detomidine	0.02	Routine dental treatment 3/10 fell down	28
Detomidine	0.04	After a period of maximal exercise To effect recommended	33
Detomidine	0.01	Diagnostic anesthesia lameness examination Can be antagonized by 0.1 mg/kg atipamezole IV	63
Romifidine		Tooth rasping:	28
	0.08	1/10 fell down	
	0.12	1/10 fell down	
Romifidine	0.04-0.08	Stimulation	99

Note: The horse's disposition and physical status will affect dose requirements. If α_2-adrenoreceptor agonists are used alone, even if horses appear deeply sedated they may react violently to stimulation; therefore, α_2-adrenoreceptor agonists are frequently combined with opioids or acepromazine. For painful procedures, local or regional analgesia and systemic analgesics should be added.

TABLE 22-2. Recommended Sedation Protocols

Drugs	Dosage (mg/kg) and Route (IV)	Comments	References
α_2-ADRENORECEPTOR AGONIST + OPIOID			
Xylazine	0.7	Unstimulated horses	44
Methadone*	0.1		
Xylazine	0.7	Unstimulated horses	44
Buprenorphine†	0.004, 0.006		
Xylazine	0.4	Clinical cases	Personal experience
Levomethadone‡	0.05		
Xylazine	0.5	Clinical cases	Personal experience
Butorphanol§	0.02		
Detomidine		Nociceptive threshold test	37
Butorphanol	0.025		
Detomidine	0.01-0.03	Laparoscopy with local/regional anesthesia	100, 101
Butorphanol	0.01-0.02		
Detomidine	0.013	Clinical cases	45
Butorphanol	0.026		
Romifidine	0.045	Clinical cases	46
Butorphanol	0.02	Experimental study; pain test	40
Romifidine	0.08	Experimental study; pain test	41
Butorphanol	0.025		
Romifidine	0.08	Experimental study; pain test	41
Levomethadone	0.1		
Romifidine	0.044	Experimental study; pain test	40
Morphine¶	0.1		
Detomidine	0.01	Nociceptive threshold test	37
Levomethadone	0.1		
α_2-ADRENORECEPTOR AGONIST + ACEPROMAZINE			
Xylazine	0.55	Experimental study; pain test	49
Acepromazine	0.05		
Detomidine	0.01	Experimental study for endoscopic evaluation of laryngeal function and effects of sedation on laryngeal function	25
Acepromazine	0.05		
Xylazine	2.2	After period of maximal exercise	33
Acepromazine	0.04	To effect recommended	
Xylazine	0.4		Personal experience
Acepromazine	0.02		
Levomethadone	0.05		
Xylazine	0.4		Personal experience
Acepromacine	0.02		
Butorphanol	0.02		
ACEPROMAZINE + OPIOID			
Acepromazine	0.05	Mild sedation	44
Buprenorphine	0.006	Arousable by loud noise	

Note: The responses of individual horses may vary in specific situations. For painful procedures local or regional analgesia and systemic analgesics should be added.
*Methadon Streuli.
†Temgesic.
‡L-Polamivet ad us. vet.
§Alvegesic 1% forte ad us. vet.
¶Morphin HCl sintetica 10 mg.

with guttural pouch mycosis) the IM route may be preferable. Also in fractious horses, where intravenous injection may be difficult, the IM or oral route may offer an advantage.

Other Effects and Side Effects

Administration of α_2-adrenoreceptor agonists is associated with dose dependent potent cardiovascular side effects including significant changes in blood pressure (BP) and vascular resistance, decreases in cardiac output (CO), and rapid and significant decrease in heart rate (HR) with development of first- and second-degree atrioventricular (AV) block (Figure 22-1) and occasionally other arrhythmias. Changes in BP are characterized by an initial hypertension followed by hypotension.[4,7,9,16-20] The use of atropine (Atropinsulfat 0.1%) to treat AV blocks is normally not necessary. As a matter of fact, its use is controversial because of dose-dependent tachycardia, hypertension, and side effects on the gastrointestinal tract.[21,22]

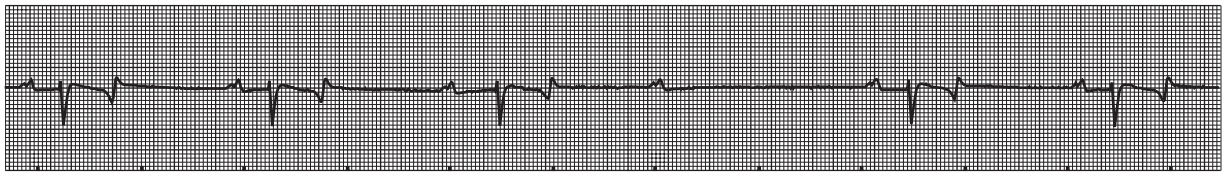

Figure 22-1. Base-apex electrocardiogram (paper speed 25 mm/sec) showing a second-degree atrioventricular block (Wenckebach, Mobitz type I) in a horse. This is a typical arrhythmia observed in horses sedated with α_2-adrenoreceptor agonists. (Courtesy Dr. C. C. Schwarzwald, University of Zurich.)

Respiration is depressed, but the effect is usually not apparent unless other drugs are co-administered or anesthesia is induced. Some horses may demonstrate signs of increased inspiratory effort or initiation of snoring, probably as a result of facial and nasal edema caused by a lowered head position and relaxation of the muscles in the larynx and nares.[12,23,24] The influence of α_2-adrenoreceptor agonists on laryngeal function need to be considered during diagnosis and evaluation of laryngeal hemiplegia.[25]

Other side effects of α_2-adrenoreceptor agonists are reduced gastrointestinal function, hyperglycemia with decreased serum insulin levels, and increased urine output.[9] The penis is relaxed and extended in males and the uterine tone increased in females.[9] The use of α_2-adrenoreceptor agonists has not been associated with an increased risk of abortion in horses.[26,27] Normally horses do not become recumbent, but ataxia may be severe and some cases of tumbling down have been described after α_2-adrenoreceptor agonist administration[28] and after a combination of high doses of α_2-adrenoreceptor agonists with opioids.[29-32]

Intracarotid injection of α_2-adrenoreceptor agonists results in excitement, disorientation, ataxia, violent collapse, recumbency accompanied by paddling, and possibly seizures. Normally horses recover spontaneously within a few minutes. If seizure activity does not stop within a few minutes, symptomatic therapy should be initiated. Benzodiazepines, guaifenesin (Myolaxin 15% ad us. vet.), and thiopental (Pentothal) have been used to control persistent seizures. Oxygen administration may be beneficial.

Special Considerations and Contraindications

Failure to achieve sedation with α_2-adrenoreceptor agonists is often caused by preexisting stress, fear, excitement and pain. These conditions increase endogenous catecholamine levels, which can interfere with the mechanism of action of the α_2-adrenoreceptor agonists. α_2-Adrenoreceptor agonists should be administered to calm horses in quiet surroundings with minimal environmental stimuli. The horse should not be stimulated (clipping, cleaning or injection of local anesthesia) until sedation is appropriate. Repeating or administering large doses may not increase the degree of sedation.[9]

Dosages of α_2-adrenoreceptor agonists that are effective in horses at rest are frequently ineffective when administered to horses after maximal exercise. Twice the standard dosages have been used effectively and safely in healthy Thoroughbred horses after maximal exercise (see Tables 22-1 and 22-2).[33,34] However, lower doses of sedatives may be effective in some horses, and when possible, sedatives should be administered in smaller doses to effect, possibly starting with the standard dose.[33] Draft horses are very susceptible to α_2-adrenoreceptor agonists, and lower dosages must be considered. In patients with impaired cardiovascular function, cardiac disease or arrhythmias caused by other pathologies (e.g., hyperkalemia), α_2-adrenoreceptor agonists should be used with caution.

Potentiated sulfonamides should never be administered IV following α_2-adrenoreceptor agonists,[35] as fatal arrhythmias might occur.

Foals

α_2-Adrenoreceptor agonists are not recommended in very young foals (less than 14 days). In very sick foals younger than 3 months, α_2-adrenoreceptor agonists should be avoided. In these animals benzodiazepines or benzodiazepines combined with opioids provide safe and effective sedation. In foals older than 30 days without systemic disease, presented for wound revision or casting, α_2-adrenoreceptor agonists can be used safely if doses are given to effect (Table 22-3).

Combinations with Other Drugs

Horses sedated with α_2-adrenoreceptor agonists may appear profoundly sedated but react violently to stimulation, especially to touch with rapid and well-directed kicks. Afterwards, if

TABLE 22-3. Recommended Sedation Protocols for Foals

Drug	Dosage (mg/kg)	Comments
AGE YOUNGER THAN 2 MONTHS OR 2-3 MONTHS AND BAD GENERAL CONDITION		
Climazolam	0.1-0.2-0.4 IV	No analgesia
Midazolam	0.05-0.1-0.2 IV	No analgesia
Diazepam	0.05-0.2-0.3 IV	No analgesia
AGE 2-3 MONTHS AND GOOD GENERAL CONDITION		
Xylazine	0.2-0.5 IV	
Levomethadone	0.05 IV	
Xylazine	0.2-0.5 IV	
Butorphanol	0.01-0.02 IV	
ANTAGONIST BENZODIAZEPINES (CLIMAZOLAM, MIDAZOLAM, DIAZEPAM)		
Sarmazenil	0.04 IV	
Flumazenil	0.04 IV	

Note: For painful procedures, benzodiazepines should be combined with an opioid, for example, butorphanol 0.02 mg/kg IV, and local or regional anesthesia should be added whenever possible. If young foals are sedated for prolonged periods, hypoglycemia and hypothermia may be important concerns requiring antagonization with the help of benzodiazepines.

otherwise undisturbed, they become well sedated again.[9] The combination of opioid drugs with the α_2-adrenoreceptor agonists appears to reduce this response and a synergistic effect regarding sedation and antinociception has been reported.[30,31,36-41] Therefore α_2-adrenoreceptor agonists are often combined with opioids (see Table 22-2).[30,31,36-46] The addition of opioids does not further impair cardiovascular function.[36,40,43] Changes in blood gases have been observed after the addition of opioids to α_2-adrenoreceptor agonists probably because of increased respiratory depression, but not to a clinically relevant extent.[36,47] If high doses of opioids are used, side effects such as excitement (muscle twitching, muzzle tremors, head pressing, increased locomotor activity and circling) can be observed. [30,31,36,38,48] Also ataxia may be increased after the addition of higher doses of opioids to α_2-adrenoreceptor agonists.[29-32]

Acepromazine (Prequillan) has also been combined with α_2-adrenoreceptor agonists or added to a α_2-adrenoreceptor agonist/opioid combination to improve sedation (see Table 22-2).[49,50] Unfortunately, only few studies are available regarding the effect of acepromazine on horses already sedated with α_2-adrenoreceptor agonists, and no clinical study evaluating the addition of acepromazine exists. Very high doses of a combination of xylazine with acepromazine have been used in horses after exercise.[33]

The addition of acepromazine to an α_2-adrenoreceptor agonist prevents the initial increase in arterial blood pressure seen when α_2-adrenoreceptor agonists are used alone.[49,51] Also, an improved cardiac output and a decreased drop in HR have been reported.[51,52] Vasoconstriction induced by α_2-adrenoreceptor agonists is overcome by acepromazine's vasodilative properties, but the exact mechanism by which acepromazine maintains circulatory variables closer to baseline values during sedation with an α_2-adrenoreceptor agonist remains unclear.[51] Arterial oxygenation is better if acepromazine is added.[51]

Uses as a Constant-Rate Infusion

The use of an α_2-adrenoreceptor agonist as a CRI is becoming more and more popular because of more constant sedation and fewer side effects compared to repeated bolus administration.[4] Detomidine, medetomidine, xylazine, and romifidine CRI have been described for standing sedation of horses (Table 22-4).[4,32,53-56]

Opioids, acepromazine, and ketamine (Narketan 10 ad us. vet.) have also been used in combination with CRIs of α_2-adrenoreceptor agonists (see Table 22-4).[6,55-57] Although described in the literature, ketamine should never be given as a bolus or loading dose to awake standing horses, and only low CRI dosing regimens should be used.

Whenever CRI is used, the level of sedation should be assessed frequently and drug delivery adjusted to effect by decreasing or discontinuing the infusion temporarily if weakness or instability occurs or by giving small additional boluses of the α_2-adrenoreceptor agonist or opioid if sedation or analgesia is insufficient. If upper airway obstruction is severe, the head should be elevated. To treat mucosal edema, nasal drops containing phenylephrine (5 mL of 0.15% phenylephrine [Phenylephrin HCl 1.5 mg/mL] into each nostril of a 500-kg horse) can be used or long nasal tubes inserted into the nostrils to maintain patent airways (Figure 22-2).

Antagonists

α_2-Adrenoreceptor agonist can be antagonized by α_2-adrenoreceptor antagonists. The antagonists used in equine practice are yohimbine (Yohimbine hydrochloride), tolazoline (Tolazolin HCl 120 mg/mL) and atipamezole (Antisedan) (Table 22-5). Atipamezole is the most selective α_2-adrenoreceptor antagonist. Severe side effects can occur and antagonists should

TABLE 22-4. Protocols for Prolonged Sedation using a Constant Rate Infusion (CRI)

Drugs	Loading Dose (µg/kg) IV	CRI (µg/kg/hr) IV	If Insufficient Sedation (µg/kg) IV	Comments	Reference
Medetomidine	5	5	0.6	Exploratory laparoscopy	6
Morphine	after 10 min: 50	30			
Detomidine	10	9.6		Laparoscopy	57
Buprenorphine	6				
Detomidine	7.5	36; dose halved every 15 min	6 detomidine or 19 butorphanol	Different clinical procedures	56
Detomidine	10-20	20	2.2-4.4	Different clinical procedures	102
Morphine	100-150	25-50	16.7-34		
Ketamine	No loading dose	600			
Detomidine	10-20	20	2.2-4.4	Different clinical procedures	102
Butorphanol	11-16	22	5-7		
Ketamine	No loading dose	600			
Acepromacine	55-88	No CRI		Different clinical procedures	102
Morphine	100-150	25-50			
Ketamine	No loading dose	600			
Medetomidine	5	3.5		Undisturbed horses	4
Romifidine	80	30		Undisturbed horses	55
Romifidine	80	29		Undisturbed horses	55
Butorphanol	18	25			
Xylazine	1100 (= 1.1 mg/kg)	690		Undisturbed horses	32

Note: For painful procedures, local or regional analgesia and systemic analgesics should be added.
CRI, Constant-rate infusion.

TABLE 22-5. Dosages of α_2-Adrenoreceptor Antagonists Used in Horses

Drug	Dosage (IV)	Comments	References
Atipamezole	Moderate sedation: 0.1 mg/kg	After detomidine 10 µg/kg	62
	Profound sedation: 0.16 mg/kg	After detomidine 20 µg/kg	62
	0.1 mg/kg	After 20 µg/kg detomidine; not complete reversal	61
	0.08 mg/kg	After 10 µg/kg medetomidine	103
	0.06 mg/kg	After medetomidine CRI	4
	0.15 mg/kg	After xylazine 1 mg/kg	104
Tolazoline	4 mg/kg	After detomidine 40 µg/kg; side effects are common	60
	4 mg/kg	After 20 µg/kg detomidine; apprehension in 2/6 horses	61
	7.5 mg/kg	After xylazine CRI	59
Yohimbine	0.12 mg/kg	After xylazine CRI; 2/4 horses had agitation, muscular tremors, and mild excitement	59

Note: Selection of drug and dosage should depend on type and dose of α_2-adrenoreceptor agonist used and time elapsed since administration. The dosage should also be adapted to the degree of sedation. The drugs used should be administered very slowly IV and to effect.
CRI, Constant-rate infusion.

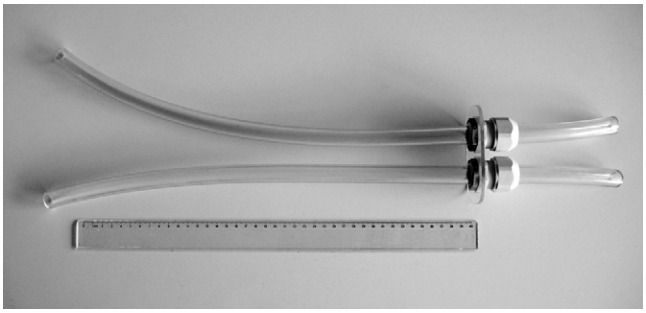

Figure 22-2. Nasal tubes that can be used to facilitate breathing in case of upper airway obstruction caused by nasal mucosal swelling (e.g., during prolonged sedation with low head position). The longer portion is inserted into the ventral meatus.

only be used as a rescue (if possible, slowly to effect), if an exaggerated or unexpected pharmacological or behavioral response occurs, or in case of a severe overdose of α_2-adrenoreceptor agonist.[58] By administering α_2-adrenoreceptor antagonists, the analgesic effect of α_2-adrenoreceptor agonists will be abolished, and cardiovascular and behavioral side effects can occur. Agitation, stress response and overreaction have been noted after all three α_2-adrenoreceptor antagonists listed earlier.[59-62] Dosages of antagonists should be reduced if more selective α_2/α_1 agents are used (medetomidine), if low dosages of α_2-adrenoreceptor agonists where used, and if the agonists were administered more than 45 minutes previous to reversal.

After small dosages of detomidine (10 µg/kg IV) to facilitate anesthetic blocks for lameness diagnosis, sedation can be antagonized with atipamezole 0.1 mg/kg IV to allow continued evaluation.[63]

Phenothiazines

Mechanism of Action

The phenothiazines exert their tranquilizing effect primarily by blocking dopamine receptors in the basal ganglia and limbic system. They produce a calming effect, indifference, and decreased locomotor activity.[64] Phenothiazines do not produce analgesia, and sedation is less than after α_2-adrenoreceptor agonists, rarely achieving reliable restraint in horses.[65]

Uses

The most commonly used phenothiazines in horses are promazine and acepromazine. Acepromazine is a potent phenothiazine derivate and is extensively used in equine practice. Chlorpromazine, propiopromazine (Tranvet Injectable Solution), propionylpromazine (Combelen) and promethazine are no longer used because of their unpredictability and side effects.[64]

Because phenothiazines have no potent tranquilizing effect they should be combined with opioids, α_2-adrenoreceptor agonists (see earlier), or both for more reliable sedation, or they should be used as a sole agent for nonpainful procedures like shoeing, assisted training in young horses, or tranquilization for transport. The phenothiazine acepromazine is a frequently used premedicant before general anesthesia because it reduces MAC,[66] has an antiarrhythmic and antifibrillatory effect,[67] and decreases the anesthetic risk in horses.[1,68]

Acepromazine increases digital blood flow,[69,70] and because of this it has been incorporated into the treatment and/or prevention of laminitis.[71] It has also been used in horses to induce extrusion of the penis from the sheath for examination.[72]

Acepromazine can reduce or inhibit opioid-induced excitement and manic behavior.[73] A positive effect in treatment and prevention of malignant hyperthermia has also been reported.[64]

Administration, Dosages, Routes

Acepromazine can be administered orally, IM, or IV. The degree of tranquilization is related to the dosage and route of administration, with IM injections having a lower maximal effect but longer duration of action than a similar IV dose.[74]

Recommended dosages are 0.02 to 0.05 mg/kg IV or IM. Some cases of excitement and extrapyramidal signs have been reported after higher doses.[64,75] Oral bioavailability is high, and it may be as high as for the IM route. The maximal recommended oral dosage is 0.1 mg/kg.[76,77]

Peak effects are reached after 10 to 30 minutes, 20 to 40 minutes, and 1 hour after IV, IM, and oral administration, respectively. Therefore, acepromazine needs to be administered several minutes before faster-acting drugs such as α_2-adrenoreceptor agonists or opioids if a simultaneous effect is

desired. The effects of acepromazine may last as long as 6 to 10 hours depending on the horse, or even longer with very high doses.[74]

Side effects seem to be less severe after oral or IM administration compared to the IV route, although this may be dose-dependent.[74,76]

Other Effects and Side Effects

Phenothiazine tranquilizers produce hemodynamic effects predominantly related to their α_1-adrenergic blocking activity and resultant arterial hypotension.[49,65,74,78,79] The route and dosage of acepromazine have some influence on the degree and duration of hypotension.[74] After administration via the IM route, hypotension is of slower onset but longer duration compared to the IV route.[74] Hypotension is less of a problem in fit and healthy animals, but horses that are stressed or anxious may be more susceptible to the hypotensive effect, probably because of increased circulating concentrations of epinephrine. Acepromazine blocks α_1-adrenoreceptors receptors, and this combined with β_2-adrenoreceptor vasodilating effects caused by epinephrine may result in "epinephrine reversal," resulting in further decrease in blood pressure.[64] If necessary, hypotension can be successfully treated with fluids. If fluids are not sufficient, the α_1-adrenoreceptor agonist phenylephrine (Phenylephrin HCl 1 mg/mL) should be used to counteract the vasodilation; epinephrine (Adrenalin, Sintetica) should never be used (see earlier "epinephrine reversal"). The effect of acepromazine on HR is controversial; some authors report a decrease,[79] others no change[49] or even an increase in HR.[65,74] The overall effect on the HR does not seem to be clinically important, and the increase observed in some horses may be compensation for hypotension.[74]

Acepromazine produces little respiratory depression and the effect is primarily on respiratory rate with no effect on pH or blood gas tensions.[49,65,74,79,80]

Acepromazine has an important effect on packed cell volume (PCV).[72,79,81] After clinically relevant doses, the PCV decreases by around 20% from presedation values or even more. The decrease is attributed to α-adrenergic receptor blockade leading to splenic capsular relaxation with subsequent red blood cell sequestration, and dilution by interstitial water entering the vascular compartment as a result of hypotension.[64] The decrease in PCV is independent of the route of administration. At dosages less than 0.01 mg/kg, the magnitude of PCV decrease is dosage dependent, whereas at dosages more than 0.01 mg/kg there is only an effect on duration of the decrease.[72,81]

Dosage dependent penile protrusion and penile prolapse have been observed in horses treated with dosages of 0.01 mg/kg and higher.[72] Priapism and paralysis of the retractor penis muscle have been attributed to the use of phenothiazine tranquilizers.[82-84] However, cases also have been described in horses anesthetized without the use of phenothiazines.[85] Whether this complication is dosage dependent is unknown. Also, administration of a phenothiazine derivative without subsequent priapism in an individual horse does not eliminate the possibility of priapism in the same horse after repeated administration of the same drug in the future.[86] Priapism and paralysis seems to be more frequent in stallions than in geldings,[83] although cases of priapism in geldings have been described.[82,84,85,87]

If priapism is observed, blood should immediately be forced out of the penis by manual compression, with the goal to reposition the penis back into the prepuce where it can be retained with a purse-string suture around the prepuceal orifice.[82] Benztropine mesylate (Cogentin) (0.01 to 0.02 mg/kg slowly IV) may be effective in resolving priapism.[84,85] Surgical treatment by creating a shunt between the corpus cavernosum penis and the corpus spongiosum penis has also been described.[86,88] Until the penis is completely retracted, frequent cooling and massage may be beneficial. Overall, treatment should be oriented toward protecting the penis and preventing or reducing swelling. Additional information on this subject can be found in Chapter 60.

Other effects of acepromazine including loss of thermoregulatory control and decreased gastrointestinal motility have been described. The loss of thermoregulatory control should be considered in small horses (ponies, foals), particularly when ambient temperatures are very high or very low.

Because phenothiazines may reduce the seizure threshold, they should be avoided in epileptic patients.[89] However, this contention has been challenged and is discussed controversially in the literature.[90]

The same effects as with α_2-adrenoreceptor agonists are observed after accidental intracarotid injection of phenothiazines (see "α_2-Adrenoreceptor Agonists," earlier).

Special Considerations and Contraindications

The use of phenothiazines is restricted to "healthy" animals, because reduction in blood pressure could impair tissue perfusion, particularly in systemically compromised horses.

Phenothiazines are contraindicated in patients with hypovolemia, horses with low PCV, patients at risk of bleeding because of type of surgery or of bleeding disorders, and in young or debilitated foals. The risks and benefits of their use should be weighed very carefully in breeding stallions and animals with a history of seizures. Acepromazine should be used cautiously in stressed horses.

It is important to remember before sedating a horse with acepromazine that the negative effects on blood pressure and PCV can persist for more than 10 hours[74,81] and there is no antagonist available.

Foals

Foals may not be able to compensate for the hypotension induced by phenothiazines. Also hypothermia may be a problem. Acepromazine is acceptable in older, healthy foals if long-term sedation is necessary. However, it is not recommended in very young or compromised foals.

Combinations with Other Drugs

Acepromazine can be combined with opioids and α_2-adrenoreceptor agonists or both to produce more predictable and prolonged sedation (see Table 22-2).[25,44,49,50,52] Cardiovascular function is good after adding acepromazine to α_2-adrenoreceptor agonists and it even seems to prevent some cardiovascular side effects induced by α_2-adrenoreceptor agonists.[49,52]

A combination of a high dosage of α_2-adrenoreceptor agonists with 0.04 mg/kg acepromazine has been used successfully in horses after exercise (see Table 22-2).[33]

Antagonists

No specific antagonist exists.

Butyrophenones

Butyrophenones are not recommended in horses because of their lack of predictability and severe side effects, especially on behavior. Droperidol (Inapsine), lenperone (Elanone-V), and azaperone (Stresnil ad us. vet.) have been studied in horses.[64]

Benzodiazepines

The benzodiazepines diazepam (Valium 10 mg), midazolam (Dormicum), climazolam (Climasol ad us. vet.), and zolazepam (in combination with tiletamine) (Zoletil ad us. vet.) have been used in horses.

Mechanism of Action

Benzodiazepines produce their pharmacologic effects by facilitating the actions of γ-aminobutyric acid (GABA), the principal inhibitory neurotransmitter in the central nervous system.[91] These drugs produce minimal calming effects with excellent muscle relaxation and minimal to no cardiovascular or respiratory depression in awake horses.[92] Benzodiazepines also have an anxiolytic, hypnotic, and anticonvulsive action but no analgesic effects.[64]

Uses

Benzodiazepines should not be used for standing sedation in adult horses because of unreliable sedation and severe ataxia.[92] On the other hand, for neonatal foals, they are reliable and good sedatives (see Table 22-3).

Benzodiazepines are also used as adjuncts to general anesthesia and as anticonvulsants in equine practice (see Chapters 18 and 19).[93]

Very low dosages of diazepam (up to 0.01 mg/kg IV) have been used to produce short-term tongue or jaw relaxation for dental treatments[94] and to increase libido in stallions.[95] Benzodiazepines can produce severe ataxia at higher dosages.

Administration and Routes

Diazepam, midazolam, and climazolam can be administered by the IV route. If only IM administration is possible, midazolam should be used.

Other Effects and Side Effects

After benzodiazepine administration, healthy adult horses may become anxious, apprehensive, and agitated. Also severe ataxia and recumbency have been observed after benzodiazepine administration in adult horses.[92] Benzodiazepines can stimulate food intake in horses.[96]

Foals

Foals become recumbent and reliably sedated with benzodiazepines. Benzodiazepines provide no analgesia, and therefore these drugs should be combined with opioids and/or local/regional anesthesia whenever surgical intervention is planned. If young foals are sedated over prolonged periods, hypoglycemia and hypothermia may be a concern. Because of this, antagonism of benzodiazepines is often recommended at the end of a procedure. Antagonists should also be used if dangerous respiratory depression develops.

Antagonists

Flumazenil (Anexate) (0.01 to 0.04 mg/kg slowly IV) and sarmazenil (Sarmasol) (0.04 mg/kg slowly IV) produce rapid reversal.

Opioids

Opioids are never used as sole sedatives in healthy pain-free horses because dosage-dependent excitement or dysphoria can occur. During standing restraint for surgical procedures opioids are used in combination with sedatives to improve sedation and analgesia (see Table 22-2). Opioids are discussed in detail in Chapter 23.

Mechanism of Action

Opioids act via different opioid receptors (see Chapter 23).

Uses

The combination of opioids with other sedatives such as acepromazine and α_2-adrenoreceptor agonists provides very good quality standing restraint in horses (see Table 22-2). In foals, opioids are frequently administered in combination with benzodiazepines for sedation.

Opioids can also be used for balanced general anesthesia protocols (see Chapter 18) and as systemic or local/regional analgesics (see Chapter 23).

Other Effects and Side Effects

When used alone, opioids can produce restlessness, agitation, shivering and increased sympathetic activation, motor activity, and ataxia in horses.[97] These excitatory effects are less common when α_2-adrenoreceptor agonists or acepromazine are coadministered. The characteristic dosage-dependent increase in "spontaneous" locomotor activity has been blocked or dampened by the administration of naloxone (Narcan) or acepromazine.[73]

Opioids can produce important side effects on the gastrointestinal tract of horses, dosage-dependent effects on cardiopulmonary system, and histamine release.[64] For more details see Chapter 23.

ADJUVANTS

Different adjuvants such as lidocaine (Lidocain HCl 5%) or ketamine CRI can be used in combination with sedatives to improve quality of restraint and analgesia, although no clinical studies exist.

LONG-TERM TRANQUILIZATION

Sometimes injured horses or horses recuperating from major surgery need to be rested in a box stall for long time periods, which may be very difficult, depending on the character of the

individual horse. Long-acting sedatives have been used to tranquilize nervous or fractious horses for several weeks. Drugs like reserpine, fluphenazine, and chlorpromazine have been considered for this purpose, although they may be accompanied by several important side effects and these drugs are not sufficiently studied in the equine species.[93]

CONCLUSION

There is no perfect drug for a specific situation or a specific horse and therefore normally a multimodal approach is beneficial. Sedatives should be combined with local/regional anesthesia and analgesics (e.g., opioids and nonsteroidal anti-inflammatory drugs) whenever painful interventions are planned.

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CHAPTER 23

Equine Pain Management

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PHYSIOLOGIC CONSEQUENCES OF PAIN

The assessment and alleviation of pain in animals is an important part of the veterinary surgeon's role for medical, ethical, and welfare reasons. Pain is a complex, dynamic, physiological, and emotional phenomenon that can be extremely challenging to manage and causes suffering and distress to the animal. It can produce many undesirable effects in both the short and long term. Damage to body tissues as a result of trauma, surgery, or disease processes stimulates neuroendocrine responses, namely increased sympathetic outflow from the spinal cord, increased release of hormones (including adrenocorticotropic hormone, cortisol, and renin), and decreased release of insulin and testosterone. Clinically, this may result in adverse effects on wound healing and predispose to infection. Weight loss because of decreased food intake and increased protein catabolism may be dramatic and the change in feeding behavior, coupled with a significant reduction in gastrointestinal motility secondary to pain, may result in the development of colic. The increase in sympathetic tone upregulates heart rate, which in turn increases myocardial oxygen consumption; causes vasoconstriction, elevating blood pressure, and may lead to potentiation of arrhythmias. An increase in blood pressure may have deleterious effects, for example by upregulating intraocular pressure following intraocular surgery or by increasing bleeding at a surgical site.

Horses' "fight or flight" nature means that they often react to severe pain by behaving violently. This can make them difficult and dangerous to handle with the potential for serious injury to both handler and horse. Even in less extreme cases, horses may still act unpredictably, making thorough examination and treatment a challenge.

One significant long-term adverse consequence of acute pain is the development of chronic or maladaptive pain; a disease process in itself. In this situation, pain persists after the injury has apparently healed. Chronic pain may also be associated with a long-standing pathological process, such as laminitis, although the severity of the clinical condition may not correlate with the degree of pain experienced by the animal. Chronic pain is incredibly difficult to manage because it is often not responsive to "traditional" analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids.

There are different types of pain: physiological, inflammatory, and chronic. Horses may experience more than one type of pain simultaneously.

ACUTE PAIN

Acute pain, following tissue trauma or application of a noxious stimulus, is the result of activation of peripheral sensory nerve endings acting as sensory receptors (nociceptors). Nociceptors have been described in most structures of the body, including the skin and viscera. They transduce and encode noxious stimuli and send impulses to the dorsal horn of the spinal cord via small myelinated (Aδ) and thinly and nonmyelinated (C) afferent nerve fibers. After reaching the dorsal horn of the spinal cord, the Aδ and C fibers from superficial pain pathways (e.g., somatic structures, such as skin) are transmitted via the spinocervicothalamic tract,[1] whereas those from the deep pain pathways (e.g., viscera and periosteum) are transmitted via the spinoreticular tract.[2] Pain is perceived by the animal when the signals reach the somatosensory cortex.

The mechanisms of nociception are complex and dynamic, and modulation, either facilitation or inhibition of the signals, can occur within the central nervous system (CNS). Descending pathways originate in the brain within areas such as the periaqueductal grey matter and the ventromedial medulla and interact with the ascending pathways, particularly at the level of the spinal cord, to form a "negative feedback loop." Neurotransmitters involved in descending inhibitory pathways include glutamate, norepinephrine, serotonin, γ-aminobutyric acid (GABA), and endogenous opioids. The premise behind the *gate theory*, described by Melzack and Wall in 1965,[3] is that the input to the brain is the sum of the excitatory and inhibitory inputs. Therefore, facilitation of inhibitory pathways can be exploited to provide analgesia.

Peripheral Sensitization

Prolonged stimulation of the pain pathways can result in alterations often referred to as peripheral and central sensitization. Peripheral sensitization occurs at the level of the injured tissue. Tissue damage results in release of inflammatory mediators including prostaglandins, bradykinin, histamine, hydrogen ions, potassium ions, and substance P. These mediators may stimulate peripheral nerve endings (nociceptors) directly, or they may increase the sensitivity of the nociceptor to other stimuli. Clinically, peripheral sensitization may result in an avoidance of or aggressive response to a stimulus that would normally be considered innocuous (e.g., light touch). This is referred to as *allodynia*.

Central Sensitization

Plasticity within the spinal cord and brain means that nociceptive pathways may be upregulated in response to acute pain and inflammation. The physiological mechanisms behind central sensitization are complex but ultimately result in enhanced pain transmission by neurons. One of the most important mechanisms is thought to be related to *N*-methyl-D-aspartate (NMDA) receptor stimulation by the neurotransmitter glutamate. The result is hyperalgesia; an exaggerated response to a noxious stimulus. Secondary hyperalgesia occurs in the surrounding noninjured area. When these changes have occurred, effective pain control is more difficult and higher doses of analgesics are required.

CHRONIC PAIN

Central sensitization plays an important role in the development of chronic pain. The mechanisms behind chronic pain are

pathological processes and do not benefit the animal. Chronic pain has significant welfare implications because it can be difficult to diagnose and treat effectively. Following surgery in people, the incidence of the development of chronic pain may exceed 50%.[4] Many procedures associated with the development of chronic pain in people, such as dental surgery and hernia repair, also are commonly performed in horses, so it is reasonable to assume that chronic pain following surgery may also occur in this species. Preemptive and multimodal analgesia—the preoperative administration of analgesics acting on different parts of the nociceptive pathways—may attenuate both peripheral and central sensitization, reduce postoperative pain (compared to when the same analgesics are administered after surgery), and decrease the incidence of chronic pain.

A more commonly recognized cause of chronic pain in horses is laminitis. Laminitis is associated with peripheral sensory nerve damage, which results in chronic pain with a neuropathic component.[5] Pain associated with laminitis can be severe, and failure to adequately control pain may necessitate humane destruction of the horse.

PAIN ASSESSMENT

It is essential to be able to assess and quantify pain to determine when intervention is required and to assess the efficacy of administered analgesics. Physiological variables, such as heart rate and respiratory rate, are easily measured and are objectively quantifiable. However, they have been poorly correlated with subjective assessments of pain in horses[6] so, rather than relying on them in isolation, they should be considered in light of the animal's clinical condition and drug treatments and used as part of a holistic pain assessment.

The development of pain scales for use in horses lags behind progress made in the field in small animals. The reproducibility, specificity, and sensitivity of several parameters selected from a review by Ashley et al[7] have been established in the development of a composite orthopedic pain scale in horses.[8] This scale requires further validation in a clinical setting, but it has the potential to be a useful tool and a helpful basis for further work in this area.

In the meantime, accurate quantification of a horse's pain remains very difficult, but implementing a standardized approach to assessing each individual horse will assist in identifying of signs of pain and has the potential to significantly improve pain management.

Ideally, the same trained person should perform all assessments for each horse to minimize interobserver variability. Demeanor is commonly used for subjective pain assessment in horses[9] and may be influenced by the type of pain that the horse is experiencing. For example, horses with severe abdominal pain may appear excited and violent, whereas horses in significant pain due to laminitis may appear to be very quiet and depressed. The horse's demeanor, posture, and activities should be evaluated first, preferably with the observer outside the sight of the horse, although this is often not possible. Behavioral signs that may indicate pain include low head carriage, horse positioned at the back of the stable, vocalization (groaning, neighing), agitation, restlessness, weight shifting, lifting a limb, tail swishing unrelated to the presence of flies, abnormal distribution of body weight (e.g., uneven weight-bearing on limb), a "tucked-up" appearance, and looking at a painful body part. Interaction between horses housed in groups can also give the observers useful information about the horse's emotional state. More subtle signs that may not be visible from a distance include bruxism, increased jaw tone, sweating, and muscle fasciculations.

In an attempt to get a more complete view of the pain that the animal is suffering, the pain assessment protocol should incorporate dynamic and interactive components.[10] The observer should then approach the horse while noting the horse's response and willingness to interact. In some cases, tissue damage and inflammation is obvious, and wherever possible the examination should include observation of the horse's response to palpation of both the injured and surrounding areas. It is also often helpful to palpate tissue at a site remote from the injury to see how the horse responds to painless touch. In cases where the source of pain is not immediately apparent, palpation may elicit a pain response and aid diagnosis.

Food and water consumption should be monitored. Watching horses eat may give useful information in cases with dental pain and indicate whether a horse is willing to bear weight on its thoracic limbs and lower its head to the ground to eat.

In appropriate cases, the willingness to walk as well as the gait itself should be assessed. Lameness may be immediately apparent in horses with limb pain, but gait is often influenced by pain in other parts of the body. For example, pain following castration may result in reluctance to move and/or a short pelvic limb stride length.

Chronic pain can be even more challenging to diagnose than acute pain, and behavioral signs and changes may be more subtle. In the long term, loss of weight and muscle wasting may be evident. The use of an algometer (Figure 23-1) to assess induced back pain in horses by quantification of the pressure required to elicit a withdrawal response has been described.[11] This objective measure could potentially be a useful method to help monitor the response to treatment in horses with chronic musculoskeletal conditions. Pain intensity and nature change over time. Regularly reassessing the animal is very important to help monitor progression and to ensure that the selection and dosages of analgesics are appropriate.

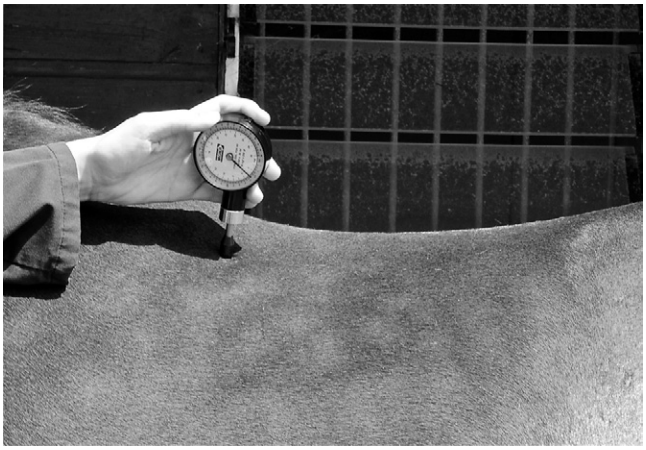

Figure 23-1. Pressure algometry in a horse. The algometer is applied to the horse's skin in the area of interest and pressure is applied until a response (skin twitch, local muscle contraction, or movement of the horse) is observed: "threshold pressure." The algometer is removed from the skin as soon as the response is observed.

ANALGESIA PROTOCOLS

Drugs commonly used for analgesia in horses include nonsteroidal anti-inflammatory drugs (NSAIDs), α_2-adrenoceptor agonists, opioids, ketamine, nitrous oxide, and local anesthetics. Horses are perhaps fortunate in that combinations of these drugs are used frequently in sedative and general anesthetic protocols, and that their simultaneous administration (multimodal analgesia) has a potential additive or synergistic effect on analgesia. Preemptive analgesia can also be accomplished by relatively minor adjustments to protocols, for example administration of NSAIDs on the morning of surgery, and ensuring that all other analgesics are administered before the surgical procedure begins. This approach can optimize the effectiveness of the analgesic technique, thereby reducing the likelihood of the development of central sensitization as well as reducing the postoperative requirements for analgesics.

The surgical procedure and presence of pain preoperatively should be considered when selecting an analgesic protocol; a horse presented for fracture repair requires a greater degree of analgesia than a horse undergoing repair of a small, superficial skin laceration. It is also important to monitor each individual horse's response to analgesics so that drugs and dosages can be adjusted as appropriate.

In addition to pharmacological therapy, other factors may assist with pain management, such as immobilization of a fractured limb or surgery to remove painful focus (e.g., enucleation). The importance of nursing care should not be underestimated and can greatly influence the rehabilitation process.

Nonsteroidal Anti-Inflammatory Drugs

NSAIDs play an important role in equine pain management protocols. They are particularly effective against inflammatory pain because they work by inhibiting cyclooxygenase (COX) enzymes, which are necessary for prostaglandin production during the inflammatory response. Traditionally, COX-1 was considered to be a constitutive enzyme and COX-2 inducible following tissue trauma. It was thought that many of the adverse effects associated with NSAID administration were due to inhibition of COX-1. This led to the development of COX-2–selective NSAIDS. Theoretically, these should have had a favorable side-effect profile in comparison to nonselective NSAIDs. Unfortunately, COX-2–selective NSAIDs are associated with an increased risk of adverse cardiovascular events in people, and it has become apparent that the delineation between the two forms of the enzymes is not as unambiguous as initially thought.[12]

Phenylbutazone, flunixin meglumine, and ketoprofen are the most commonly used NSAIDs in horses[13] and are potent COX-1 inhibitors.[14] Adverse effects on the gastrointestinal tract mucosa are potential side effects of NSAID administration to horses.[15] It has been speculated that COX-2–selective drugs, such as firocoxib (Equioxx), will be associated with fewer gastrointestinal adverse events,[16] and it will be interesting to see if this is verified in clinical cases.

Other NSAIDS used in horses include vedaprofen (Quadrisol), suxibuzone (a precursor of phenylbutazone) (Danilon Equidos), carprofen (Rimadyl), and meloxicam (Metacam).

A liposome-based diclofenac cream (Surpass Topical Cream) has FDA approval for use in horses in the United States. The cream is applied topically, for example over a joint, and exerts a local anti-inflammatory effect with minimal systemic absorption. There are conflicting reports in the literature on the efficacy of this cream in experimental models of synovitis and osteoarthritis,[17,18] although more positive results were reported in a clinical field trial.[19]

Opioids

Opioids produce analgesia by binding to mu (μ), kappa (κ) or delta (δ) receptors in the spinal cord and brain. Peripheral opioid receptors are also found in inflamed tissue.

Opioids may be administered by intravenous, intramuscular, epidural, or intraarticular injection. Transmucosal and transdermal routes of administration may be alternative options in "needle-shy" horses.

The use of opioids for analgesia is commonplace in small animal practice, but fear of side effects may have traditionally limited the use of opioids, particularly full μ-agonists, for analgesia in horses. Excitement, thought to be caused by CNS stimulation, is often observed when partial and full agonist opioids are administered alone to pain-free horses.[20,21] In contrast, excitement and locomotor stimulation are rarely observed when opioids are administered to horses undergoing surgery or in combinations with sedatives.[22-24] Morphine has been implicated in the etiology of postanesthetic colic in one institution.[25] However, results from another center demonstrated an absence of effect from perianesthetic morphine as a risk factor for colic in horses.[26] No adverse and some beneficial effects of morphine administration to horses undergoing general anesthesia (0.1 mg/kg[22] and 0.15 mg/kg followed by 0.1 mg/kg/hr[27]) have been reported, although the analgesic effects remain to be evaluated.

Butorphanol is the most commonly used opioid in equine practice in the United Kingdom[9] and is incorporated frequently into sedative and general anesthetic protocols. It is a synthetic opioid, which is thought to act as an agonist at κ-receptors and an antagonist at μ-receptors.[28] The dosage recommended for analgesia (0.1 mg/kg) is approximately 5 to 10 times that recommended for sedation. The short duration of action (30 to 60 minutes) may account for the lack of discernable analgesic effect following castration in ponies,[24] so frequent redosing or administration by continuous rate infusion has been recommended.[28] After exploratory laparotomy, horses that received butorphanol by constant rate infusion (CRI) (0.013 mg/kg/hr) for 24 hours after surgery had lower plasma cortisol levels, lost less weight, and had lower pain scores compared to horses that did not receive butorphanol.[29]

Despite the lack of evidence either way regarding the analgesic efficacy of butorphanol, administration during anesthesia has been recommended by some authors because it appears to blunt sympathetic stimulation during surgery[30] and may improve the subjectively assessed quality of anesthesia.[31]

The analgesic potential of buprenorphine, a μ-agonist opioid, has been investigated in horses. Administration of 10 μg/kg IV provides antinociception to a thermal stimulus for approximately 7 to 11 hours[32,33] and provides long-lasting analgesia without significant side effects following castration in ponies.[34] Buprenorphine is well absorbed from oral mucosa in cats,[35] and a recent case report suggested that sublingual administration of buprenorphine may be a potential option for pain management in horses.[36] Sublingual administration of 6 μg/kg

buprenorphine provided analgesia for approximately 12 hours in a 5-month-old Thoroughbred filly with a suspected fracture of the first cervical vertebra.[36]

Transdermal fentanyl patches have been used as part of analgesic protocols in horses.[37] Dosing recommendations appear to be empirical at present, and for an adult horse two or three 10 mg or 100 μg/hr patches are placed on a shaved area of skin. Marked variations in plasma levels have been reported,[38,39] so monitoring each individual horse's response to treatment is essential.

α_2-Adrenoceptor Agonists

Xylazine, detomidine, romifidine, medetomidine (where allowed), and dexmedetomidine are commonly included as part of sedative and general anesthetic protocols, and their use in these contexts are discussed in Chapters 18 and 22. The mechanisms behind the analgesic effects of the α_2-agonists are not understood fully but are thought to involve receptors in the brain and spinal cord.[40] The sedative effects of α_2-agonists usually outlast the analgesic effects[41] and, although useful as part of a preemptive, multimodal analgesic technique, the profound sedation produced by IV administration usually precludes the use of infusion techniques for long-term pain management. The epidural route can be considered for longer-term pain management because, in the case of xylazine, analgesia can be achieved at lower doses compared to IV administration.

Local Anesthetics

Local anesthetics act by blocking sodium channels in excitable membranes and preventing the transfer of nociceptive information. Local anesthetics are used frequently to assist with diagnosis of lameness and can also be used for therapeutic purposes by topical administration, infiltration, and regional anesthesia. In addition to playing an important role in multimodal pain management strategies, they reduce sedative and anesthetic requirements and also facilitate surgery by improving muscle relaxation and reducing the likelihood of a response to surgical stimuli. The recent trend toward performing surgical procedures, such as selective fracture repair, dental extractions, sinus surgery, and laparoscopy, on standing, sedated horses, rather than under general anesthesia, means that the role of local anesthetic techniques is increasing in importance. Local anesthetic techniques for surgery in the head region have been reviewed,[42] and common perineural injection sites are described in Chapter 30.

Paravertebral anesthesia is commonly used for flank laparotomies in cows but is also a very useful technique for laparoscopy (and flank laparotomies) in horses. The skin, muscles, and peritoneum of the flank region are desensitized by anesthesia of the dorsal and ventral branches of the eighteenth thoracic and first and second lumbar spinal nerves.[43] An alternative approach is segmental thoracolumbar anesthesia with local anesthetic administered via a catheter inserted into the subarachnoid space at the lumbosacral intervertebral space. This requires special equipment and strict adherence to aseptic technique. Ataxia of the pelvic limbs is a potential adverse effect.[44]

Anesthesia of the tail and perineum can be achieved by epidural administration of local anesthetics at the first coccygeal interspace and is described in the section on epidural analgesia (see later in this chapter).

Transdermal lidocaine patches and cream are being used to provide analgesia in small animals, although there are limited data on efficacy. A recent study in horses found that application of 5% lidocaine cream to the carpus reduced the horse's subsequent response to needle insertion, although no effect was evident after application of a lidocaine patch.[45]

A technique has been developed for long-term analgesia of the distal thoracic limb by continuous perineural blockade of palmar nerves.[46] This has the potential to be very useful but requires further development and refinement before introduction into clinical use, because limb swelling developed following infusion of lidocaine, bupivacaine, and mepivacaine over 1 to 2 days.

Lidocaine Infusions

Intravenous administration of lidocaine is used to reduce ileus following intestinal surgery in horses[47] and as part of a balanced anesthetic protocol (see Chapter 18). Intravenous lidocaine administration during anesthesia reduces postoperative morphine requirements and pain in people,[48] although the analgesic effects have not yet been comprehensively evaluated in horses in the clinical arena. In conscious experimental horses lidocaine infusion (2 mg/kg followed by 0.05 mg/kg/min) produced an antinociceptive effect to a thermal stimulus, suggesting that lidocaine may produce somatic analgesia. Further investigations into these analgesic effects are warranted.

Drugs for the Treatment of Central Sensitization

Many drugs developed for indications other than the treatment of pain, such as antidepressants, anticonvulsants, and antiarrythmics, have analgesic or antihyperalgesic properties that make them useful for the management of pain, particularly chronic pain, that is poorly responsive to traditional analgesics.

Ketamine is the only drug used for the treatment or prevention of central sensitization that has a marketing authorization for administration to horses, although the indication is for induction of anesthesia rather than pain management. It is an *N*-methyl *D*-aspartate (NMDA) receptor antagonist, and subanesthetic doses are thought to have an antihyperalgesic effect.[49] No adverse effects on behavior were detected during infusion of ketamine at 0.4 and 0.8 mg/kg/hr in conscious horses.[50] High concentrations of ketamine may also block sodium channels, and this may explain the short duration of analgesia (15 minutes) when used for an abaxial sesamoid nerve block.[51]

Tramadol is a synthetic codeine analogue. It binds weakly to the μ-receptor and inhibits neuronal reuptake of norepinephrine and 5-hydroxytryptamine. The effective dosage of tramadol in horses has not been established; 2 mg/kg IV did not produce any adverse effects but did not result in antinociception to a thermal stimulus.[52] The oral bioavailability of tramadol is very poor, so this route of administration is unlikely to be effective in horses.[53]

Gabapentin was originally developed as an anticonvulsant and is used to treat neuropathic pain in people. The precise method of action is not clear but it is thought to modulate the activity of presynaptic voltage-gated calcium channels, which are upregulated in chronic pain states. It does not act specifically

as an analgesic but can reduce established hyperalgesia. Gabapentin at a dosage of 2.5 mg/kg orally every 12 hours was used as part of an analgesic protocol for the treatment of neuropathic pain caused by femoral neuropathy in a pregnant mare.[54] The pharmacokinetics of gabapentin have been described in horses, and the plasma elimination half-life following a dosage of 5 mg/kg orally is 3.4 hours.[55] Further work is required to determine the clinical efficacy of gabapentin in horses.

Intraarticular Analgesia

Intraarticular administration of opioids and local anesthetics following arthroscopy in horses is increasing in popularity. Opioid receptors are present in equine synovial membranes[56] and this may account for the reported analgesic and anti-inflammatory effects of intra-articular morphine. In a lipopolysaccharide-induced synovitis model, the effects have been reported to last approximately 20 hours after a dose of 0.05 mg/kg[57] and at least 24 hours following 0.05 to 0.1 mg/kg.[58]

Intra-articular local analgesics can provide effective analgesia in the short term. In people, concerns have been raised over the potential toxic effects of local anesthetics on chondrocytes following prolonged postoperative infusions. This effect appears to be dosage- and time-dependent. A recent editorial suggested that in people, a single dose appears to be safe but prolonged administration of high concentrations of local anesthetics should be avoided.[59] In the absence of data relating to the horse it would appear prudent to limit intra-articular administration of local anesthetics to a single dose, until more studies are available.

The use of intra-articular corticosteroids, hyluronic acid, and polysulfated glycosaminoglycans are discussed in Chapter 79.

Epidural Analgesia

Epidural injections can be performed at two sites in horses: the lumbosacral space (cranial) and the first coccygeal interspace (caudal). Cranial epidural injections are rarely performed in horses because they can be technically difficult, carry the risk of dural puncture, and are more likely to result in motor blockade to the pelvic limbs than the more commonly performed and simpler caudal epidural.

Indications for caudal epidural administration of drugs include obstetric manipulations; surgery of the tail, perineum, or genitourinary tract; intraoperative analgesia; and postoperative pain management. Infection at the site of needle placement is a contraindication for the use of this technique. Other relative contraindications include coagulopathies (risk of bleeding within spinal canal), ataxia (risk of recumbency), neurological diseases, and septicemia (risk of spread of infection into epidural space). In addition, the safety of the person performing the procedure should be considered carefully.

The horse should be restrained adequately and, if possible, evenly bearing weight on its pelvic limbs. The first coccygeal interspace may be located by digital palpation (area approximately 5 cm (2 inches) craniad to the start of the tail hairs on the midline) during dorsoventral movement of the tail. Locating the site for injection may be difficult in obese or heavily muscled horses. The site should be clipped and strict aseptic technique should be used. Desensitization of the tissues along the proposed needle path with local anesthetic will reduce the horse's response to subsequent spinal needle placement. An 18 to 20 gauge (G) spinal needle (bevel facing craniad) is inserted through the skin (Figure 23-2). The angle of insertion may need to be altered to ensure that the needle passes through the interspinous and interarcuate ligaments and into the epidural space. A change in resistance to needle advancement may be appreciated as the epidural space is entered. The needle should be withdrawn approximately 5 mm if the floor of the vertebral canal is encountered. Needle placement can be verified by the "hanging drop" technique; a drop of saline or local anesthetic placed on the hub of the needle will be drawn into the epidural space upon its penetration with the needle tip. The syringe containing the drugs for injection should be attached to the needle hub, and no blood should be aspirated into the syringe when gentle suction is applied. The drugs should be injected very slowly and minimal resistance to injection should be encountered if the needle tip is in the epidural space.

For longer-term pain management an epidural catheter may be inserted (Figure 23-3). Careful attention to the maintenance of the catheters and use of aseptic technique is important to avoid introducting pathogens.

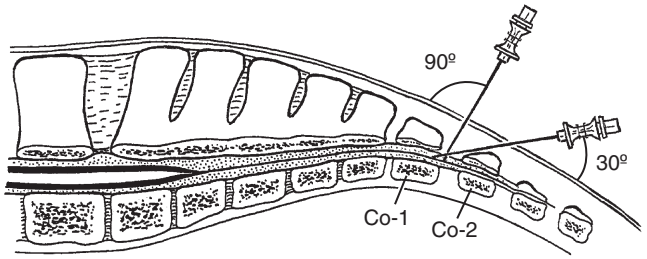

Figure 23-2. Diagram showing needle placement for caudal epidural injections in horses.

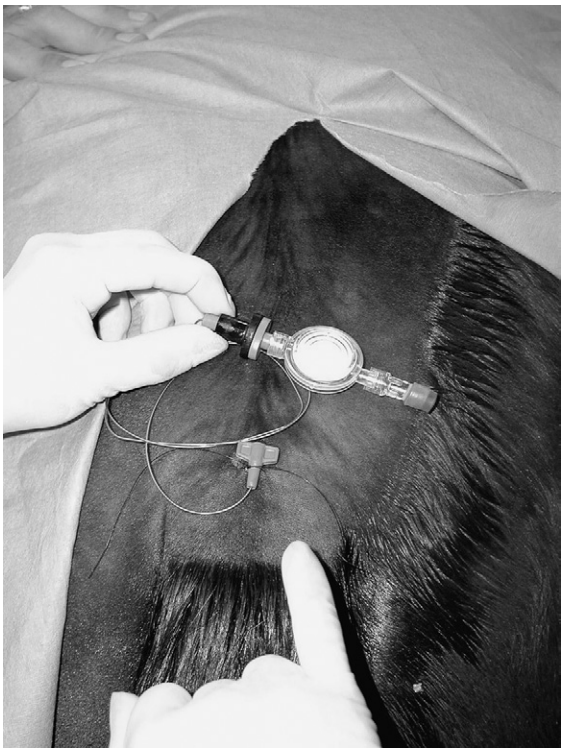

Figure 23-3. Epidural catheter being placed for long-term use.

TABLE 23-1. Doses of Drugs Administered via a Caudal Epidural Injection to Produce Perineal Analgesia in Horses*

Drug	Dosage	Volume	Weight	Onset	Duration	Extent of Analgesia
Lidocaine (2%)[60]	0.22 mg/kg	–	352-482 kg	4 min	1.5 hr	
Ropivacaine (0.5%)[61]	–	8-9 mL	475-565 kg	10 min	3 hr	S2 dermatome in 3/10 mares
Bupivacaine (0.5%)[62]	0.06 mg/kg	3.7 mL (mean)	240-350 kg	6 min	5.3 hr	
Xylazine[63]	0.25 mg/kg	8 mL (with 0.9% NaCl)	420-500 kg	13 min	165->180 min	S3 dermatome
Detomidine[63]	0.06 mg/kg	8 mL (with 0.9% NaCl)	420-500 kg	12 min	160 min	T15 dermatome in one horse, most commonly S1-S2
Methadone[64]	0.1 mg/kg	20 mL (with 0.9% NaCl)	495-593 kg	15 min	5 hr	T13 dermatome
Morphine[65]	0.1 mg/kg	20 mL (with 0.9%NaCl)	511 kg (mean)	5 hrs	6 hr	Thoracic region
Ketamine[66]	0.5, 1, and 2 mg/kg	9 mL	400 kg	5-10 min	30-75 min	Upper pelvic limb
Tramadol[65]	1 mg/kg	20 mL	511 kg (mean)	30 min	4 hr	Sacral region
Lidocaine (2%) + xylazine[60]	0.22 mg/kg + 0.17 mg/kg	6 mL	454 kg	5 min	330 min	

*The maximum extent of analgesia (where investigated) is shown.

Drugs Used

Drugs that have been administered into the epidural space in horses include local anesthetics, opioids, α_2-agonists, ketamine, and tramadol. These have been used as sole agents and in combinations and are summarized in Table 23-1.[60-66] Whenever possible, solutions of drugs used for epidural administration should be preservative-free.

Local anesthetics administered into the epidural space can provide anesthesia of the perineal region. Severe ataxia and recumbency are possible consequences of motor blockage of the pelvic limbs following cranial spread of local anesthetics. It is recommended that the volume for injection be limited to 8 to 9 mL of local anesthetic in a 500-kg horse.[61]

Morphine, methadone, and buprenorphine are available in some countries as preservative-free solutions and have been reported to produce relatively long-lasting analgesia after epidural administration. Adverse effects reported following epidural morphine administration in horses include self-limiting pruritus[67] and wheals in the perineal region.

Epidural administration of xylazine (0.17 mg kg^{-1})[68] or detomidine (0.06 mg/kg)[69] in 10 mL of saline produces analgesia lasting 2 to 3 hours, in contrast to medetomidine[70] and romifidine,[71] which appear to be less efficacious. Sedation may be apparent following epidural administration of α_2-adrenoceptor agonists.

Ketamine has been reported to produce analgesia following epidural administration in horses. The use of epidural ketamine in people is controversial because of concerns about neurotoxicity, although it is not clear if the nerve damage is caused by ketamine itself or by the preservative (benzethonium chloride).[72]

Complementary Therapy

Acupuncture and electro acupuncture are being used more frequently as adjuncts to pain management in horses. They are most commonly used as a treatment for musculoskeletal and visceral pain. Experimental studies have investigated the analgesic efficacy in visceral[73,74] and somatic[75] pain models with mixed results. Acupuncture and electro acupuncture do result in an increase in β-endorphin concentration within cerebrospinal fluid, and this may be involved in mediating the analgesic effects.

Extracorporeal Shock Wave Therapy

Extracorporeal shock wave therapy (ESWT) is increasingly being used in the management of orthopedic conditions in horses. Anecdotal reports suggest that ESWT may produce a short-term analgesic effect. However, one study failed to demonstrate this in horses with navicular disease.[76] One study investigating the effect of this type of treatment in 16 horses suffering from proximal palmar metacarpal or plantar metatarsal pain showed that after 72 hours the weight-bearing discrepancy between affected and contralateral forelimb decreased significantly.[77] Further investigations into the short- and long-term analgesic effects of ESWT in the management of specific conditions are required. It is however interesting to learn that at certain racetracks in the United States these devices are banned.

Therapeutic Ultrasound

Therapeutic ultrasound, magnetic, and laser therapy are also used for pain management, although studies are required to objectively evaluate responses to treatment.

SUMMARY

In summary, a thorough and systematic approach to pain assessment and implementation of a multimodal approach to pain management has the potential to significantly improve patient welfare and outcome after illness, injury, or surgery.

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CHAPTER 24

Principles of Plastic and Reconstructive Surgery

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Many of the reconstructive and plastic surgery principles and techniques outlined in this chapter can be used on a routine basis in the execution of elective procedures, such as tissue biopsy, whereas others will be useful in the repair of more extensive, traumatic injuries. Skin has unique physical properties, which can be manipulated to the advantage of the surgeon and patient. Understanding the general process of wound healing, as well as the differences that are unique to the horse, is the basis for formulating a treatment plan (see Chapter 5). The type of injury and its location on the body, degree of contamination, age of the wound, integrity of the local blood supply, and amount of skin loss must all be considered when deciding on the most appropriate method for managing a particular wound.[1-3] In addition to biological factors, the physical size of equine patients and the environment in which they are kept present unique management challenges not encountered in the treatment of soft tissue injuries in other species, including humans.

The overall goal in the management of any wound, regardless of whether it is treated by primary closure or by delayed primary or secondary closure or is allowed to heal by second intention is to achieve a result that is as functional and as cosmetically appealing as possible. Primary closure of traumatic wounds in many instances is not possible or recommended. Only wounds that are classified as clean or clean contaminated with adequate blood supply should be considered for immediate closure. Premature closure of heavily traumatized or contaminated wounds is one of the biggest mistakes made by equine practitioners during early wound management. Wounds managed for 1 to 3 days with treatment directed at reducing edema, contamination, and infection become better candidates for closure. Management of wounds by delayed primary closure (before the appearance of granulation tissue) or secondary closure (after the appearance of healthy granulation tissue) heal more rapidly and with fewer complications. Delaying closure to the fourth day or later has been associated experimentally with reduced wound sepsis.[4] Primary, or even delayed primary, closure may not be a viable option when the contaminated wound is associated with a significant skin deficit. Many of these wounds are best treated by secondary closure or as open wounds. Unfortunately, second-intention healing is often prone to development of exuberant granulation tissue and delays in contraction and epithelialization (see Chapter 5). To get the best cosmetic and functional results, open granulating wounds should be managed in such a way as to allow maximal contraction and optimal epithelialization.

Carefully planned reconstructive and skin-mobilizing techniques can be used in conjunction with primary, delayed primary, or secondary wound closure and as part of an overall strategy of open wound management to minimize the potential cosmetic and functional shortcomings of excessive epithelialization (scarring). Besides traumatic wounds, there are other clinical situations, such as the planned excision of skin lesions, in which reconstructive procedures can be used to facilitate closure of surgical incisions. The extent to which some of these techniques can be used depends on the presence of adequate tissue surrounding the wound. The benefits of successfully using reconstructive procedures include an increased percentage of the wound covered with full-thickness skin, improved cosmetic appearance and function, and a reduction of overall healing time. Ultimately the combination of skin loss and the amount of redundant or loose skin surrounding a wound influences the clinician's ability to close a wound and the extent to which wound contraction will contribute to the final result.

In the quest to minimize wound morbidity, many reconstruction techniques have been developed. Methods vary from use of simple tension relieving sutures, to incorporation of skin mobilization and expansion techniques, to the use of skin flaps. Understanding the principles and limitations of these procedures, adhering to the basic principles of equine wound management, and providing appropriate postoperative care all contribute to a successful outcome.

CUTANEOUS BLOOD SUPPLY

Successful reconstructive surgery relies heavily on an adequate blood supply. In mammals, two types of vessels supply the skin: perforating musculocutaneous vessels and direct cutaneous vessels. Perforating musculocutaneous arteries pass through the muscle to supply the skin. This arrangement is found in both people and pigs. Horses' skin, similar to that of dogs and cats, is supplied by direct cutaneous arteries, which reach the skin by passing through the fascial septa between muscle bodies.[5] Direct cutaneous vessels run subdermally, run parallel to the skin surface, and are closely associated with the panniculus muscle in areas where this structure is present. In the distal extremities,

where no panniculus muscle exists, the direct cutaneous arteries run beneath and parallel to the dermis. In the dog (and hypothesized to occur in the horse), smaller vessels branch off these cutaneous arteries and arborize in the dermis, forming three closely interconnecting plexuses—the deep subcutaneous plexus, the middle cutaneous plexus, and the superficial subpapillary plexus—which together supply the dermis and adnexal structures of the skin.[6] The superficial plexus is an important component of the thermoregulatory system, and all three are crucial in maintaining the integrity of the blood supply and are thus of importance when manipulating skin in reconstructive procedures.

In dogs and cats, a number of skin flaps have been developed based on the major cutaneous arteries of the trunk.[7] These include axial pattern flaps, island arterial flaps, and vascularized free flaps. A skin flap based on the cutaneous portion of the caudal branch of the deep circumflex iliac artery has been described and used in experimental studies of a vascularized free tissue transfer in the horse.[8] More detailed descriptions of the vascular supply to the equine skin, which could be reliably used for the reconstruction of skin deficits, are required to advance development of additional options.

PHYSICAL AND BIOMECHANICAL PROPERTIES OF SKIN

Skin has three structural components, which impart tension and viscoelasticity: collagen, elastic fibers, and ground substance (primarily proteoglycans). It is important to note that these components vary considerably between individuals and between different sites of the body. In humans, they are known to be affected by aging, solar radiation, disease, and chemical and physical trauma.[9] Viscoelastic materials are those for which the relationship between stress and strain depends on time. If the stress is held constant, the strain increases with time (creep); and if strain is held constant, the stress decreases with time (relaxation). Tension is the magnitude of force skin can exert. Understanding these physical and biomechanical properties inherent to skin allows the clinician to manipulate them to his or her advantage.

Lines of Skin Tension

The normal tension that exists in skin is a result of the elastic fibers in the dermis and is the reason skin edges retract when incised. In 1861, Langer mapped lines of tension (Langer's lines or lines of maximal tension) over the surface of the human body but did not take into consideration body or limb position or movement. However, because skin is anisotropic (lacking similar properties in all directions), skin tension lines are influenced by muscle contraction, movement of joints, and other external forces. Thus, Langer's lines are more accurately referred to as relaxed skin tension lines.[10] Incisions made parallel to lines of maximal tension will gap to a lesser extent and heal with a finer scar than incisions made at right or oblique angles to them. Incisions made at oblique angles to the lines become curvilinear whereas incisions made at right angles to lines of tension gape widely, require more sutures for apposition, and heal with scars that, over time, become stretched and less cosmetically acceptable. Tension lines have been described in dogs, but they have been investigated to only a very limited extent in the horse (Figure 24-1).[11,12] Diagrams showing the orientation of tension lines in the dog resemble the color patterns seen on the tiger and zebra, and it has been suggested that the lines of the skin color pattern in these animals closely mimic lines of tension.[10] In the absence of detailed information, these generalities can be used to advantage in planning reconstructive procedures in horses.

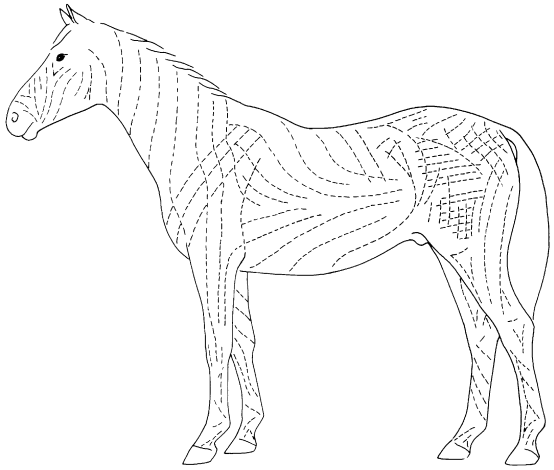

Figure 24-1. Cleavage lines of equine skin. (Redrawn from Hrudka F, Popesko P, Komarek V: Principal Morphology of Domestic Animals, Bratislava, 1962, Slovak Publishing House of Agricultural Literature, based on work by Najbrt.)

Skin Extensibility

Skin extensibility is another important physical property of the dermis that is used to advantage in reconstructive surgery. It refers to the skin's normal stretching capacity while an anatomic part is at rest and represents the physical property that allows wounds to be sutured closed following removal of skin. Lines of maximal extensibility have been shown to run at right angles to Langer's lines, which logically could also be referred to as lines of minimal extensibility.[13] Pinching skin into folds at the site of a proposed reconstructive procedure can be used to roughly determine the extent of orientation of skin extensibility and to assess whether or not the edges of the remaining defect can be brought into apposition after excision of a lesion or the revision of a scar.[13] When excising an elliptical piece of skin, closure is facilitated by ensuring that the long axis of the ellipse is aligned to Langer's lines. Because the lines of maximal extensibility are at right angles to Langer's lines, the width of the ellipse can be wider if the long axis is parallel rather than perpendicular to Langer's lines. Tension lines and lines of extensibility in human skin are well documented, and diagrams showing their locations can be found in most plastic and reconstructive surgery texts. In the horse, lines of maximal skin extensibility were determined for the carpus, which led to the surgical recommendation for making longitudinal versus transverse incisions over the dorsal, medial, and lateral aspects of the carpus.[14]

Creep and Stress Relaxation

Two types of creep can occur within the skin. *Mechanical creep* is a biomechanical property of skin, which allows it to stretch beyond its normal limits of extensibility under a constant load.

This results as the normally convoluted collagen fibers become straight and aligned parallel to the stretching force. *Biological creep* differs from mechanical creep in that the skin does not stretch but instead increases area of coverage by increasing epidermal mitotic activity, upregulation of blood vessels, and increasing dermal cell numbers, resulting in skin development. This phenomenon is seen with pregnancy, growth of subcutaneous masses, body wall hernias, and, to the advantage of plastic surgeons, with tissue expanders.

Stress relaxation occurs when the strain imposed on a piece of skin is kept constant. Over time the amount of strain required to maintain elongation of the skin is decreased as a result of the collagen fiber alignment and lengthening, which occurs with mechanical creep. The process of stress relaxation explains why the tension on a suture line decreases to some extent a few minutes to hours after surgery.

A number of surgical techniques in reconstructive surgery utilize these viscoelastic properties of skin; however, there are only limited documented accounts of the application of these procedures in horses. Used alone or in combination with the other techniques described in the following paragraphs, they provide interesting possibilities for the management and reconstruction of skin wounds in horses.

PRINCIPLES OF WOUND CLOSURE

Incision Orientation

Whenever possible, a plan of attack should be made prior to creating an incision or closing a traumatic wound. The location of the injury or lesion, the elasticity of the surrounding tissue, the available blood supply, and the character of the wound bed should all be considerd when planning reconstructive surgery. The extensibility of the skin should be assessed with the affected region of the body in a relaxed position and during movement. This can be done by grasping the skin surrounding the wound or lesion with a thumb and forefinger while moving the body part. Careful assessment will identify areas from which skin may be mobilized and areas that are under tension. The skin can be marked with an alcohol-resistant marker prior to surgery, outlining the surgical approach.

Whenever possible, planned removal of lesions involving the limb should involve incisions that are parallel to the long axis of the limb. Repair of wounds that are located obliquely or transversely to the limb's axis often require postoperative casting, splinting, or bandage support to prevent excess tension during flexion and extension of the limb. Placement of incisions for the management of lesions of the upper body are site dependent. Positioning the horse's body during surgery so that the affected region is under the greatest tension facilitates selection of closure techniques that minimize the risk of postoperative dehiscence.

Sutures and Patterns

Sutures are used to approximate skin edges, reduce tension, and reduce dead space. The proper placement and use of appropriate suture patterns are important technical considerations in reconstructive surgery. The selection of a suture pattern is often based on personal preference; however, in some instances, there are definite advantages and disadvantages associated with particular patterns that should be taken into consideration. The choice of suture material is of less importance.[7,10] Manipulation of the tissue when placing sutures should be performed as atraumatically as possible. If instruments are used to stabilize or manipulate the skin edges, crushing and abrading tissue, which is detrimental to future healing, should be avoided; using one's gloved fingers is probably the least traumatic method. Most reconstructive procedures can be performed using a standard instrument set; however, the addition of a limited number of special instruments will greatly assist the surgeon. The addition of sharp skin hooks or Lahey traction forceps aids in minimizing trauma when stabilizing tissue. Sterile Backhaus towel clamps are an acceptable alternative. Stabilization of the skin using an instrument that penetrates the tissue is less damaging to the skin and its microvasculature than using an instrument that has a crushing action.

To facilitate optimal healing, accurate apposition of the skin edges with minimal interference to the blood supply should be the goal when suturing wounds. The pattern and technique of suture placement, the number of sutures, and suture line support are all important in ensuring an optimal outcome. The spacing of sutures plays an important role in maintaining optimal apposition of the skin edges. As a rule, sutures should be placed at a distance from the skin edge equal to the thickness of the skin itself.[15] However, other factors, such as the tension on the wound edges and the thickness and stiffness of the skin, must also be taken into consideration. The collagenolytic and inflammatory processes that take place during early wound healing serve to weaken the suture-holding ability of skin, and as a result, sutures should be placed at a distance of at least 0.5 cm from the skin edge for improved security.[16] Clinical experience with equine wounds treated by secondary closure shows that wound edges where newly epithelialized tissue exists have little or no holding strength, making it important to place the sutures well back from the skin edge to ensure optimal holding strength. In laboratory animals, the levels of matrix metalloproteinases (MMPs) have been shown to be higher in keratinocytes during re-epithelialization, and in a porcine model, the levels of these collagenolytic enzymes have also been shown to be higher and to persist longer in granulating wounds than in sutured wounds.[17,18] Neutrophil-derived MMPs have been associated with nonhealing wounds in human patients, and overexpression of this particular collagenase may be part of the pathogenesis of chronic ulcers.[19] Tissue strength and not suture size or number are more important determinants as to whether or not a wound will dehisce. A sound working knowledge of the biology of wound healing improves clinical judgment and reduces the risk of failure of repair (see Chapter 5).

Sutures should be placed as close together as is necessary to approximate the skin edges accurately. Wounds along tension lines close with fewer sutures than do those perpendicular to the line of tension. More sutures are required in thinner skin than in thicker skin.[20] For wounds under tension, increasing suture numbers is preferable to increasing material size.[20] In human surgery, placing interrupted sutures 0.5 cm apart provides tissue apposition and minimizes tension on individual sutures.[16] The suggestion that the distance between individual sutures should be twice the thickness of the skin may apply, although in some instances, this procedure may result in an excessive number of sutures being used, leading to impaired healing as a result of excessive tissue reaction.[15]

The tension applied when placing and tying skin sutures should be sufficient to appose the skin edges, but overtightening

of sutures can result in failure of the suture line. Suture tension is a summation of intrinsic and extrinsic forces. *Intrinsic tension* is the constricting effect within the suture loop, which, if excessive as a result of the suture being tied too tightly, causes strangulation of the blood supply to tissue within the loop of the suture. A higher degree of intrinsic tension can be generated with heavier suture material, and any postoperative swelling will increase the intrinsic tension of a suture. *Extrinsic tension* results from opposing forces exerted by the surrounding skin on the suture when drawing the skin edges together. Extrinsic tension is related to the size of the defect being sutured and to the amount of redundant or loose skin in the area.[21] Tension on sutures can lead to tissue necrosis by interfering with local blood supply, increasing edema, and decreasing tissue perfusion. The clinical result of this can include wound dehiscence, sloughing of tissue, visible scar formation, and delayed healing, which may have catastrophic consequences depending on the location of the suture line.

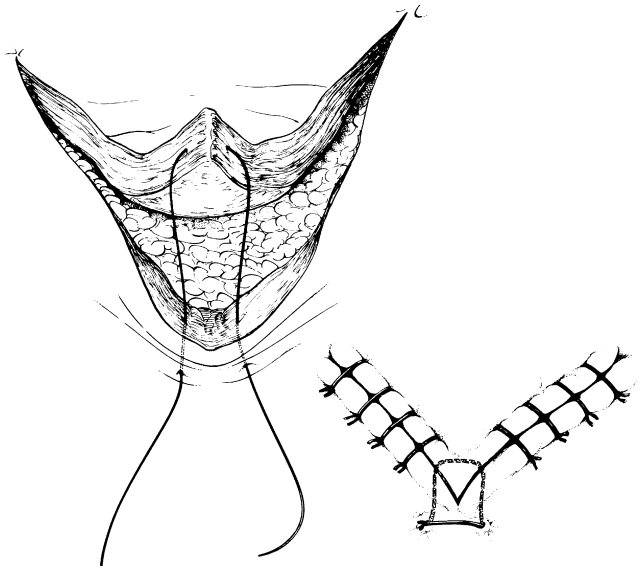

Figure 24-2. Corner suture used to minimize vascular compromise to the tip of the flap.

Basic Patterns

SUBCUTICULAR AND INTRADERMAL SUTURES

Subcuticular sutures are those placed in the subcutaneous tissue layer. They can be placed in a continuous or an interrupted pattern. Bites into the underlying soft tissue minimize the dead space. Subcuticular sutures reduce the tension between the wound margins prior to placement of skin sutures.[20] This pattern is inappropriate as a standalone closure pattern.

Intradermal sutures can be continuous or interrupted and provide excellent skin apposition and eliminate the need for percutaneous skin sutures in some instances. This can be advantageous in some patients, eliminating the need for suture removal. Other advantages are the elimination of suture tract sinuses and possible scarring associated with the use of skin sutures.

Simple interrupted sutures

Simple interrupted sutures (see Figure 16-7, *A*) can provide excellent tissue apposition if they are not placed too far back from the skin edge or pulled too tightly, both of which will cause inversion of the skin edges. Placement with slight eversion is preferred to inversion.[22] The ability to vary the width of their placement in irregularly shaped wounds and those with variable skin thickness facilitates better alignment of the skin edges than do continuous patterns. Tension across individual sutures can also be easily adjusted. Skin wounds closed with interrupted patterns have been shown to have greater tensile strength and less compromise of the microvasculature than those closed with simple continuous sutures.[23,24] If tension exists across the suture line, the attributes of simple interrupted sutures can be combined with other tension suture patterns.

Simple continuous sutures

As with any continuous pattern, knot failure or suture breakage results in breakdown of a large portion or all of the incision line. Continuous patterns (see Figure 16-7, *H*) are quickly placed, but it is not possible to vary the tension on the suture line to the same degree as with an interrupted pattern. Continuous patterns have been shown to result in increased edema and compromised circulation and to have a prolonging effect on the inflammatory phase of wound healing.[23,24] Although a simple continuous pattern has some advantages, the lack of precise skin apposition makes it less appropriate for use in plastic or reconstructive procedures.

Corner suture

Also called a three-point or half-buried mattress suture, the corner suture is used to secure the sharp intersecting point of a Y-shaped incision (Figure 24-2).[15] The intersecting apex of a Y-incision is predisposed to ischemia, and the design of this suture pattern is such that it will not further compromise the blood flow. The three-point suture can also be used in other situations in which the angle of the skin edge, when sutured, would predispose it to ischemia. The corner suture pattern begins like a routine horizontal mattress suture, but instead of penetrating the full thickness, only a partial-thickness bite is taken. The suture is then passed horizontally through the dermis at the point of the V and completed as it was started with a partial-thickness bite.

TENSION-RELIEVING PATTERNS

Tension along suture lines can result from attempting to close wounds where there has been loss of skin, swelling in the area, or loss of elasticity in the surrounding tissue of more chronic wounds.[25] Closing wounds under tension should always be performed with caution, but the relationship between the tension on sutures and the incidence of necrosis of skin flaps has been challenged in a study, which concluded that the vascular damage resulted primarily from the process of undermining the tissue, and that skin with a good blood supply could be closed under considerable tension without fear of necrosis.[26] Careful judgment is required when increasing stress on suture lines; however, clinical experience suggests that equine wounds can be closed under a reasonable amount of tension and, provided there is a good blood supply and the suture line is well supported by some form of coaptation afterwards, healing is not delayed. If the force required on individual sutures to appose the skin edges increases to the point where vascular compromise is becoming a threat, tension sutures can be used to minimize that risk. Tension sutures can be used either alone or in combination with appositional patterns such as simple interrupted sutures and they are

often used in conjunction with other skin-mobilization techniques such as undermining or release incisions.

Tension sutures can usually be removed within 7 to 10 days, depending on the skin tension and provided that the site can be bandaged for additional suture line support.

Mattress sutures

Mattress sutures are described as vertical (see Figure 16-7, *E*) or horizontal (see Figure 16-7, *G*), depending on their orientation to the skin edge. These patterns when placed well away from the skin edges can be used when there is mild to moderate tension, and both provide reasonable apposition but have a tendency to evert the skin edges. They are placed 2 to 4 cm away from the wound margin and prior to sutures used to appose the skin edges. The vertical pattern is stronger under tension than the horizontal pattern and, as with a simple interrupted suture, results in little if any compromise to the microcirculation.[27] Horizontal mattress sutures, because of their configuration, may result in ischemia of the tissue within its suture loop, which may compromise subsequent efforts, if necessary, to reconstruct the defect. The number of tension sutures used in a wound is wound dependent. Buttons, plastic or rubber tubing, or gauze can be used beneath the skin suture loop to distribute pressure and prevent the suture from cutting through the skin (see Figures 16-9, *B* and *C*). These are known as *quilled support*, *quilled*, or *stented sutures*.[27] Although quilled sutures are particularly useful on the body where pressure bandages cannot be used to support the suture line, care should be taken when they are used beneath bandages or casts because external pressure can cause additional skin damage.[28]

The corium vertical mattress pattern results in minimal trauma (see Figure 16-7, *F*). The suture penetrates the skin and exits at the wound level through the corium (dermis). On the opposite side of the skin defect to be closed, the suture is placed entirely in the corium, where it exits intradermally at the skin edges, penetrates the dermis again at the other side of the defect, and exits near the initial penetrating suture, where the knot is tied. This results in a cosmetically superior closure because the sutures exit on only one side of the defect, which ensures perfect alignment of the skin edges without inversion or eversion.[29]

Far-near-near-far suture

The far-near-near-far suture pattern (or its modified form, the near-far-far-near pattern) combines a tension suture, the *far* portion, and an appositional suture, the *near* portion, with the name denoting the order and relative distance the sequential suture bites are taken from the skin edge (see Figure 16-9, *D*). This pattern has a higher tensile strength than either a simple interrupted or a mattress pattern, but it has the disadvantage of requiring that a large amount of suture material be placed in the wound.[27,29]

Walking suture

Walking sutures have been described for closing skin defects in small animals, particularly in areas where the skin fits loosely, such as the trunk (see Figure 16-9, *E*).[29,30] For additional information on this technique, see Chapter 16.

Support for Suture Lines

Supplying adequate support for sutured wounds can make the difference between success and failure in reconstructive procedures. The method of choice is determined to some extent by the location of the lesion and the amount of tension on the suture line. In many areas, bandages may be used to support a suture line, immobilize adjacent skin and joints, minimize edema formation, and obliterate dead space.[31] Pressure bandages are frequently used to support suture lines; however, a subtle balance exists between providing adequate support of the suture line and causing interference with regional blood flow. As a result of absorption of moisture and compression of the material, pressure bandages constructed from cotton or linen materials soon lose their compressive effect and hence their support.[31] To maintain effectiveness, it is necessary either to change these bandages at frequent intervals or to apply tension with an elastic bandage. (A more in-depth discussion on this topic can be found in Chapter 17.) When dealing with repairs on the upper limb or body, the clinician is often limited to the use of stent bandages (tie-over bandages). Stent bandages can provide effective support of suture lines and help obliterate dead space, but they are less effective in preventing or minimizing edema formation (Figure 24-3).

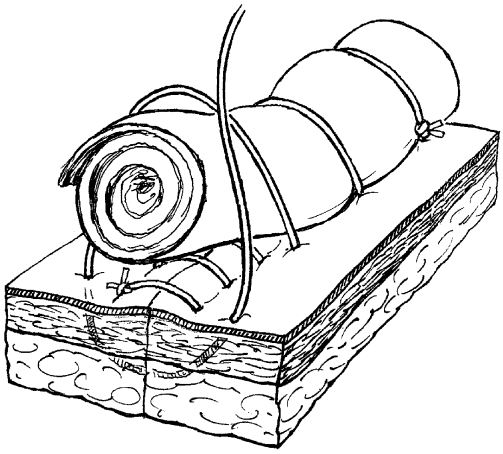

Figure 24-3. A stent bandage consists of rolled up sterile gauze sponges sutured with a large simple continuous suture pattern over an incision closed with simple interrupted sutures.

Cast or bandage removal is dictated by the amount of tissue mobilization and the anticipated tension on the sutured wound secondary to movement, swelling, and location when it is removed. In general, casts are removed between 2 and 3 weeks. Bandages, provided the dressing is clean and dry, are changed at 3- to 5-day intervals and maintained for 4 to 5 weeks.[32] Despite skin healing, tissue strength is only 20% of normal tissue at 3 weeks, 50% at 3 months, and can remain 20% to 30% weaker than the original tissue at 1 year (see Chapter 5). Prematurely removing the bandage or returning the horse to activity has resulted in failure of many wounds that were thought to be healed.

TENSION-RELIEVING AND SKIN-MOBILIZATION PROCEDURES

Tension on suture lines can result in discomfort for the animal, increased scarring, ischemia of the skin margin, and suture line breakdown with partial or complete wound dehiscence. As mentioned previously, suture line tension is influenced by the anatomic location of the lesion. On the lower limb, where there is essentially no redundant skin, excessive tension on suture

lines may create a tourniquet effect around the limb, leading to vascular compromise.[30] The consequences of tension across suture lines can be attenuated by using tension sutures, undermining the surrounding skin, or by using tension-relieving incisions or skin-mobilization techniques. These techniques may be used alone or in combination.

Skin-mobilization techniques allow many wounds with skin loss to be sutured closed rather than relying on contraction and epithelialization. Early wound coverage avoids complications associated with second-intention healing and results in a more rapid return to function. Although many of the reconstructive techniques that have been described for humans and small animals have obvious practical limitations in horses, certain techniques are applicable.

Undermining Skin

Undermining the skin edges around the wound or excision site prior to closure is the simplest tension-relieving procedure (Figure 24-4). Undermining is accomplished by either blunt or sharp dissection with careful attention to the blood and nerve supply of the given region. Blunt dissection has the advantage of minimizing damage to the cutaneous blood supply over sharp dissection. Opening the blades of scissors that have been inserted closed into fascial planes or using the handle of a scalpel in a back-and-forth motion are both accepted methods for bluntly undermining skin. Sharp dissection using a scalpel or scissors, however, is often necessary to separate the skin edge from the underlying tissue planes in chronic, fibrotic wounds. When sharp dissection is used there is an increase in bleeding, which may lead to hematoma formation. Placement of a drain prior to skin closure or a pressure bandage after closure may be necessary.

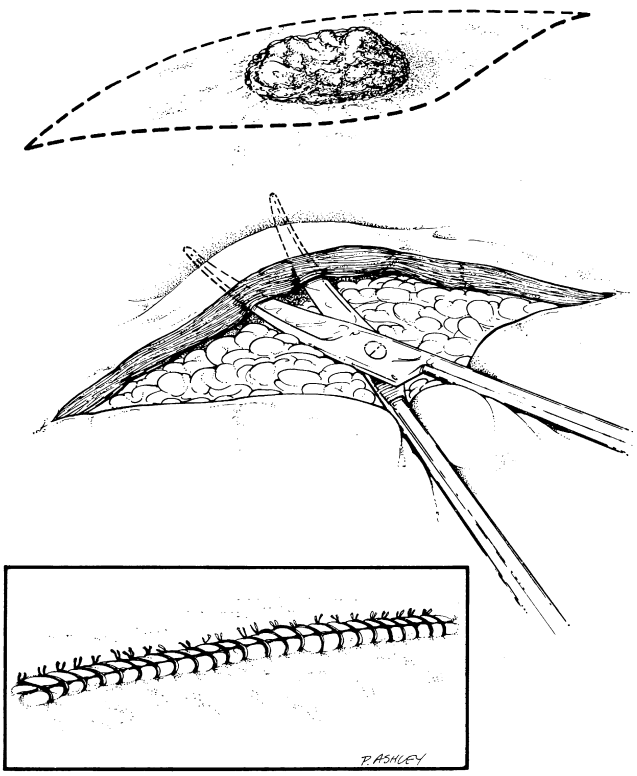

Figure 24-4. Fusiform excision. After excision, the skin is undermined to facilitate closure of the defect. Optimal length-to-width ratio of the fusiform defect is 4:1.

The depth at which skin is undermined depends on the vascular supply to the skin and the presence of superficial nerves in the particular area. Skin on the trunk should be elevated below the level of the panniculus muscle to preserve the small direct cutaneous vessels. On the distal limbs, where no panniculus muscle exists, the dissection plane should be as deep as possible between the subcutaneous tissue and the deep fascia.[21,29] Fascial planes are easy to identify where an elective incision has been made and primary closure is being used. In older wounds, however, because of scar tissue that develops as a result of the inflammatory process, these planes are not as readily identified until the dissection has been continued some distance from the skin edge. In these situations, it seems appropriate, from a clinical perspective, to begin the dissection at a level that approximates the original thickness of the skin.

The degree to which skin needs to be undermined can be estimated by drawing the skin edges together with towel forceps. As a rule, in a fresh wound, a distance equal to the width of the defect itself should be elevated on each side of the wound. If this is not sufficient, undermining can be extended half as much again. The extent to which skin can be undermined without significantly damaging the blood supply and causing necrosis has not been determined. In small animals, it is thought that undermining can be quite extensive without causing necrosis, provided there is limited interference with the blood supply. Clinical experience suggests the same is true for horses.[29,30,33] Used in conjunction with a mesh-expansion technique on the limbs of dogs, 360-degree undermining was not associated with any major complications.[34]

The benefits derived from undermining skin are the result of freeing the skin from its underlying subcutaneous attachments, which allows the surrounding skin to be drawn toward the defect and makes use of the elastic properties of the surrounding skin. The degree of benefit depends on the location of the wound and any other factors that alter the biomechanical properties of skin. In chronic wounds, as the process of wound healing proceeds, first the inflammatory edema and later the development of fibrous tissue will both lead to a loss of skin elasticity, which may necessitate undermining the adjacent skin more extensively to achieve the desired effect. Having an appreciation of the lines of maximal tension and extensibility in different regions of the body will allow the surgeon to take full advantage of the gains made by undermining.

Tissue Debulking

When performing delayed secondary closure or scar revision, removal of exuberant granulation tissue or fibrous tissue reduces the volume of the tissue in the wound, which allows closure under less tension. To determine the plane of dissection for débridement, the incision should extend beyond the perimeter of the wound into normal skin. The thickness of the normal skin can be used as a guide to determine the appropriate plane to undermine the skin adjacent to the wound site or scar. The granulation tissue or scar tissue within the wound site should then be removed until the normal subcutaneous tissue plane is encountered. When debulking a chronic wound, careful dissection is required to avoid inadvertent transection of vessels or nerves, trauma to tendons, and penetration of synovial structures.

Tension-Release Incisions

Tension-release incisions are longitudinal incisions made adjacent to the wound margin, which aid in advancing skin to cover the wound. When the tissue between the wound edge and the incision is undermined, the elevated skin acts as a bipedicle advancement flap. This technique is used to allow the movement of a limited amount of skin in those areas where simple undermining alone does not adequately relieve tension, yet the size of the defect to be closed does not justify more elaborate plastic procedures. A release incision can be made on either side of the wound or on both sides. When making a single release incision the incision should be placed at a distance of approximately the width of the wound away from the wound's edge over healthy tissue. When using two release incisions the placement of each incision is similar to that of a single incision. Release incisions are generally not closed but are allowed to heal by second intention. The wound that is created when using a single relief incision is similar in area to that of the original wound. When creating two release incisions the resultant size of each new wound is approximatley half of the original wound area, which is preferred over one release incision. Creation of tension release incision(s) is justified when it is necessary to provide skin coverage to a more vital area such as bone or tendon or ligament.

Mesh Expansion

Mesh expansion is a simple technique that when combined with undermining can increase the amount of skin available for mobilization and therefore wound coverage. The mesh incisions are created when the skin has been freed from attachments to the underlying fascia. The small incisions are made in staggered rows, parallel to the wound edge (Figure 24-5). Applying traction to the skin edges provides a good indication for the optimal location and orientation of the stab incisions to gain maximal relief of tension.[35] As a guideline, in fresh wounds, the distance between the individual stab incisions and between the adjacent rows should measure approximately 1 cm.[33] Placing either the rows or the incisions too closely should be avoided to prevent the possibility of the adjacent stab incisions becoming confluent or causing focal vascular compromise. The appropriate length of the stab incisions varies depending on the degree of elasticity and pliability of the undermined skin. In fresh wounds, more expansion can be obtained with smaller incisions, whereas for those wounds where the surrounding skin is thickened and fibrotic, longer incisions are required to achieve the same result. In some older wounds, the stab incisions do not readily open at the time of suturing, but clinical experience has shown that expansion does occur within a few hours postoperatively as a result of the viscoelastic nature of skin. Stress relaxation results in the undermined skin's becoming fatigued over time and in the stab incision's opening, which together result in reduced tension on the suture line.

The small mesh expansion wounds heal faster and more cosmetically than longer tension release incisions; however, they provide less skin relaxation than the larger release incisions. A combination of the methods of undermining, tension-release incisions, and mesh expansion can be used successfully to facilitate primary and delayed primary and secondary wound closures on the head, trunk, and limbs of horses.

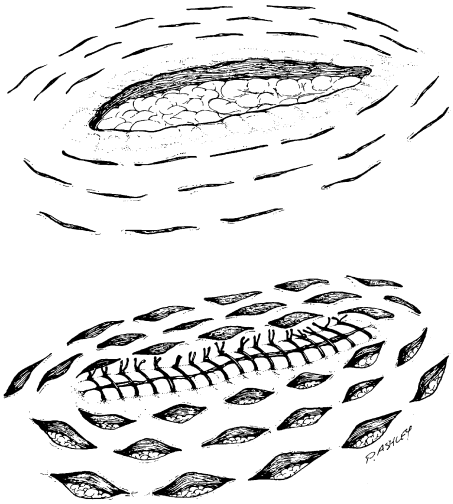

Figure 24-5. The mesh expansion method of closure. After undermining of the skin around the defect, two or three staggered rows of stab incisions are made parallel to the skin edges. After closure of the defect, the stab incisions gape open to provide both tension relief and drainage.

V-to-Y-Plasty and Y-to-V-Plasty

The V-to-Y-plasty technique for relieving tension requires a V-shaped incision with the point of the V directed away from the defect to be closed. When closure of the original defect has been accomplished, closure of the V incision is done by converting it to a Y (Figure 24-6). The longer the legs of the V, the greater will be the degree of tension relief; however, this method provides only a limited amount of skin movement and is not suitable for management of large defects.

This technique has been advocated for scar revision of the eyelid (see Chapter 56) and for relieving tension when closing elliptical defects.[28,29] Conversely the Y-to-V-plasty may be used if additional tension is required, such as for the surgical correction of an entropion.

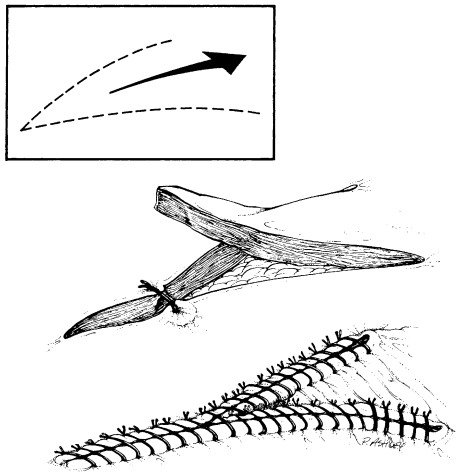

Figure 24-6. V-to-Y-plasty for tension relief. With this technique, tissue is mobilized in the direction of the arrow.

z-Plasty

Z-plasties can be used to relieve tension along a linear scar, to change the orientation of an incision line or a scar, or to relieve tension when closing a large defect.[13,29,36] These effects are achieved by recruiting loose tissue from the sides of the surgical site, and the success of the procedure depends on the presence of sufficient loose skin in the area to permit mobilization of tissue.[23,29] Where this situation does not exist, as in the lower limb of the horse, other procedures may be more suitable.

A Z-plasty represents a modification of a transposition flap. The basic Z-plasty is performed by making a Z-shaped incision with subsequent undermining and transposition of the two triangular portions of skin formed by the dissection (Figure 24-7). This technique results in a change in the orientation of the central limb of the Z and a gain in its length. The gain in length achieved by this procedure is in the direction of the original central limb—that is, before transposition occurs. Commonly, the Z is symmetric, with the three limbs of equal length and the same angle between the two arms and the central limb. An angle of 60 degrees is most frequently used, which results in a 75% gain in length. Varying the angles of the Z and the length of the arms influences the amount of elongation, and decreasing the angles results in a decreased gain in length. Maintaining a 60-degree angle and lengthening the limbs of the Z can achieve an increased gain in length; however, the greater the increase in length, the greater will be the resultant tension in the surrounding tissue. Using multiple small Z-plasties in the place of a single large procedure can minimize this problem.[21,29] Although the biomechanics and mathematics of Z-plasties have been examined extensively, this has been done using laboratory models, which are not influenced by the same forces as those found in living tissue.[37]

When Z-plasties are used to realign scars in humans, the limbs of the Z are oriented to follow the lines of minimal tension in the area, and the angles between the limbs of the Z vary according to the obliquity of the scar in relation to the lines of tension. If the procedure is to be used successfully for scar revision in horses, more information on the lines of tension and extensibility in the equine integument is needed.

In the horse, Z-plasties have been recommended as a relaxation procedure for closing elliptical defects and for revising scars involving the eyelids that have resulted in an acquired entropion.[38] A double Z-plasty was used to correct a stenotic naris in a filly following trauma.[39] Although other uses undoubtedly exist, the procedure does have some limitations and potential complications. The most common complication encountered with the use of a Z-plasty is ischemia and necrosis of the tips of the triangular skin flaps. The risk of this problem occurring can be reduced by making the base of the flap as broad as possible, and by minimizing tension on the suture line. Use of a modified three-point (half buried horizontal mattress) suture at the tips of the flaps is also recommended.

W-Plasty

W-plasties are designed to improve the cosmetic appearance of a scar. Scars that are at right angles to lines of maximal tension tend, over time, to widen and become cosmetically less acceptable. By using a W-plasty, such a scar is excised by making a series of opposing zigzag incisions in such a way that, when sutured, the incisions will interdigitate, with the resulting final suture line resembling a series of Ws. In this technique, the orientation of the single scar is changed from being at right angles to the lines of maximal tension to a series of connected smaller scars that are better aligned with the force of maximal tension and are thus not inclined to widen. Because this procedure involves the removal of tissue, with a subsequent increase in tension on the surrounding skin, and because it is primarily aimed at cosmetic improvement, careful consideration should be given to the need to undertake the surgery in the first place. If the technique is to be used, a template should be fashioned and the incision lines drawn carefully on the skin to ensure that the two opposing incisions are adequately aligned.

SKIN STRETCHING AND EXPANSION TECHNIQUES

Presuturing

Presuturing is based on the phenomenon that skin held in tension will gradually stretch. It is an ideal method to consider when elective procedures, such as skin biopsy, scar revision, or delayed wound closure, are being considered. It relies on the physiologic response of mechanical creep, which results in skin

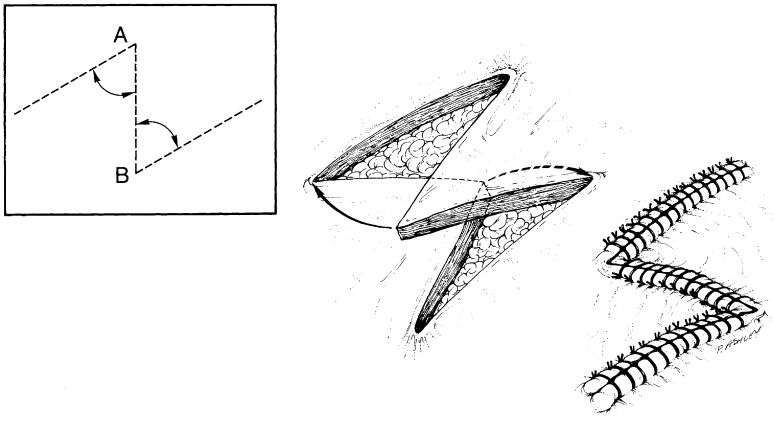

Figure 24-7. *Z*-plasty. The length of the central limb *(A to B)* is equal to the length of the two arms, and tissue movement occurs along the plane of the central limb. After closure, the orientation of the *Z* incision changes.

expansion and can be achieved when sutures are placed several hours prior to removal of a skin lesion. Using local anesthesia, sutures are placed to plicate skin over an area of planned excision.[40] The direction of suture placement is chosen according to the anticipated direction of primary closure. Wounds of the body and upper limb are generally the most amenable to presuturing. Large, nonabsorbable sutures (No. 1 or No. 2, polypropylene or nylon) are placed through the skin perpendicular and 2 to 6 cm to either side of the lesion. The sutures are then tightened to elevate and fold the skin over the lesion or wound. If presuturing is used in conjunction with tumor excision, the surgeon should be careful to place the sutures distant to the lesion to avoid iatrogenic seeding of tumor cells into healthy tissue.

The gain in tissue expansion persists for some time following removal of the preplaced sutures. This gain in tissue then facilitates closure of the wound site. In a pig model, the tension required to close a standardized wound was decreased by 40% when using this technique. Compared with simply undermining the surrounding skin, presuturing did result in some tissue gain and an initial decrease on closing tension, although this advantage diminished over time.[41] The advantages of presuturing diminish as edema and skin stiffness increase when sutures are left in place for too long. In the horse, when preplaced sutures were left in place for 24 to 30 hours, moderate edema resulted that was not seen when the sutures were removed after 4 to 8 hours.[42,43] Periods as short as 2½ hours have been shown to reap the benefits of presuturing.[40]

The original description of this procedure suggests that presuturing avoids the potential complications that can be associated with undermining.[40] However, the combination of presuturing and undermining may provide additional advantages.

Intraoperative Tissue Expansion

Intraoperative tissue expansion has been advocated as a means of decreasing incision closure tension.[44] Expansion can be obtained by techniques as simple as the placement of towel clamps and pulling the wound edges together for a short period of time prior to closure or the insertion of a Foley catheter in which the balloon is then inflated. Others have temporarily implanted balloon devices.[45] Variations in technique have been described ranging from tension being applied in three or four cycles of 3 to 5 minutes, to a single loading cycle applied for 20 minutes. Although expansion was initially thought to occur because of the viscoelastic property of creep, this explanation has been challenged.[46,47] Histologic studies fail to confirm the dermal changes associated with mechanical creep.[48] It is more likely that expansion results from the inherent elastic property of skin (extensibility), which is site dependent. [46,49-52] Although there are no published reports of using this technique in equine reconstructive surgery, there are sufficient reports of the potential benefits in other species to justify considering this procedure as part of a strategy to manage tension across a wound closure.

External Skin-Stretching Devices

Several external skin stretching devices are commercially available. These attach onto or into the normal skin on each side adjacent to the wound site or proposed incision and take advantage of the creep and stress-relaxation properties of skin. A maximum tension force of 3 kg/cm^2 can be safely applied across the wound to stretch the surrounding skin. Depending on the system used, tension can be gradually increased until this limit is reached.

The successful use of a relatively simple system using adherent skin pads and elastic connecting cables has been described in dogs.[53] The placement of the adherent pads relative to the skin edge can be varied according to the amount of skin recruitment required. The amount of tension was increased every 6 to 8 hours. The greatest gains in recruitment of surrounding skin appeared to be achieved 48 to 72 hours after application. The technique was reported as being well tolerated, and no biologic complications were encountered. Separation of the adhesive pads from the skin as tension was increased was the most common technical complication. Although the greatest gains in skin recruitment are seen between 48 and 72 hours, stretching can be achieved in as short as 20 to 30 minutes in wounds where the skin and subcutaneous tissues are normal. Interspersing periods of relaxation between periods of tension application results in greater elongation of tissues.

Elastomers

Silicone elastomer tissue expanders have been used in other species, but reports of their use in horses are limited.[54] Elastomers, which consist of a silicone pouch that can be gradually inflated percutaneously with sterile saline are available in various sizes and shapes to allow flexibility in placement and amount of tissue expansion. An elastomer with a base size 2½ times that of the defect that is to be reconstructed is selected and implanted below the skin adjacent to the lesion site. After the implant incision has healed, the pouch is expanded every 4 to 7 days through a dedicated self-sealing portal, thereby stretching the overlying skin. The optimal inflation pressure of the elastomer has not been determined in large animals. In a canine study, 20 to 40 mm Hg was initially used, but pressures approaching capillary pressure have been shown to be safe.[55] When sufficient skin expansion has been achieved, the elastomer is removed and the expanded tissue is undermined and used as a skin flap to cover the defect.

This procedure takes advantage of both mechanical and biological creep and can expand the area of the skin two- to threefold. The epidermis responds to the gradual inflation by an increase in mitotic activity and a net increase in epidermal tissue. The dermis thins in response to the elastomer expansion. However, skin flap thickness remains relatively the same because of the fibrous tissue response, which develops around the elastomer.[48] The initial thickening of the epidermis and thinning of the dermis seen immediately after expansion has been shown to return to normal 24 weeks after transfer of expanded skin in dogs, and similarly, the capsule that forms around the expander disappears.[56] However, this fibrous capsule results in an overall thickening of the raised flap and loss of elasticity of the expanded skin.[55] This increased stiffness may compromise the surgeon's ability to manipulate the flap in the reconstructive procedure. Removing the fibrous capsule may resolve this problem to some extent, and the effect of capsulectomy on the blood supply to the expanded skin has been investigated.[57] Although some controversy surrounds this issue, it appears that removing or incising the capsule to facilitate advancement of the flap does not critically affect the survival of the expanded tissue.[57]

The use of expanders may be of substantial benefit where limited skin is available for reconstructive procedures. Use in

dogs is well tolerated and has been associated with increases of 35.9% and 37.3% in the surface areas of skin on the antebrachium and crus following controlled expansion.[55,58] Skin flaps made from expanded skin are twice as likely to survive as those created from acutely raised skin.[59] This increased viability is attributed to similar effects seen in the delay phenomenon.[59,60] Despite increased survivability and the added benefits of presence of hair follicles, similarity of haircoat color of the donor skin to the surrounding area, the absence of a donor site defect, and the quality of the skin that is obtained, use of silastic elastomers in horses has been limited.

Complications include possible pain during saline distention, pressure necrosis of overlying skin, implant failure, wound dehiscence, and premature exposure of the expander necessitating its removal before adequate expansion had been achieved. Tissue expanders represent an additional dimension to the treatment and repair of skin defects in horses.

COSMETIC CLOSURE OF SKIN DEFECTS OF VARIOUS SHAPES

When undertaking scar revisions or the closure of traumatic wounds, the size of the lesion, the amount of redundant skin in the area, the elasticity of the surrounding skin, and the inherent lines of extensibility are all factors that must be considered during the planning process. Sound clinical judgment remains the cornerstone of making the required surgical decisions. Complex mathematical analyses have been developed to assess the optimal closure patterns of wounds with complex shapes, and these analyses may provide helpful information in the future.[61] The amount of skin that can be excised, the extent of undermining of the surrounding skin, the type of tension-relieving techniques used, the type of suture patterns to be applied, and postoperative support of the repair are other details that should be carefully contemplated when reconstructive or skin mobilization procedures are undertaken.

Fusiform Defects

Fusiform defects have sometimes been incorrectly referred to as elliptical defects; the latter do not have the tapered or spindle-shaped appearance of a fusiform excision and do not close as well.[10,29] Fusiform excisions can be used for elective scar revision, with the long axis of the defect being oriented parallel to the lines of skin tension (see Figure 24-4). A 3 : 1 or 4 : 1 length-to-width ratio will allow closure of the defect with a fairly even distribution of tension along the length of the suture line.[21,29] Shorter, broader defects result in uneven tension and in the formation of dog-ears at the end of the suture line.[62] In fusiform incisions, dog-ears can be prevented by tapering the ends of the excised portion of skin (described later). As with any reconstructive procedure, careful marking of the proposed incision facilitates excision of a piece of skin that has the optimal orientation and length-to-width ratio.

Triangular, Square, and Rectangular Defects

Where sufficient movable skin is present to permit closure, triangular defects should be closed from the points of the triangle toward the center, which will result in the formation of a Y-shaped suture line. There is a tendency for the skin at the center of the Y to develop ischemic necrosis. A half-buried horizontal mattress suture in a three-point closure (see Figure 24-2) can be used to secure this central point with minimal interference to the blood supply.[22,29]

Square and rectangular defects can also be closed by beginning to suture at the corners of the defect and closing toward the center.

V-Shaped Defects

V-shaped defects are fairly commonly encountered in veterinary medicine, and often part of the V-flap is lost or requires débridement prior to closure. After débridement, the resulting chevron-shaped defect can be closed with a Y-pattern in a fashion similar to that described for triangular defects. If the chevron-shaped defect is long and narrow, provided there is sufficient movable skin in the surrounding area, a fusiform incision could be made around it, which is then closed like a linear defect.

Circular Defects

Closure of circular defects commonly results in the formation of dog-ears, and several techniques have been described to prevent their occurrence. It is possible to close the defect without modifying its circular appearance by orienting the suture line along the relaxed skin lines of tension, followed by removal of any dog-ear as described later. A circle can be converted into either an X- or a Y-shape by tightening sutures placed at three or four points equidistant to each other around the circumference of the defect. The closure can then be completed, although this method also results in the formation of dog-ears.

The simplest method of facilitating closure of a circular defect is to excise two triangles on opposite sides of the circle, thereby creating a fusiform defect (see Figure 24-4). From a geometric point of view, the height of each triangle should be at least equal to the diameter of the circle; this will result in the removal of normal skin equivalent to 1½ times the area of the original defect.[63] The implications of removing this amount of normal skin must be carefully evaluated.

The double S-shaped incision with a bi-winged excision, the bow tie, and the combined V-incision are alternative methods of modifying circular defects and have been well described.[36] These methods are all designed to minimize scarring and dog-ear formation in human cosmetic surgery and are best applied to small circular defects.

Management of Skin Puckers (Dog-Ears)

Skin puckers or dog-ears often develop at the end of the suture line when closing oval or irregularly shaped defects or when using skin flaps as part of a reconstructive procedure. These dog-ears, if ignored, will flatten out to some extent over time and may be of little consequence. If the pucker is small and the cosmetic appearance is of limited importance, they can be managed by placing small sutures through half the thickness of the skin. In some locations, however, this conservative approach may result in an unsightly blemish. Because correction of dog-ears requires lengthening of the incision, it is worthwhile to assess the extent to which the cosmetic result will be compromised if the pucker is left alone.

To remove the puckered skin, the dog-ear can be stabilized with an appropriate instrument and removed by incising around the base, which results in a lengthening of the incision line

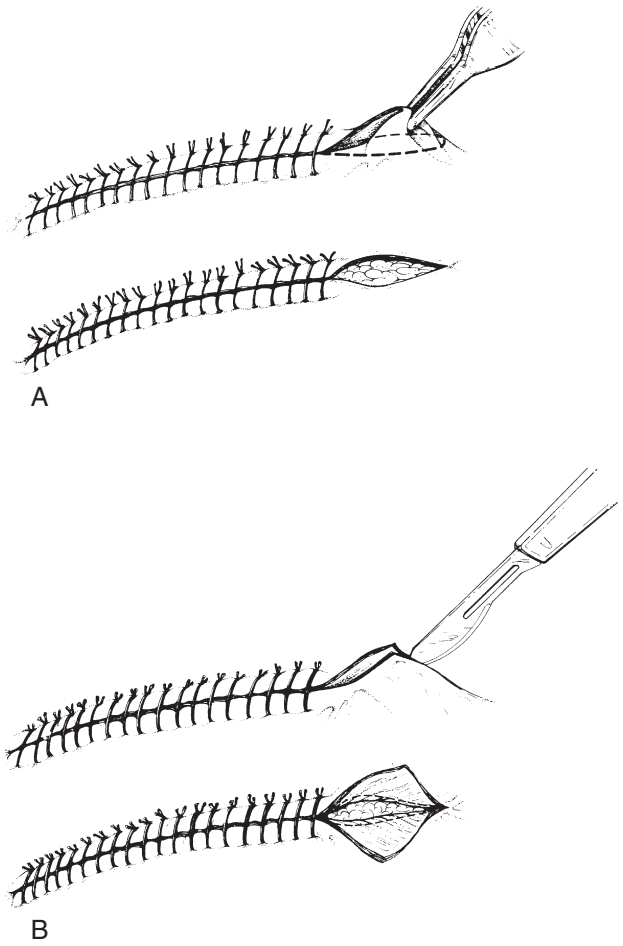

Figure 24-8. Methods for removal of dog-ears. **A,** The dog-ear is held securely, elevated and is excised level with the surrounding skin, leaving a small fusiform defect to close. **B,** The dog-ear is bisected to form two small triangular flaps, which are resected level with the skin surface.

(Figure 24-8, *A*). An alternative method is to close the incision until the dog-ear becomes well defined, and then to extend the initial incision through the middle of the dog-ear. This converts the pucker into two small triangular flaps of skin (Burow's triangles), which can be excised at their base (see Figure 24-8, *B*).

SKIN FLAPS

A skin flap or pedicle graft is a portion (or flap) of skin that, when created, can be moved from its original location to a second location on the body while, by virtue of its attachment (or pedicle), it maintains its vascular supply.[62] The survival of these grafts, unlike that of free skin grafts, is not dependent on successful revascularization from the recipient site. Pedicle grafts, because they have an attached blood supply, can be used to cover areas that, because of an inadequate blood supply, would be unsuitable for free skin grafts. When successful, pedicle grafts reduce healing time, minimize wound contraction and epithelialization, and provide an acceptable cosmetic result.[64] In contemplating the use of a skin flap, the clinician should evaluate the amount of loose skin in the area. It is preferable to be able to close the secondary defect that is created when the flap is raised, so sufficient skin must be available to be moved without producing excessive tension, either on the donor site or on the suture line at the new location. For this reason, large wounds on the distal limb may not be suitable candidates for a local skin flap, although some success with the use of advancement flaps and rotating flaps has been reported in treating wounds on the dorsal aspect of the tarsus and in the area of the metacarpophalangeal joint.[54,65,66] Pinching the adjacent skin into folds or pushing it toward the center of the primary defect can be used to assess availability of surrounding skin.

Flaps can be classified as local or distant, depending on the relationship of the donor site to the recipient site. Distant flaps usually involve either multiple surgical procedures or a degree of postoperative immobilization and patient compliance that makes them impractical in equine surgery. Local flaps (including rotation flaps, transposition flaps, and interpolating flaps) are usually one-stage procedures and can be simple advancement or rotating flaps. These types of flaps have been used successfully in the horse (discussed in more detail later).

Skin flaps can also be categorized as random or axial pattern flaps according to the nature of their vascular supply. Random pattern flaps, which do not have a defined vascular pattern, depend on the subdermal plexus for their survival and have been referred to as subdermal plexus flaps.[7,29] Axial pattern flaps, on the other hand, contain at least one major direct afferent and efferent cutaneous vessel and are usually named in accordance with that vessel. This enables a larger area of skin to be mobilized. In the dog, the omocervical, thoracodorsal, and caudal superficial epigastric flaps are well recognized and established axial pattern flaps. In horses, local random pattern flaps have greater potential use than axial pattern flaps.

Survival of a flap depends on the survival of its intrinsic vasculature. Random pattern flaps have traditionally been defined by the length-to-width ratio. Although the detailed differences in regional blood supply to the skin have not been studied in the horse, differences have been reported in other species, and those studies show that the optimal ratio for a flap varies with the location on the body. Length-to-width ratios from 1 : 1 to 3 : 1 for single-pedicle and 1.5 : 1 to 3 : 1 for bipedicle flaps have been reported in the veterinary literature. However, studies in dogs and pigs have shown that flaps of a standard length with similar vascularity survive regardless of the flap width.[67] In horses, a correlation of flap width to viable length in random pattern pedicle flaps of the trunk has been shown.[64] Length-to-width ratios of 1.5 : 1 and 3 : 1 have been used successfully on the limb and head, respectively.[54] The narrower the pedicle of the flap, the more maneuverable it will be, but the greater will be the chances of compromising its blood supply. On the other hand, a wide-based flap will have a good vascularity, but mobility, particularly rotational, is limited.

The *delay phenomenon* has been used to enhance the blood supply to the skin to increase the chances of flap survival. This two-stage technique involves incising and undermining the skin and subcutaneous tissue of the proposed flap and then leaving it sutured in its original location for a period of time. The incision may be on three adjacent sides of the flap or on two opposite sides, and undermining may extend to involve all or part of the flap. The most appropriate period of delay varies among species: in rabbits and pigs, 8 to 10 days is adequate, whereas circulation in single-pedicle flaps in dogs rose to 150% of normal in 3 weeks.[62,68] Although the mechanism by which flap survival improves using this technique is not well understood, both denervation and ischemia have been shown to play a role.

Both contribute, with a resultant increase in the number and diameter of the vessels as well as an increase in blood flow into the flap through its primary attachments.[62] Although this technique is not commonly used in equine reconstructive surgery, as the horizons of plastic surgery in the horse are advanced, specific studies are required to determine the most appropriate method and optimal time delay.

Tension applied to pedicle grafts has been shown to decrease blood supply and thus increase flap necrosis in pigs.[69] However, a study assessing the effect of tension on perfusion of axial and random pattern flaps in foals concluded that adequate perfusion was maintained in equine axial pattern flaps even when subjected to high tension.[70] Assessment of flap viability based on clinical criteria, such as temperature and color, is probably adequate. Other, more sophisticated techniques, such as radioisotope tests, histamine wheal, and fluorescein tests, have been described but are not routinely used.[21,29] It is debatable whether venous compromise or arterial compromise, or a combination of both, is of prime importance in flap necrosis; however, tension on the pedicle from stretching or over-rotating should be avoided to help prevent flap necrosis. Several methods for improving flap survival, in addition to the delay phenomenon, have been suggested; these include the use of vasodilators, massage, dimethyl sulfoxide, and hyperbaric oxygen, but all are of questionable clinical value and require further investigation before they can be recommended.[21,29]

Attention to the specific anatomy of local cutaneous vasculature is important in flap elevation. Flaps raised on the trunk should be elevated beneath the cutaneous musculature, whereas on the limb, dissection should be performed deep in the subcutaneous tissue. Careful design of the flap and good postoperative care greatly improve the success rate achieved. Planning should include determining which type of skin flap is to be used as well as mapping and marking the borders of the piece of skin to be elevated. This can be done using a paper towel or a cloth template of the defect to be covered. The flap should be slightly larger than the defect. Sharp corners, which may be prone to ischemia, should be avoided.

Advancement Flaps

Advancement flaps are random pattern flaps that can be of either the single or the bipedicle type. They have also been referred to as sliding flaps because there is no rotation of the elevated skin. A V-Y-plasty is an example of an advancement flap.

Simple bipedicle advancement flaps are performed either by making a single longitudinal incision on one side of the wound or by incising on each side of the defect. These incisions are made parallel to the wound edge to create skin flaps that are approximately the same width as the original defect. The bipedicle nature of this flap (Figure 24-9) provides two sources of blood supply to ensure survival of the mobilized portion of skin. The flaps and surrounding skin can be undermined and the initial defect closed. Depending on the nature of the surrounding skin, the new defects can be either closed or left to heal by contraction and epithelialization. As with many types of flaps, moving a single or bipedicle advancement flap may create some laxity in the adjacent skin. This can be managed by removing small triangular portions of skin called Burow's triangles, thereby preventing the formation of a dog-ear at the end of the suture line.

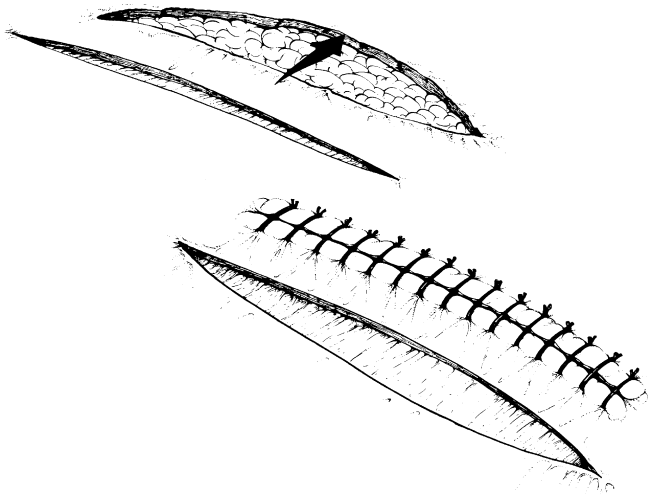

Figure 24-9. Simple bipedicle flap. This can be used with a single incision, as depicted here, or a second incision can be made on the opposite side of the primary defect. By using two relaxing incisions, it may be possible to close the secondary defects.

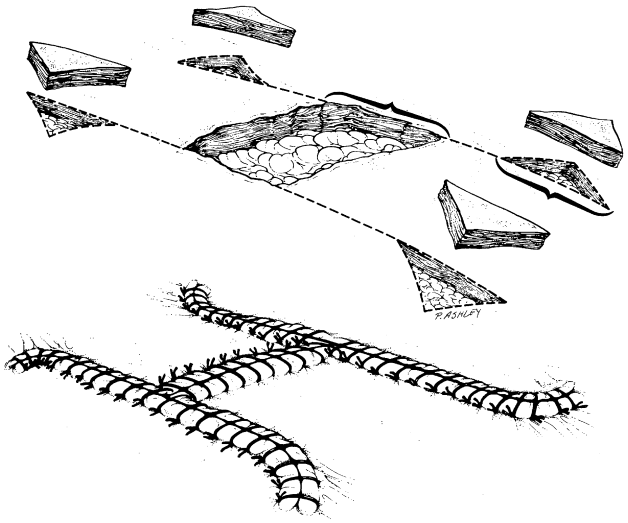

Figure 24-10. Sliding H-plasty. Burow's triangles are created and excised to prevent dog-ear formation after mobilization of the skin.

An H-plasty is a modification of a single-pedicle flap and is used to mobilize tissue during closure of a rectangular defect. Two single-pedicle flaps are used on opposite sides of the defect to advance the skin, and Burow's triangles are excised at the base of both pedicles to avoid dog-ear formation (Figure 24-10).

Rotating Flaps

Three types of rotating flaps are described: rotation, transposition, or interpolating flap. A *rotation flap* requires creation of a semicircular incision and moving the tissue laterally to cover the defect (Figure 24-11). A rectangular, single-pedicle flap that is created adjacent to a defect and subsequently rotated on its pedicle is called a *transposition flap* (Figure 24-12). These can be rotated up to 180 degrees, but increasing the amount of rotation has the effect of shortening the flap, which must be considered when planning the surgery. Moving a similar rectangular flap

Figure 24-11. Rotation flap. This pedicle flap is semicircular and can be used to cover small defects.

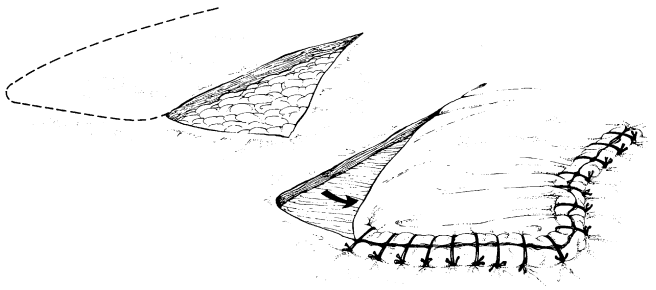

Figure 24-12. Transposition flap. This rectangular pedicle flap can be rotated up to 180 degrees to cover a defect.

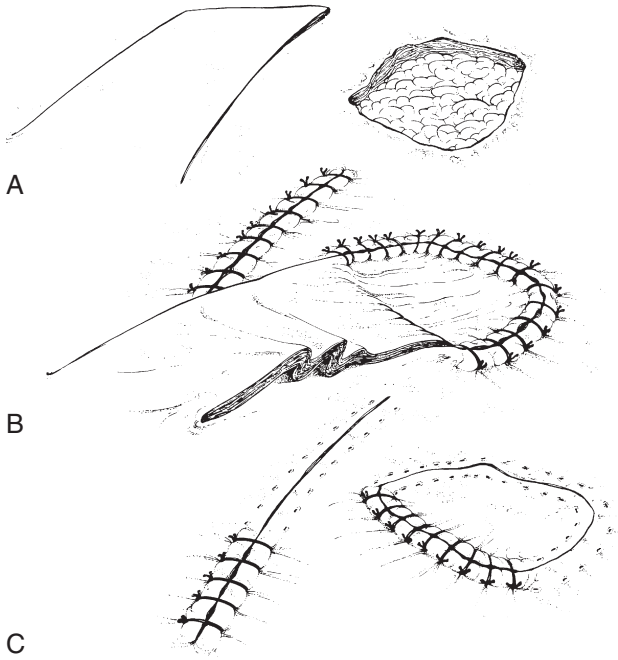

Figure 24-13. Interpolating flap. **A,** A pedicle flap is lifted from the skin adjacent to the defect. **B,** The flap bridges normal tissue to cover the defect. **C,** After the defect has healed, the pedicle is resected and the incised edges are sutured.

onto an immediately adjacent defect (i.e., the pedicle bridges an intact portion of the skin) creates an *interpolating flap* (Figure 24-13). The successful use of a rotating flap has been described in the management of a lesion on the craniolateral aspect of the tarsus of a horse.[65]

VASCULARIZED FREE TISSUE TRANSFERS

Vascularized free tissue transfers developed as a natural progression to axial pattern flaps with their well-defined arteriovenous supply. Microvascular techniques enable the skin/vessel unit to be transferred to remote sites and survive. An excised axial pattern graft can be reattached to its original perfusing vessel (orthotopic flap) or to vessels at a different location (heterotopic flap). Although the latter grafting technique is attractive as a useful technique in horses for the repair of limb wounds, successful protocols have remained currently elusive. Identified axial pattern flaps (based on dedicated direct cutaneous arteries) in the horse include those perfused by the caudal superficial epigastric artery, the medial saphenous artery, branches of the deep circumflex iliac artery, branches of the superficial temporal artery, dorsal and lateral nasal arteries, thoracodorsal artery, suprascapular artery, and linguofacial artery. Attempts to transfer a large axial pattern flap based on the deep circumflex iliac artery and vein, and a smaller one based on the saphenous artery and the medial saphenous vein, to distant defects in experimental horses have been unsuccessful.[8,71] In two separate studies, none of the heterotopically transferred flaps survived for longer than 4 to 6 days. The reasons for failure are not fully understood but may include poor perfusion as a result of vasospasm, a species tendency toward vascular thrombosis, or ischemia-reperfusion (IR) injury. Neutrophils are a significant source of the oxygen free radicals that mediate IR injury. A study to assess the role that IR injury plays in the failure of vascularized free tissue transfer in the horses showed no difference in neutrophil accumulation between control myocutaneous flaps and those subjected to a period of ischemia and reperfusion.[72]

Although the initial results are disappointing, further studies may resolve the difficulties encountered and make this technique a viable option in the future.

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CHAPTER 25

Skin Grafting

James Schumacher

INDICATIONS FOR GRAFTING

The most common indication for applying a skin graft to a horse is a wound so large that it cannot heal by any other means, but the presence of any open wound that cannot be sutured is an invitation for grafting (Figure 25-1). The rapid healing achieved with a skin graft may make grafting the most economical choice for managing some wounds that cannot be sutured. A healed skin graft contains some epidermis, dermis, and some adnexa, including hair follicles, so it is likely to be more cosmetic and resilient than a wound healed primarily by epithelialization. Skin grafting should not be regarded as an option of last resort and should not be avoided because of fear of failure. Many techniques of skin grafting require little experience and only basic surgical equipment.

GRAFT CLASSIFICATION

The two basic types of skin grafts are the pedicle graft and the free graft.[1-3] A *pedicle* graft remains connected to the donor site, at least temporarily, by a vascular pedicle that sustains the graft, ensuring its viability. A pedicle graft is useful for covering a poorly vascularized wound, because the graft does not depend on the vascularity of the recipient site. The cosmetic appearance of wounds healed by application of a pedicle graft is good, because all components of skin are transferred. Cutaneous wounds of horses are rarely covered with a pedicle graft because the inelastic nature of equine skin makes mobilizing an adequate amount of skin for advancement difficult.[4] In humans and dogs, skin flaps that incorporate a direct cutaneous artery and vein (i.e., axial-pattern flaps) can be transferred to adjacent sites, or even to remote sites, by using microsurgical techniques to anastomose the flap's vessels to local recipient vessels.[5] Similar transfers in horses are unsuccessful, apparently because of reperfusion injury to the graft.[6,7]

A *free* skin graft is a piece of skin that has been completely separated from its local blood supply and transferred to a wound at another site, where it must establish new vascular connections to survive.[8,9] Free grafts can be categorized in several different ways, one of which is by their source. Most skin grafts applied to wounds of horses are *autografts* (or isografts), which are grafts transferred from one site to another on the same individual (Table 25-1).[10] The recipient of an autograft mounts no detrimental immune response against the graft because the recipient and donor are the same. Autografting is the most common and practical type of grafting of wounds of horses.

A graft transferred between different members of the same species is an *allograft* (or homograft), and a graft transferred from one species to another is a *xenograft* (or heterograft). The recipient of an allograft or xenograft mounts an immune response against the graft, causing the graft to be eventually rejected.[8,11] A second allograft from the same donor applied to the wound survives only a few days, but an allograft from a different donor survives for about the same time as the first allograft.[8] Because the immune mechanism is highly specific, an allograft from a different donor is not affected by circulating lymphocytes sensitized against the first allograft. Although allografts and xenografts are rarely applied to wounds of horses, these grafts are occasionally useful as a biological bandage.[12-14]

Free skin grafts can be categorized according to their thickness. Full-thickness grafts are composed of epidermis and the entire dermis; split-thickness (or partial-thickness) grafts are composed of epidermis and only a portion of the dermis. The percentage of dermis within the graft influences the graft's

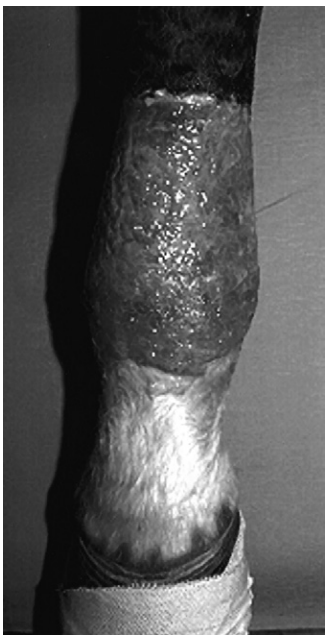

Figure 25-1. A granulating wound on the fetlock of a horse. This wound is so extensive that it cannot heal by any means other than skin grafting.

TABLE 25-1. Graft Terminology*

Greek	Latin	Definition
Autograft	Isograft	A graft transferred from one site to another on the same individual
Allograft	Homograft	A graft transferred between different members of the same species
Xenograft	Heterograft	A graft transferred from a member of one species to a member of another species

*To avoid confusion, Greek and Latin prefixes that identify the source of the graft should not be used interchangeably in discussions.

Boykin JV, Molnar JA: Burn Scar and Skin Equivalents. p. 523. In Cohen IK, Diegelmann RF, Lindblad WJ, (eds): Wound Healing. Saunders, Philadelphia, 1992

acceptance, durability, and cosmesis at the recipient site. The thickness of dermis within the graft is directly proportional to the graft's durability and cosmesis but inversely proportional to the graft's ability to survive.

Full- or split-thickness free skin grafts can be applied to the surface of wounds as solid or meshed sheets, or they can be embedded in granulation tissue as pinch, punch, or tunnel grafts. Each method of free grafting has its merits and drawbacks, and the method selected depends on circumstances, such as the size and location of the wound, the necessity for cosmesis, the financial constraints imposed by the owner, the equipment available, and the expertise of the surgeon.

PHYSIOLOGY OF GRAFT ACCEPTANCE

Recipient Bed Characteristics

A prime requisite for free grafting is a recipient bed that is vascular and free of infection and devitalized tissue. A free graft cannot be expected to survive if placed on bone denuded of periosteum, tendon denuded of paratenon, or cartilage denuded of perichondrium, but practically any other tissue is capable of supporting a free graft.[15-18] Joint capsules, ligaments, and adipose tissue are capable of accepting a skin graft, but they do not accept a graft readily. Fresh surgically created wounds or fresh clean wounds caused by accident are vascular and free of infection and so readily accept a graft.[18,19] Granulating tissue is also capable of accepting a graft, although not as readily as is fresh tissue.[2,19,20] Granulation tissue healthy enough to allow epithelium to proliferate at its margin is generally healthy enough to accept a skin graft[18] (Figure 25-2).

Adherence

The graft is initially adhered to the recipient site by fibrin, which binds to collagen within the graft.[9,15,21,22] Vessels and fibroblasts invade the fibrin matrix by the fourth or fifth day, and the graft becomes firmly united to the recipient bed by around the 10th day.

Figure 25-2. Proliferating epithelium at the margin of a wound on the hip of a horse. Proliferating epithelium, represented by the pale ring around the edge of the wound, indicates that the wound is healthy enough to accept a skin graft.

Serum Imbibition (Plasmatic Circulation)

The newly applied graft is nourished by plasma-like fluid that it passively imbibes, by capillary action, into its open vessels, a process referred to as *plasmatic imbibition* or *plasmatic circulation*.[9,15,23] Oscillating movement of fluid into the vessels keeps the vessels open for revascularization. The graft becomes edematous and remains so until vascular connections to the graft are restored.

Revascularization

After about 48 hours, new capillaries generated in the recipient bed traverse the fibrin layer to anastomose with those in the graft, a process called *inosculation*.[9,15,23,24] Simultaneously, new capillaries from the recipient bed invade preexisting vessels within the graft, and others cut new vascular channels into the dermis (called *neovascularization*). The graft revascularizes between the fourth and fifth day after grafting, and reestablishment of circulation and lymphatic drainage brings about resolution of edema.[24]

Vascular bridging is a phenomenon that may enable a portion of the graft overlying a relatively avascular portion of the wound to revascularize.[17,25] With vascular bridging, capillaries enter the portion of the graft overlying the avascular portion of the wound from the relatively highly vascular aspects of the surrounding recipient bed to create a vascular bridge across the avascular portion of the wound. For vascular bridging to occur, the avascular area of the wound must be small, and the surrounding area must be highly vascular. Full-thickness grafts bridge avascular areas better than split-thickness grafts do, because collateral circulation within the dermis of a full-thickness graft is less interrupted than that within the dermis of a split-thickness graft.[25]

Organization (Organic Union)

The epidermis of the graft becomes hyperplastic after grafting and can double in thickness during the first 2 weeks.[18,26] The epidermis, especially that of a full-thickness graft, may die and slough in some areas, exposing pale dermis beneath it[26] (Figure 25-3). The exposed dermis may closely resemble granulation tissue, so a surgeon may remove the upper layer of the wound in preparation for a second graft, not realizing that a portion of the dermis has been accepted. Dermis can be differentiated from granulation tissue by its paler color. Exposed dermis becomes covered with epithelial cells that migrate from the hair follicles and the eccrine glands within the dermis.

Split-thickness skin grafts of humans usually begin to develop pigment about 4 weeks after grafting,[2,15,16] and the same is true of split-thickness grafts of horses.[26] Split-thickness grafts often remain scaly for several months, until the eccrine glands regenerate.[2,19,26] Split-thickness grafts of horses, no matter the thickness at which the grafts are harvested, are likely to be devoid of sweat glands,[27,28] and even full-thickness grafts of horses may be devoid of sweat glands.[27] Hair begins to appear in split-thickness grafts at between 4 and 6 weeks[26] and often grows to a greater than normal length, at least on grafts applied to limbs.[29,30] The growth of longer hairs may be related to a difference in temperature between the grafted limb and the donor site.

Reinnervation returns more rapidly, but to a lesser extent, in split-thickness grafts than in full-thickness grafts.[15,31] Sensation

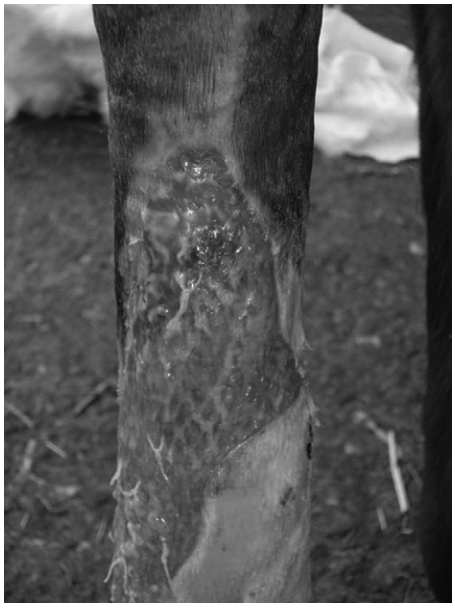

Figure 25-3. Grafted wound of a horse. The epidermis has sloughed from all areas of the graft, exposing pale dermis beneath it. This wound contained enough dermis to allow rapid healing. (Courtesy Justin Perkins, BVetMed, University of London)

in split-thickness grafts of humans usually first appears between the seventh and ninth weeks but occasionally is present as early as the tenth day.[32] Sensation in split-thickness grafts remains incomplete and patchy. Regional nerves enter the base and margins of the graft and, by following vacated neurolemmal sheaths, reconstruct the innervation pattern of the grafted skin.[31] Humans sometimes suffer from hyperesthesia of the grafted wound,[16,31] and this may occur in horses also.

Graft Contraction

After being harvested, grafts immediately shrink because of recoil of the elastic fibers within the deep dermal layers.[18] This shrinkage is referred to as *primary contraction*. Primary contraction is greatest in full-thickness grafts and decreases as grafts become thinner. Full-thickness grafts from humans contract to nearly half their original size after being harvested. Split-thickness grafts that are about half the thickness of the whole skin contract to about three quarters of their original size, and grafts composed solely of epidermis do not shrink at all. The consequences of primary contraction are minor, because it is reversed when the graft is stretched and sutured or stapled to the recipient site.

Although grafts appear to contract after being accepted, this contraction is probably caused by contraction of the recipient bed, not by the graft itself.[18] Open wounds heal, at least in part, by contraction, which is brought about by the action of contractile fibroblasts, or myofibroblasts, within the wound.[18,33] Skin grafts inhibit wound contraction, to various extents, by accelerating the life cycle of the myofibroblasts within the wound.

The extent to which a skin graft is capable of inhibiting wound contraction is influenced by the relative thickness of the graft's dermis and the stage of healing of the recipient bed at the time of grafting.[18,33,34] The total percentage of the dermis grafted is more important than the absolute thickness of the skin graft in preventing wound contraction. Fresh or granulating wounds of humans and rats receiving a full-thickness graft tend to contract less than wounds receiving a split-thickness graft, even when the full-thickness graft is thinner than a split-thickness graft, indicating that physical or biochemical differences between the upper and lower portions of the dermis may strongly influence the effect of the skin graft on wound contraction.[33-35]

In a study on the effects of grafts on wound contraction in horses, fresh or granulating wounds to which a split-thickness graft was applied contracted at the same fractional rate as fresh or granulating wounds to which a full-thickness graft was applied.[36] The fractional rate of contraction of grafted fresh wounds, however, was less than that of grafted, granulating wounds.

Contraction of wounds of humans can sometimes lead to considerable cosmetic defects and to *contracture,* which is deformity of one or more joints caused by contraction of the wound.[8] Contraction of wounds of humans is sometimes intentionally inhibited by autografting or allografting to prevent cosmetic and functional defects caused by contracture.[2] Skin of horses is more mobile than that of humans and so is more capable of healing by contraction. Contraction of wounds of horses rarely, if ever, causes contracture of joints, so contraction of wounds of horses, before or after grafting, should be considered beneficial because it results in more rapid healing and smaller scars. If applied to a wound after contraction has commenced, skin grafts, regardless of whether they are full thickness or split thickness, appear to have little effect on inhibiting contraction in horses.[37] In one study, application of Meek micrografts (see "Modified Meek Grafting Technique," later) to naturally occurring wounds of horses stimulated, rather than inhibited, contraction of the wounds.[30]

GRAFT FAILURE

Reasons for graft failure include fluid accumulation beneath the graft, movement, and infection.[16,18,22,23,29] Grafts applied to horses fail most frequently because of infection.[38]

Fluid Accumulation

The graft must contact the wound intimately to prevent fluid from becoming interposed between the graft and the wound.[23] A hematoma, seroma, or exudate beneath the graft prevents fibrin from attaching the graft to the wound and acts as a barrier to the ingrowth of new vessels.[17,23] A graft can survive for only several days by imbibing nutrients from the hematoma or seroma, and if budding capillaries from the recipient bed are unable to traverse this barrier within this time, the graft expires. To avoid formation of a hematoma or seroma beneath a graft, grafting of a hemorrhagic wound should be delayed until hemorrhage has ceased.

Infection

Granulation tissue always has resident bacteria at its surface, but it contains abundant blood vessels and phagocytic cells that act as a partial barrier to bacterial invasion.[8,15,17,22,27,39] The wound becomes infected when the concentration of bacteria in the tissue exceeds the ability of humoral and cellular defenses to destroy the bacteria, and for most types of bacteria, this concentration seems to be about 10^5 organisms per gram of tissue.

Survival of a skin graft is better correlated to the concentration of bacteria in the recipient bed than to any other single factor.[40,41] If a wound is found, by quantitative bacterial analysis, to contain more than 10^5 bacteria per gram of tissue, the wound should not be grafted.

Because quantifying the concentration of bacteria in a wound is time-consuming and often impractical, at least for most veterinary surgeons, and because the concentration of bacteria in the wound is sometimes not as important as the *type* of bacteria,[22] the bacterial status of a wound to be grafted is usually assessed qualitatively. A wound that shows signs of inflammation, such as redness, swelling of the surrounding area, and formation of exudate, should be assumed to be infected. Bacteriologic culture of a wound that appears to be infected should precede grafting, and isolates should be tested for antimicrobial susceptibility. Unfortunately, even a wound that has no signs of inflammation and appears to be healthy may have more than 10^5 bacteria per gram of tissue.[41]

The concentration of some bacteria, most notably β-hemolytic streptococci and *Pseudomonas* spp, required to infect a wound is much less than 10^5 per gram of tissue.[22,42] β-Hemolytic streptococci produce proteolytic enzymes, including streptokinase and staphylokinase, that are destructive to both the graft and its recipient bed. These bacterial enzymes, and others, destabilize the fibrin network between the graft and the recipient wound by catalyzing the conversion of plasminogen to plasmin, which digests fibrin.[22,43] *Pseudomonas* species also critically weaken a graft's fibrinous attachment by producing elastase, which specifically degrades elastin in the dermis of the graft to which fibrin attaches.[22]

Inflammation

Granulating wounds on the distal portion of the limb of horses are inherently chronically inflamed,[44,45] which causes the granulation tissue to be poor in quality and to produce a moderate amount of exudate, factors that impair acceptance of a graft. Granulating wounds of ponies are less apt to be chronically inflamed,[45] and consequently, a graft applied to a granulating wound of a pony may be more likely to be accepted than would a graft applied to a granulating wound of a horse.

Motion

The graft must remain immobilized long enough to become vascularized. Shearing forces between the graft and the underlying recipient bed dislodge the fibrin seal, impairing plasmatic imbibition, inosculation, and capillary ingrowth. Shearing forces occur when the recipient wound is located in a highly mobile area, such as over a joint, or when the grafted area is inadequately immobilized. Fibrin glue has been used successfully to promote acceptance of skin grafts applied to wounds of human patients by increasing the strength of the attachment between the graft and the wound.[43] A trial using fibrin glue to attach skin grafts to wounds of horses, however, did not decrease the incidence of graft failure.[46]

GRAFTING TECHNIQUES

Preparation of the Recipient Site

Preparation of the recipient site is the most important contributor to survival of a free graft.[4] Factors to consider when determining if a wound is prepared sufficiently to accept a graft are the wound's vascularity and whether the wound appears to be infected.[15] Grafting should be delayed if the wound appears to have inadequate vascularity to support a free graft or if it appears to be infected or to have a higher than ordinary vulnerability for developing infection.

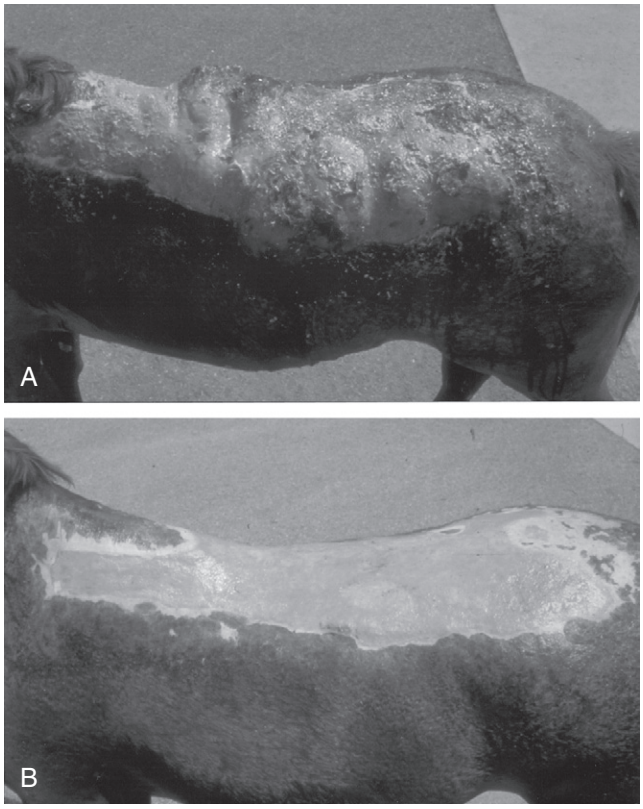

Figure 25-4. Burn wound on the back of a horse. **A,** The wound contained exuberant, relatively avascular granulation tissue that was excised to skin level, with the horse standing, in preparation for grafting. **B,** About 75% of a meshed, split-thickness skin graft, applied several days later, was accepted.

Highly vascular granulation tissue readily accepts a graft, but as the granulation tissue matures, its vascularity and ability to accept a graft diminish[42] (Figure 25-4). Fibrous, poorly vascularized granulation tissue, or granulation tissue plagued by unremitting infection, should be excised to a level below the margin of the wound to allow the wound to form fresh, vascular granulation tissue free of infection before a graft is applied.[29] Granulation tissue can usually be excised with the horse standing, because granulation tissue has no innervation.

Delaying grafting of a fresh wound for several days may decrease the time required for the graft to revascularize.[47] Sprouting capillaries capable of rapidly revascularizing the graft develop in the wound within 48 hours, so, by delaying grafting for 2 days after wounding, the time before the graft begins to revascularize (i.e., the phase of plasmatic imbibition) can be reduced by about one half. Wounds that are allowed several days to develop sprouting capillaries before being grafted are referred to as prepared wounds.

Although eliminating all bacteria from a wound is difficult, removing streptococci is relatively easy, because streptococci are nearly always susceptible to penicillin.[42,48,49] Occasionally, penicillin or other β-lactam antibiotics are ineffective in resolving a

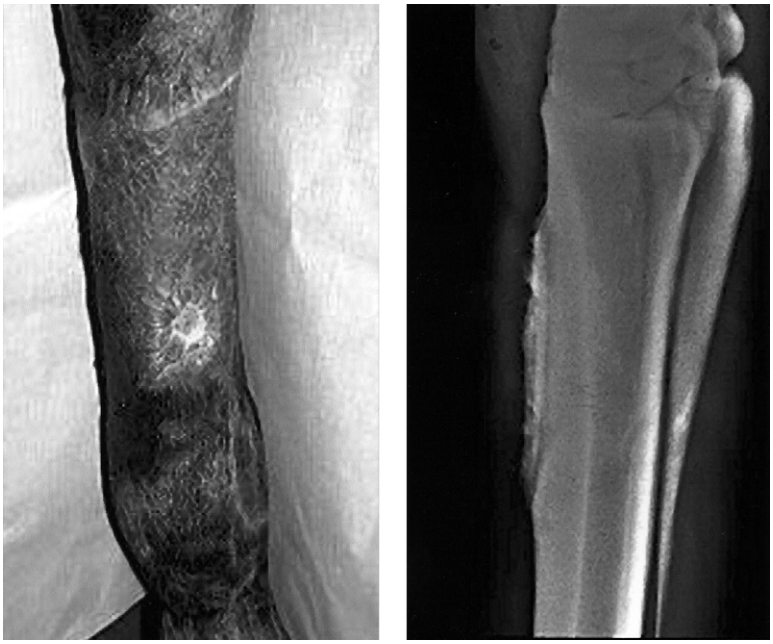

Figure 25-5. A graft applied to a healthy-appearing, granulating wound on the dorsal aspect of a metacarpus was completely accepted, but a draining tract soon appeared in the healed wound. Drainage was caused by infection of bone beneath the wound and the development of a bone sequestrum.

streptococcal infection, because other resident bacteria, particularly staphylococci and gram-negative bacteria, produce β-lactamase, an enzyme that inactivates β-lactam antibiotics.[48] The efficacy of the β-lactam antibiotic against streptococci can be preserved if the β-lactam antibiotic administered is effective against β-lactamase–producing bacteria as well as streptococci. The activity of the β-lactam antibiotics can also be preserved by administering clavulanic acid, a potent inhibitor of β-lactamase, in conjunction with the β-lactam antibiotic.[48] Clavulanic acid has no therapeutic value when administered alone. Pseudomonads are usually sensitive to topically applied mafenide acetate, aminoglycosides, or polymyxin-B sulfate.[18,50]

Topical application of an antimicrobial drug to an infected, granulating wound is often more effective for eliminating infection than is systemic administration of the same antimicrobial drug, because systemically administered antimicrobial drugs often fail to reach therapeutic concentrations within granulation tissue.[15,51] Fibrin within granulation tissue appears to prevent adequate penetration of the antimicrobial drug.

Before a wound is grafted, it should be evaluated thoroughly for evidence of damage to synovial structures, ligamentous or tendinous tissue, or bone. Bone beneath slowly healing wounds or beneath wounds with draining tracts should be examined radiographically for evidence of a foreign body and for evidence of infection or sequestration of bone (Figure 25-5). Granulation tissue within chronic, slowly healing wounds, particularly those wounds that appear to be pruritic, should be examined histologically for the presence of larvae of the equine stomach worm *Habronema*. Some neoplasms, such as sarcoids or carcinomas, sometimes resemble granulation tissue.

Preparation of the recipient bed immediately before grafting should include clipping or shaving the skin around the wound. A surgical scrub solution can be used to cleanse the skin around the wound, but the wound itself should be cleansed only by rinsing it with physiologic saline solution or a balanced electrolyte solution. Contact between the wound and the detergent found in surgical scrub solutions increases the wound's susceptibility to infection.[52]

Preparation of the Donor Site

The area selected as the donor site depends on the technique of grafting selected and on whether the graft is to be harvested with the horse anesthetized or standing. Because harvesting skin creates a wound, the donor site, especially the donor site of a split-thickness graft, should be at an inconspicuous location.

Hair is removed from the donor site by clipping with a No. 40 clipper blade, by shaving, or by applying a chemical depilatory agent. Although chemical depilatory agents may irritate the donor skin, they have no clinically apparent detrimental effect on acceptance of grafts (my observation). When hair is removed completely from the surface of the donor site by shaving or by depilation, the graft should be marked in a way that ensures that when the graft is applied, the direction of its hair growth matches that of the skin surrounding the recipient site. The direction of hair growth on the graft remains visible when the donor site is clipped with a No. 40 clipper blade. After the donor site is scrubbed, it should be rinsed thoroughly with water or physiologic saline solution so that no residue detrimental to the graft, such as isopropyl alcohol, remains.

Full-Thickness Sheet Grafting

The site most suitable for procuring a full-thickness graft is the cranial pectoral region, where the skin is relatively mobile (Figure 25-6).[53,54] The graft can be removed, and the donor site sutured, with the horse anesthetized or with the horse standing and sedated after desensitizing the donor site with local anesthetic solution. If a sutured wound in this region should dehisce, healing occurs rapidly by contraction with little scarring. The scar is less obvious if the donor site spans the midline of the pectoral region.[54a] The graft should be cut slightly larger than the recipient site, even though the shrinkage caused by recoil of elastic fibers is largely reversed when the graft is stretched and fixed to the margin of the wound.[3,18]

Subcutaneous tissue attached to the undersurface of the graft acts as a barrier to plasmatic imbibition and ingrowth of new

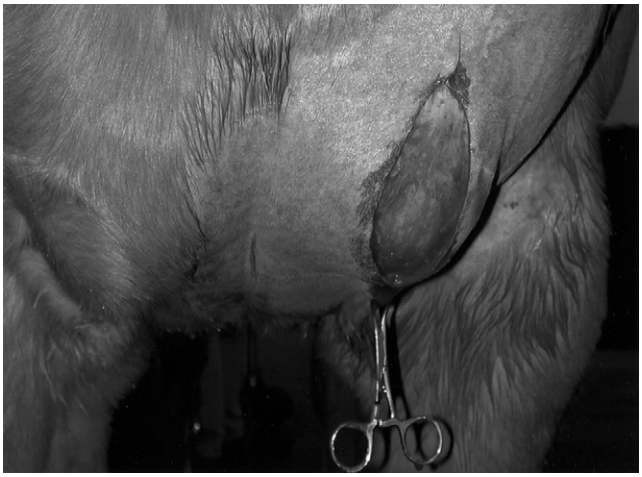

Figure 25-6. Cranial pectoral area from which a full-thickness graft was obtained. The graft was obtained with the horse standing. The incision was closed with skin staples.

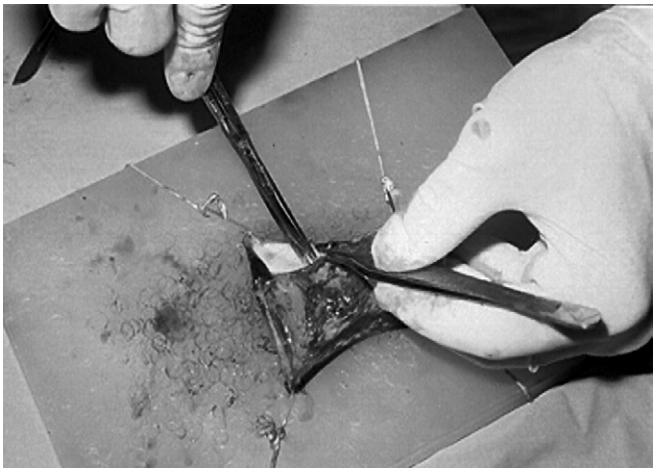

Figure 25-7. Removing fascia and fat from a full-thickness graft using sharp dissection. A scalpel blade can also be used.

vessels, so it must be removed to expose the dermis and its vasculature.[1,3,23] Subcutaneous tissue can also be excised from the graft as the graft is being excised, or it can be excised after stretching and fixing the graft, epidermal side down, to a sterile piece cardboard, Styrofoam, plastic sheet, or polypropylene block. Fascia and fat should be removed by sharp dissection, because scraping the subcutaneous tissue from the graft can injure the dermis and its vasculature (Figure 25-7). The graft should be attached to the recipient site with slight tension to keep the small dermal vessels open for inosculation.[55]

Full-thickness sheet grafting requires no sophisticated equipment for harvesting and can be performed with the horse standing. Full-thickness skin grafts resist trauma better than split-thickness skin grafts, and because they contain all the properties of the surrounding skin, full-thickness skin grafts provide the best cosmetic appearance and function of any type of free graft.[2,3,8,38,56] A full-thickness skin graft can be easily procured to cover a cutaneous defect created by excision of a cutaneous lesion and is particularly advantageous for treating horses undergoing removal of a cutaneous malignancy, such as a sarcoid, of the distal portion of a limb because closing the wound with a graft enables excision of a wide area of tissue surrounding the neoplasm, thereby decreasing the likelihood of recurrence of neoplasia.[57]

Full-thickness grafts are not accepted as readily as split-thickness grafts, because they have fewer exposed blood vessels than split-thickness grafts and because their requirement for nourishment is greater.[8,23,56,58] Circumstances for full-thickness grafting must be ideal, because blood vessels in a full-thickness graft that are available for diffusion of nutrients or for inosculation by vessels in the recipient wound are few in number and large in diameter. Although healthy, highly vascular granulation tissue is capable of accepting a full-thickness skin graft,[36] full-thickness grafting is usually reserved for fresh, uncontaminated wounds.[2,8,15,26,42] Desquamation of full-thickness grafts, even those applied to fresh wounds, has been reported to occur frequently after application to wounds of human patients[59] and to wounds of horses.[57] The entire epidermis may become necrotic and slough without adversely affecting the final appearance of the graft because re-epithelialization occurs from the dermal adnexa and from the epithelial edges of the wound (see Figure 25-3).[57,60]

A considerable disadvantage of full-thickness grafting of wounds of horses is the horse's lack of redundant donor skin. This disadvantage makes full-thickness grafting practical for covering only relatively small wounds, provided that the surgeon intends to suture the donor site after the graft is harvested. The largest elliptical graft that can usually be harvested from the cranial pectoral region of a horse of average size is about 8 cm (3 inches) at its widest point. A full-thickness graft can be used to cover a wound larger than the graft itself by meshing and expanding the graft, but full-thickness skin of horses is too thick to be easily meshed using some commercial meshgraft dermatomes, such as the Zimmer mechanical skin mesher (see "Meshing Sheet Grafts," later).

Split-Thickness Sheet Grafting

A split-thickness graft is composed of epidermis and a portion of the dermis. The thickness of a split-thickness graft is determined by the relative amount, rather than the absolute amount, of dermis included in the graft, because the thickness of the dermis varies between individuals and between regions.[2,27,29] To obtain a split-thickness graft, a portion of the dermis with its overlying epidermis is harvested with a free-hand knife, a drum dermatome, or a power-driven dermatome. Relatively inconspicuous donor sites for obtaining a split-thickness graft from horses are the ventral portion of the abdomen and the ventrolateral aspect of the thorax, caudal to the elbow.[53,61-63]

Hand Instruments

Scalpel blades, double-edged razor blades, and surgical straight razors are sometimes used to harvest small, split-thickness grafts from humans, but these types of dermatomes are of little use for harvesting skin grafts from horses, because they can be used to harvest only relatively small grafts and because their use requires considerable technical expertise to obtain a graft of consistent thickness.

Large grafts necessary to cover wounds of horses can be cut with a variety of knives designed specifically for harvesting skin. The Watson skin grafting knife, a modification of the

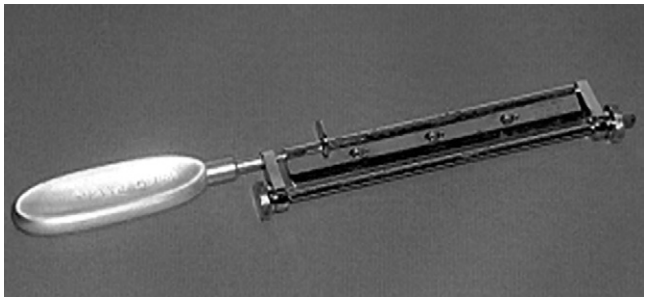

Figure 25-8. The Watson skin grafting knife. Using this dermatome, a surgeon can harvest a graft 100 mm (4 inches) wide. The knife uses disposable blades and is fitted with an adjustable roller in front of the blade to control the depth of the cut.

Braithwaite knife, can harvest a graft 100 mm (4 inches) wide, uses disposable blades, and is fitted with an adjustable roller in front of the blade to control the depth of the cut (Figure 25-8). Because the thickness of the graft is controlled by the adjustable roller, only a moderate amount of practice is required to develop the expertise necessary to achieve a uniform, split-thickness graft of the desired thickness. The position of the adjustable roller is controlled by a screw marked with calibrations at one end and a lock at the other. The thickness of graft represented by each calibration is learned by experience. The thickness of the graft depends not only on the position of the roller but also on the angle of incidence at which the skin is cut and on the pressure applied to the knife while cutting.[42] The heavier the pressure and the greater the angle of incidence, the thicker is the harvested skin.

Drum Dermatomes

A clinician with only minimal training can harvest a uniform section of skin with a drum dermatome.[2,15,23] The drum dermatome uses a knife that oscillates back and forth on a piston to precisely split the dermis of a section of skin that has been glued to the drum of the dermatome (Figure 25-9). The depth of cut is predetermined by the caliber of shims placed next to the blade.

The drum dermatome allows the surgeon to harvest a graft of the exact dimensions to fit the wound for which the graft is intended.[15] Using a template from the recipient site, glue is applied only to a similar area on the drum, so that as the drum is rotated, only that portion of skin that adheres to the drum is cut. The drum dermatome requires no external power source. It is considerably more expensive than a free-hand knife but not as expensive as power-driven dermatomes.[8] A major drawback to the use of the drum dermatome to harvest grafts for wounds of horses is that the length of the graft is limited by the circumference of the drum. An example of a drum dermatome is the Padgett Manual Dermatome (Z-PD-100R).

Power-Driven Dermatomes

Power-driven dermatomes use a rapidly oscillating blade, driven by an electric motor or gas turbine to harvest a split-thickness skin graft with predictable precision. The depth and width of cut can be adjusted precisely. Commonly used power-driven dermatomes are the Stryker electric dermatome and the Brown pneumatic or electric dermatome (Figure 25-10). The cutting

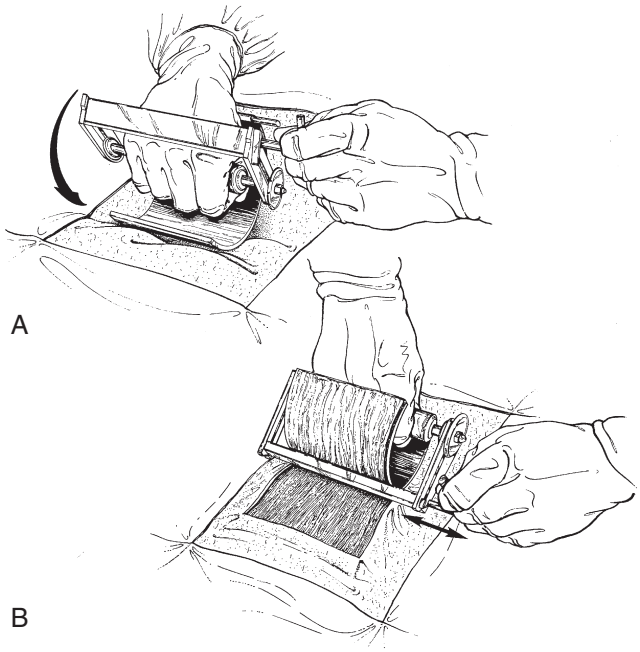

Figure 25-9. Harvesting a skin graft with a drum dermatome. **A,** Rotating the drum of a drum dermatome elevates skin to which it has been glued. **B,** The skin attached to the drum is cut at a precise depth with a knife that oscillates back and forth on a piston.

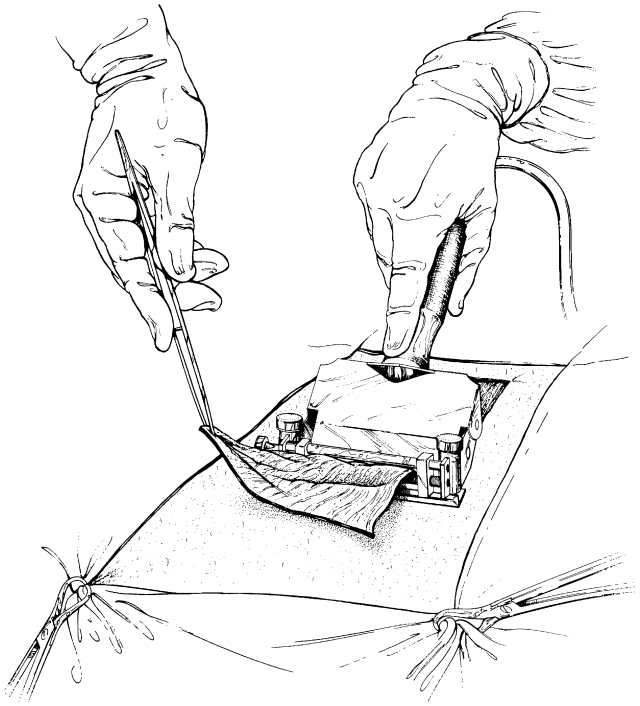

Figure 25-10. Harvesting a split-thickness skin graft with a pneumatic dermatome. Although they are expensive, power-driven dermatomes allow the cutting of very uniform grafts by minimally experienced operators.

head of a pneumatic dermatome oscillates more rapidly than that of an electric dermatome, making harvesting smoother.[23]

The Davol-Simon skin graft dermatome is a relatively inexpensive dermatome powered by a rechargeable handle, similar to the handle of an electric toothbrush.[17,18] A disposable, nonadjustable cutting head harvests a split-thickness skin graft 33 mm (1½ inches) wide and 0.38 mm (0.015 inch) thick. Grafts obtained with this skin graft dermatome are too thin to impart a cosmetic appearance to wounds of horses, but they may be useful for covering relatively small wounds when cosmetic appearance is not important.

Although a power-driven dermatome provides precision harvesting, the widest graft that can be harvested with many power dermatomes is only 76 mm (3 inches). A surface firmer than the ventral aspect of the abdomen, the least conspicuous donor site on the horse, is often necessary when harvesting with a power-driven dermatome. Although operating a power-driven dermatome requires only a minimum of experience, a power-driven dermatome requires skilled maintenance.[8] Because of their expense, power-driven dermatomes are found primarily only at large equine surgical referral centers.

An advantage of the free-hand knife over the power-driven dermatome is that consistently uniform grafts more than 100 mm wide (4 inches or greater) can be harvested using a free-hand knife. Grafts can be harvested from the ventral aspect of the abdomen with a free-hand knife, which is a difficult feat for a power-driven dermatome. A free-hand knife is far less expensive than a power-driven dermatome, requires less maintenance, has fewer parts to malfunction, and is easier to sterilize and to transport.

Technique of Harvesting a Split-Thickness Graft

A split-thickness skin graft can be harvested from any convex surface of the body, but the cosmetic appearance of the horse should be considered when selecting a donor site, because a split-thickness graft of acceptable durability and hair coverage cannot be obtained without creating a large epithelial scar at the donor site. Harvesting a split-thickness skin graft is painful to the horse and can be accomplished only when the horse is anesthetized.

To harvest skin from the ventral aspect of the horse's abdomen using a free-hand knife, the horse is positioned in lateral recumbency with its abdomen protruding beyond the edge of the table. The ventral portion of the abdomen must protrude over the edge of the table to accommodate the handle of the hand-held knife. The donor site is prepared for aseptic surgery, but draping is not necessary. Harvesting usually begins at the umbilicus and extends craniad. The donor site can be stabilized, if necessary, by assistants stretching the skin with towel clamps. The knife is applied to the skin at an acute angle of about 5 to 10 degrees, and the skin is cut using a regular sawing motion, concentrating on moving the blade back and forth, rather than pushing it forward (Figure 25-11). Lubricating the donor site with physiologic saline solution and the blade with a light coating of mineral oil reduces friction. Mineral oil does not adversely affect acceptance of the graft.[8,18] Slight, uniform tension should be applied to the cut end of the graft as the graft is harvested.

After several centimeters of graft have been cut, the graft and donor site should be inspected to ensure that the graft is the desired thickness before harvesting is continued. The thickness

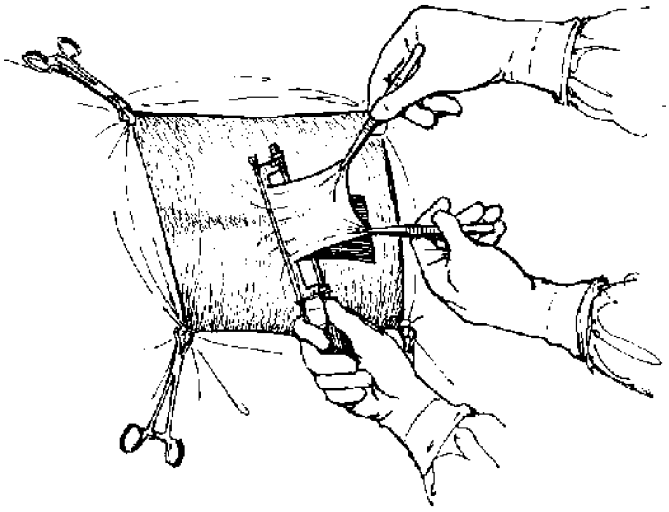

Figure 25-11. Harvesting split-thickness skin from the ventral aspect of the abdomen using a free-hand knife. The ventral portion of the abdomen must protrude over the edge of the table to accommodate the handle of the hand-held knife.

of the graft can be assessed subjectively by examining the graft for translucency and the donor site for the pattern of bleeding.[17,18] A shallow cut through the dermis produces a translucent graft and exposes many small bleeding vessels, whereas a deep cut produces a more opaque graft and exposes fewer but larger bleeding vessels. If subcutaneous fat is exposed, the graft is full-thickness and therefore much too thick. The depth of cut can be changed by repositioning the adjustable roller, by changing the pressure applied to the knife, or by raising or lowering the knife's angle of incidence. When the desired length of graft has been cut, the knife is tilted upward to sever the graft. If the graft is to be meshed and fully expanded, the length of graft should be considerably longer than the wound to which it is applied, because expanding the graft's width also shortens the graft's length.

The split-thickness graft is the most useful type of graft, because it can be used to cover defects too large to be covered by a full-thickness graft and because it survives more readily than does the full-thickness graft.[8,20,42,58] The cosmetic appearance of a wound healed by split-thickness skin grafting is inferior to that of a wound healed by full-thickness skin grafting, but it is superior to that of a wound healed by island grafting (see "Island Grafts," later). A split-thickness graft is less conveniently procured than a full-thickness graft because, to obtain a split-thickness graft, the horse must be anesthetized.

Meshing Sheet Grafts

A split-thickness or full-thickness sheet graft can be applied to a recipient bed as a solid or as a meshed sheet. A graft is meshed by uniformly fenestrating it manually with a scalpel blade or mechanically with a meshgraft dermatome. The primary reason for meshing a sheet graft is to allow the graft to uniformly cover a wound larger than the graft itself.[2,15,17,23,29,53,61-66] Grafts are also meshed to prevent serum, blood, or exudate from mechanically disrupting a newly applied graft from its delicate fibrinous and vascular attachments to the recipient bed. Fenestrations also enable a topically applied antimicrobial agent to contact a large

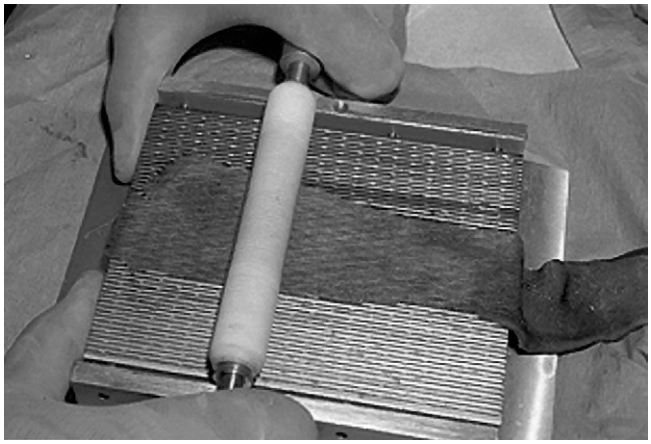

Figure 25-12. A Padgett mechanical skin mesher. The mesher consists of an aluminum block with staggered, parallel rows of blades and a Teflon rolling pin. The graft is positioned on the block, dermal side down, and pressed into the cutting blades with the rolling pin.

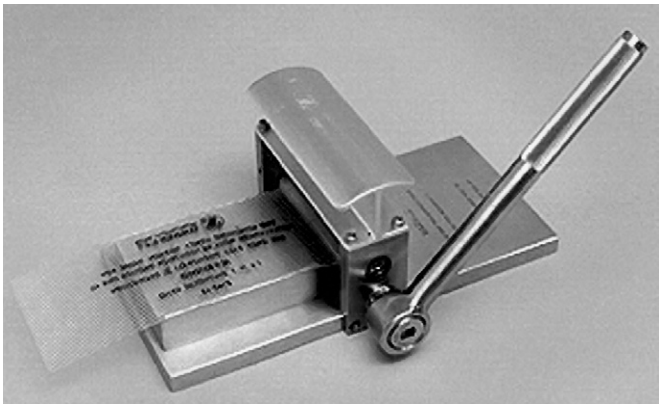

Figure 25-13. A Zimmer mechanical skin mesher. The mesher is capable of expanding the graft to many times its original area. The graft is positioned on a specially grooved plastic carrier, and the graft and carrier are fed through the mesh cutter by turning a hand crank.

portion of the recipient wound. A meshed graft conforms to an irregular surface better than does a nonmeshed graft, and its ability to expand allows it to better tolerate motion. Fenestrations in the graft fill with fibrin, which increases the graft's stability on the recipient bed.[38]

An example of a meshgraft dermatome is the Padgett mechanical skin mesher (Mesh Skin Graft Expander, No. Z-PD-170). This instrument consists of an aluminum block with staggered, parallel rows of blades and a Teflon rolling pin. The graft is positioned on the block, dermal side down, and pressed into the cutting blades, in the direction of the blades, with the rolling pin (Figure 25-12). The rolling pin should first be rolled lightly across the graft to secure the graft to the blades of the mesher and then firmly so that the blades pierce the graft. The staggered cuts produce a meshed pattern that allows the graft to be expanded to three times its original width.

The Zimmer mechanical skin mesher (Zimmer Meshgraft Dermatome) is a more elaborate and expensive meshing instrument, capable of expanding the graft to one and one-half, three, six, or nine times its original area. The graft is positioned dermal side down on a specially grooved plastic carrier selected according to the degree of expansion desired, and the graft and carrier are fed through the mesh cutter by turning a hand crank (Figure 25-13). The pattern engraved onto the plastic carrier determines the distance between cuts. The graft must be placed on the ridged surface of the carrier, because placing the graft on the smooth side of the carrier causes the graft to be shredded into spaghetti-like strips.

An important consideration when purchasing a meshgraft dermatome is the width of grafts routinely harvested with the skin-graft dermatome. Grafts up to 100 mm wide are harvested routinely with a free-hand skin graft knife, but a graft wider than 76 mm is difficult to mesh with a Zimmer mechanical skin mesher. Grafts up to about 110 mm wide can be fitted onto the cutting surface of the Padgett mechanical skin mesher. Although the Padgett mechanical skin mesher accommodates a graft only 13.5 cm (5.3 inches) long, a longer graft can be meshed in sections.

Full-thickness sheet grafts of horses are difficult to mesh on commercial meshing instruments and often must be meshed manually. Meshing a graft manually is tedious, especially if the graft is large or if it must be greatly expanded. The skin is fixed to a cutting board, such as sterile sheet of cardboard or Styrofoam, with the dermal side up, and after the subcutaneous fascia is removed staggered, parallel rows of incisions are created using a scalpel blade. The longer and the more numerous the incisions, the greater the expansion.

Figure 25-14. An expanded, meshed graft on the back of a foal. The graft has the appearance of chain-link fencing and uniformly exposes portions of the wound within the fenestrations. These exposed portions of the wound must heal by contraction and epithelialization.

An expanded, meshed graft has the appearance of chicken wire or chain-link fencing and uniformly exposes portions of the wound within the fenestrations (Figure 25-14). The amount of wound exposed within the fenestrations depends on the degree to which the graft is expanded. Portions of the wound covered by the graft heal primarily, but each portion of the wound exposed within the fenestrations must heal by contraction and epithelialization. The exposed portions of the wound epithelialize rapidly because of the enormous increase in the border of the graft from which epithelial cells migrate.[61,62] The epithelium that eventually covers the expanded fenestrations has no adnexa, causing the wound to heal with abundant, diamond-shaped epithelial scars. The more a graft is expanded at application, the more apparent are the diamond-shaped scars.[2,42,61,62] Greatly expanded grafts are difficult to apply, and

the greater the expansion, the longer the period of epithelialization. The size of the epithelial scars can be reduced by attaching the graft to the wound so that the fenestrations within the graft are parallel with the skin lines (i.e., parallel to the long axis of the limb).[63] The diamond-shaped epithelial scars within the grafted wound diminish in size as the grafted wound contracts.

Applying Sheet Grafts

Excess granulation tissue should be excised before the graft is harvested, to allow time for hemorrhage to stop before the graft is applied. Excising exuberant granulation tissue 24 hours in advance of grafting ensures hemostasis and provides time for budding capillaries to develop at the surface of the wound for rapid vascularization of the graft[47] (see "Preparation of the Recipient Site," earlier). If granulation tissue is not exuberant, the surface of the wound should be rubbed with a gauze sponge or gently scraped with a scalpel blade held 90 degrees to the wound, until serum exudes from the wound's surface.

The graft should be situated on the wound so that the direction of its hair growth conforms to that of the surrounding skin. A graft harvested with the horse anesthetized can be sutured or stapled to the wound's margin before the horse is allowed to recover from anesthesia. To avoid constant disturbance of the fibrin seal at the edge of the graft when securing the graft with sutures, the suture needle should pass from the graft to the surrounding skin, rather from the surrounding skin to the graft.[23] To suture or staple the graft to the wound's margin with the horse standing, the margin of the wound must first be desensitized using local or regional anesthesia. The graft can be fixed to the margin of the wound, with the horse standing, without using local or regional anesthesia, by overlapping and gluing the margin of the graft to the margin of the wound with cyanoacrylate glue (Superglue), provided that sufficient skin has been harvested to allow overlap of the graft (Figure 25-15). Applying the graft with the horse standing reduces the time of general anesthesia and eliminates the possibility of damage to the graft that may occur while the horse recovers from general anesthesia.

A graft need only be fixed to the wound's margin, because fibrin produced by the wound fixes the graft to the wound's surface within minutes after the graft is applied.[2,26] If the wound is large or is in an area that is difficult to immobilize, however, such as the dorsum of a fetlock, the graft can be further secured with simple interrupted sutures placed through the fenestrations in the meshed graft. To suture a graft to the bed of a fresh wound with the horse standing, the wound must first be desensitized with regional or local anesthesia. A graft can be sutured to granulation tissue with the horse standing without anesthesia because granulation tissue has no innervation (Figure 25-16). Sutures can be removed in 6 or 7 days when acceptance of the graft is certain. Catgut is convenient to use as a suture because it swells, causing the knot to loosen. If tied with a square knot, the suture can usually be removed by applying gentle traction to one end of the suture with a hemostat. Removing sutures in this fashion is easier than cutting the sutures.

Acceptance and Cosmesis

The thickness of a graft greatly influences its acceptance at the recipient wound.[8,17,20,23,27,28,58] The thinner the graft, the less

Figure 25-15. Attaching a graft to the margin of a wound, with the horse standing. The graft is attached by overlapping and attaching the margin of the graft to the margin of the wound with cyanoacrylate glue.

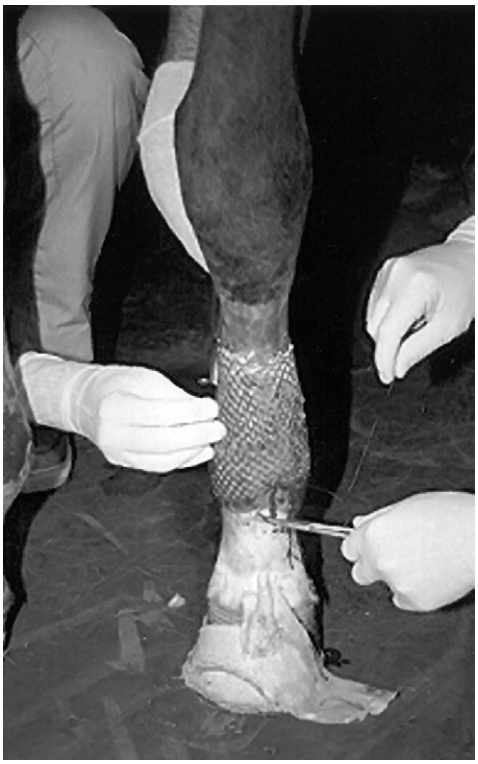

Figure 25-16. Suturing a graft to granulation tissue with the horse standing. Anesthesia is not necessary because granulation tissue has no innervation.

its metabolic demands and the less its demands for vascularity at the recipient bed. Blood vessels branch as they ascend the dermis, so the thinner the graft, the greater the number of exposed vessels. The greater the number of exposed vessels, the better the absorption of nutrients from the recipient wound and the more rapid revascularization from inosculation. The thinner the graft, the poorer its durability and cosmetic appearance.

Split-thickness grafts obtained from the hip, gaskin, or ventral aspect of the abdomen of horses that are 0.5 mm (0.020 inches) or less thick lack durability and have sparse or no hair coverage.[20] Split-thickness grafts harvested from these areas and from the ventrolateral aspect of the thorax that are between 0.63 mm (0.025 inches) and 0.76 mm (0.030 inch) thick have moderate to good piliation and good durability.[20,29] The thickness to which the graft should be harvested is a matter of judgment, because the thickness of the dermis varies between horses and between donor sites.[29] Although one of the aims of split-thickness grafting is to provide adequate piliation at both the donor and recipient sites, occasionally piliation is inadequate at either site.[56]

When donor skin is in short supply, such when a burn wound covers a large portion of the trunk, a "two-layer" technique of harvesting split-thickness grafts may provide sufficient skin to cover the wound. Using this technique, two thin split-thickness grafts are harvested from the same site. The first split-thickness graft is composed of the epidermis and a thin portion of the dermis. The second split-thickness graft, harvested from the same site, is composed only of dermis. Although the second is graft composed only of dermis, epithelialization proceeds from adnexa contained within to resurfaces the dermis.[67]

Island Grafts

An island graft is a small piece of full-thickness or split-thickness skin placed either into or onto a granulating wound.[68-70] Other names for this type of graft include implantation graft and seed graft.[3,4] Implanting island grafts into granulation tissue of horses, rather than applying them to the surface of the wound, avoids shearing forces between the graft and the bandage. The purpose of island grafting is to increase the area of epidermis from which epithelialization can proceed; consequently, wounds that receive island grafts heal primarily by epithelialization. Types of island grafts used to graft wounds of horses are the punch graft, the pinch graft, and the tunnel graft.

Punch Grafts

Punch grafts are small full-thickness plugs of skin that are harvested and implanted into granulation tissue using skin biopsy punches. Punch grafts can be taken directly from the horse or from an excised full-thickness sheet of skin.

Common donor sites for obtaining punch grafts directly from the horse are the ventrolateral aspect of the abdomen, the perineum, and the portion of the neck that lies beneath the mane. The donor site is clipped, scrubbed, and desensitized with a local anesthetic agent. The perineal area can be desensitized using caudal epidural anesthesia. Removing punch grafts directly from the horse with a skin biopsy punch creates small blemishes at the donor site (Figure 25-17). Therefore, the grafts should be harvested in a symmetrical pattern about 1 cm apart to improve cosmesis.[38] The small wounds created by the biopsy punch need not be closed, but suturing or stapling each wound may produce less-obvious blemishes.

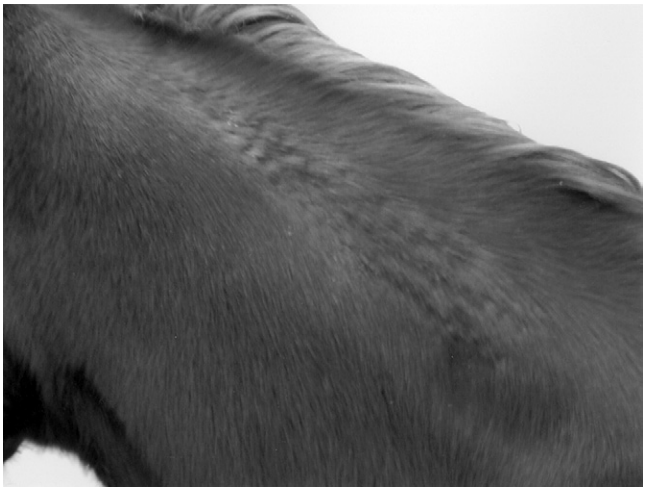

Figure 25-17. Blemishes on a horse's neck created with a skin biopsy punch.

Subcutaneous fascia and fat should be sharply excised from each individual graft before it is implanted to facilitate plasmatic imbibition and re-vascularization.[71] Removing subcutaneous tissue from each small plug of skin is a tedious process. One method is to stretch the subcutaneous tissue between one thumb forceps grasping the top of the plug and another thumb forceps grasping the subcutaneous tissue. A second person excises the taut subcutaneous tissue from the dermis with scalpel blade. Another method is to excise subcutaneous tissue from the dermis while the cut disc remains attached to the horse. The biopsy punch is rotated until it penetrates the skin, and the graft is elevated, using thumb tissue forceps, to expose the attached subcutaneous tissue, which is excised from the dermis using a scalpel blade.[72]

Punch grafts can also be harvested from an elliptical, full-thickness sheet of skin, which is usually harvested from the cranial pectoral area, where the skin is relatively mobile.[1] A 10-cm–long by 4-cm–wide (4-inches–long by 1½-inches–wide) section of skin provides enough punch grafts to cover most wounds and allows easy primary closure of the donor site.[54] The section of skin is stretched and secured, epidermal side down, to a sterile piece of cardboard or Styrofoam or to a sterile polypropylene block. After all subcutaneous tissue is sharply excised from the section of skin, full-thickness plugs are cut from the skin using a 6- to 7-mm–diameter skin biopsy punch (Figure 25-18). The grafts are stored on a gauze sponge moistened with physiologic saline solution until they are implanted, and the wound created at the donor site is closed in one or two layers. Applying a stent bandage to the sutured wound decreases tension on the suture line.

Exuberant granulation tissue at the recipient site should be excised to the level of the margin of the wound 24 to 48 hours prior to grafting. The recipient holes in the granulation tissue should be created before the grafts are harvested to allow hemostasis to occur prior to implantation. The depth of the recipient holes should correspond to the thickness of the grafts to be inserted.[1] To avoid obscuring the wound with hemorrhage that occurs when the recipient holes are created, creation of the recipient holes should begin at the distal end and proceed proximad.[71] A cotton-tipped applicator inserted into

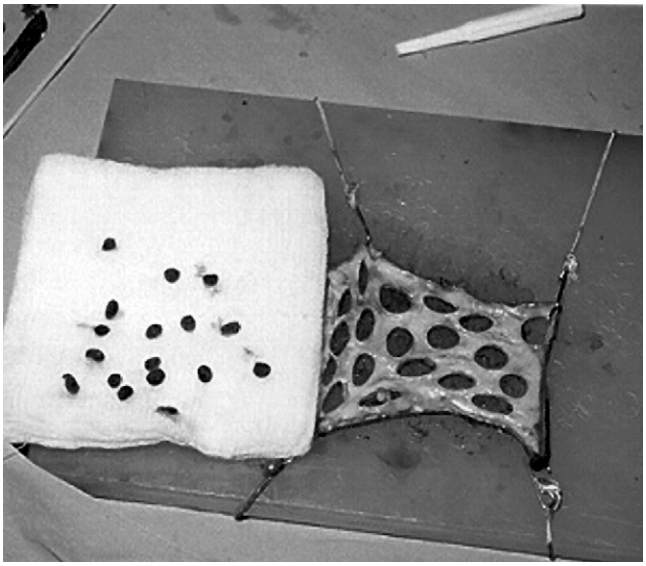

Figure 25-18. Punch grafts harvested from a full-thickness section of skin. The section of skin was obtained from the pectoral region. Full-thickness plugs are cut from the skin using a skin biopsy punch.

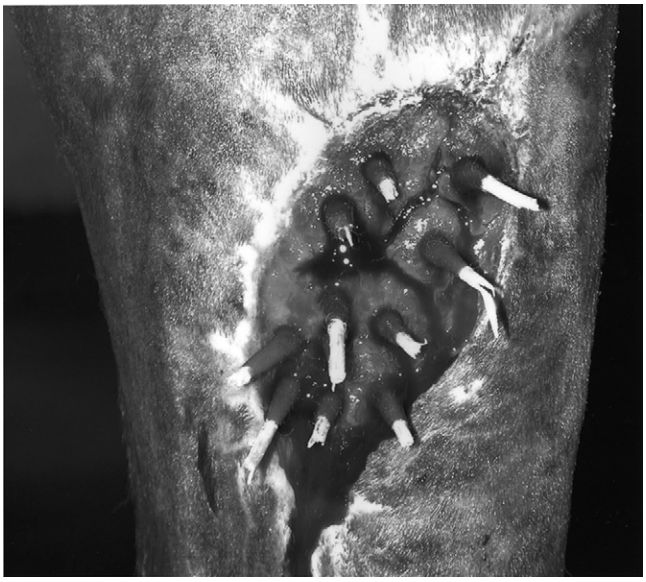

Figure 25-19. Cotton-tipped applicators inserted into recipient holes in a granulating wound. An applicator enables a hole to be located easily and prevents a blood clot from forming in the hole.

the recipient holes enables each hole to be located easily for insertion of a graft and prevents a blood clot from forming in the hole (Figure 25-19). Spraying the wound with phenylephrine before creating the recipient holes may improve visibility by decreasing hemorrhage. Creating the recipient holes hours in advance of implantation ensures a hemorrhage-free wound for grafting.[54] The holes should be placed about 6 mm (about ¼ inch) apart in a symmetrical pattern.[1]

The recipient holes are created with a slightly smaller biopsy punch than that used to harvest the grafts, to allow for contraction of the graft. For example, if the grafts are harvested with a 7-mm–diameter biopsy punch, the recipient holes should be

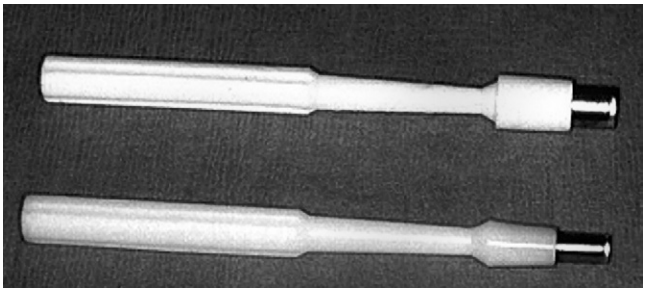

Figure 25-20. Skin biopsy punches used for harvesting punch grafts and for creating recipient holes. The recipient holes for punch grafts are created with a slightly smaller skin biopsy punch than that used to harvest the grafts. The larger punch is used to harvest punch grafts, and the smaller biopsy punch is used to create recipient holes.

created with a 5-mm biopsy punch (Figure 25-20). The grafts are inserted into recipient holes, and the grafted wound is covered with a non-adherent dressing and a bandage. Consideration can be given to the direction of the hair growth on the graft when the graft is inserted, but the cosmetic outcome may be little affected by properly orienting the direction of the hair.

The pigmented, superficial portions of the grafts frequently slough, exposing non-pigmented deeper layers. By 3 weeks, each plug of skin is surrounded by a red ring, which represents migrating epithelium.[1] Acceptance of the grafts is ensured if the grafts remain within the recipient hole.[38] Survival of 60% to 75% of the grafts is a realistic expectation.[54] The time required for the wound to completely epithelialize is inversely proportional to the amount of wound covered by viable plugs. Punch grafting requires no expensive or sophisticated equipment and little expertise, and the grafts are accepted into recipient beds that are unsuitable for sheet grafting.[1] Punch grafting can be performed with the horse standing, making this an inexpensive method of grafting wounds. Punch grafting is usually reserved for small wounds where cosmesis is not important. Wounds healed by punch grafting are covered primarily by an epithelial scar from which grow sparse tufts of divergent hair (Figure 25-21).

Pinch Grafts

Pinch grafts are small discs of skin, harvested by excising an elevated cone of skin, that are laid onto or implanted into granulation tissue.[26,49,55,68-70] The pinch graft is sometime referred to as a Reverdin graft.[70] The donor sites for obtaining pinch grafts from the horse are the same as described for punch grafts; small blemishes created at these donor sites are relatively inconspicuous. A disc of optimal size is approximately 3 mm in diameter, which approaches full thickness toward its center, but its thickness diminishes toward its periphery.[69]

Preparation of the donor site is identical to that described for punch grafts. A cone of skin is elevated using a tissue forceps, a suture needle, or a hypodermic needle with a bent point, and it is excised with a scalpel blade (Figure 25-22).[49,55,68-70] A No. 11 scalpel blade is most suited for excising the cone of skin. The grafts are stored on a gauze sponge moistened with physiologic saline solution until they are implanted.

The recipient site is prepared in the same way as for punch grafts. The recipient pockets can be created hours in advance of implantation to ensure a hemorrhage-free wound for grafting,

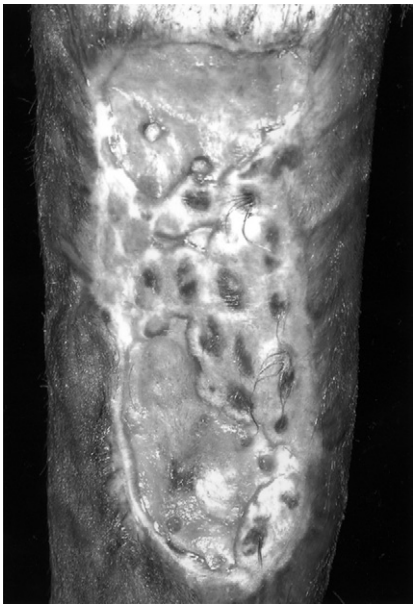

Figure 25-21. A nearly healed, punch-grafted wound. The nonpigmented tissue between the pigmented punch grafts is epithelium. This epithelium will eventually become pigmented.

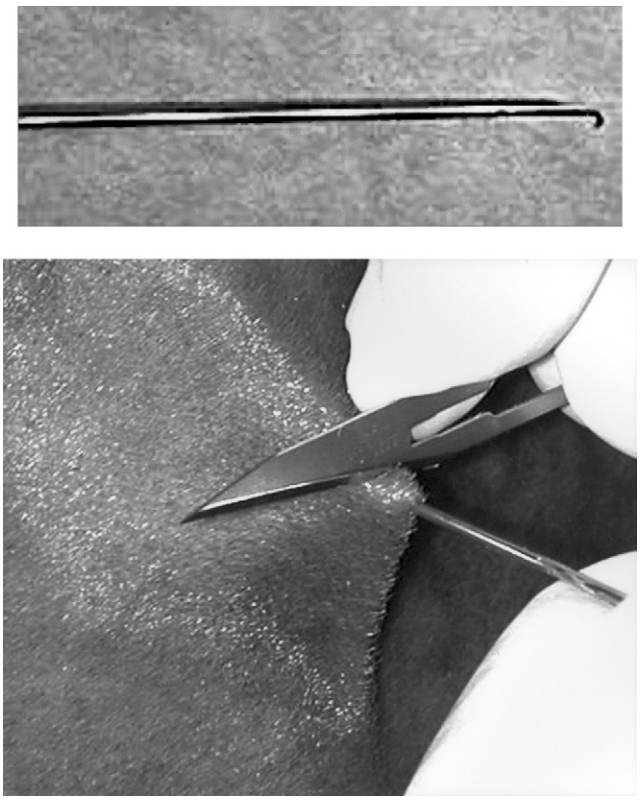

Figure 25-22. Harvesting a pinch graft. A cone of skin is elevated using a hypodermic needle with a bent point, and the elevated cone of skin is excised with a scalpel blade.

Figure 25-23. A wound covered with pinch grafts.

but locating the pockets after hemorrhage has ceased may be difficult.

To implant a graft, a No. 15 scalpel blade is stabbed into the granulation tissue of the recipient bed at an acute angle to create a shallow pocket into which the graft is inserted. Implantation should begin at the distal end and proceed proximad, because the pockets are obscured by hemorrhage after they are created. The grafts should be implanted about 3 to 5 mm apart.[69] Each graft is laid onto the wound, epidermal side up, proximal to the pocket, and slid into the pocket using a hypodermic needle, a straight suture needle, or the scalpel blade used to create the pocket. Consideration can be given to the direction of the hair growth on the graft when the graft is inserted, but the cosmetic outcome is little affected by properly orienting the direction of the hair. To speed implantation, three or more grafts can be laid onto the granulation tissue at once, each 15 to 20 mm (6 to 8 inches) proximal to its proposed site of implantation. Hydrostatic pressure prevents the grafts from falling from the wound. The surgeon creates a pocket distal to a graft and inserts the graft into the pocket using the same scalpel blade that was used to create the pocket. The surgeon can create and implant the pockets in rapid succession without looking away from the wound.

Wounds created at the donor site are left to heal by second intention or closed with a single suture or staple. The grafted wound is covered with a nonadherent dressing and a bandage.

The thin layer of granulation tissue covering each pinch graft usually sloughs between the first and second week after grafting.[69] Grafts initially appear as dark spots on the wound's surface (Figure 25-23), but frequently the superficial pigmented portion of the graft sloughs, causing the graft to appear as a white spot surrounded by pink granulation tissue. Within 3 weeks, each disc of skin is surrounded by a red ring, which represents migrating epithelium. Epithelium migrates from the margin of the wound, as well as from the margin of each graft, so that eventually the entire wound is covered with epithelium. Even under adverse conditions, one can expect at least 50% to 75% of the grafts to survive.[69]

Pinch grafting, like punch grafting, is a relatively inexpensive technique of grafting, because the procedure can be performed with the horse standing and because only basic instruments are required. Little expertise is necessary, and the grafts can survive in a granulation bed that is less than ideal.[3,68] Pinch grafting is tedious, however, and imparts a poor cosmetic appearance, because the wound is left covered by an epithelial scar from which grow sparse tufts of divergent hair.

Tunnel Grafts

Tunnel grafts are strips of split-thickness or full-thickness skin implanted into tunnels created in granulation tissue.[4,73,74] The grafts are exposed days later, when they have re-vascularized, by excising the overlying granulation tissue that forms the roof of the tunnel. Donor skin can be harvested from various sites on the horse, using various techniques, and implanted with the horse anesthetized or sedated.

Split-thickness or full-thickness strips of skin can be harvested conveniently from the portion of the neck that lies beneath the mane or the ventral aspect of the flank.[4,73,74] In preparation for harvesting strips of skin, linear wheals, 2 to 3 cm wide and slightly longer than the wound to be grafted, are created along the longitudinal axis of the donor site by subcutaneously injecting local anesthetic solution (or physiologic saline solution if the graft is to be harvested with the horse anesthetized).

A straight intestinal forceps is applied to the base of each wheal so that skin protrudes slightly above the jaws of the forceps.[4,73,74] If a strip of skin longer than the jaws of the forceps is required, two intestinal forceps are applied end-to-end to the base of the wheal. The thickness and width of the graft is determined by the amount of skin that protrudes above the jaws. The protruding skin is severed with a scalpel (Figure 25-24). If the excised strip of skin is full thickness, subcutaneous tissue should be removed from it to expose the dermis, and the wound at the donor site should be closed with staples or sutures. If the strip of skin is split thickness, the donor site can be closed or left open.

Full-thickness strips of skin can also be harvested from the neck or ventral portion of the abdomen by making a series of parallel incisions 2 mm apart.[74] Strips are excised, and subcutaneous tissue attached to the dermis is sharply excised. The donor sites are closed with sutures or staples. Split-thickness strips of skin can be cut from a split-thickness sheet of skin usually harvested from the ventral aspect of the abdomen or ventrolateral aspect of the thorax, using techniques previously described (see "Technique of Harvesting a Graft," earlier). The donor site of the split-thickness sheet graft heals by epithelialization.

To implant a strip of skin in the granulation bed, the end of a long, thin, rat-tooth alligator forceps is inserted into granulation tissue at the edge of the wound and advanced through the granulation tissue at a depth of about 5 or 6 mm until it emerges at the opposite margin of the wound.[4,74] One end of the graft is grasped in the jaws of the forceps, with the epidermis positioned toward the surface of the wound, and pulled through the tunnel. The grafts should be embedded at right angles to the convex surface of the wound to aid entry and exit of the forceps.[74] A wound that is not convex is more easily grafted if granulation tissue is allowed to proliferate so that it protrudes slightly above the margin of the wound.[4] The strips should be embedded about 2 cm apart.[74]

The strip of skin is embedded in two steps if the alligator forceps is not long enough to completely span the wound, or if the wound is too convex for the alligator forceps to completely span it.[4,73,74] In either case, the forceps is advanced through the granulation tissue as far as possible and exited, one end of the graft is grasped, and the forceps is pulled back through the tunnel, with the graft in tow (Figure 25-25). The

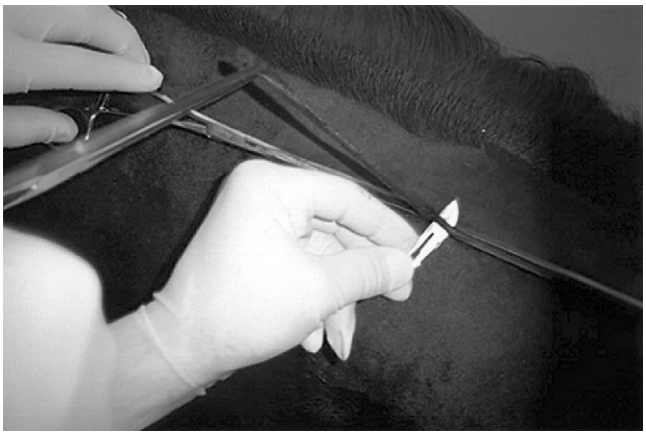

Figure 25-24. Harvesting a tunnel graft. One or two straight intestinal forceps are applied to the skin, so that skin protrudes slightly above the jaws of the forceps. The protruding skin is severed with a scalpel.

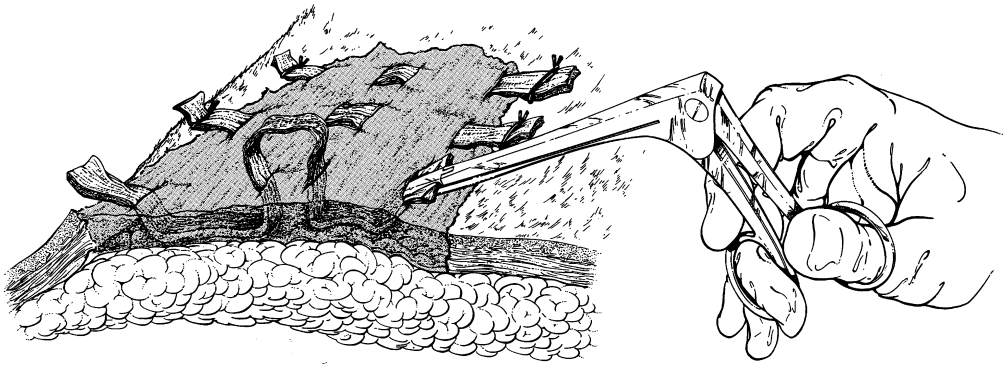

Figure 25-25. Implanting a tunnel graft using an alligator forceps. The strip of skin is embedded in two steps if the forceps is not long enough to completely span the wound.

end of the forceps is reinserted into the granulation bed at the opposite margin of the wound and advanced beneath the surface of the granulation tissue until it emerges at the site of exit of the graft. The end of the graft is grasped in the jaws of the forceps, and the forceps, with graft in tow, is pulled back through the second tunnel.

The graft can also be embedded by positioning the strip of skin onto a strip of adhesive tape, with the epidermis next to the tape.[73] The purpose of the adhesive tape is to prevent the edges of the strip from folding inward and to act as a guide when the grafts are later exposed. The tape should extend 5 cm beyond one end of the strip, and excess tape at this end is folded over the skin so that about 1 cm of the end of the strip is covered by tape. Excess tape along the graft's margin is trimmed with scissors. The tape-covered end of the graft is fitted through the eye of a 10- to 12-cm (4- to 5-inch) cutting needle with a flat curve, and the graft–tape composite is implanted by advancing the needle beneath the surface of the granulation bed. If the needle is not long enough to span the wound completely, or if the wound is too convex for the needle to completely span it, the needle is advanced beneath the bed as far as possible, exited, and reentered at the point of exit for a second bite. Another method of creating the strips of graft and tape is to lay a sheet of split- or full-thickness skin, epidermal side down, onto the sticky side of a similarly sized sheet of adhesive tape; strips of skin and tape are cut from this composite.[54]

The exposed ends of the embedded strips are attached to the margin of the wound with a suture, staple, or cyanoacrylate glue, and the wound is covered with a non-adherent dressing and a pressure wrap. Six to 10 days after grafting, the roof of each tunnel is removed, with the horse sedated or anesthetized, to expose the grafts, and if necessary, to remove adhesive tape.[4,73,74]

To remove the roof of a tunnel, a smooth, malleable probe is inserted into the tunnel superficial to the graft, and a V-shaped strip of granulation tissue is carefully excised over the probe with a scalpel blade to expose the graft. The roof of each tunnel can also be sawed off using a twisted doubled piece of fine wire threaded through the tunnel.[73] Accidentally removing a graft while attempting to expose it or failure to locate buried grafts can detract from the success of tunnel grafting.[74] These complications can best be avoided by applying a tourniquet proximal to the wound to enhance visibility or by exposing the grafts with the horse anesthetized. Placing the grafts closer than 2 cm apart may affect the viability of granulation tissue surrounding the grafts. About 60% to 80% acceptance of the strips can be expected.[73,74]

Tunnel grafting is recommended primarily for wounds located in areas where immobilizing other types of grafts would be difficult, such as the dorsal aspect of the hock.[73,74] Tunnel grafts are unaffected by movement in highly mobile areas, because the grafts are circumferentially encompassed and immobilized by granulation tissue. Tunnel grafting does not require expensive equipment and can be performed with the horse standing, but the technique requires the presence of a granulation bed, and aspects of this technique, especially removing the top of the tunnels, make tunnel grafting relatively tedious. The tedious job of removing the granulation tissue over the graft may not be necessary; however, if the graft is embedded so that only 1 to 1.5 mm of granulation tissue covers the graft. The shallow covering of granulation tissue sloughs, usually within a week, exposing the graft.

Modified Meek Grafting Technique

The modified Meek technique of skin grafting is a combination of island grafting and split-thickness sheet grafting.[30] Using this technique, 3-mm^2 islands of split-thickness skin, or micrografts, are applied to the surface of the wound. The technique is particularly useful for grafting large wounds because the donor skin can be expanded more with micrografts than with meshed sheet grafts.[75] This technique of grafting is particularly useful when the donor skin is in short supply, as may be the case for horses with extensive burns.

Meek micrografting equipment consists of a frame, a cutting block, and a pneumatic or hand-driven motor. A split-thickness section of skin of the desired thickness is harvested with the horse anesthetized (see "Split-Thickness Sheet Grafting," earlier) and the split-thickness graft is placed, dermal side down, onto a 42 × 42 mm cork plate and trimmed to the size of the plate. The cork plate, containing the graft, is placed onto a cork holder, which is placed in a cutting block (Figure 25-26). The cork, covered with graft, is then passed through a machine that contains 13 rotating, circular blades that cut the graft, but not the cork, into 14 3-mm wide strips (Figure 25-27). The cork and

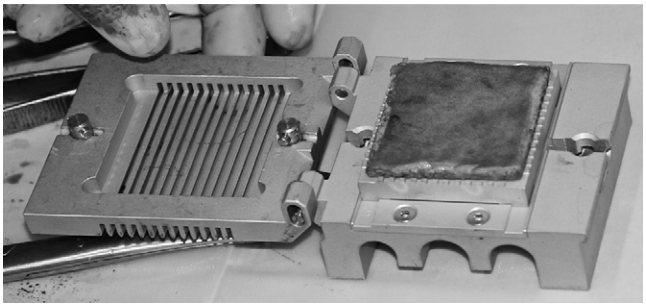

Figure 25-26. To obtain Meek micrografts, a split-thickness graft is placed with the dermal side down, on a cork template and trimmed exactly to fit the template. The cork template with graft is placed on a cork holder, which is then placed in the cutting block. (Courtesy Jacintha Wilmink, Woumarec, Wageningen, NL.)

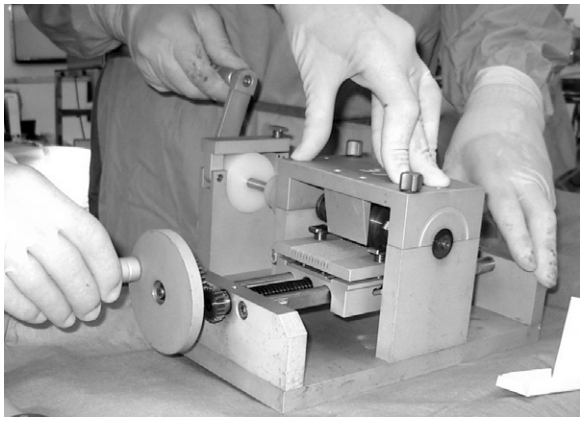

Figure 25-27. A Meek micrograft machine shown with a hand-drive (a machine with pneumatic motor for turning the knives is also available). The cutting block with the cork template and graft are passed through the Meek micrograft machine, which contains 13 circular knives that cut the graft into 14 3-mm wide strips. The cork holder is rotated 90 degrees and replaced onto the cutting block, and by passing it through the machine again, the strips of graft are cut into 196 (14 × 14) 3 × 3 mm micrografts. (Courtesy Jacintha Wilmink, Woumarec, Wageningen, NL.)

graft are rotated 90 degrees, replaced onto the cutting block, and again passed through the machine, which cuts the strips of graft into 196 (14 × 14) 3 × 3 mm grafts.

The epidermal surface of the graft is sprayed with an adhesive, which is allowed to dry until it becomes tacky. The cork plate, covered with graft, is pressed onto a pleated polyamide gauze backed by aluminum foil, which when unfolded expands the graft in a ratio of 1:3, 1:4, 1:6, or 1:9 (Figure 25-28). A pleated gauze with the smallest expansion ratio (1:3) is used for grafting most wounds of horses because most horses have an abundant amount of skin available for harvesting. The cork is carefully removed, leaving the postage stamp–sized islands of graft on the gauze. The pleats are unfolded, separating the grafts at a set uniform distance, by applying traction to all of its four sides (see Figure 25-28), and the supporting aluminum backing is peeled off and discarded, leaving the expanded gauze with the separated islands of graft. The expanded gauze to which the grafts are attached is trimmed appropriately, applied graft side down to the wound, and secured to the margin of the wound with staples or sutures (Figure 25-29). The grafted wound is covered with a bandage or a cast. The gauze can be removed after about 6 days, when the grafts have become attached to the wound.

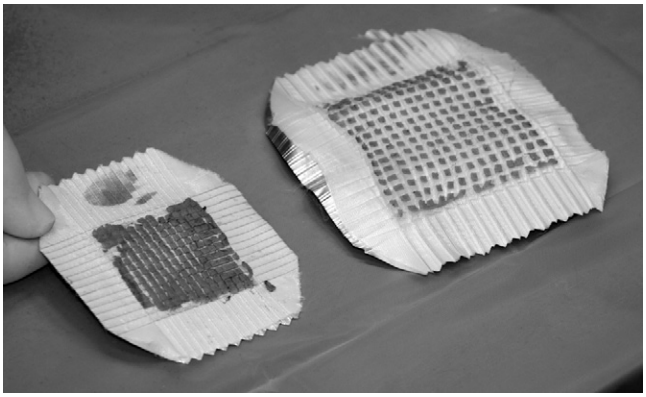

Figure 25-28. *Left,* The epidermal side of the Meek micrografts was sprayed with an adhesive, and the graft was glued to a prefolded pleated polyamide gauze (e.g., plissé), after which the cork template was removed. *Right,* The plissé was unfolded in two directions, separating the Meek micrografts at set uniform distances. (Courtesy Jacintha Wilmink, Woumarec, Wageningen, NL.)

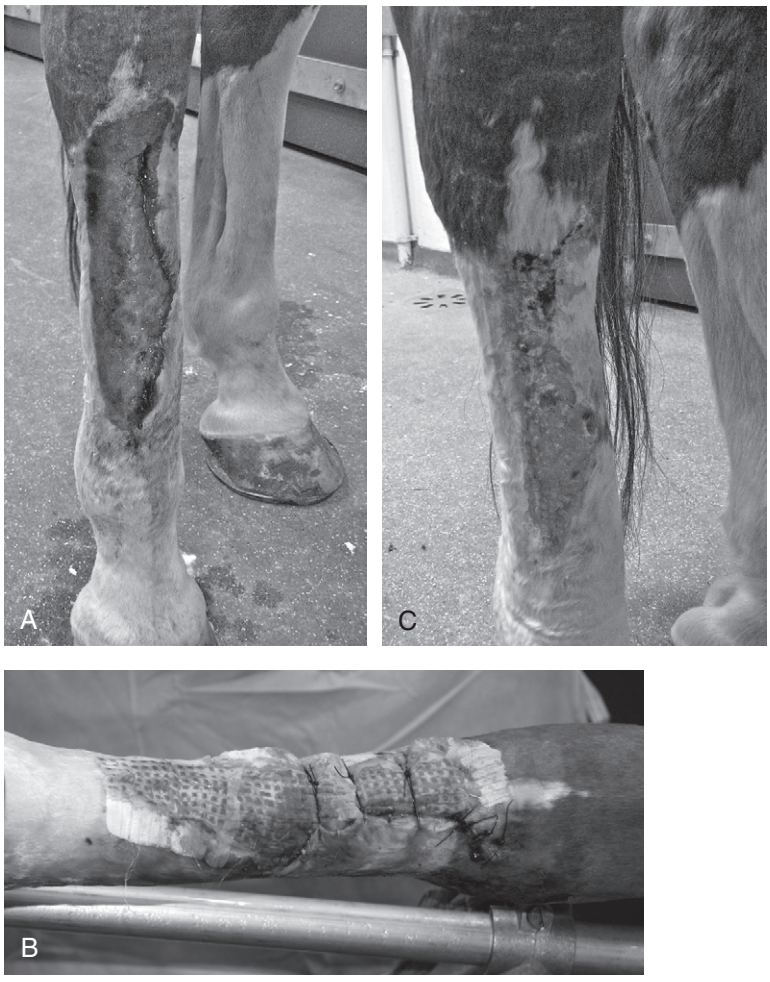

Figure 25-29. A, A large chronic wound at the time of admission. The horse was caught in a fence several weeks earlier and acquired a large loose skin flap on the medial side of the metatarsus; **B,** One month later grafting was performed on the prepared wound. The expanded gauze with Meek micrografts was applied to the wound, and the gauze was trimmed to the size of the wound and secured with staples and sutures. The loose skin flap that was retracted was mobilized and secured with three big sutures to the lateral wound margin; **C,** The same horse 1 month later. Nearly all micrografts are accepted, and the wound is almost healed. (Courtesy Jacintha Wilmink, Woumarec, Wageningen, NL.)

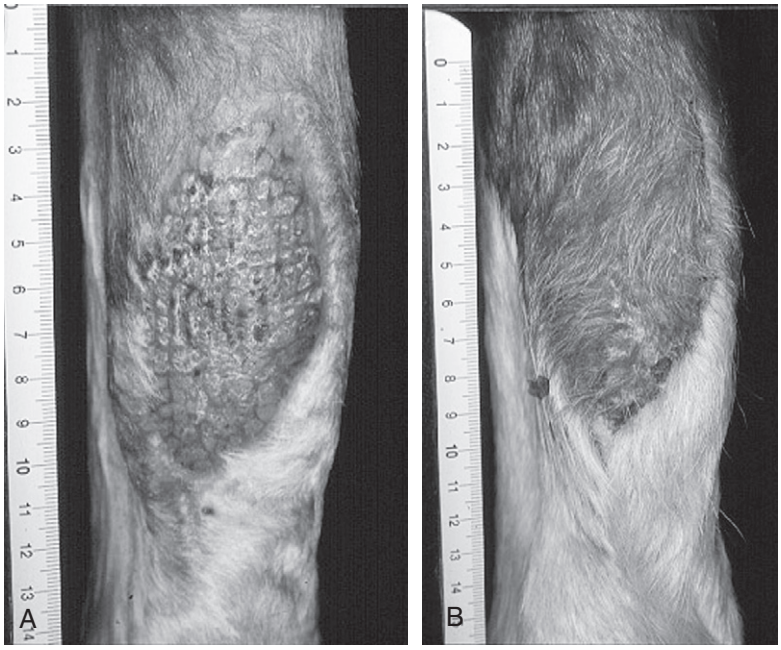

Figure 25-30. A, A nonhealing wound on the palmar aspect of a metatarsus is almost healed after receiving Meek micrografts 24 days earlier. The original square Meek micrografts from which epithelialization proceeds can still be seen. Only tiny areas remain to be epithelialized. **B,** Five months after grafting, the grafted site is covered with hair the same color as that at the donor site. The hair coat is somewhat sparser than the surroundings because the newly formed epithelium between the micrografts does not produce hair. (Courtesy Jacintha Wilmink, Woumarec, Wageningen, NL.)

As the wound contracts, the distance between the grafts decreases. Most wounds that receive Meek micrografts are healed within a month, regardless of the wound's size and location.[30] Hair growth is regular, but thinner and longer than normal (Figure 25-30).

The modified Meek technique of skin grafting provides uniform distribution of properly oriented islands of split-thickness skin. Detachment of one Meek micrograft, such as from infection or movement has no direct effect on surrounding micrografts because the micrografts are not connected to each other. Detachment of a portion of a sheet graft, on the other hand, may propagate into other areas of the graft, resulting in complete loss of the graft.[30,75,76] Meek micrografts are, therefore, more easily accepted by poor-quality wounds than are sheet grafts and can be applied with greater ease and in a higher concentration than can other types of island grafts, such as pinch, punch, or tunnel grafts. Acceptance of Meek micrografts in horses appears to be consistently better than what can be achieved using other techniques of skin grafting, and in one study, acceptance approached 95%.[30]

A disadvantage of the modified Meek micrografting technique over other methods of island grafting is that the horse must be anesthetized to harvest the split-thickness donor skin. Equipment to harvest the split-thickness skin and the micrografting machine are relatively expensive but no more so than much of the mechanized equipment available for harvesting and meshing sheet grafts.

AFTERCARE OF THE DONOR SITE

The donor site of a split-thickness graft retains a portion of the dermis and is comparable to a deep abrasion.[15,18,29] Blood and fibrin form a scab over the abrasion, and beneath this scab, epithelial cells from the wound's edge and epithelial cells produced by adnexa in the remaining dermis migrate to cover the surface of the abrasion. The wound at the donor site is usually covered by epithelium within 1 to 3 weeks, depending on the depth of the cut (Figure 25-31).[20,62] The new pink epithelium covering the donor site begins to develop pigment within a month after it forms, causing the epithelial scar to become black. The quantity of adnexa is greatest in the upper layers of the dermis, and consequently, healing of the donor site proceeds more quickly when a thin skin graft is harvested.[15,20] When grafts between 0.63 mm and 0.76 mm thick are harvested from the horse, the donor site heals with a noticeable epithelial scar.[20,61]

Figure 25-31. The donor site of a split-thickness graft 2 weeks after the graft was harvested. The donor site is nearly covered by epithelium. The new, pink epithelium will begin to develop pigment within a month, causing the epithelial scar to become black.

Pain caused by exposure of nerves at the donor site of a split-thickness graft can be reduced by bandaging the donor site,[15] but split-thickness grafts of horses are virtually always taken from an area that is difficult to bandage. Pain can be decreased by covering the donor site with fine-mesh gauze or a biological bandage, such as a stored allograft or xenograft (see "Allografts and Xenografts" and "Storage of Grafts," later). The gauze or biological bandage falls off when the donor site is completely

epithelialized. An uncovered donor site develops a scab, and this scab should not be removed. Any attempt to remove the scab is met with violent resistance from the horse, and its removal interferes with epithelialization that proceeds beneath it. An analgesic drug, such as phenylbutazone, should be administered before the graft is harvested and for at least several days after surgery.

AFTERCARE OF THE RECIPIENT SITE

The grafted wound should be covered with a sterile, nonadherent dressing, such as cotton nonadherent film dressing (Telfa Sterile Pads), rayon polyethylene dressing (Release), or petrolatum-impregnated gauze dressing (Adaptic). Petrolatum-impregnated gauze dressings can be prepared by steam-autoclaving a container filled with gauze sponges topped with a dollop of petrolatum. The petroleum impregnates the gauze as it melts. The wide weave of a gauze sponge, however, makes the sponge more likely to adhere to the grafted wound than would a commercially available petrolatum-impregnated gauze dressing with a close weave.

The primary dressing is secured to the grafted wound with sterile elastic, conforming, rolled gauze. If the grafted wound is in an area that is difficult to immobilize, such as the dorsal aspect of the hock, securing the primary dressing with staples[38] or with elastic adhesive tape, rather than rolled gauze, may help to decrease shearing forces between the graft and the bandage. The secondary layer of the bandage should be bulky, to decrease motion of the limb, and absorbent, to wick bacteria and destructive enzymes from the grafted wound. Immobilizing the limb with a cast is usually not necessary unless the grafted wound is located in a highly mobile area, such as the dorsum of the fetlock. A splint or a Robert Jones bandage applied to the limb usually immobilizes the grafted wound sufficiently.

The bandage should not be changed for 4 or 5 days after grafting, unless virulent nosocomial infections are a common problem in the hospital, because changing the bandage may disturb the graft's delicate attachment to the wound. If such infections are a common problem in the hospital, however, the bandage should be changed daily, or even twice daily, for at least 5 days. Changing the bandage daily allows exudate, which contains destructive enzymes, to be removed from the wound, and it allows application of an appropriate antimicrobial drug to the wound. Complete loss of a sheet graft, from infection or motion, later than 5 days after grafting is uncommon.[38] Streptococci or pseudomonads can quickly destroy a recently applied graft (Figure 25-32), so prompt recognition of infection and application of an appropriate antimicrobial drug to the grafted wound are necessary. Infection caused by a *Pseudomonas* species is characterized by the presence a bluish-green exudate on the surface of the wound and an odor similar to that of grape juice. Exudate that develops on a grafted wound should be cultured for bacterial growth, and isolates should be tested for antimicrobial susceptibility. Until the results are known, a broad-spectrum antimicrobial agent, effective against both β-hemolytic streptococcus and *Pseudomonas,* such as a combination of ticarcillin disodium and clavulanate potassium,[50] should be applied topically to the wound.

If the limb has been immobilized in a cast, and if nosocomial infection from streptococci or pseudomonads is a common problem in the hospital, an antimicrobial drug should be applied periodically to the wound through an infusion tube fixed to the grafted wound and exited through the top of the cast. A serviceable infusion tube can be prepared from silicone tubing by sealing its distal end and perforating it (with a 25-gauge needle) in multiple places along the portion that lies adjacent to the wound.

Figure 25-32. A recently applied graft destroyed by streptococcal infection.

Even a docile horse should be heavily sedated during the initial bandage changes to help avoid movement that may disturb the delicate vascular connections forming between the recipient bed and the graft. Adherence of the primary dressing to the grafted wound by fibrin that exudes through a meshed graft usually indicates that the graft is well adhered to the wound by a fibrin seal. Soaking the primary dressing with physiologic saline solution may ease removal of the dressing. The dressing should be removed by grasping it at one end and pulling it parallel to the wound; pulling the dressing perpendicular to the wound places more traction on the graft. If the primary dressing cannot be removed easily, it should be left in place.

Exuberant granulation tissue that inhibits epithelial migration may grow through the latticework of a meshed graft, especially if the graft was applied to the wound fully expanded. A corticosteroid applied to the grafted wound causes the exuberant granulation tissue within the graft to regress, allowing epithelial migration to proceed, even if at a slower than normal pace.[29,77-79]

Ideally, grafted wounds on limbs should be protected by a bandage until epithelialization is complete. If bandaging becomes impractical before epithelialization is complete, small, nonepithelialized areas within the grafted wound can be allowed to heal beneath a scab, which soon forms when the wound is left exposed.

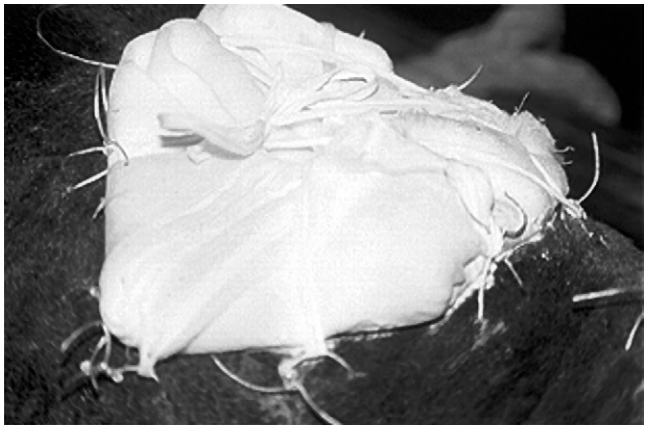

Figure 25-33. A stent bandage applied to a grafted wound on the back of a horse. A grafted wound in an area that is difficult to wrap can be protected with a stent bandage. To apply a stent bandage, the grafted wound is covered with a nonadherent dressing followed by a bolus of gauze. Long ends of interrupted sutures, preplaced around the margin of the grafted wound, are tied tightly over the bolus.

A grafted wound in an area that is difficult to wrap, such as the abdomen or thorax, can be protected with a "tie-over" bolus dressing, sometimes referred to as a stent bandage.[4,29,71] To apply a stent bandage, the grafted wound is covered with a nonadherent dressing followed by a bolus of gauze. Long ends of interrupted sutures, preplaced around the margin of the grafted wound, are tied tightly over the bolus (Figure 25-33).

Pressure on a graft from a bandage is not necessary for the graft to be accepted, so, for areas that are difficult to bandage or for wounds that are chronically plagued with infection, the grafted wound can be left uncovered.[2,23] "Open" grafting avoids shearing forces imposed by a bandage and avoids maceration of the graft by allowing constant drainage of exudate. To successfully employ open grafting, precautions, such as tying the horse or applying a neck cradle, must be taken to prevent the horse from disturbing the exposed graft.

Some horses probably suffer from hyperesthesia at the grafted wound, so when bandaging is discontinued, temporarily applying a neck cradle or tying the horse may be prudent to prevent the horse from mutilating its grafted wound. Drying and scaling of the healed donor and recipient sites of a split-thickness graft, caused by reduced concentration of eccrine glands, can be lessened by periodically applying an ointment containing lanolin until enough glands regenerate that scaling is no longer a problem.[8]

ALLOGRAFTS AND XENOGRAFTS

Cutaneous allografts (i.e., skin grafts taken from one animal and transplanted to another of the same species) or cutaneous xenografts (i.e., skin grafts taken from one animal and transplanted to another of a different species) can be used as a biological dressing on wounds of horses. Cutaneous allografts and xenografts, usually pigskin, have been used extensively since the middle of the 19th century to temporarily dress large wounds on humans. Although xenografts have been used to dress wounds of horses,[12,13,80,81] the use of cadaveric, cutaneous allografts to dress their wounds has only recently been investigated.[14] Viable cutaneous allografts can be obtained from refrigerated cadavers 24 hours or longer after death.[82] Allografts applied to wounds of horses seem to survive between 2 and 3 weeks, but the length of survival can be difficult to determine, because even when the superficial portion of the graft appears to be desiccated, the deeper portion of the graft may still be viable and attached to the recipient wound by strong vascular connections.

A cutaneous allograft or xenograft can be used to temporarily cover a wound if autografting is physically unfeasible or economically impractical. An allograft or xenograft encourages healing by promoting epithelialization and angiogenesis at the recipient bed.[83] The allograft or xenograft also retards formation of exuberant granulation tissue and acts as a bacterial barrier to protect the wound from infection.[12,80,82] A cutaneous allograft or xenograft revascularizes, and this vascularization may explain, at least in part, the ability of the cutaneous allograft or xenograft to resist infection.[11] Acceptance of a graft is not essential for bacterial destruction, however, because bacterial colonization decreases even beneath nonviable grafts, perhaps because the fibrin that forms between the graft and the wound enhances phagocytosis.[12,13,15,39,84] Adherence of an allograft or xenograft to a wound is a useful indicator that the wound is sufficiently healthy to accept an autograft.

The use of cutaneous porcine xenografting in humans arose from the need for a substitute for allografts as a biological dressing because of the short supply of cadaveric skin, but clinical comparisons in humans have shown that cutaneous porcine xenografts are inferior to cutaneous allografts.[11] Cutaneous porcine xenografts are more poorly adhered than allografts, allow higher bacterial counts in the wound, and cause a more intense immunological rejection.

STORAGE OF GRAFTS

Autografts or allografts can be applied successfully to wounds after being stored for several weeks at refrigeration temperature, in gauze that has been soaked in either physiologic saline solution or lactated Ringer's solution.[8,17,23] Skin can be stored in a refrigerator in a nutrient medium, such as McCoy's 5A Medium to which serum has been added, for much longer periods.[82,85-87]

McCoy's 5A Medium is a tissue-culture medium composed of a balanced electrolyte solution to which amino acids, vitamins, dextrose, and a pH indicator (phenol red) have been added. Adding vitamins, amino acids, dextrose, and serum to the electrolyte solution greatly extends the time that a graft can be stored. Skin from humans has been stored successfully in refrigerated nutrient media for 6 to 8 weeks.[82,85] Wounds of horses have been grafted using split-thickness skin grafts refrigerated at 4° C in a solution of McCoy's 5A Medium and horse serum for 3 weeks, with consistently successful results,[87] and wounds of some horses have been grafted successfully using skin stored for as long as 12 weeks.

The concentration of serum in the storage medium should be between 10% and 33%.[88] A concentration of serum greater than 33% stimulates the metabolic activity of the graft. Antigenic reaction of the serum to the graft is avoided by using a commercially available antibody-free serum (GG-Free Horse Serum), pooled homologous serum, or the horse's own serum.[85] The stored grafts should be allowed access to air.[82]

To prepare a meshed split-thickness skin graft for storage, the graft is laid on a sterile gauze swab or gauze dressing, with the epidermis of the graft next to the gauze. The gauze–graft

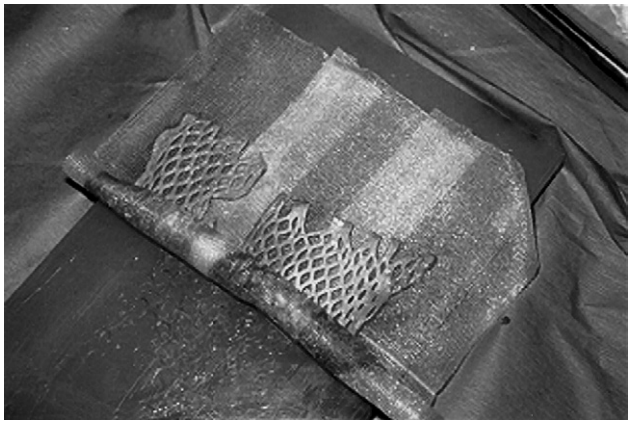

Figure 25-34. Preparing the graft for storage. The gauze–graft composite is rolled up, with the gauze to the outside of the roll.

Figure 25-35. Storing the graft. The gauze-graft composite is stored, in a refrigerator, in a sterile container containing McCoy's 5A Medium to which horse serum has been added.

composite is rolled up, with the gauze to the outside of the roll (Figure 25-34), and placed in a sterile container containing approximately 1 to 2.5 mL of the storage medium for each square centimeter of stored graft[85,88] (Figure 25-35). The storage medium should be examined occasionally for a color change brought about by a change in pH. A color change in the McCoy's 5A Medium from cherry-red to orange-yellow indicates excessive accumulation of catabolites and the need for immediate application of the graft or replacement of the medium. Only half of the volume of medium should be replaced, because total replacement affects the graft deleteriously.[85]

A split-thickness skin graft, harvested while the horse is anesthetized for treatment of a wound, such as removal of a bone sequestrum, can be stored until the wound's condition has improved enough to permit grafting.[23,89,90] A graft can be stored for delayed grafting of an excisional defect accompanied by excessive hemorrhage. Storing the graft, so that it can be applied with the horse standing, shortens the anesthetic period and eliminates the risk of the graft's being disturbed during recovery. A stored graft is often more readily accepted than a fresh graft, because grafts stored for 24 hours or more release anaerobic metabolites that stimulate rapid revascularization of the graft.[47,91] For this reason, a stored graft is sometimes referred to as a "prepared graft."

A stored graft is useful for covering a defect created when all or a portion of a primary graft sloughs, and for this reason, harvesting more skin than is required to cover a wound is often prudent, especially when grafting has a greater than usual possibility of failure. A stored graft not required for autografting can be used as a biological dressing for a wound of another horse, when circumstances permit.

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CHAPTER
26 Management of Superficial Wounds

Dean A. Hendrickson

The skin is the largest organ in the body and is very adaptable to the environment. The main functions of the skin are to protect against wear and bacterial invasion, to aid in thermal regulation, and to prevent water loss.[1,2]

ANATOMY

The anatomy of the equine skin was first completely described by Talukdar and colleagues in 1972.[3] Using normal horses of ages 2 to 17 years, they collected skin from 49 different areas. The average skin thickness of the body skin was 3.8 mm, the skin near body openings 3.3 mm, and the skin at the main and tail 6.2 mm. Skin was thicker in older geldings than in younger horses and at the extensor surface of the legs than at the flexor surface of the legs. In general, the skin of the horse is thicker than that of swine, goats, and sheep; thinner than that of beef cattle; and similar in thickness to that of dairy cattle.[3]

The epidermis is composed of four layers: the stratum basale, stratum spinosum, stratum granulosum, and stratum corneum (Figure 26-1). The stratum corneum slows water loss and functions as a barrier to harmful substances. Other cells found in the epidermis include melanocytes for production of melanin, Merkel cells for mechanoreception of light touch, and Langerhans' cells to help fight infection by engulfing foreign material. The dermis is separated into two layers, the superficial papillary layer and the deep reticular layer, and it provides support for the epidermis. The dermis in the lumbar, sacral, and gluteal regions has a third layer of collagenous fibers. The dermis also contains a rich vascular network that supplies the skin with a continuous blood supply. The vascular system is very important in temperature regulation, access of nutrients to the skin, absorption of topical medications, delivery of systemic medications, and the healing of wounds (see Chapter 5). The main vascular supply comes from the subcutaneous area. In the thinner-skinned areas, and the hair angle is more acute. The hair follicles in the general skin areas where the hair length changes with the season are shorter than the hair follicles in the areas of mane and tail. In most cases, two sebaceous glands are associated with each hair follicle. The sweat glands are apocrine.[2,3] The hairs are surrounded by nerves that provide sensation by responding to pressure on the hair shaft. The primary nerve supply comes from the subcutaneous region.

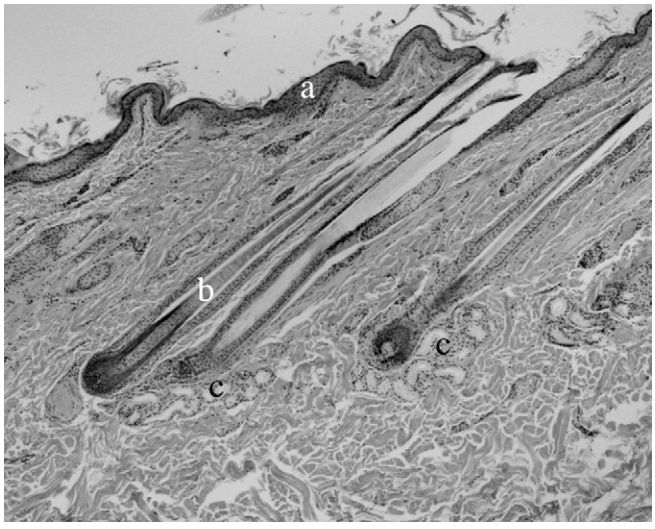

Figure 26-1. Photomicrograph of normal equine skin epidermis *(a)*, hair shaft and follicle *(b)*, and sweat glands *(c)*.

Cleavage lines (Langer's lines of tension, see Chapter 24) have been described in many species. The pattern is determined by placing an awl through the epidermis into the dermis and observing the orientation of the split or cleavage formed.[4] In a review, Rooney cites the work of Schouppe, who studied cleavage lines.[4,5] Schouppe found that horses had cleavage lines whose orientation was parallel to the predominant orientation of the collagen fibers. These cleavage lines changed very little between adult horses, and wounds that healed best were parallel to the cleavage lines. In general, the cleavage lines were parallel to the long axis of the legs, head, and torso, and perpendicular to the long axis of the neck and flank. This is different from the cleavage lines found in dogs, which look more like stripes on a tiger.[6] In my opinion, cleavage lines on horses more closely approximate those of dogs than was reported by Schouppe (see Figure 24-1). When possible, incision lines should be made parallel to the cleavage lines.

FIRST AID

Appropriate wound care is always a balance between improving the wound environment and harming the cells that are integral to the healing process. Consequently, the veterinarian must carefully weigh the benefits and the detriments of any particular action, not only for the immediate results but also for the long-term healing process. Additional information on first aid can be found in Chapter 73.

ASSESSMENT

Wound assessment is one of the most important steps in wound therapy. More repaired wounds fail because of improper preparation and assessment than because of improper therapy. Properly preparing the wound provides an accurate assessment of the wound and deeper structures. Wound preparation begins with placing the animal in a location and environment that will allow the clinician to best determine the status of the wound. If the environment is not adequate to perform all of the tasks in the following paragraphs, it may be a good idea to move the horse to a place where these procedures can be done more efficiently and easily. In general, the best option is to perform any lifesaving therapy at the site where the horse was injured, and then to transport the horse as quickly as possible to a location where definitive therapy can be performed.

INITIAL MANAGEMENT

Restraint and Analgesia

For the horse with a wound, many types of restraint are appropriate, from simply holding its head to general anesthesia. Before using sedation, the horse's systemic status should be determined. It is rare for a horse to lose enough blood to cause problems with sedation, but if this is the case, the horse may collapse because many commonly used sedative agents induce hypotension. Physical examination, blood work including packed cell volume, and skin turgor testing can help determine the systemic status of the horse and allow the clinician to decide if sedation is advisable (see Chapters 1 and 4). Alternatives to systemic sedation include manual restraint with a twitch and/or local anesthesia of the affected area. In many cases, local anesthetic agents provide the best opportunity to prepare and examine the affected area and to eventually treat the wound.[7]

Initial Preparation

After the horse has been restrained appropriately and the area has been desensitized with local anesthetic, the clinician decides whether to treat the wound under aseptic conditions or if a simple clean technique is adequate. Simple clean technique should always include the use of clean exam gloves. If there is a possibility of cross-contaminating a wound while preparing and exploring, aseptic technique should be used. The next step is to clip the hair over and around the wound site. To minimize the amount of clipped hair that gets into the wound, it can be covered with water-soluble, sterile, lubricating gel prior to clipping. It is best to put on a pair of sterile gloves, apply the gel onto the fingers, and rub the gel into place (Figure 26-2). This technique generally ensures that the gel will remain in contact with the wound bed. Some of the clipped hair is is trapped in the gel. At the end of clipping, the gel with the trapped hair can simply be rinsed from the wound with water or saline solution. Only when the entire wound area has been clipped and prepared can the clinician begin to fully appreciate the complexity of the wound. In many cases, the object(s) causing the wound, even seemingly insignificant ones, will have invaded synovial structures, and because accurate assessment was not made, appropriate aggressive therapy was not instituted, leading to the eventual euthanasia of the horse.

DÉBRIDEMENT

Débridement is an effective way to reduce the bacterial load within a wound and to minimize necrotic tissue. The benefits of débridement have been widely discussed in the field of human wound care.[8] Necrotic tissue and bacterial infection are major roadblocks to effective and cosmetic wound healing. The presence of foreign material reduces the number of bacteria necessary for infection by a factor of 10 (i.e., from 10^5 to 10^4 bacteria per gram of tissue). Débridement is used to alter the wound classification from infected to contaminated to clean contaminated to clean. Each type of débridement has positive and negative consequences for wound healing, and it is important to select the technique that provides the best benefit-to-detriment ratio. The most common types of débridement are sharp, mechanical, chemical, biological, and autolytic. Sharp and autolytic débridement should be used primarily in equine wound care. The other types of débridement tend to create more trauma to the wound bed, consequently increasing the healing time required.

Sharp Débridement

Sharp débridement is one of the least traumatic methods available to the equine clinician, yet it is often underutilized. The major drawback to sharp débridement is that when the tissue is removed, it cannot be put back, so it is wise to be conservative in the removal of tissue. If the margin between viable and nonviable tissue is not clear, it is often better to leave some tissue for removal at a later time if necessary. Many skin flaps, especially on the head, retain viability even though they are cool at presentation. Tools for sharp débridement include a scalpel, scissors, and lasers. Scalpel débridement is the least traumatic of these techniques (Figure 26-3), and it should be used

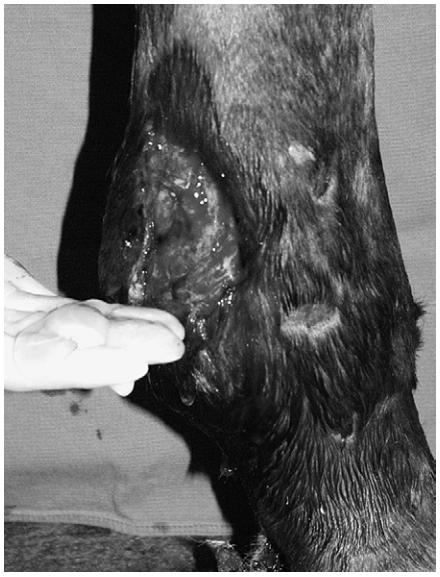

Figure 26-2. Application of sterile water-soluble gel to wound before clipping.

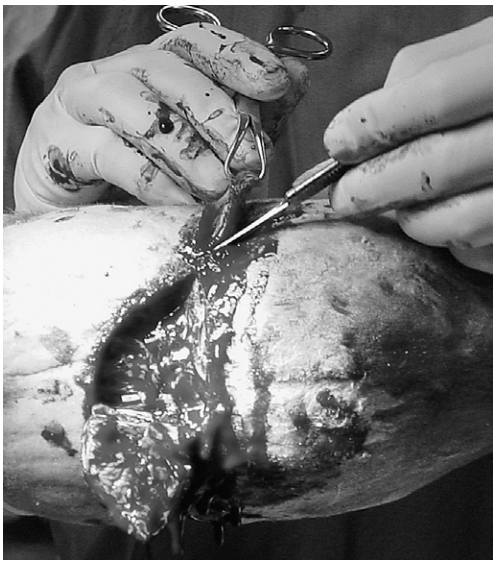

Figure 26-3. Sharp débridement of a wound on the dorsal and lateral aspect of the pastern region.

whenever possible in the initial wound treatment stages. Other types of sharp débridement play a more secondary role.

Mechanical Débridement

Mechanical débridement is more traumatic, and unfortunately it is probably the most commonly used method in the horse. It can be performed using woven gauze (Figure 26-4), lavage (Figure 26-5), wet-to-wet, or wet-to-dry dressings. Mechanical débridement can be a useful tool, and it can be beneficial in the healing process, but it can also be very traumatic. Often, when using gauze, too much pressure is applied and trauma to the wound bed ensues. Only gentle pressure should be used. If more aggressive débridement is necessary, sharp débridement should be selected. Woven gauze provides mechanical débridement characteristics superior to those of nonwoven gauze (Figure 26-6).

Lavage can be used successfully for mechanical débridement. It involves two critical components, the selection of a noncytotoxic cleansing solution and the delivery of that solution to the wound surface with appropriate pressure and volume to wash away the necrotic debris without pushing it further into the tissue planes of the wound.[9] The pressure should be between 10 and 15 pounds per square inch (psi). One way to achieve this pressure is to attach a 19-gauge needle or catheter to a 35-mL syringe.[10] Many devices are available. Showerheads are safer and more effective than single-jet lavage systems. Therefore, devices that produce high pressures (e.g., the WaterPik) should not be used. A simple gentle, low-pressure lavage system can be made by using a 16-gauge needle to punch four to eight holes in the cap of a 1-L bottle and then squeezing saline out (Figure 26-7, and see Figure 26-5).

The fluids most commonly used in veterinary medicine are dilute antiseptics. However, there is no evidence that dilute

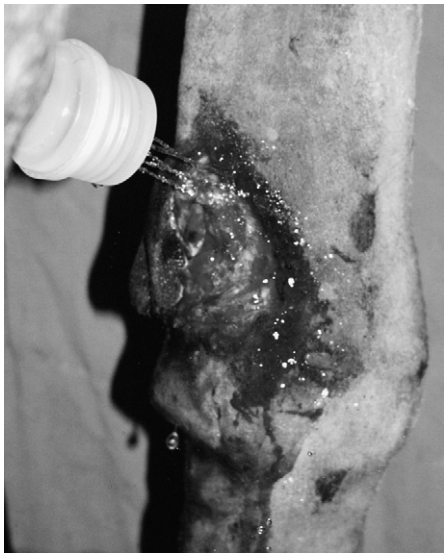

Figure 26-5. Mechanical débridement with sterile saline.

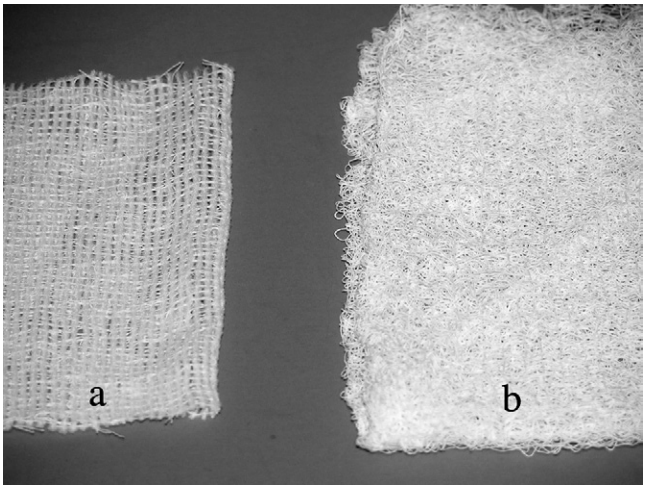

Figure 26-6. Nonwoven *(a)* and woven *(b)* gauze.

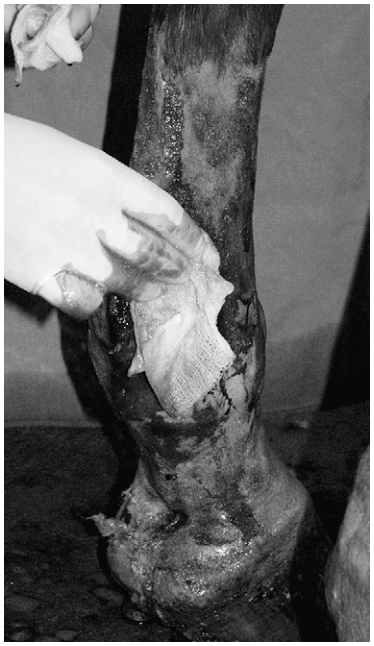

Figure 26-4. Mechanical débridement with woven gauze.

Figure 26-7. Saline bottle, with 16-gauge holes in the cap, used for lavage (see Figure 26-5).

antiseptics successfully kill bacteria when there is necrotic tissue in the wound. To achieve bacterial killing in necrotic tissue, high concentrations of antiseptic are necessary, and high concentrations have a negative effect on the cells necessary for wound healing. Antiseptics are best used around the wound but not in the wound.[11] Saline has been shown to be effective in reducing bacterial counts in an infected wound but to have no effect on wound healing in clean wounds.[12,13] In one study comparing saline solution and water in an infected wound in a rat model, saline solution reduced the bacterial counts by 81.6%, whereas tap water reduced bacterial counts by 82%.[14] However, work by Buffa and coworkers suggests that tap water is very toxic to fibroblasts.[15] In the presence of exudate, surfactant-based wound cleansers, such as Constant-Clens (Kendall/Covidien, Healthcare, Mansfield, MA), are more effective than saline or polyionic fluids.[10,16-18] In summary, the surgeon should avoid putting something in a wound that would not be tolerated in the eye or should not be consumed.[16,18] If wound infection is present, sharp débridement together with systemic antibiotics is necessary to rid the host of bacteria. Local antibiotics, either by intravenous or intraosseous perfusion or topical application, are also helpful.

Wet-to-wet or wet-to-dry dressings can be used for mechanical débridement. Wet-to-wet dressings are intended to stay wet, whereas the wet-to-dry dressings are intentionally designed to dry out between bandage changes. Wet-to-wet dressing may have to be remoistened up to six times a day. These dressings are much more effective at removing necrotic tissue than is mechanical débridement using gauze, while causing less damage to the fibroblasts and epithelial cells. However, it is very time consuming and is generally available only in an intensive care hospital environment. Wet-to-dry dressings are performed by moistening the primary dressing (the dressing against the wound bed) with saline while leaving the rest of the dressings dry. The concept is that the absorbent secondary dressing will pull the fluid from the primary dressing, pulling the wound exudate into the primary dressing also. Because it is difficult to keep the primary dressing moist unless a large amount of exudate is produced by the wound, the primary dressing often becomes dry between bandage changes. When the dried primary dressing is removed, it effectively débrides the wound bed, but this débridement is very nonselective. Thus, the usefulness of a wet-to-dry bandage is questionable. Primary dressings should never be allowed to become dry, as this reduces the effectiveness of autolytic débridement (see later) and leads to indiscriminate removal of epithelial cells and fibroblasts.

Chemical and Enzymatic Débridement

Chemical débridement has been used in many different forms in wound care. Dakin's solution, a diluted sodium hypochlorite (bleach) solution, was originally used during World War I. Other chemical agents include hydrogen peroxide, acetic acid, and, more recently, hypertonic saline. Chemical débridement is nonselective and should be reserved for very contaminated wounds. Hypertonic dressings provide an effective chemical débridement with minimal damage to the wound in the early stages of healing (see "Second-Intention Healing," later). While hypertonic saline débridement dressings are very effective in selected cases, sharp débridement is probably a better technique for removing large amounts of necrotic debris.

Enzymatic débridement involves placing enzymes directly onto the wound bed. The most commonly used enzymes are streptokinase/streptodornase, collagenase, DNase/fibrinolysin, papain/urea, and trypsin. Enzymatic débridement is limited when large amounts of necrotic tissue have to be removed and should be used only after an initial sharp débridement. For enzymatic débridement to be effective after a rigid eschar has formed, the eschar must first be scored with a scalpel blade.

Biological Débridement

Biological débridement has been used since antiquity by applying larvae of *Lucilia sericata* (greenbottle fly) into wounds. The Larvae digest only the necrotic tissue and pathogenic bacteria, leaving the healthy tissue unharmed. More recently, sterile medical grade larvae have been made avialable. The larvae provide rapid and relatively selective débridement. Unfortunately, there is often a problem with client compliance as there is a reluctance to allow the larvae to inhabit the wound. There is some evidence that secretions of the larvae may be able to be used for débridement, antibacterial effects, and promotion of angiogenesis.[19]

Autolytic Débridement

Autolytic débridement is the least traumatic of these techniques. It is achieved by leaving wound fluid (containing white blood cells and enzymes released from dead white blood cells) in contact with the wound bed. The white blood cells and enzymes affect only the dead and necrotic tissue, leaving healthy cells intact for wound healing. This can occur only in moist wounds (Figure 26-8). When wounds are allowed to dry, autolytic débridement is slowed significantly. Autolytic débridement

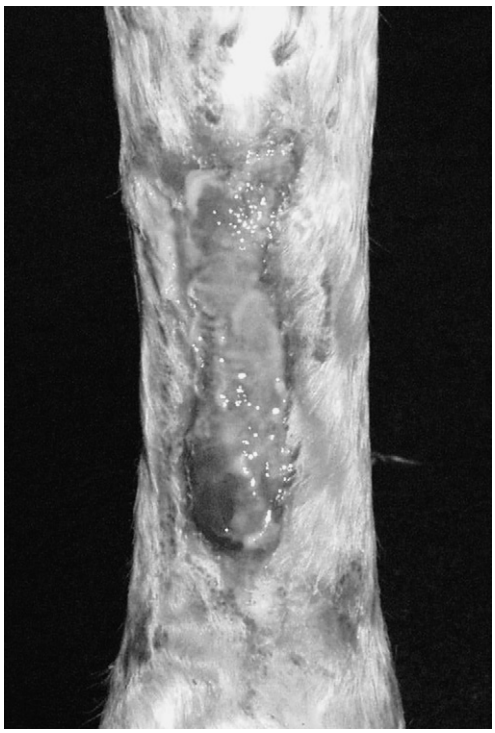

Figure 26-8. Wound undergoing autolytic débridement. This is a normal amount of exudate between bandage changes.

reduces the bacterial count by allowing access of white blood cells to the wound bed without causing continued trauma to the wound bed. However, sharp débridement should be performed before using autolytic débridement because the latter technique is ineffective in the presence of large volumes of necrotic material.

OPTIONS FOR WOUND CLOSURE

The options for wound closure can generally be categorized as primary closure, delayed primary closure, and second-intention healing. To choose the most appropriate technique, the stages and classification of wounds must be understood.

Stages of Wound Healing

There are four stages of wound healing: the inflammatory/cellular reaction stage, the débridement stage, the tissue formation/proliferation stage, and the maturation/remodeling stage. However, these stages overlap during the wound healing process (see Chapter 5). The duration and intensity of the inflammatory stage are generally determined by the extent of injury. For example, a surgical wound has a much shorter inflammatory stage than a severe degloving wound. The inflammatory stage is prolonged in the presence of necrotic debris, foreign material, or infection. The inflammatory stage should be reduced to the shortest duration possible. Extension of the inflammatory stage results in a delayed repair process.[8]

The débridement stage begins early in the inflammatory stage and is marked by migration of neutrophils and macrophages into the wound site. The white blood cells phagocytize bacteria and enzymatically remove necrotic tissue. The length of this stage depends on the size of the wound and the amount of necrotic debris present.

The tissue formation (or proliferation) stage involves fibroblast migration and proliferation as well as epithelialization of the wound. The fibroblasts migrate into the wound along the fibrin scaffold. They secrete the ground substance of the wound as well as the collagen that will eventually provide tensile strength in the wound. Granulation tissue, which is highly vascular and important in wound contraction, quickly becomes evident in a noninfected wound. Wound contraction is the process by which full-thickness skin is drawn together by specialized fibroblasts known as myofibroblasts, which are similar to smooth muscle cells. Myofibroblasts cause thinning of the surrounding skin as they pull the wound edges together. Wound contraction, which is most effective in areas with excess skin, stops when cells of the same type are brought together, or when skin tension equals the ability of the myofibroblasts to contract. Epithelialization, which is part of the repair phase of the wound and begins early in the wound-healing process, can be stopped with infection, drying of the wound surface, exuberant proliferation of granulation tissue, and repeated dressing changes.

The maturation stage of wound healing occurs when there is an equilibrium between collagen production and collagen destruction. It is during this stage that the collagen fibers are realigned along the lines of tension and when wound tensile strength increases. The number of fibroblasts is also reduced.

Wound Classification

Wounds are typically classified on the basis of degree of contamination. Clean wounds, usually seen only in surgical situations, are not infected and do not involve the respiratory, alimentary, or urogenital tract. Clean contaminated wounds, generally seen in surgical situations, involve the lumen of the respiratory, alimentary, or urogenital tract. Contaminated wounds are generally traumatic in nature and may have gross contamination and necrotic debris. Infected wounds generally involve large numbers of bacteria, inflammation, edema, and suppuration.[20] For additional details on infection, see Chapter 7.

Another wound classifciation technique is the color evaluation or RYB color code.[21] The concept is that if you evaluate the wound colors and the percentage of surface area of the different colors in the wound, healing progress can be better determined. The "R" refers to red, and indicates granulation tissue. The "Y" refers to yellow, and indicates purulent debris. The "B" refers to black, and indicates necrotic tissue. Some wound care specialists add a "P," which refers to pink and indicates epithelialization, and others will add a "G," which refers to green and indicates gangrenous tissue. The wounds are evaluated at each dressing change, and a "color" scale is determined. In general, as healthy wounds progress, the black and yellow colors decrease, and the amount of red and pink color increases. If a wound deteriorates, it will have proportionately more black and/or yellow. Infection should always be suspected if this occurs.

In summary, in the early stages of wound healing (i.e., in the inflammatory or débridement stage), wounds that are clean or clean contaminated are the best candidates for primary or delayed primary closure. Wounds that are in the later stages of healing or are contaminated or infected heal best by second intention.

BACTERIA IN WOUNDS

All wounds contain bacteria, but not all bacteria in wounds are cause for concern. Bacterial contamination describes the presence of bacteria in a wound without active multiplication or trauma to the host. Bacterial colonization indicates that the bacteria have attached to the tissue and are multiplying but are not necessarily causing trauma to the host. High numbers of bacteria in the absence of a host response may indicate an impaired immune response. Bacterial infection occurs when bacteria invade healthy tissue and actively multiply, overwhelming the host's immune response.[22] Frequent bandage changes have been implicated in an increase in bacterial numbers as the wound is left exposed to the environment during the dressing change.

Bacteriologic Assessment

Two methods of bacteriologic assessment used in equine practice are described: qualitative and quantitative. Qualitative assesssment involves determining the types of bacteria in the wound, coupled with sensitivity testing. These methods can guide the clinician in choosing antibiotics for treatment. Quantitative bacteriology is rarely performed in veterinary medicine, but it should be considered when a wound is not progressing as anticipated or when a skin graft fails. Bacterial counts greater

than 10^5 per gram of tissue generally indicate an active infection. The number of bacteria needed to produce an active infection is reduced if foreign material (e.g., suture, necrotic debris, a foreign body) is present, if the virulence of the bacteria is high, or if host resistance is decreased. Clinical observation is generally adequate for determining if a wound is infected. Signs such as discolored granulation tissue, edema in and around the wound, purulent exudate, odor, and lameness can indicate an infection. However, a wound can be infected without these overt signs. If infection is suspected, the wound should be cultured.

Technical Considerations in Contaminated Wound Closure

The three main strategies for preventing bacterial infection of wounds are effective wound cleansing and débridement (see "Débridement," earlier), appropriate use of advanced dressings and procedures for dressing changes (see "Second-Intention Healing," later), and appropriate use of topical antimicrobial agents.[9]

Antiseptic agents, in use since the 1800s, are effective against a broad range of gram-positive and gram-negative bacteria. However, they do not penetrate necrotic debris well and are unlikely to reduce bacterial populations deep in a wound bed.[10] Many investigators have looked at the use of antiseptic and antimicrobial agents in the wound.[23-29] In one study, the use of saline in combination with gauze was more effective in reducing bacterial numbers and encouraging wound healing than silver sulfadiazine or povidone-iodine.[28] In a review, Rodeheaver noted that the following antiseptics have been used in the false hope of killing bacteria without negatively affecting the wound bed: acetic acid, alcohols, aluminum salts, boric acid, chlorhexidine, formaldehyde, gentian violet, hexachlorophene, hydrogen peroxide, hypochlorite, iodine, povidone-iodine, Merthiolate, permanganate, and silver nitrate.[10] A recent paper concluded that practitioners should reconsider the ritualistic use of chlorhexidine of any concentration on open wounds until such time that research actually proves that the benefits outweigh the ill effects.[30] Although the evaluation of daily bathing with chlorhexidine to reduce the acquisition of methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant enterococci in human intensive care units has been shown to be beneficial,[31] it is clear that antiseptics should be reserved for use on the normal skin surrounding the wound and not on the wound bed itself.

Unlike antiseptics, topical antimicrobial agents provide efficacy against bacteria within the wound bed, and, depending on the vehicle, they have minimal negative side effects on wound healing. In one study in horses with experimentally created leg wounds, silver sulfadiazine used alone without wound dressings resulted in less formation of granulation tissue than wounds covered with pressure bandages.[32] Antiseptics cannot target specific bacteria, but topical antibiotics can. However, antimicrobial resistance has rendered some antimicrobials ineffective, so the use of systemic antibiotics alone has been advocated to control the appearance of resistant organisms. In some chronic infections, the blood supply to the surface is diminished, and topical antimicrobials are required. Antimicrobials should always be selected on the basis of culture and sensitivity or on the basis of a judgment on the most likely bacteria to be found in the wound. When possible, to reduce concerns about antibiotic resistance, the antimicrobial chosen for topical use should be one that is unlikely to be used systemically. Wound débridement is still the best way to reduce the bacterial count in wounds with necrotic debris, and it should be performed before either an antiseptic or a topical antimicrobial is used.

Systemic antibiotics should be used for chronic or acute wounds that have a large degree of trauma or are close to critical structures such as bones or joints. If possible, cultures and sensitivity test results should be obtained to determine an appropriate antibiotic. Triple antibiotic ointment has been shown to be one of the most effective topical antibiotics in wound healing. Nitrofurazone, at least in current vehicles, is quite toxic to wounds and cannot be recommended.

WOUND CLOSURE TECHNIQUES

After the wound has been successfully débrided, cleaned, and examined, the veterinarian needs to employ some method to achieve an epithelial covering. The options are suture closure, healing by second intention, skin grafting, or some combination of these to provide a continuous epithelial surface over the wound. The type of closure technique to use depends on what caused the wound, the time from injury, the degree of contamination, the extent of the injury, the potential dead space, and the veterinarian's surgical skills. The "golden period" of 6 hours from the wounding, after which the wound is considered to be infected, is no longer deemed correct, and it behooves the clinician to examine the wound carefully to determine which of the following techniques to use for wound closure. The primary goal is to have a functional and cosmetic end result.

Primary Closure

Primary closure is a technique whereby the wound is closed immediately and completely, using strict aseptic technique. This is the technique most likely to provide the best functional and cosmetic result. Unfortunately, primary closure is acceptable only in wounds with minimal tissue loss, minimal bacterial contamination, and minimal tension on the wound edges after closure.

Regardless of whether primary closure or delayed primary closure (see later) is performed, the wound needs to be cleaned and prepared for closure, because excess bacteria in the wound increase the possibility of wound dehiscence. There are many suturing techniques for wound closure (see Chapter 16) and many reviews of suture types for primary closure.[33-37] For areas of tension, complex suture patterns (such as the near-far-far-near, vertical mattress, and horizontal mattress patterns) provide more tension reduction than simple patterns such as a simple interrupted or simple continuous patterns (see Chapters 16 and 24). The near-far-far-near suture pattern (see Figure 24-4) is most satisfactory, as it provides apposition of the skin edge at the same time as tension relief. Additionally, the mattress patterns can be used with stents to reduce pull-through at the skin–suture interface (see Chapter 16). A large-diameter suture material should be used if tension exists. Small-diameter suture material can be used between tension-relieving sutures for tissue apposition and cosmetic purposes. The wound edges should be undermined whenever possible, preserving blood supply, to reduce tension on the closed wound.

When closing a wound, the management of dead space is very important. Failure to obliterate dead space can lead to the development of a hematoma or a seroma, providing an excellent medium for bacterial growth. One or a combination of four techniques can be used to manage dead space in a wound: suture, meshing the skin, passive or active drains, and pressure bandages. Each technique has benefits and risks that need to be weighed. Suture material, although very useful in wound closure, can also act as a foreign body. Excess suture use (too many sutures, a too-large diameter, or too many knots) can potentiate infection.[38] Consequently, the clinician should use the smallest-diameter suture material possible, monofilament, absorbable suture material, and only surgeon's knots (or interrupted sutures if absolutely necessary when the suture material must be buried). Good surgical technique also benefits the patient, as it reduces trauma to the wound site. If dead space cannot be managed by suture placement, meshing the skin (see Chapter 25) or suction drains (passive or closed) should be employed (see Chapter 17). However, drains not only allow evacuation of dead space but also can act as a conduit for bacteria to enter the wound. Drains should be left in place only as long as necessary to reduce the possibility of infection. Pressure bandages can and should be used whenever possible in addition to sutures, meshing, or drains, or sometime in place of them to reduce dead space. A properly applied bandage closes dead space without adding any foreign material. However, if a bandage is too tight, the blood supply to the wound may be compromised, leading to wound failure.

Delayed Primary Closure

In a delayed primary closure, the wound is initially treated openly to allow débridement and reduce bacterial contamination. Next, the wound is closed primarily. In some cases, only a portion of the wound can be completely closed (Figure 26-9). Delayed primary closure is reserved for wounds that have mild to moderate bacterial contamination, minimal tissue loss, and minimal tension on the wound edges after closure. Drains may be placed to evacuate fluid after closure. Delayed wound closure is very useful in the management of abdominal incisions after colonic rupture in humans.[39-41] These studies showed as much as a twofold increase in incisional infection with primary closure of contaminated abdominal wounds. Delayed primary closure after 3 to 5 days did not result in an increased hospital stay.

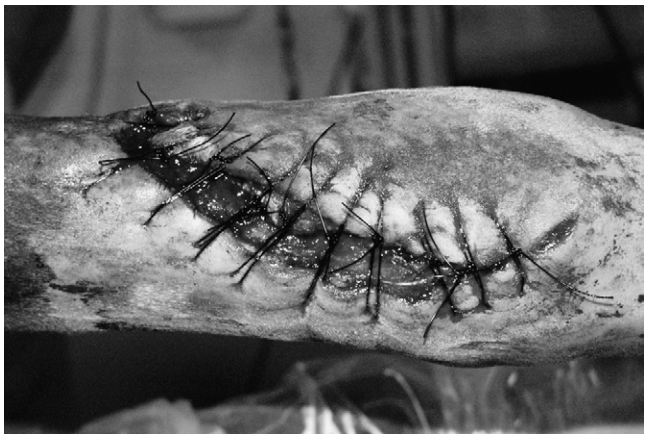

Figure 26-9. A chronic wound that has been débrided and partially closed with near-far-far-near sutures.

Wounds destined for delayed primary closure should be débrided and cleaned to reduce the bacterial burden. Hypertonic saline dressings (see "Second-Intention Healing," next), topical antimicrobials, and systemic antimicrobials can be very useful in treating wounds prior to closure.

Second-Intention Healing

Second-intention healing occurs when primary or delayed primary closure cannot be accomplished. In most cases, these wounds have gross contamination and moderate to severe tissue loss that would make closure difficult. They must heal completely through the process of contraction, granulation, and epithelialization.

In the relatively new concept of moist wound healing, the wound exudate is purposely left in contact with the wound bed. In 1962, George Winter showed that in both swine and humans, full-thickness skin wounds kept in a moist environment re-epithelialized in approximately 12 to 15 days, whereas similar wounds exposed to the air took 25 to 30 days to heal.[42,43] Moist wound healing resulted in wounds that were less inflamed, caused less itching, had less eschar formation, and were more likely to heal without scarring. However, the earliest reports of moist wound healing were by Bloom and Bull in the mid 1940s. Bloom, an army surgeon, sterilized cellophane to treat burn wounds at a World War II prisoner of war camp. His primary goal was to reduce the risk of bacteria entering the wound. Although the dressing reduced bacterial infection, unanticipated benefits included movement with less pain, and less plasma loss.[44] Bull and colleagues studied a transparent nylon that stopped bacterial and fluid penetration yet allowed wound exudate to escape from the dressing. Benefits included faster healing and fewer dressing changes.[45]

The concept behind moist wound healing is that the wound exudate provides the necessary cells and a substrate rich in enzymes, growth factors, and chemotactic factors; controls infection; and provides the best environment for healing. Enzymes come from the breakdown of white blood cells and metalloproteinases. Occlusive dressings keep the wound fluid in contact with the wound bed to encourage autolytic débridement. This in turn provides a good foundation for the rest of the wound-healing process. Local growth factors and cytokines (see Chapter 5) provide a stimulus for the fibroblasts, epithelial cells, and angiogenesis. Chemotactic factors stimulate more neutrophils and macrophages for continued bacterial control and débridement. A moist environment allows better migration of neutrophils and macrophages than a dry wound environment. It has also been shown that wound fluid, even without white blood cells, has some antimicrobial action.[46] Another benefit of occlusion is constant thermal regulation. Concerns with moist healing include bacterial colonization, folliculitis, and the possibility of trauma to periulcer borders. Although bacterial colonization is a concern, a wound that is not infected before occlusion is unlikely to become infected afterward. Another benefit of moist wound healing is that the reduced frequency of dressing changes means that the wound is less exposed to the environment, so it is less likely to become contaminated. Last but not least, moist wound healing is cost effective for the owner.

DRESSINGS, GROWTH FACTORS, AND DRUGS

The open wound changes as it heals, and different dressings provide better results when used during specific stages of the healing process.[47] Consequently, dressing manufacturers have developed many different dressings. Under the following headings, some of the more common dressings are described, and the wounds for which they are best used are mentioned. The ideal dressing keeps the wound bed moist, but not overly so, and the surrounding skin dry. The clinician needs to learn how to manage the amount of exudate present while making sure the wound does not dry out. Experience will quickly guide the use of the different dressing types. The dressings are described in the order a surgeon might use them when treating a wound.[48]

Hypertonic Saline Dressing

Most contaminated and infected wounds contain necrotic tissue and need some form of débridement. Hypertonic saline dressings (Curasalt, 20% hypertonic saline on a Kerlix gauze) have been designed for use on necrotic or heavily exuding wounds. They work by osmotic action to remove necrotic tissue and bacteria. However, they provide a nonselective chemical débridement and consequently need to be monitored carefully. Dressings need to be changed every 24 to 48 hours at the beginning of the treatment period to maintain the hypertonicity and thus the effectiveness. These dressings can be used effectively in most abscess situations. This dressing is available in a 15 × 15 cm (6 × 6 inch) loosely woven gauze and a 1.2 cm (½ -inch) cotton tape for packing small draining tracts. After the necrotic tissue or bacterial infection has been removed, another dressing type should be used.

Honey

Honey has been used in wound healing for centuries because of its ascribed bacteriocidal effects. Also, "growth factor–like" properties of honey have the capability to improve wound healing. In one study comparing honey and sugar, honey was noted to be more effective in reducing bacterial numbers and promoting wound healing.[49] The patients treated with honey were also in slightly less pain during dressing changes and when exercising. More recent research would indicate that Manuka honey from the nectar of the Manuka bush *(Leptospermum scoparium)* in New Zealand is superior at reducing bacterial numbers than are other types of honey.[50] When comparing Manuka honey to hydrogel in a prospective study, the honey group had increased incidence of healing, effective débridement, and lower incidence of infection than the control group.[51] Although the exact reasons for the benefits are unknown, simple hypertonicity does not appear to be the sole cause, especially in light of the previously mentioned study with Manuka honey.[49] There are no scientific reports on the use of honey in equine wounds.

Antimicrobial Dressings

Antimicrobial dressings have been designed to stop bacterial penetration into open as well as closed wounds. The agents are very effective against bacteria and minimally traumatic to the wound. A preferred dressing is called Kerlix AMD (Figure 26-10). The active agent is polyhexamethylene biguanide (PHMB), and it is available bound to Kerlix Super Sponge, Kerlix roll gauze, and other types of dressings. PHMB belongs to a class of cationic surface-active agents that have been used as preservatives in aqueous solutions and as disinfectants and antiseptics (e.g., in cosmetics, contact lens solutions, baby wipes, and pool sanitizers). Increased concentrations, when impregnated into fabric, have shown the capability to suppress microbial growth and penetration. Microbial death occurs by destabilization and disruption of the cytoplasmic membrane, resulting in leakage of macromolecular components. This response is irreversible, and the microbe cannot adapt or become resistant to the PHMB. These dressings are particularly useful in preventing bacterial infection in surgical incisions or where wounds are close to synovial structures and subsequent deep penetration of bacteria would be catastrophic. Although the dressings have been designed to prevent bacterial penetration, they have also been useful in reducing the bacterial load in the wound (Figure 26-11). As these dressings are dry, and made with woven gauze, they should be premoistened before being placed in a wound. They can also be used to cover surgically closed incisions. Silver has recently been impregnated into dressings as well, and they

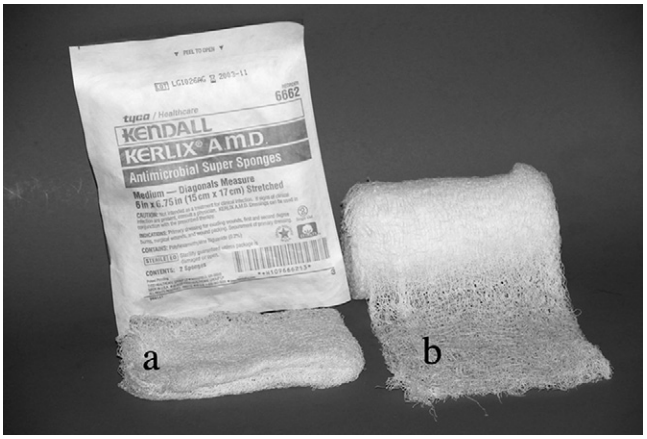

Figure 26-10. Antimicrobial dressing, 6′6-inch woven gauze *(a),* and roll gauze *(b).*

Figure 26-11. Saline-moistened Kerlix AMD (Kendall/Covidien, Mansfield, MA) antimicrobial dressing roll gauze used for chronic back wound after dorsal spinous process removal.

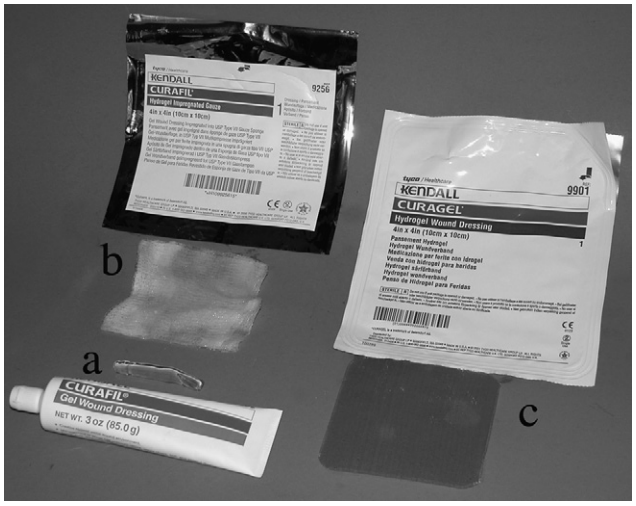

Figure 26-12. Gel dressings (Kendall/Covidien). *a,* Curafil as an amorphous gel. *b,* Curafil as a gel-impregnated gauze dressing. *c,* Curagel, a gel pad.

can be used in a similar fashion. An *in vitro* evaluation of a silver chloride–coated nylon wound dressing against common equine wound pathogens identified an effective reduction in bacterial numbers.[52] The dressings should be changed every 3 to 7 days (the shorter times when used for open wounds and the longer times for surgically closed wounds). After the wound has been cleared of infection, the dressing should be replaced with one of the following dressings.

Hydrogels

Hydrogel dressings were designed to provide moisture to a wound that has dried out. Hydrogels are medical-grade gels composed of water, glycerin, and polymers. They conform to the wound, are nondrying, and provide a bacterial barrier and, eventually, a moist environment. They are available as amorphous gels, in gel-impregnated gauze, or in a mesh-reinforced pad (Figure 26-12). They are completely occlusive and provide an excellent environment for autolytic débridement, white blood cell migration, and thermal regulation, and they thus result in improved wound healing. The gel dressing should be changed every 4 to 7 days, depending on the amount of exudate. When the wound is moist, a different dressing should be used.

Calcium Alginate Dressings

Calcium alginate dressings are used primarily for the granulating phase of wound repair. They are made from alginate, a derivative of seaweed. The calcium in the dressing interacts with sodium in the wound, providing a wound exudate that stimulates myofibroblasts and epithelial cells and speeds wound homeostasis.[53] The calcium also modulates epithelial cell proliferation and migration. Alginate dressings come in nonwoven pads and rope dressings. They conform well to the wound, have excellent vertical wicking properties, and are designed for moderately to heavily exudative wounds. If a wound has a granulation tissue defect and does not have a moderate amount of exudate, the pad should be premoistened with saline before use. The dressings can be changed every 3 to 7 days, depending on the amount of exudate.

Topical Dressings: Collagens and Maltodextrins

Topical dressings such as collagens and maltodextrins are designed for use in the granulating stage of wound repair. They are hydrophilic and should maintain a moist wound bed. They are available in powder or gel form. If the wound does not have a lot of exudate, it is probably best to use the gel form to prevent drying out of the wound bed. Maltodextrins may provide nutrition to the wound bed.[54,55] As soon as adequate granulation tissue is present, a semi-occlusive foam dressing should be used.

Biological Dressings

Various substances have been used as biological dressings. These dressings are intended to provide a framework over which other cells migrate, and as a stimulant to those cells to form the tissue that is desired. Some of the more commonly used biological dressings include porcine small intestinal submucosa (SIS), porcine bladder basement membrane, equine amnion, and various skin products. The porcine small intestinal submucosa and bladder products are not rejected by the host as other xenografts are. From clinical experience, they appear to be best if they are kept moist. In one study, SIS dressing was applied to fresh and chronic wounds. Compared with similar wounds treated without SIS, exuberant granulation tissue was reduced and drainage was facilitated, which reduced wound exudation and improved epithelialization. Overall, wound healing was faster and costs for bandages and hospitalization were reduced.[56] Other studies have shown that the use of equine amnion reduces wound retraction and granulation tissue formation while improving epithelialization.[57] Amnion has been shown to be beneficial as a nonadherent dressing in skin grafting.[58]

Growth Factors

Growth factors (see Chapter 5) have become very popular in experimental studies of wound care. The benefit of platelet-derived growth factor (PDGF) in decreasing wound-healing times has been shown.[59,60] It acts as a chemotactic agent and mitogen for fibroblasts, smooth muscle cells, and inflammatory cells.

Other growth factors have been studied in horses to determine their effects on granulation tissue formation. In one study, it was determined that transforming growth factor-β (TGF-β) has a profibrotic function in wound healing, in that it seems to encourage formation of granulation tissue. It was also found to be more upregulated in limb wounds than in trunk wounds.[61] A human study has shown the benefit of leaving wound fluid in contact with the wound bed by stimulating fibroblast and endothelial cell growth.[62] Various cytokines have been found useful in treatment of experimentally infected wounds.[63] More studies need to be performed to look at different combinations of growth factors, used sequentially through the wound healing process to determine the true effectiveness of exogenous growth factors. The growth factors may be most useful in the granulating and epithelialization stages of wound healing and may be used in conjunction with other synthetic dressings.

Semiocclusive Foam Dressings

Semiocclusive foam dressings are designed for use on mildly exudative wounds (Figure 26-13). They are best used after the

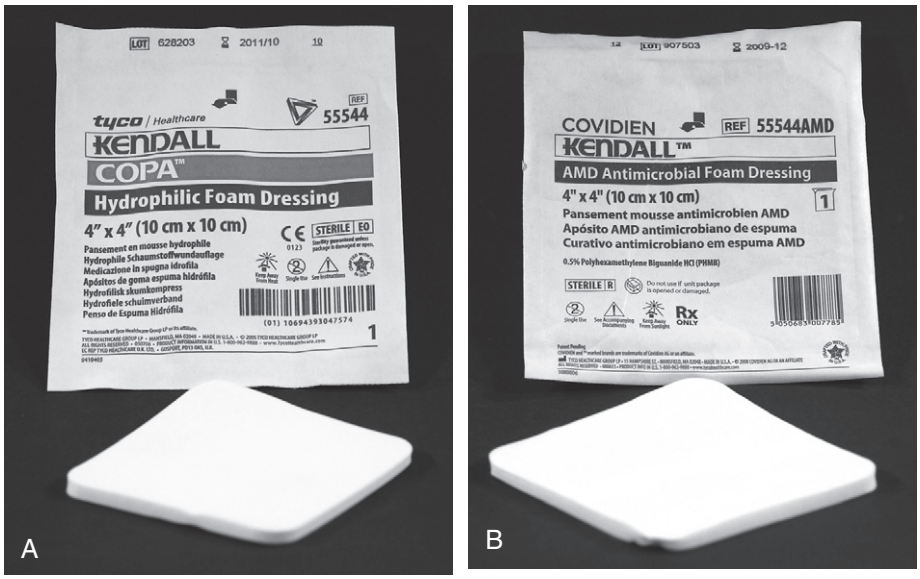

Figure 26-13. Semiocclusive foam dressings. **A,** COPA (Kendall/Covidien), **B,** AMD Antimicrobial dressing (Kendall/Covidien).

wound has been cleaned up, necrotic debris has been removed, bacteria are brought to a count below that needed for infectivity, and healthy granulation tissue is present. The dressings should not be used in the presence of infection. Foam dressings provide a moist environment and thermal regulation that will enhance epithelialization while minimizing exuberant granulation tissue. Newer foam dressings have smaller pore sizes that reduce the chance of tissue growing into them, thereby reducing the chance of the dressing's adhering to the wound. Polyurethane layers prevent strikethrough while still allowing moisture-vapor transmission reducing skin maceration. It is also possible now to use a semiocclusive foam dressing impregnated with an antimicrobial (AMD Foam). It is best to surgically débride exuberant granulation tissue prior to applying a foam dressing. Nonadherent foam dressings have also been useful in skin grafting. They should be changed every 4 to 7 days, depending on the amount of exudate.

Negative Pressure Wound Therapy

Negative pressure (vacuum) wound therapy has been shown to be beneficial in both acute and chronic wound care. A foam dressing is cut to the shape of the wound, placed into the wound cavity, and covered with a transparent adhesive drape (see Figures 17-8 and 17-9). In horses, the skin should be prepared by clipping, shaving, degreasing with alcohol, and applying an adhesive spray. A small hole is made in the drape, and a rigid drain is inserted. The drain tube is attached to a vacuum canister. In one study on humans, it was shown that while the cost of negative pressure wound therapy is more expensive to begin with, it allowed a more rapid definitive therapy when compared to moist wound healing alone (average of 20 days less therapy). The hospitalization cost was approximately 60% less for the negative pressure group than the moist wound healing group.[64] While information in horses is limited, it would appear to be a valuable asset in a wound-healing regimen. Possible benefits include increased blood flow, increased angiogenesis, increased rate of granulation tissue formation, increased flap survival, and decreased bacterial number and edema formation.

Corticosteroids

Corticosteroids are commonly used in equine wound care to reduce the volume of, or to inhibit the formation of, exuberant granulation tissue. However, *in vitro* studies have shown that hydrocortisone upregulates plasminogen activator inhibitor-1 and downregulates plasminogen activators. The plasminogen activators play an important role in wound homeostasis. This change probably inhibits proteolytic matrix degradation and reepithelialization, which are both necessary for rapid and efficient wound repair.[65] Dexamethasone has been shown to interfere with the synthesis and degradation of types I and III collagen in rats. Type III collagen plays a major role in the induction of wound healing and is affected more dramatically by dexamethazone.[66] Triamcinolone has been shown to decrease vascular growth and consequently granulation tissue formation in rabbits. Topical administration was more severe in reducing angiogenesis than was systemic therapy. Testosterone has been shown to delay wound healing in mice.[67] Based on this information, steroids should not be used to inhibit excess granulation tissue formation. There are better ways to accomplish this, such as effective wound débridement and the use of moist wound healing principles.

Nonsteroidal Anti-Inflammatory Agents

Inflammation is an important part of the wound healing process. However, to achieve the most cosmetic and functional results, the inflammatory stage should be minimized.[8] At therapeutic doses, studies have shown that there are few deleterious effects of using anti-inflammatory drugs during wound healing.[68,69] In time, the selectivity of new antiprostaglandin drugs may provide better efficacy without potential side effects, which will make the use of these drugs even more beneficial.

Antiseptics

Generally, antiseptics should not be used in superficial wound. However recently octenidine dihydrochloride (Octenivet Solution, Schülke & Mayr GmbH, Norderstedt, Germany),

a cationic antiseptic belonging to the bispyridine class of chemicals and being effective against gram-positive and gram-negative bacteria[70] was introduced to the veterinary market in Europe. Effectiveness could be shown in oral hygiene by preventing plaque and gingivitis, as a whole body wash for MRSA decolonization,[71] and for skin disinfection of pre-mature newborn infants.[72] Currently, no veterinary literature is available but clinical studies are under way.

TETANUS PROPHYLAXIS

Tetanus, caused by *Clostridium tetani*, should be prevented if at all possible. Adequate protection can be achieved by vaccination, but therapy after infection is often unrewarding.[73,74] Although it has been shown that horses given an initial vaccination and a single booster should be protected for 5 years, it is probably better to have an annual booster.[74-76] This is especially true in horses with an uncertain vaccination history. Unvaccinated horses should receive both a tetanus toxoid and a tetanus antitoxin.

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CHAPTER 27

Management of Deep and Chronic Wounds

Dean A. Hendrickson

The first part of this chapter is devoted to deeper wounds of the head, neck, thorax, abdomen, and extremities; and the second part discusses chronic wounds. Please see Chapters 5 and 26 for more in-depth information on specific wound-healing events.

DEEP WOUNDS

Wounds in different parts of the body heal at different rates and often produce disparate cosmetic results.[1,2] Wounds on the head, neck, and body often appear to heal better than wounds on the extremities. In a review on the topic, Knottenbelt discussed reduced vascularity in the extremities as a possible cause for prolonged wound healing there.[2] However, increased infection, increased inflammatory response, and reduced regional temperature are all involved in the equation as well.[3-5]

Head and Neck

The head and neck have an extensive vascular supply that provides nutrition, oxygen, and white blood cells to the wound for healing. There is good soft tissue support to most areas of the head and neck, which also provides a good basis for healing.[2,6] Unfortunately, many other important structures, such as the cranium, eyes, ears, sinuses, salivary ducts, nerves, and nares, can be involved in lacerations, and exploring them to ensure that nothing is missed requires a thorough knowledge of their anatomy. For example, one horse hit its head on a feeder, causing a laceration in front of the ear. At the initial presentation, the wound looked very superficial; however, a more thorough examination revealed that it involved the cranium and actually penetrated into the brain (Figure 27-1). Initially, the horse had no obvious neurologic signs indicating the puncture. Thus, a thorough and complete physical examination is necessary to adequately assess wounds to the head and neck. Often, horses with head wounds should be heavily sedated before wound preparation and exploration, unless neurologic signs are present.

Diagnostic aids for evaluating head and neck wounds include radiographs, ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI). Radiographs of the head can be difficult to evaluate because of all of the overlapping bones.[7] CT (Figure 27-2) and MRI provide excellent renderings of abnormalities, but they are expensive, not always available, and require general anesthesia. Therefore, if applied, surgical

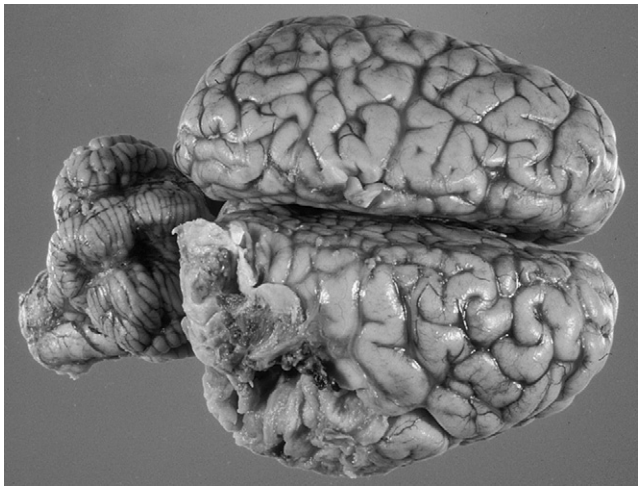

Figure 27-1. The brain of a horse with a penetrating injury to the head showing trauma to the right temporal lobe.

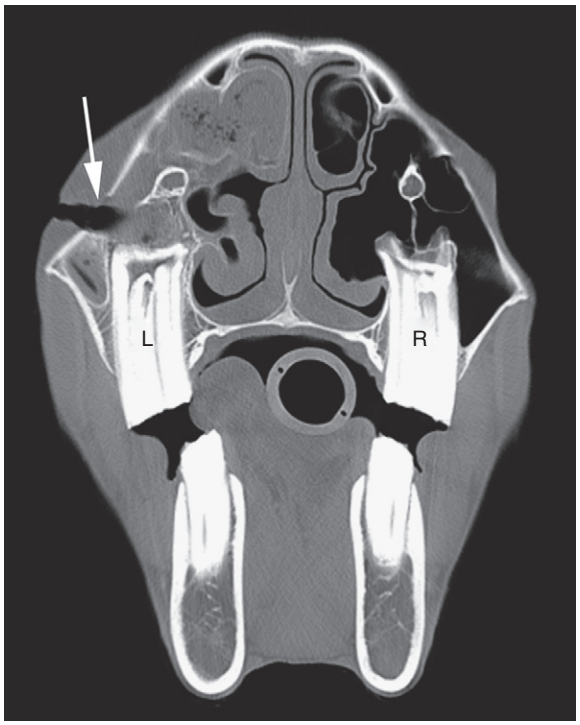

Figure 27-2. Computed tomography scan of a horse with trauma to the left maxillary sinus and subsequent filling of the sinus with blood. The *arrow* shows the entry wound.

wound management should follow the evaluation, if needed. Fortunately, good wound preparation and digital examination often provide enough information on the amount of bone involvement in a head laceration. Fractures should be considered whenever there is a laceration to the head. When cranial fractures are suspected, a thorough neurologic examination should be performed to help determine the extent of the laceration. If the cranium is involved, aggressive therapy should be instituted to reduce the possibility of bacterial penetration and eventual septic encephalitis.

Lacerations around the orbit should be examined to determine involvement of the bone and the globe of the eye (see Chapter 55). To avoid trauma to the cornea, special care is needed when cleaning around the eye. Chlorhexidine should not be used in this area.[8] If possible, lacerations around the eye should be closed primarily to reduce functional problems of the lids. Lacerations to the lids should be closed using small-diameter, absorbable suture material in many layers (see Chapter 56). Multiple layers using small-diameter suture materials provide more stability in areas of increased motion yet will not unnecessarily add to the foreign body volume of the wound. Orbit fractures can be treated using bone reconstruction plates (Figure 27-3) (see Chapter 102).

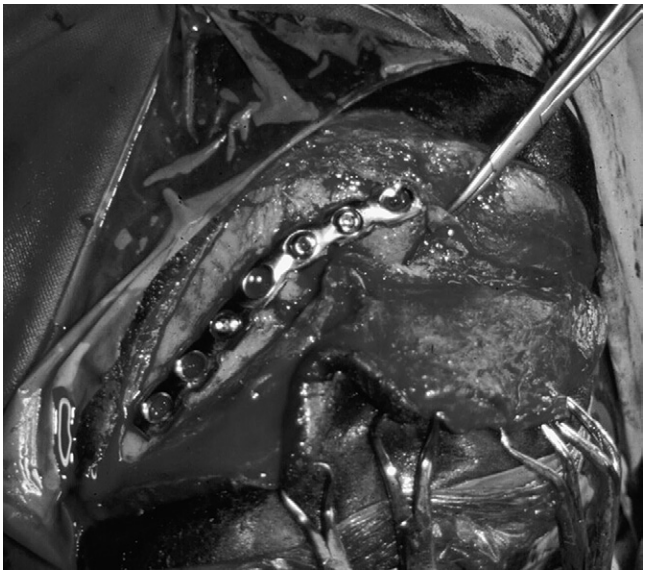

Figure 27-3. Intraoperative photograph of a horse with an orbital fracture repaired with a reconstruction plate.

Repair of ear lacerations can be particularly difficult because ears have considerable mobility. Their range of motion is nearly 270 degrees, and they are supported by cartilage. Ear lacerations, like eyelid lacerations, should be closed primarily whenever possible for the best cosmetic and functional results. Because of the location and blood supply of the ear, lacerations there are rarely too contaminated to close primarily, but sometimes the wounds are too old to hold sutures and must heal by second intention, with revision at a later date. General anesthesia should be used to repair ear lacerations for the best cosmetic result. Rolled gauze or radiographic film can be used as a support to minimize ear movement and improve healing. The gauze is placed inside the pinna and the pinna taped around the gauze. Some horses resent the gauze and shake their head. Placement of radiographic film on both the inner and outer portion of the pinna in combination with the introduction of mattress sutures through the films and the ear represents another option.

Lacerations that involve the frontal and maxillary sinuses may lead to bone sequestration and eventual fistula formation.[9] Prompt intervention can make the difference between a wound that can be closed primarily and one that requires multiple surgeries and wound revisions. Stable bone fragments should be elevated back into position, whereas loose fragments devoid of periosteal coverage should be removed. Rotational skin and periosteal flaps may be necessary to close defects in this area. If there is a large bone defect, the addition of a periosteal flap may

improve the possibility of bone formation in the area. Other dressings, such as ACell, have been suggested for use in this type of wound to encourage bone formation.

Mandibular lacerations can involve other structures, such as the salivary ducts or even the mandibular incisors. Depending on the cause of the laceration, evaluation of the ducts can be very difficult, especially if extensive trauma has occurred. When possible, the integrity of the duct should be confirmed by feeding the horse and observing salivary fluid loss. If transected ends of the parotid salivary duct can be visualized and anastomosed, the result will be better, but this is rarely achievable. If the ends cannot be anastomosed, ligating the salvary duct may be all that is necessary to stop salivary secretion because the gland will atrophy. Sometimes the salivary gland has to be chemically ablated.

Lacerations of the nares are common in equine practice. Successful reconstruction depends on thorough wound débridement, good case selection, and using multiple layers of suture closure to reduce the chance of incisional dehiscence. Fresh wounds are easiest to treat if there is not a large volume of tissue missing. When horses present with wounds that are 7 to 20 days old, the wounds should probably be allowed to heal by second intention and be reconstructed at a later date when they are more likely to hold sutures. In all cases of nares lacerations, multiple layers of closure are necessary for a successful closure.[10]

Thorax

Trauma or lacerations to the thorax can involve the ribs and/or can penetrate the pleural cavity and cause a pneumothorax. Consequently, the wounds must be carefully examined to be absolutely certain of the depth of penetration. The area should be clipped and aseptically prepared to allow a thorough examination of the site (see Chapter 26). Aseptic technique should be used for all thoracic wounds, because involvement of the pleura cannot be ruled out until after the manual examination. If the examination area is quiet, the surgeon may be able to hear air being sucked into the wound during inspiration. Thoracic auscultation should also be performed to help rule out pneumothorax. Radiography, ultrasonography, and thorocoscopy can be helpful adjuncts to the examination (Figure 27-4). If the wound has entered the pleural cavity, a chest tube should be placed to evacuate the air from the chest (see Chapter 48). If the surgeon cannot place a chest tube, the wound should be closed in as many layers as possible, bandaged, and then referred (see Chapter 48). As an alternative to a chest tube, a teat cannula can be attached to a 60-mL syringe with a three-way stopcock to remove air from the chest cavity. With time, it is possible to evacuate most of the air from the pleural cavity with this technique, but a suction pump is more efficient. The chest tube or the teat cannula should be placed in the upper third of the chest to enable removal of the largest volume of air. In case of pleural penetration, broad-spectrum antibiotics should be instituted. The wound care instituted should be appropriate for the stage of healing. Stent bandages can be used to cover the primary dressing. Thoracoscopy can serve as an aid to diagnose pleuritis secondary to thoracic lacerations (see Chapter 48).

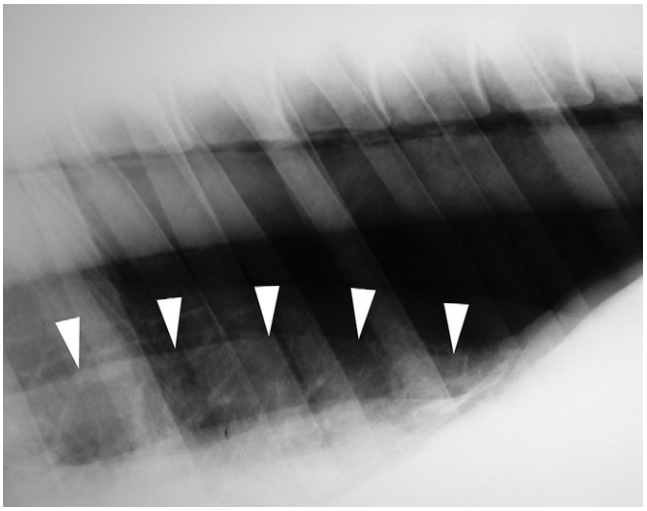

Figure 27-4. Lateral radiographic view of a horse with a thoracic injury and subsequent pneumothorax. Note the dorsal edge of lung outlined by *arrows.*

Abdomen

Lacerations to the abdominal wall offer a diagnostic challenge. The abdominal wall has numerous layers that vary depending on the location. The hair should be clipped, and aseptic technique should always be used on abdominal wounds until peritoneal penetration has been ruled out. If the entry point is small, it may be useful to enlarge the wound to better explore the deeper layers, and the horse may have to be sedated to allow thorough examination. The skin is generally more resistant to tearing than the fascial planes and muscles of the abdominal wall. All tissue planes should be examined for involvement. It is possible for the tissue planes to move, allowing overlap of the layers. An abdominocentesis may help, but in many cases, the cell counts and total protein are within normal limits. Confirmation of abdominal penetration may not be possible, so the veterinarian must carefully monitor the patient for signs of peritonitis after the wounding.

If the wound does not involve the peritoneal space, it should be débrided and closed in multiple layers if at all possible. A drain may be helpful in evacuating dead space. Wounds that have a large amount of tissue loss may be predisposed to herniation. However, it is not advisable to place a mesh in a contaminated wound. Therefore, the wound should be treated until healed, and then a mesh can be implanted to reduce the chance of hernia formation. Abdominal bandages can be useful to help support the body wall. The need for abdominal support may be minimal, but the bandage often reduces edema formation at the wound site and encourages more rapid wound healing. Broad-spectrum antibiotics are often indicated in abdominal wounds.

If peritoneal penetration has occurred, an abdominal bandage should be placed on the animal and broad-spectum antibiotics administered for transport to a surgical facility for an abdominal exploration. Whenever peritoneal penetration has occurred, it is possible that there has been trauma to the viscera. Abdominal lavage using copious amounts of polyionic fluid should be performed, and broad-spectrum antibiosis should be continued. Laparoscopic exploration should be considered to evaluate the peritoneal cavity (see Chapters 13 and 34). It is not possible to visualize all portions of the bowel, but it can be possible to evaluate the dorsal-most structures in a standing animal and the ventral-most structures in a dorsally

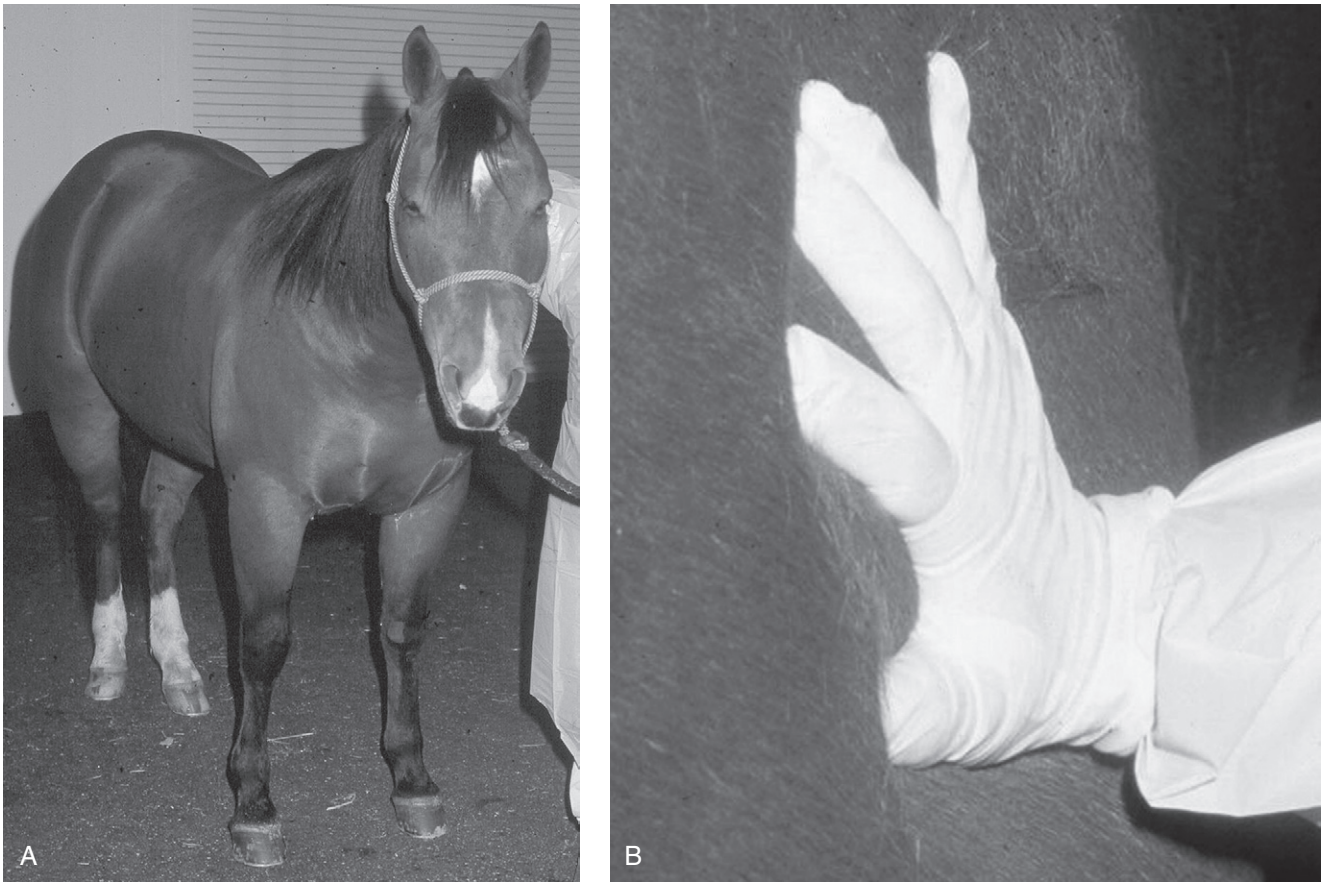

Figure 27-5. A, A horse with an axillary wound and subsequent subcutaneous emphysema. **B,** Close-up showing how the skin indents over the shoulder with manual pressure.

recumbent animal. Directed lavage can be very helpful in reducing the contamination of peritoneal penetration.

Extremities

Axillary Lacerations

Horses that present with lacerations of the axillary region often have subcutaneous emphysema (Figure 27-5). The emphysema is created when the horse moves its leg forward, opening the wound and filling it with air. When the horse moves forward and the leg is positioned caudal relative to its forward position, the air is trapped and forced into the surrounding tissues. In some cases, a pneumomediastinum occurs, which can lead to a pneumothorax.[11] The hair over these wounds should be clipped, and the wounds should be cleaned and débrided. Packing the wound is difficult, but it reduces the accumulation of air in the surrounding tissues. The best way to reduce subcutaneous air accumulation is to limit the movement of the horse. When the air has accumulated, it must be reabsorbed, and there is no efficient way to remove it. Calcium alginate dressings may encourage formation of granulation tissue, speeding the sealing of the wound.

Lacerations Involving Synovial Structures

Lacerations into synovial structures must be identified and aggressively treated as early as possible. The veterinarian must know the locations of the synovial structures—repeated and thorough reviews of equine anatomy are helpful. Whenever a laceration occurs that is close to one of these structures, synovial involvement must be determined. The best way to confirm the involvement of a synovial structure is to place a needle into the structure at a site distant from the wound and distend the structure with sterile saline (Figure 27-6). Strict aseptic technique is a prerequisite for this injection. If the wound involves a synovial structure, aggressive therapy, including lavage, intravenous antibiotics, local perfusion, and effective wound dressings, should be performed (see Chapter 83). Synovial structures should be closed primarily if the wound can be débrided and the synovial membrane apposed.

Heel Bulb Lacerations

Lacerations to the heel bulb area are common in equine practice and can involve anything from the skin to the coffin joint, so the structures involved must be identified. Synovial involvement of either the digital flexor tendon sheath or the coffin joint should be ruled out before treatment. If there is no synovial involvement, heel bulb lacerations are often best repaired using delayed primary closure. Because the foot is in contact with the ground, most heel bulb lacerations are very contaminated. Effective wound preparation, débridement, and treatment are critical (see Chapter 26). After the wound is cleaned, it can be sutured and a slipper cast applied.[12] The

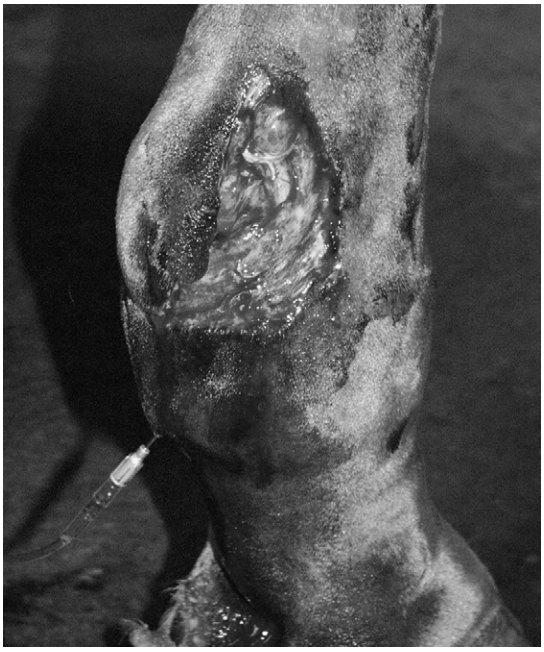

Figure 27-6. The digital tendon sheath is filled with sterile saline solution to determine if communication exists with the laceration on the lateral aspect of the fetlock region.

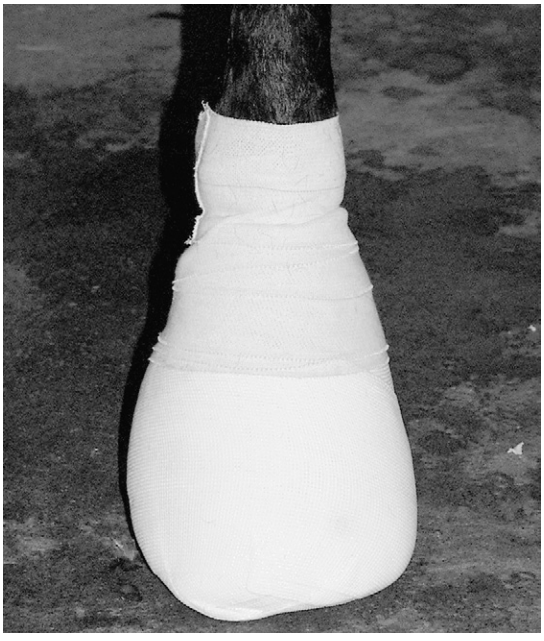

Figure 27-7. A horse in a slipper cast after reconstruction of a coronary band laceration. (See Chapter 17 for cast application.)

slipper cast reduces movement in the area and allows much more rapid and cosmetic healing (Figure 27-7). Additional information of the management of heel bulb wounds is found in Chapter 90.

CHRONIC WOUNDS

Chronic wound care can be challenging and demanding. In human medicine, "a chronic wound is a window to underlying disease. Each wound is a symptom of underlying infirmities that undermine the potential for healing."[13] The local wound environment is part of the larger milieu of the body, and chronic, nonhealing wounds can indicate a larger problem in the macroenvironment of the body.[13] Consequently, it is critical to examine the entire animal when a nonhealing or chronic wound is present. Chronic wounds in both people and horses are commonly found on the extremities. In people, they are often secondary to chronic metabolic diseases, whereas horses often have some type of underlying infection.

Much work has been done on the local wound environment of nonhealing wounds in humans. Studies have shown that wound fluid found in chronic wounds inhibits the growth of fibroblasts by affecting the cell cycle.[14,15] The negative effect of the wound fluid can be reversed by high temperatures. Tumor necrosis factor-α (TNF-α) may play a role in the reduction of fibroblast growth rates, whereas interleukin-1β (IL-1β) and transforming growth factor-β1 (TGF-β_1) do not significantly affect growth rates.[15] Other studies have shown an increase in fibroblast proliferation when these cells are exposed to wound fluid from chronic wounds.[16] Differences in experimental conditions may have caused the difference in results, which underscores the importance of further research into the effects of chronic wound fluid.

Another difference in wound fluid obtained from acute versus chronic wounds is in proteolytic activity. In acute wound fluids, the plasminogen activator system is active, and in chronic wound fluids, the urokinase-type plasminogen activator and urokinase-type plasminogen activator receptors are active.[17] Different proteins are expressed at different levels during the wound healing process. Heat shock protein, platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), vascular endothelial growth factor (VEGF), and their receptors were increased in the wound within 24 hours of skin injury. The levels declined to normal levels by day 7 to day 14, coinciding with healing. The expression of these factors in chronic wounds was delayed or inhibited, suggesting that addition of these agents might improve wound healing in chronic wounds.[18]

Sarcoids are the most common cutaneous tumor in the horse. Frequently sarcoids develop at the location of previous wounds.[19] A study designed to look at the response of cells—isolated from sarcoids, granulation tissue, and normal dermal fibroblasts grown from primary cell cultures—to growth factors showed different morphologic features for each cell type. Sarcoid-derived and granulation tissue–derived cells grew more slowly than the normal dermal cells. When growth factors (epidermal growth factor, acidic fibroblast growth factor, and basic fibroblast growth factor) were added, the sarcoid-derived cells and normal cells were stimulated, but the granulation tissue–derived cells were inhibited. However, when TGF-β_1 was added, the granulation tissue–derived cells were not preferentially inhibited.[20] Continued research in wound healing should help to identify ways to change chronic wounds to acute wounds, thereby improving the healing response.

Chronic wounds often heal with a poor functional and cosmetic result. For the most functional and cosmetic healing, the best environment for wound healing should be provided early. The best way to treat a chronic wound is to not allow the wound to become chronic in the first place. Because this is not always possible, the surgeon is required to treat horses with chronic, nonhealing wounds.

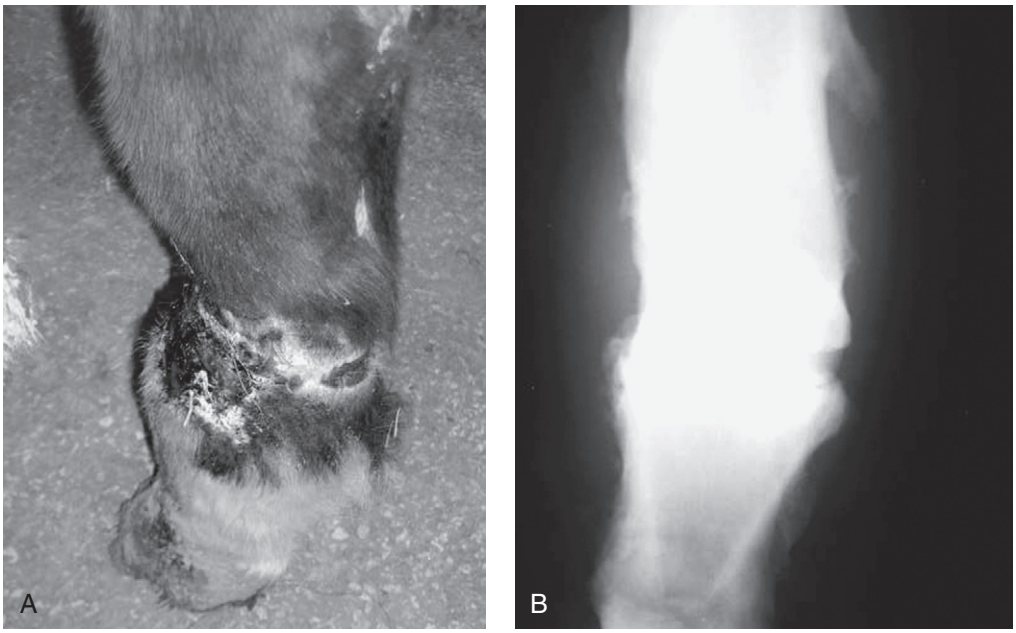

Figure 27-8. A, A chronic wound near the fetlock. **B,** Radiographic evidence of osteomyelitis and new bone production.

As with an acute wound, the first step in treating a chronic wound is to prepare the wound for examination (see Chapter 26). The examination generally starts with digital exploration. The wound should be explored for pieces of foreign material (e.g., bone, wood, metal, sand, suture material). In some cases, draining tracts are found that guide the exploration (see Chapter 28). In others, the foreign material is walled off and difficult to palpate. In those cases, ancillary methods such as radiography, ultrasonography, CT, or MRI can be useful. Radiography is best for bone and other radiodense objects. Ultrasonography can be very useful for radiolucent objects such as wood. CT and MRI may be very useful but cost is often prohibitive.

Infection should be suspected whenever a wound does not heal in the anticipated time frame. In a review of wound infection, Dow identified parameters that show the effect of chronic wound infection on wound healing.[21] He also identified the best ways to diagnose wound infection, the most common being by the clinical appearance of the wound. Inflammation, edematous granulation tissue, discolored granulation tissue, draining tracts in the granulation tissue, and odor are clinical signs that should increase suspicion of infection (Figure 27-8).

A second (and the absolute) way to determine infection in a wound is by bacterial culture. However, all wounds contain bacteria. A wound with a bacterial burden of 10^5 or 10^6 bacteria per gram of tissue is generally considered to be infected.[21] Quantitative bacteriology involves submitting a defined amount of tissue (at least a gram) for bacterial culture and calculating the number of bacteria per gram of tissue. This is rarely done in equine medicine, but it should be considered when wound healing does not progress as expected. Qualitative culture should be performed to determine the types of bacteria in the wound, and antibiotic sensitivity testing should be conducted to determine the most effective antibiotic. (See Chapter 26 for information on topical antibiotics, antiseptics, and systemic antibiotic therapy.) As mentioned repeatedly, adequate débridement and removal of foreign material represents the most important steps in reducing wound infection is.

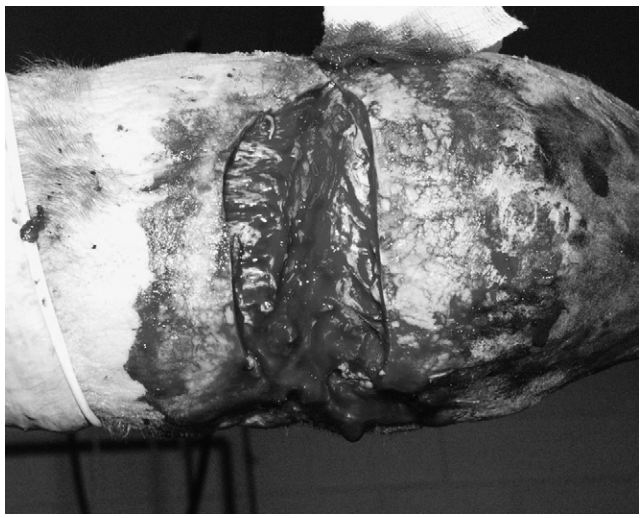

Figure 27-9. A chronic wound after débridement to encourage wound healing.

After examining and exploring the wound, the clinician decides the next step in wound care. In many cases, the wound needs to be "freshened" to stimulate it to heal. Surgical débridement, commonly used to accomplish this (Figure 27-9), can essentially turn a chronic wound into an acute wound.

Surgical débridement is a vital adjunct for wound care in humans with diabetic foot ulcers, and it is an important part of chronic wound therapy.[22,23] Any infected bone or tissue should be removed by the débridement, so that no foreign material is left behind.

As previously described, growth factors hold promise for wound healing. An understanding of the wound environment

is necessary before growth factors are routinely used.[24] Purified growth factors may be useful only in combination.

After surgical débridement, wound closure may be attempted (see Chapter 26). A benefit of working with a chronic wound is that the granulation and scar tissue often stretch the skin. After the scar tissue and granulation tissue are removed, there is ample skin to close the defect. In some cases, only a portion of the wound can be closed, and the rest is allowed to heal by second intention or skin grafting (see Figure 26-11). The skin within approximately 1 cm of the skin edge is usually not healthy enough to hold sutures, and it is often best to remove this tissue during wound débridement.

Sometimes, wounds do not respond in an anticipated fashion, and keloids or excess scar tissue results. Keloids are distinguished from hypertrophic scars in that keloids extend beyond the original wound and rarely regress.[25] The pathogenesis of keloid or hypertrophic scar formation is unknown, but it has been linked to an increased number of epidermal Langerhans' cells.[26] The epidermal immune barrier may play an important role in the development of hypertrophic scars, which have been noted to have increased blood flow in humans evaluated with laser Doppler.[27] In the normal wound healing process, the vasculature diminishes, allowing the completion of healing. Hypertrophic scars may represent an interruption in this healing process. Possibly, vasoconstrictive agents employed in the maturation stage of healing would reduce the proliferation of vasculature, which might inhibit hypertrophic scarring. In humans, there is a familial disposition to keloid formation. Steroid injection, surgical excision, radiation therapy, compression, and tension reduction have been used for treatment of keloids.[28,29] *In vitro* culturing of fibroblasts from keloids with the dietary compound quercetin (a flavonol) has shown some promise in inhibiting proliferation and contraction of the cells.[30]

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CHAPTER
28 Management of Sinus Tracts and Fistulas

John A. Stick

Sinus tracts are distinct from fistulas, although the terms are often used interchangeably. A fistula is an abnormal passage or communication, usually between two internal organs or leading from an organ to the surface of the body.[1] Examples include communications between a tendon sheath and a joint (synovial fistula), from the intestine through the abdominal wall (enterocutaneous or parietal fistula), and between the mouth or esophagus and the surface of the body (orocutaneous or esophageal fistula).[2,3] A sinus tract, on the other hand, is defined as a cavity or channel. It may be normal (e.g., a venous sinus), or it may be a pathologic condition (e.g., an abnormal channel or fistula that permits the escape of pus through the skin).[1] In this chapter, the discussion is limited to the management of sinus tracts. Fistulas are described in chapters dealing with the specific affected anatomic organ or space.

ETIOLOGY

Chronic sinus tracts in horses commonly occur secondary to trauma and foreign bodies. Those involving the lower limbs are frequently associated with bone sequestra (Figure 28-1). Many of these become self-limiting, as the sequestrum undergoes natural débridement or will resolve immediately after sequestrectomy. However, chronic sinus tracts associated with foreign bodies have been described in many areas of the body and are sometimes quite difficult to resolve, especially if the foreign body is inert and not easily degraded by lysosomal enzymes released from white blood cells. Foreign bodies can be wood, metal, sand or small rocks, and even plastic.[4] Frequently, nonresorbable suture material results in a chronic sinus tract.[5]

Sharp foreign bodies have a tendency to migrate through tissues, especially when one end is blunt and the other is sharp, and can be found at some distance from the original site of entry. Examples of this include wood and metal foreign bodies with one blunt and one pointed end and can include surgical instruments.

DIAGNOSIS

Because foreign bodies are suspected at any time a chronic sinus tract is encountered, identification of the type of foreign body prior to surgery is always attempted. Foreign bodies are frequently noted as radiographic abnormalities on survey films, and, therefore survey films should always be taken. Metallic foreign bodies are easily identified this way. Other radiographic abnormalities include soft tissue swelling, thickening, gas densities, and soft tissue and periosteal reactions near bone, any of which may allow the foreign material to be located and identified.

Positive-contrast sinography is easily performed with water-soluble contrast medium (Figure 28-2). It is particularly useful for identifying of radiolucent foreign bodies, which frequently appear as space-occupying lesions in the center of the contrast

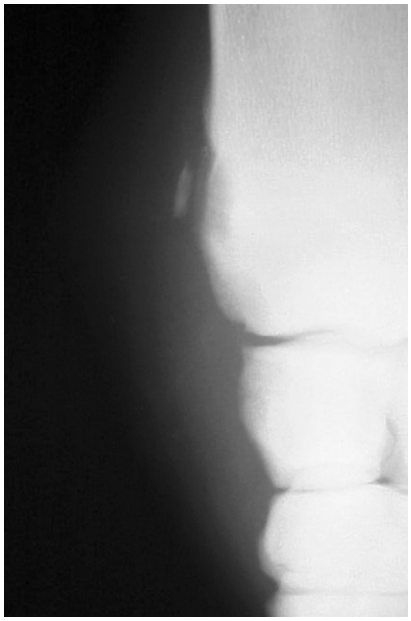

Figure 28-1. Radiograph of the carpal region of a horse that had a draining sinus tract just proximal to the carpus. Note the sequestrum on the distal radius.

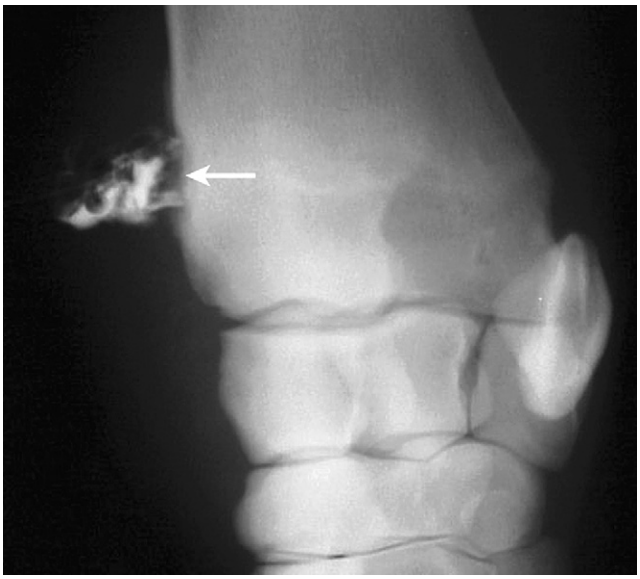

Figure 28-2. Positive-contrast sinography (performed on the horse shown in Figure 28-1) outlines the extent of a sinus tract. Note that the contrast material outlines the sequestrum *(white arrow).*

material. Ultrasonography is also useful in the diagnosis of foreign bodies such as wood, especially those embedded in muscle. Water-bath studies have shown that bone, wood, and large tendons all demonstrated acoustical shadowing in equine muscle.[6-8] Differences in hyperechogenicity and acoustical shadows help detect and identify a foreign body.

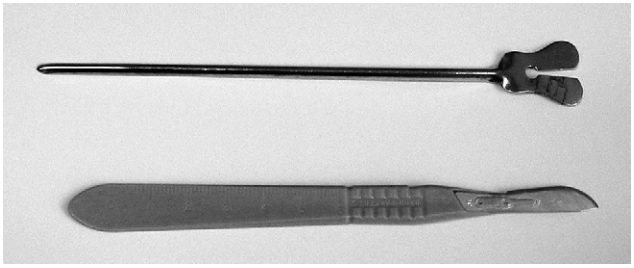

Figure 28-3. A groove director (next to a scalpel blade). When the scalpel blade is placed within the groove director with the point ventrad, the groove director guides the blade into the tract, and the incision can be made from the sinus tract outward. (See Figure 28-6.)

EXPLORATION

Extirpation of the foreign body is the treatment of choice for a chronic sinus tract. Exploration of the sinus is carried out with a malleable probe and a groove director (Figure 28-3). A malleable probe is used to determine the direction in which the exploration should occur (Figure 28-4), and a groove director allows sharp dissection of the superficial portion of the sinus tract, which can be opened rapidly without bypassing the tract. Frequently, sinus tracts travel at varying angles and need to be opened in several directions. Occasionally, they even need to be opened a fair distance from the opening in the skin.

After exploration with a malleable probe, the groove director is inserted through the opening in the skin and guided along the probe. The probe is removed and, with the point of a No. 10 scalpel blade placed in the groove, the tract is opened toward the skin (Figure 28-5).

This process is repeated if the tracts continue in other directions. When sinus tracts are quite deep, Weitlaner retractors are useful to hold open the incision. The tract itself usually has a dark purple membrane, which allows easy identification. Initial

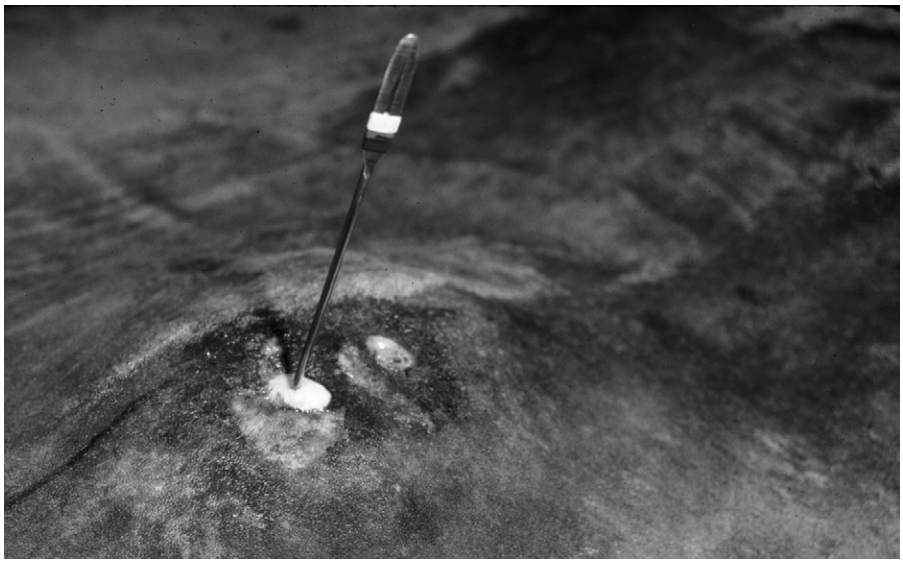

Figure 28-4. A malleable probe is inserted into one of the draining tracts in the ventral abdomen of a horse to localize the foreign body (nonabsorbable suture material in this case).

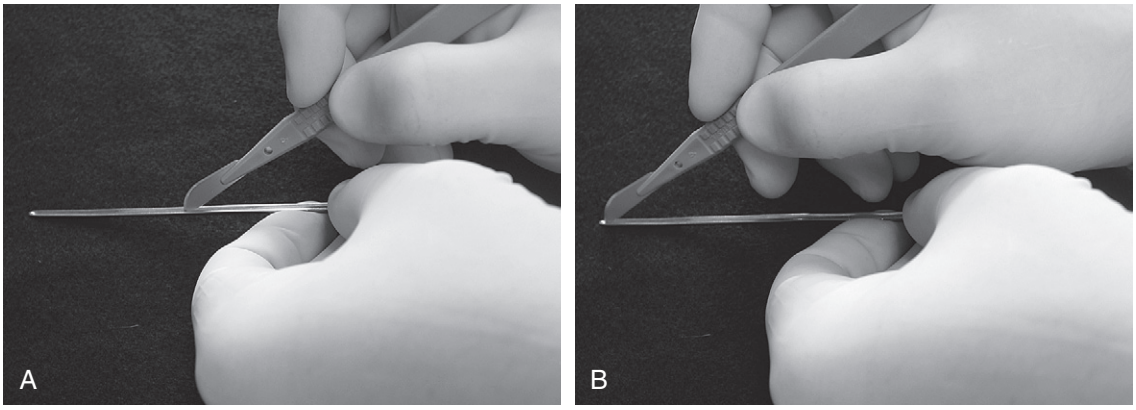

Figure 28-5. The groove director is inserted through the cloaca into the sinus tract. This is followed by sliding a No. 10 scalpel blade point in the groove of the tract **(A)** and opening toward the skin. As the scalpel blade reaches the end of the groove director, it has a stop **(B);** hence, the incision is not made beyond the extent of the end of the groove director.

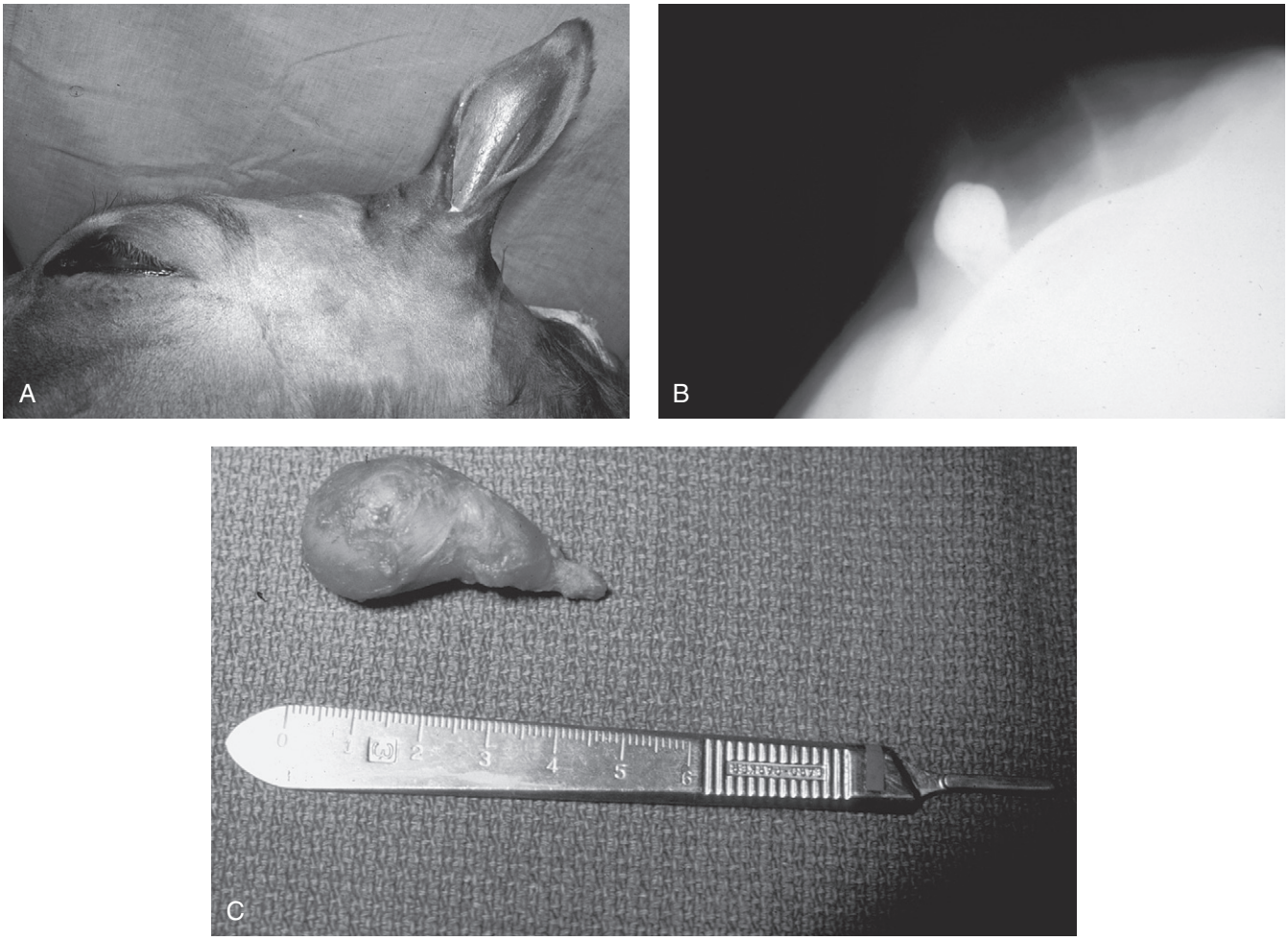

Figure 28-6. Sinus tracts located at the base of the ear usually indicate the presence of a conchal cyst. This can usually be palpated just cranial to the ear as an enlargement **(A),** radiographs confirm that it is a tooth **(B),** and the aberrant tooth with its cyst lining is removed in its entirety **(C).**

infusion of a vital dye such as Evan's blue further facilitates recognition of the tract during surgery, as it stains the tract lining dark blue.

After the foreign body has been identified and removed, all linings of the sinus tract should be curetted so that most of the bacterial contamination is removed and the sinus tract will stop draining. The membrane itself will produce purulent material for some time. The tract should be thoroughly flushed with sterile saline. Although much of the tract may be closed after this procedure, it is usually better to simply débride the cloaca rather than to close it.

SPECIAL CONSIDERATIONS

The location of the sinus tract may be an indication that the tract did not develop secondary to a foreign body. Any time a sinus tract develops on the head, a sinus infection or a dental problem should be suspected, especially if sinography outlines the tract to the apex of a tooth. A sinus tract opening found near the base of the ear is usually the result of a conchal cyst (ear tooth). This can easily be diagnosed with radiography (Figure 28-6). For details on surgical treatment of this condition, see Chapter 29. A sinus tract over the pole or the withers region may indicate the development of an infected bursa in this area (Figure 28-7). The former is called *pole evil* and the latter is

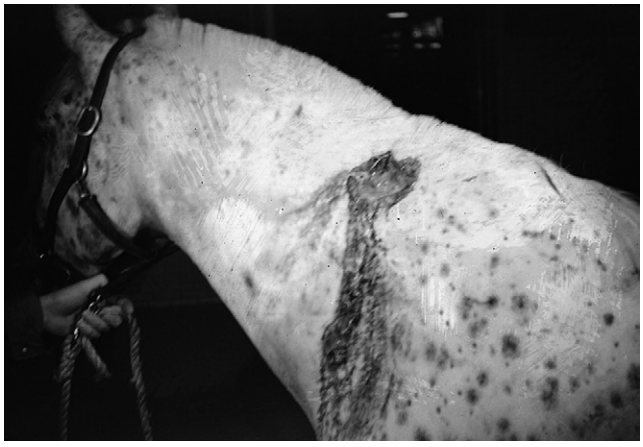

Figure 28-7. Any time a sinus tract develops along the withers, the condition of fistulous withers is suspected, and this can be confirmed radiographically. However, because this condition may be caused by a *Brucella* infection (zoonosis risk), cultures and a titer should be obtained prior to further diagnostic workup and treatment. (See Chapter 82 for more details on this condition.)

called *fistulous withers.* If either is the case, a blood sample should be taken to determine a *Brucella* titer, as brucellosis is a human health hazard.

COMPLICATIONS

Lacerations or incisions into large vessels, or even into internal organs, are possible when following a sinus tract. Therefore, caution is necessary when sinus tracts are deeply embedded in and around body cavities. Also, some sinus tracts contain more than one foreign body, especially if the foreign body is made of wood or nonabsorbable suture material. Other sinus tracts do not contain a foreign body, as it may have been absorbed or expulsed and the drainage comes from the sinus tract lining alone. Owners should be warned that recurrence is a possibility any time this exploration is attempted, and that, in fact, foreign bodies sometimes cannot be found. Another surgery may be necessary when a new cloaca forms from a new sinus tract.

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CHAPTER 29

Skin Conditions Amenable to Surgery

Elizabeth A. Carr

The majority of equine skin diseases requiring a surgical consultation or surgical intervention are neoplastic in origin. Consequently, the major focus of this chapter is on neoplasms of the skin. The biological behavior, predisposing factors, diagnosis, prognosis, and treatment options are discussed. When available, comparative reviews of treatment protocols are included. The goal of this chapter is to provide information to assist the clinician in determining the best approach to treatment of a particular neoplastic condition. In addition, a handful of other skin conditions are reviewed. This is not an exhaustive review of all skin conditions but instead a review of the more common diseases, with an emphasis on those that are amenable to surgical resolution.

SARCOIDS

The equine sarcoid was first described by Jackson in 1936.[1] The most common tumor in horses worldwide, sarcoid is a cutaneous, fibroblastic neoplasm with a proliferative epithelial component.[1] Sarcoids are frequently classified histopathologically as benign tumors because of the morphologic characteristics of the fibroblasts and because many sarcoids are slow growing and cause little if any physical problems in the affected animal. This classification is misleading, however, and ignores the large number of sarcoid tumors whose clinical behavior can only be described as malignant.[2]

Sarcoids are subtyped, on the basis of clinical appearance, into occult, verrucous, nodular fibroblastic, ulcerative fibroblastic, mixed tumors, and malevolent. This classification correlates with their biological behavior (Figure 29-1). The most aggressive subtype consists of malevolent sarcoids, which infiltrate locally along fascial planes and vessels; grow rapidly, and have a high recurrence rate after excision.[2]

Sites of predilection vary with geographic location and include the face (muzzle, ears, and periocular region), distal limbs, neck or ventral abdomen, and areas of previous injury and scarring.[3] Location has been reported to affect prognosis, with sarcoids on the distal limb and periorbital region having a worse prognosis for resolution than sarcoids in other locations.

Epidemiology

The relationship between breed and risk for development of sarcoid was evaluated in a large number of horses admitted to the New York State College of Veterinary Medicine between 1975 and 1987.[4] Quarter Horses and related stock breeds were almost twice as likely to develop sarcoid as Thoroughbreds. Standardbred horses were less than half as likely to develop sarcoid as Thoroughbred horses. A direct genetic linkage has been reported between equine leukocyte antigens (ELA) and risk for equine sarcoid. ELA alleles A3 and W13 were strongly associated with risk for sarcoids in Thoroughbreds and Swiss, French, and Irish Warmblood horses.[5-7] The absence of the W13 allele in the Standardbred population may confer relative resistance to sarcoid. Interestingly, a familial predisposition has been reported in a herd in which all five descendants of a sarcoid-affected mare developed sarcoid, whereas none of the unrelated animals on the premises developed these tumors.[8]

Spontaneous malignancies that develop as a result of serial genetic mutations are more common in older animals. Sarcoids generally develop in younger individuals, suggesting a genetic

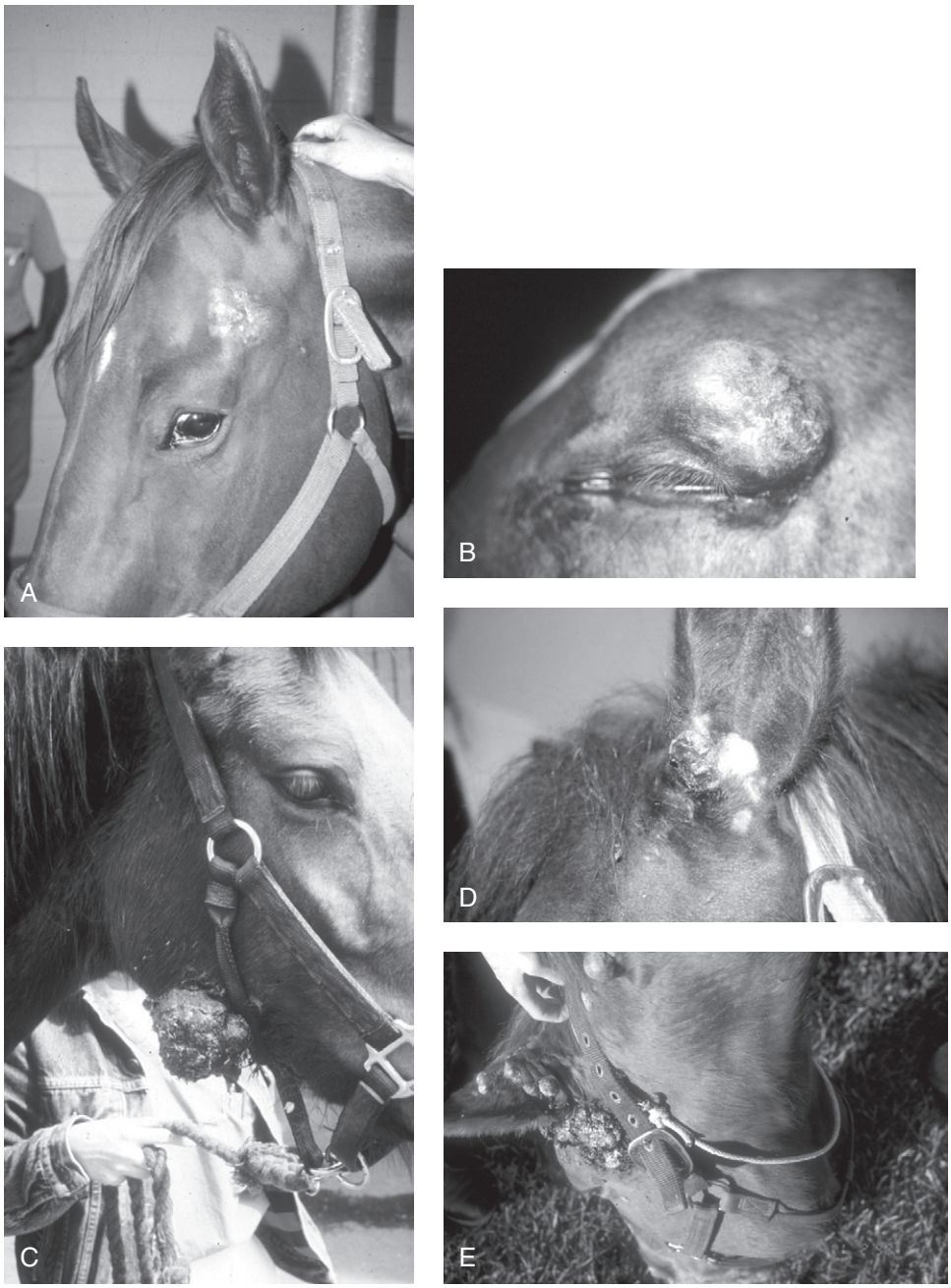

Figure 29-1. Subtypes of sarcoids. **A,** A verrucous sarcoid on the temporal region of the head. **B,** A subcutaneous fibroblastic sarcoid. **C,** An ulcerative fibroblastic sarcoid. **D,** A mixed form consisting of both verrucous and fibroblastic components. **E,** A horse with a malevolent sarcoid. The lesion had spread down the neck along lymphatic chains and to the retropharyngeal and parotid lymph nodes.

predisposition or an exogenous factor. Individuals with a genetic predisposition have an inherited gene defect that increases their susceptibility to further mutation. Consequently, these individuals develop tumors at a much earlier age. The genetic predisposition for and the age of onset of sarcoids suggest that both genetic factors and exogenous factors may play a role in development. A long-term epidemiologic study of a large herd of donkeys suggests the possibility of transmission of sarcoids between individuals housed in close contact.[9] In addition, an epizootic outbreak was reported in a small herd.[8] Incidence peaked in young horses over a 1-year period. Interestingly, only related, highly inbred animals were affected. A vector such as a fly may explain the predisposition for tumors to develop around the eye, distal limbs, and ventrum (common feeding locations for these insects).

A specific viral etiology was first proposed by Olsen and Cook.[10] While studying the transmissibility of cutaneous papillomas, they noted a striking similarity between equine sarcoid and experimental lesions induced by the injection of bovine wart extracts into the skin of individual horses. Several research groups have since confirmed the presence of bovine papillomavirus (BPV) DNA in nearly all equine sarcoid tissues examined.[11-20] Papillomaviruses play a role in oncogenic transformation in other species, but viral infection alone is not enough to trigger

transformation, as BPV DNA can be found in normal skin of sarcoid-affected horses.[20-22] Papillomavirus infection of an abnormal host (such as BPV infection in a horse) generally results in a nonproductive infection; consequently, no viral capsids are produced. Instead, the virus maintains its presence by production of several housekeeping genes, including two, *E5* and *E6*, known to be capable of transforming cells to malignancy. The production of one of these two transforming genes, *E5*, has been documented in a small number of sarcoids examined.[23] Expression of the viral E5 protein appeared to correlate with the grade of tumor malignancy. Furthermore, although viral DNA was detected in normal skin surrounding sarcoids, E5 protein expression was not. A triggering event such as trauma may be required to activate a latent viral state. This could be one explanation for the tendency for sarcoids to occur at the sites of previous injuries. Experimental inoculation of purified viral DNA has resulted in sarcoid-like tumors, but the majority of these lesions regress spontaneously.[24]

A viral role is strongly suggested by the finding of viral DNA and viral transforming proteins in all sarcoids examined. However, the mechanism of viral transformation is unclear. Given the genetic and breed predispositions, sarcoid occurrence is most likely the result of host, viral, and environmental factors.

Treatment

There are numerous articles regarding treatment of sarcoids. The number of treatment options reflects the variable success as well as the costs of certain treatments. Efficacy is difficult to critically assess; most early studies were not controlled and were based on a subset of cases sent to referral hospitals. Sarcoids commonly referred for treatment are usually more aggressive variants or tumors that have recurred after unsuccessful treatment. Sarcoids can transform to a more aggressive phenotype after incomplete or unsuccessful treatment and are then harder to resolve. In contrast to those tumors typically seen at referral centers, there is anecdotal evidence of a subset of sarcoids that remain quiescent and of others that undergo spontaneous regression.

Surgical Excision

Surgical excision without adjunctive therapy has been reported to be one of the least successful treatment options, having a recurrence rate of 15.8% to 82%.[3,25-27] The reason for this may in part be that tumor projects fronds of malignant cells into the surrounding "normal tissue."[28] Because these projections are infrequent, they are missed on histopathologic review, leading to an incorrect assumption of complete excision. In addition, the possibility exists that a viral agent may remain latent in the surrounding tissue.[20,21] Surgical trauma and the subsequent growth stimulation may trigger viral activation and expression of viral oncogenes. In a recent prospective trial, the detection of viral DNA at the surgical margin of an excised sarcoid correlated with an increased risk of recurrence.[21]

Laser Ablation

Laser ablation of tumors has been used to remove or debulk tumor mass.[25,29-32] Laser ablation causes less damage to the surrounding tissues and less spread of malignant cells to healthy regions than sharp surgical excision with mechanical tools. A large retrospective study evaluating CO_2 laser excision of 60 sarcoids documented an overall recurrence rate of 38%.[30] The use of laser ablation in conjunction with intralesional chemotherapy or other adjunctive therapy may improve overall success rates.

Cryotherapy

Cryotherapy (see Chapter 14) has historically been one of the most commonly used methods for treatment of equine sarcoid.[33-35] Success rates of 60% to 100% have been reported. However, a recent retrospective study evaluating treatment success in a large number of periorbital sarcoids reported a recurrence rate of 91% (9% success rate) in tumors treated by cryotherapy.[27] Longer follow-up time may explain the discrepancy in successful outcome. Cryotherapy has its greatest success with veterinarians who use it frequently and have experience with duration and depth of freeze, which are important for an effective response. Thermocouples can be used for more precise measurement of the tissue temperature and depth of freeze. In one report, the use of a three-treatment −30° C protocol was felt to be more effective than a two-treatment −20° C protocol, with an overall initial success rate of 68%.[33] With multiple repeat treatments, the success rate improved to 85% (85 tumors resolved of 100 retreated). Thirty-three of the 50 horses were cured of their sarcoids using a liquid nitrogen spray apparatus, although many required multiple treatments.[34] Three horses in this study had spontaneous regression of untreated tumors after cryotherapy of another lesion. This seems to indicate that destruction of one tumor can result in an immune response against other, distant sarcoids.

Hyperthermia

Hyperthermia has been reported to be successful in a small number of equine sarcoids, with spontaneous resolution of some of the nontreated tumors.[36] Differences in the metabolic rate of tumor cells compared with normal cells is thought to make the tumor cell more heat sensitive.

Radiotherapy

Radiotherapy using locally implanted iridium-192 has been reported to be successful in the treatment of sarcoids.[27,37-39] The recurrence rate was low, with 1-year tumor-free incidences of 94%, 87.5%, and 100%. Radiotherapy using a linear source of iridium-192 has been reported to have a 100% success rate in a small number of sarcoids.[40] All cases were reported to resolve after a single dose of radiation. Recurrence rate at 1 year was zero for all tumor types, but the number of cases actually followed for a full year was not reported. The 1-year progression-free survival was 86.6% in 62 sarcoids treated with iridium interstitial brachytherapy.[41] Treatment complications included hair and pigment loss as well as fibrosis, cataract formation, and corneal ulceration when treating periocular tumors. Radioactive gold-198 implants have been reported to have a high success rate for treatment of sarcoids—a rate similar to that for the other implant therapies.[42] Although reported success rates using radiation therapy are high, treatment is expensive, requires special equipment and housing, and is potentially hazardous. General anesthesia may be required to implant iridium needles or to deliver a radiation dose via a linear source. These limitations

make radiation therapy a difficult and infrequently used method of treatment and confine its use to referral centers.

Immunotherapy

Immunotherapy has been used to successfully treat sarcoids in several reports.[27,35,43-45] Several immunostimulants have been used, including *Mycobacterium* cell wall extracts, live whole-cell bacille Calmette-Guérin (BCG), and propionibacterial cell wall extracts. These products are thought to stimulate cell-mediated immunity, leading to recognition of tumor cell–specific antigens and subsequently to tumor cell destruction. Most immune stimulants require multiple intralesional injections with or without prior cytoreduction of the tumor mass. Success rates are high for smaller tumors, tumors located periorbitally, and fibroblastic tumors. Knottenbelt and Kelly reported a poorer success rate with occult or verrucous periorbital lesions and hypothesized that this was because of the inability to saturate the tumor bed effectively.[27] Multiple treatments are generally required. Local tissue swelling can be quite severe, and complications include death from anaphylactic shock, particularly after two or more injections.[45] Spontaneous regression of untreated tumors has also been reported with immunotherapy.[35] Recurrence rates vary from 0% to 40%, depending on the study and observation time.[27,35,43-45]

More specific immunotherapy in the form of *vaccination* has been used to treat equine sarcoids for decades with variable success. More recently, a vaccine consisting of tumor tissue was successfully used to treat 20 of 21 horses presenting with equine sarcoid, though multiple treatments were required in some cases.[46] Vaccination of sarcoid affected donkeys using a chimeric virus-like particle that expressed viral proteins had limited success.[47]

Intralesional Cisplatin

Intralesional cisplatin in an oily emulsion is reported to have an 87% 1-year relapse-free rate for sarcoids.[48] The repositol effect of the oily emulsion prevents significant systemic levels, avoiding systemic toxicity, and maintains effective tissue levels of the chemotherapeutic for prolonged periods of time.[48] Epinephrine can be added to the emulsion (1 : 1000, diluted 1 : 10). The resultant vasoconstriction prolongs tumor cell exposure to cisplatin. A dosage of 1 mg cisplatin per cubic centimeter of tumor mass, with a minimum of four treatments, is recommended. Cytoreduction to decrease the mass of the tumor is beneficial when treating large tumors. Intralesional cisplatin instituted at the time of surgical excision or debulking was not shown to affect wound healing.[49] The repositol effect of the oily emulsion is critical for treatment success. A 66% recurrence rate after cisplatin treatment was reported by Knottenbelt.[27] However, the concentration used was lower than that recommended by the original authors.[48]

Intralesional tumor necrosis factor (TNF) combined with a xanthate compound has also been used with success.[50]

Topical Application of Chemotherapeutics

Topical application of chemotherapeutic agents, including 5-fluorouracil (5-FU) and a series of compounded creams, AW3-LUDES and AW4-LUDES, have been reported to be successful in the resolution of sarcoids.[27,51,52] 5-FU had a resolution rate of 66% in 9 tumors treated daily for 15 days, but only small occult or verrucous sarcoids were chosen for this treatment protocol.[27] In the same report, the AW4-LUDES cream had a resolution rate of 35% (56 of 146 tumors treated) without complication. The authors reserved this treatment for small, previously untreated verrucous lesions. Six additional tumors resolved with the topical application of AW4-LUDES but had significant scarring after treatment. As with 5-FU, the authors recommend avoiding the use of these creams on tumors in close proximity to the eye. Topical application of a compounded cream containing bloodroot and zinc is anecdotally reported to be extremely effective in the treatment of equine sarcoid, but few controlled studies have been done. I evaluated one sarcoid-affected horse that was treated with this compound; in response to the compound, the tumor became very swollen and ulcerated and began to grow rapidly. More controlled studies evaluating the efficacy of bloodroot and zinc against different types of sarcoid tumors are needed.

Spontaneous regression of untreated tumors suggests that immune recognition plays a role in tumor resolution.[36] Anecdotal reports of successful vaccination therapy exist, but controlled studies are rare. In one report, successful resolution was achieved in 11 of 12 horses treated with an autogenous tumor vaccine.[53] A second vaccination protocol resulted in resolution of the one recurrence. All 12 cases had had no previous tumor treatment. A lower success rate was reported in horses with previously treated, recurrent sarcoids.

With large, aggressive, or multiple tumors, it is recommended that a combination of treatment modalities be employed. At the Veterinary Teaching Hospital at Michigan State University, surgical excision or laser ablation is commonly combined with intralesional chemotherapy or radiation therapy. Unfortunately, these combination treatment regimens are expensive and require a significant time commitment from the owner or caretaker of the patient.

SQUAMOUS CELL CARCINOMA

Epidemiology

Squamous cell carcinoma (SCC), a malignant, locally invasive neoplasia of squamous epithelial cells, is the second most common tumor in horses.[54,55] Although SCC can develop anywhere on the integument, sites of predilection include areas lacking pigmentation, poorly haired regions, and skin near mucocutaneous junctions. SCC is the most common neoplasm of the equine eye, conjunctiva, ocular adnexal structures, and external genitalia.[56] It has also been reported in the nasal cavity, paranasal sinuses, pharynx, larynx, and hoof capsule, and it should be considered in horses with chronic, refractory foot abscesses.[57-60] Ultraviolet light–induced damage is thought to predispose to SCC. Many tumors arise from precancerous lesions, including actinic keratosis, carcinoma *in situ*, squamous metaplasia of the penile epithelium, and irritant-induced chronic keratitis.[61,62] Proposed irritants include topically applied chemicals, smegma, and flies. SCC has been reported to develop in areas of chronic, poorly healing wounds and at sites of previous burn injury.[56,63,64] Breeds with poorly pigmented, pink-skinned areas, including Appaloosas and paint-colored horses, are more prone to develop SCC; 69% of all ocular SCC cases occur in individuals lacking periocular pigmentation.[65]It has been suggested that draft breeds have an increased incidence of SCC.[60]

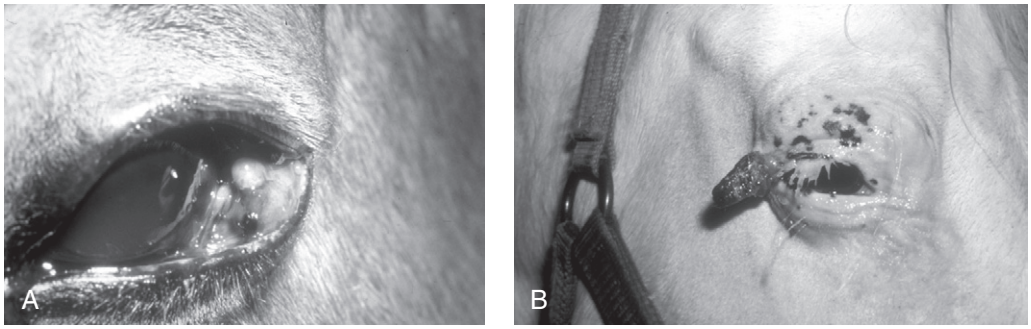

Figure 29-2. Squamous cell carcinoma. **A,** Ulcerative, proliferative lesion involving the conjunctiva of the eye in a bay Quarter Horse mare. **B,** Pedunculated lesion involving the margin of the upper eyelid in an Appaloosa mare.

Tumors are classified as ulcerative or proliferative and generally arise as solitary lesions. Ulcerative SCC may develop over time, with early lesions appearing as small nodules underlying normal-haired skin. Ulcerative lesions can be mistaken for nonhealing wounds and chronic granulation tissue, resulting in delayed treatment. Ocular lesions can begin as small ulcerative lesions on lid margins, or as keratitic plaques on the cornea, so SCC should be suspected whenever raised red lesions appear on the lid margins, sclera, or conjunctiva, particularly in unpigmented skin (Figure 29-2). Squamous cell carcinoma of the ocular structures can invade the orbit, calvarium, tear duct, and sinuses if left untreated. Proliferative pedunculated lesions on the penis often have a cauliflower-like appearance. SCC typically spreads to surrounding tissues and local lymph nodes, but distant metastasis is rare (see Chapter 60). Tumors with local or distant metastases or large, invasive tumors have a poor prognosis for cure.

Treatment

Surgical excision, radiation therapy, topical application of antimitotics, intralesional chemotherapy, cryotherapy, laser excision, hyperthermia, immunotherapy, and photodynamic therapy have all been successfully used to treat equine SCC.[42,48,66-80] Surgical excision alone should be reserved for small tumors and has a better overall success rate when combined with adjunctive therapies such as chemotherapy, cryotherapy, or radiation therapy.[49,66,67] Keratoplasty with or without adjunctive radiation therapy has been used successfully to treat corneal SCC.[68,69] Cryotherapy is reported to be most useful in small periocular lesions and lesions arising from the external genitalia. Larger lesions may benefit from surgical debulking prior to cryotherapy.[70,71] Frequent reexamination is recommended to monitor regrowth and may be the most important determinant of success. Failure to retreat small recurrences quickly may allow tumor regrowth and failure of the treatment protocol.

Irradiation

Several radiation modalities have been used to successfully treat SCC, including external beam radiotherapy, implanted radiation devices, and b-emitting wands. Strontium wands (b-radiation) can be used to treat small superficial plaques on the cornea, sclera, or conjunctiva.[72-74] Beta irradiation has a very short penetration depth, making it extremely useful for small corneal lesions; an 89% nonrecurrence rate was reported in one study.[73] Both radioactive implants and external beam therapy have been reported as successful in the treatment of SCC.[42,75-78] External beam therapy can be performed on an outpatient basis, whereas radiation implants require hospitalization and special housing during treatment protocols. Radiation therapy is generally limited to referral practices because of the licensure requirement, specialized equipment, and housing requirements.

Intralesional Chemotherapy

Intralesional chemotherapy is very effective in treating SCC, whether as an adjunctive treatment after surgical cytoreduction or as a sole treatment protocol.[48,49] Cisplatin binds directly to DNA and inhibits its synthesis in dividing cells. Mixing cisplatin in an oily emulsion creates a repositol effect, maintaining the drug in the local tissues for a prolonged period of time. Intralesional cisplatin in oily emulsion used with or without surgical cytoreduction has a 2-year local control rate of 89% for SCC. Overall success is better with SCC of the external genitalia than with periorbital SCC. Cosmetic results are generally excellent. Drawbacks include the multiple treatments needed, the expense, and potential exposure of the staff, but overall success rates are high.

Topical Chemotherapy

Topical 5-FU, with or without surgical débridement, has a reported success rate of 90% in the treatment of SCC of the male external genitalia.[78] In another small case series, three individuals with superficial SCC were successfully treated with repeated application of topical 5-FU.[79] Topical application was associated with local inflammation and swelling. The use of 5-FU as a sole treatment modality is best reserved for small superficial tumors.

Photodynamic therapy using a diode laser was used as an adjunct therapy in 9 horses (10 eyes) with periocular SCC following surgical debulking. Horses were tumor free for the duration of the study (25 to 698 months of follow-up).[80]

Successful treatment of a recurrent SCC of the lower lip using the nonsteroidal anti-inflammatory piroxicam is reported.[81] Nonsteroidal anti-inflammatory drugs inhibit cyclooxygenase (COX) enzymes that catalyze the conversion of arachidonic acid to prostaglandins. Overexpression of the COX-2 enzyme by neoplastic tissue has been reported.[82] Both the lower lip and metastatic lesions resolved with oral piroxicam therapy. No recurrence was reported at the 5-year follow-up. Successful treatment of periocular SCC using immunotherapy has also been reported, but case numbers are small.[83]

In a retrospective study of 43 horses with ocular SCC, nonrecurrence rates were 55.6% for surgical excision, 75% for radiation therapy, and 33.4% for cryotherapy, and combination therapy had a greater success rate than single-modality treatment.[66] In a retrospective study of 147 cases of ocular/adnexal SCC, factors that influenced survival included tumor location and size. Prior treatment modalities, the presence of multiple tumors, and treatment modality used at the time of examination did not influence survival.[84]

MELANOMA

Epidemiology

Equine melanoma is one of the most common skin tumors in horses. Histopathologic surveys report that melanomas account for 4% to 15% of all skin tumors.[62,85] The majority of melanomas occur in gray horses; breed predispositions are most likely caused by the incidence of the color gray within a breed. Virtually all gray horses will develop melanoma over time. In a population study of Lipizzan horses, less than 6% of horses 16 years of age or older were melanoma free.[86] Melanocytic tumors in gray horses are thought to arise because of a disturbance in melanin transfer from dermal melanocytes to follicular cells.[87] Progression from melanocyte accumulation to melanoma formation has been documented in melanoma-prone locations. Interestingly, sites of predilection are the first areas to show depigmentation changes (vitiligo) with aging.[88] There are four types of equine melanomas: melanocytic nevi, dermal melanomas, dermal melanomatosis, and malignant melanomas.[89] *Melanocytic nevi* are composed of larger pleomorphic melanocytes with an increased number of mitotic figures, binucleate cells, and variable cytoplasmic pigmentation. Melanocytic nevi are typically single or multiple discrete nodules and are found in similar frequency in both gray and nongray individuals.[89]

Dermal melanoma and melanomatosis appear benign on histopathologic examination and are composed of smaller, homogenous dendritic cells with condensed chromatin, dense pigmentation, and no visible mitosis. *Dermal melanomas* originate in the deeper dermis and are typically small singular or multiple nodules (Figure 29-3). *Dermal melanomatosis* is defined as confluent multiple large melanomas. The risk of dermal melanomatosis increases with age and its occurrence is associated with an increased risk of metastases.[86,89] True *malignant melanomas* are rare and are classified on the basis of the presence of both histopathologic and clinical characteristics of malignancy.[89] In one retrospective study, only 2 of 53 cases were classified as malignant. Both occurred in aged horses (older than 20 years), and both recurred within 10 months after surgical excision.[89] Malignant melanomas are frequently invasive and associated with a poor prognosis for complete resolution.[89-94]

Figure 29-3. Dermal melanomas on the ventral surface of the tail in a gray horse.

The majority of melanomas are located around the perineum and base of the tail, with lesions around the head (lips, eyes, parotid region) and other sites less frequently reported. Melanomas have also been reported in the foot, meninges, thorax, ocular structures, and abdominal cavity. These unusual locations appear to be associated with a poor prognosis.[89-96]

Treatment

Despite their histopathologic differences, dermal melanomas and melanocytic nevi have similar clinical characteristics, and surgical excision is curative in the majority of cases. Smaller lesions can be sharply excised; alternatively, the use of the CO_2 laser has been recommended.[97] The ability to cauterize the wound bed and control hemorrhage makes laser excision particularly useful in sites where primary closure cannot be achieved (e.g., the base of the tail).

Complete excision is difficult with larger lesions such as dermal melanomatosis, but surgical debulking can be palliative.[98] Oral cimetidine has been recommended to treat melanomas, particularly larger lesions and those exhibiting a rapid growth phase.[99-103] Cimetidine has antitumor activity and is a histamine receptor antagonist. Immunomodulation of lymphocyte activity via histamine receptor interaction is postulated to be a mechanism of antitumor activity. Reports of the effectiveness of cimetidine in the treatment of equine melanoma are variable. Response is reported to be the highest in tumors exhibiting a rapid growth phase; successful response was achieved using a dose of 2.5 mg/kg every 8 hours. The frequency of dosing may be the most critical factor in predicting clinical response. Treatment is recommended for 3 months, or for 3 weeks after cessation of tumor growth. Although a complete cure has rarely been reported, cimetidine may have some benefit in halting rapidly growing tumors and returning them to a more quiescent state.[98]

Additional treatments for equine melanoma include intralesional chemotherapy and cryotherapy. Successful treatment of smaller nodules has been reported using intralesional cisplatin, and this is beneficial in decreasing the size of larger tumors prior to surgical excision.[103,104]

A melanoma vaccine is available and has been reported to have some success.[105] Successful treatment of malignant melanoma using a cytokine-enhanced suicide gene therapy was recently reported. Repetitive treatments were performed and resolution of both treated and untreated tumors was reported with a tumor-free follow-up of 33 months.[106] The successful treatment of equine melanoma using intratumoral injections of human interleukin (IL)-12 has also been reported.[107]

In one study that examined melanomas in a population of gray horses, all patients exhibited normal quality of life regardless of tumor number and type.[86] Consequently, it is frequently suggested that melanomas be treated with benign neglect, because they are rarely the cause of significant disease in affected horses. However, with age, the risk of progression to dermal melanomatosis and metastases increases. Small nodules are

easily removed and rarely recur, but owners need to be informed that new tumors will quite likely develop over time. Although conservative management is reasonable in the majority of cases, more aggressive treatment, including early removal of smaller tumors, may decrease the risk of melanomatosis or metastases as the animal ages.

Malignant melanomas have a higher recurrence rate with simple excision. Combination therapy, including surgical debulking and intralesional chemotherapeutic injections, may offer palliation, but the prognosis for cure is poor.[88,92]

MAST CELL TUMORS

Epidemiology

Equine cutaneous mastocytosis (ECM) is less common in horses than in small animals.[108] The benign, solitary, nodular cutaneous form is the most common, although malignant ECM and congenital disseminated forms have been recognized.[109-111]It has been suggested that the benign nodular form is not a true neoplasia and is instead the result of an inflammatory reaction to dysplastic mast cells and recruited eosinophils.[112] The release of inflammatory products from accumulated eosinophils and mast cells results in necrosis, and later a granulomatous reaction develops with fibrosis and subsequent encapsulation of the nodule. The histopathologic findings of a necrotic focus, a granulomatous inflammatory response, and fibrosis surrounding well-differentiated mast cells differentiates equine cutaneous nodular mastocytosis from malignant ECM, which contains abnormal mast cells with increased nuclear-to-cytoplasmic ratios, anisokaryosis, and increased mitotic figures.[110]

Treatment

The majority of cutaneous nodular ECMs are successfully treated with wide surgical excision (margins of 1 cm or greater).[108] Reports of resolution after incomplete resection exist. Intralesional injection of glucocorticoids has also been used to successfully treat cutaneous nodular ECM. Malignant ECM is not responsive to surgical excision and is associated with a poor prognosis for cure.

LIPOMAS

Epidemiology

External lipomas are a relatively uncommon neoplasia in horses. Unlike mesenteric lipomas, they are principally found in young horses.[113-115] Subcutaneous lipomas are generally encapsulated, singular, nonpainful masses that are fluctuant to firm on palpation, although infiltrative lipomas have been reported in the horse.[113-115] Sites of occurrence include the limbs, thorax, abdominal wall, and eyelids. Histopathologic examination reveals encapsulated well-differentiated adipocytes. Infiltrative lipomas consist of similar cell types infiltrating surrounding muscle tissue.[116]

Treatment

Lipomas are generally encapsulated, slow-growing masses, and thus they are benign, clinically insignificant, and typically a cosmetic concern only. Surgical excision is reported to be curative in all cases; recurrence has not been reported even with incomplete excision of infiltrative or large lipomas.

CUTANEOUS HABRONEMIASIS

Epidemiology

Cutaneous habronemiasis is a proliferative ulcerative lesion produced by aberrant migration of the larvae of three endoparasites *(Habronema muscae, Habronema microstoma*, and *Draschia megastoma)* that inhabit the stomach of horses.[117] Eggs, shed in the feces of horses, hatch, and the larvae are ingested by maggots of the house fly, *Musca domestica*, or the stable fly, *Stomoxys calcitrans*. Third-stage infective larvae migrate to the head of the fly and are passed to the horse when the fly feeds on a warm, wet surface. The larvae are then swallowed by the horse and mature to adults in the stomach. The cutaneous form, also called summer sores, develops when larvae are attached to wound beds or aberrant moist surfaces, including the penis, prepuce, and ocular adnexa.[118,119] Lesions are most commonly seen on the penis, in cutaneous wounds, or around the eye. The proliferative, ulcerative, granulomatous lesions are thought to result from a hypersensitivity reaction to the larvae, and mild to severe pruritus may develop.[120]

Ophthalmic habronemiasis can develop in the conjunctiva or nasolacrimal ducts, resulting in third-eyelid granulomas or granulating ulcers below the medial canthus. Clinical signs include epiphora, chemosis, and photophobia. The presence of pale yellow granular material, or the finding of larvae on impression smears, may aid in the diagnosis. Histopathologic examination is recommended, because habronemiasis can develop secondary to an underlying neoplastic or infectious condition. The incidence of disease has declined dramatically since the development of ivermectin anthelmintics. Lesions occur principally in warm months and are associated with increased fly populations and poor manure collection.

Treatment

Lesions have been reported to resolve with onset of cooler weather.[119] Treatment is aimed at reducing the size of the lesions, resolving the inflammatory or allergic component, and preventing reinfestation.[119] Treatment with systemic ivermectin will kill migrating larvae but may not result in resolution of the lesion, because the dead parasite can continue to cause a severe inflammatory reaction. In some cases, oral ivermectin results in an increase in pruritus and self-trauma. Larger masses may require surgical cytoreduction. The intralesional injection of corticosteroid is recommended to decrease the allergic or inflammatory reaction to the larvae. Successful resolution using systemic corticosteroids alone has been reported.[120] Re-infestation can be prevented by application of topical organophosphates or by placing the wound under a bandage. Topical preparations that combine organophosphates, corticosteroids, dimethyl sulfoxide, and nitrofurazone have been used.[108,119] The ophthalmic form of habronemiasis can be treated with oral ivermectin, topical corticosteroid eye drops, and curettage. Careful assessment of the corneal integrity should be made prior to the use of ophthalmic corticosteroids.

PYTHIOSIS

Epidemiology

Pythiosis is a cutaneous disease caused by invasion of the organism *Pythium insidiosum*, a fungus-like oomycete.[121] The disease occurs principally in warm, tropical regions; the organism has

an aquatic life cycle and requires relatively warm temperatures for reproduction.[121] Pythiosis is commonly seen in the southern United States during the late summer and early fall. Horses are infected when invasion occurs through small wounds or skin breaks, typically in lakes, swamps, or flooded lands. Zoospores, released during reproduction, are attracted to organic debris and invade open wounds.[121] Characteristic lesions are ulcerative masses of granulation tissue. Rapid enlargement can occur, even within a matter of days. Pythiosis is often pruritic; sinus tracts are visible and often contain gritty, coral-like masses, called "kunker" or "leeches."[122] Kunker are made of necrotic vessels, inflammatory cells, and *Pythium hyphae.* Pythiosis occurs most commonly on the distal extremities and ventral body wall.[123] In chronic, untreated infections, invasion of deeper tissues, including bone, joints, tendon sheaths, and lymph nodes, can occur.[123-126] The diagnosis of pythiosis is made on the basis of the history and clinical signs, when the causative organism is found on culture or histopathologic examination.

Treatment

Surgical excision is the treatment of choice for pythiosis. If surgical excision is impossible because of the size or location of the lesion, the prognosis for resolution is poor. Systemic, topical, and intralesional antifungals have been used, but recurrence is common.[126]

Amphotericin B is administered intravenously at a dosage of 0.3 mg/kg diluted in 1 L of 5% dextrose.[127] The dose is increased by 0.1 mg/kg every third day until a maximal dosage of 0.8 to 0.9 mg/kg is achieved. Treatment can be continued daily for 30 days and then every other day until the horse is cured. Sodium iodide administered intravenously has been recommended as an adjunctive therapy.[126] A recent report showed improved *in vitro* susceptibility of 17 equine isolates using an antifungal in combination with terbinafine compared to antifungal agents alone.[128] Further work is needed to assess efficacy *in vivo*.

In addition, a topical dressing containing 50 mg amphotericin B, 10 mL sterile water, and 10 mL dimethyl sulfoxide is recommended.[125] Because amphotericin B is nephrotoxic, serum creatinine, urea nitrogen, hydration status, water consumption, and urine output should be closely monitored. Reported side effects of amphotericin B include depression, anorexia, pyrexia, and urticaria.

Immunotherapy using a vaccine made from fungal cultures is reportedly curative if administered early in the course of the disease.[129] In a study of 40 horses with pythiosis, 53% were cured with vaccination alone. Surgical cytoreduction combined with vaccination improved success rates if vaccination was performed within 2 weeks of surgical débridement.[129]

NODULAR NECROBIOSIS

Epidemiology

Nodular necrobiosis, also called collagenolytic granulomas or eosinophilic granulomas with collagen degeneration, is reported to be one of the most common skin diseases in horses.[130] The underlying etiology is unclear; insect hypersensitivity has been proposed, and the propensity for lesions to develop at pressure points (under the saddle region, in particular) suggests trauma may play a role.[131] Diagnosis is based on histopathologic findings of a granulomatous reaction containing eosinophils, lymphocytes, and histiocytes, with collagen degeneration.[132] Typical lesions are 0.5- to 1.0-cm subcutaneous nodules and are most commonly found on the withers and dorsum but can occur anywhere on the body. The differential diagnosis includes equine amyloidosis, foreign body granuloma, dermoid cysts, habronemiasis, equine sarcoid, and other neoplasia.

Treatment

Single asymptomatic lesions that are not a cosmetic concern can be left untreated. Single lesions can be surgically excised. Alternatively, intralesional injection of glucocorticoids such as triamcinolone acetonide (3 to 5 mg per lesion) or methylprednisolone acetate (5 to 10 mg per lesion) can be performed.[130] Lesions that are calcified can be difficult to inject and may require surgical excision.[108] Systemic corticosteroids have been recommended to treat multiple lesions—for example, prednisolone at 1 mg/kg orally, daily for 14 days, followed by 0.5 mg/kg orally for an additional 14 days. Lesions may recur and require multiple retreatments for resolution.

PAPILLOMATOSIS

Epidemiology

Papillomaviruses are very host- and tissue-specific and are the causative agents of the majority of warts seen in mammals. Equine papillomatosis and aural plaques are caused by equine papillomaviruses (EqPV).[133] Papillomaviruses infect the basal layer of the epidermis, resulting in an abnormal proliferation and hyperkeratosis of the epithelium. Clinical syndromes include aural plaques and juvenile papillomas (juvenile warts). Congenital neonatal papillomatosis was thought to be the result of *in utero* infection by papillomavirus; however, reports suggest that congenital papillomatosis is in reality a hamartomatous lesion and not a virus-induced growth.[134]Affected foals are born with a large wartlike lesion located anywhere on the integument.

Juvenile Papillomatosis

Juvenile papillomatosis most commonly occurs in young horses (6 months to 4 years), but older horses can be affected. Multiple small (5-mm) gray-pink, vegetative, cauliflower-like warts usually develop on the lips, muzzle, face, distal limbs, and external genitalia. Lesions can be spread by direct contact, contamination of the local environment, and vectors. Large numbers of infectious virus are shed in the superficial keratinocytes.

Aural Plaques

Aural plaques are raised, white or tan, smooth plaques arising on the internal surface of the pinnae (Figure 29-4). Fly irritation may exacerbate these lesions, making them crack and causing the horse discomfort. Equine papillomavirus DNA has been identified in both aural plaques and juvenile papillomas, but different viral subtypes are suspected.

Treatment

Congenital papillomas can be removed surgically or ligated and allowed to undergo necrosis. In the majority of cases, juvenile warts will spontaneously regress when immune recognition occurs. Immunologic compromise may result in failure to

regress; in these cases, surgical excision may be necessary. Vaccination using wart tissue has been recommended.[135] Alternative treatments include cryotherapy, intralesional chemotherapy, and radiofrequency hyperthermia.

Though historically treatment protocols for aural plaques have been unrewarding, the recently described use of topical imiquimod 5% cream resulted in resolution of aural plaques in 87.5% of horses. Duration of topical therapy was 1½ to 8 months.[136] In addition, successful resolution using laser ablation has been reported. The lesions are manually débrided with a dry gauze pad to remove keratinous debris, and then the lesions are lightly painted with the laser probe. Recurrence has not been reported.[137]

DENTIGEROUS CYSTS

Etiology

A dentigerous cyst is a congenital defect. It arises as a result of incomplete closure of the first branchial cleft during embryologic development. By definition, dentigerous cysts contain dental elements, such as enamel, dentin, and cementum.[130] The epidermal lining differentiates as does normal epidermis, and the cyst becomes nodular as it begins to fill with keratin. Lesions are usually asymptomatic. They may be present from birth, but they remain unnoticed until they enlarge.[138,139] They typically appear as a unilateral swelling at the base of the ear but can occur in other locations on the head and sinuses.[138-141] The secretory, cystic lining produces a mucoid fluid that may drain intermittently from the nodule. Diagnosis is frequently made on the basis of location, clinical history, and the presence of pale yellow, mucoid, cystic fluid.[141] Definitive diagnosis requires surgical excision and histopathologic examination.

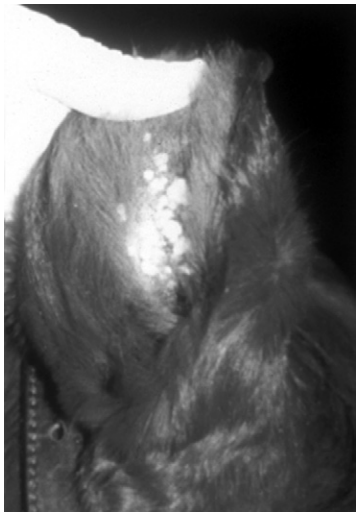

Figure 29-4. Typical appearance of aural plaques in the pinna.

Treatment

No treatment is required. However, if desired, surgical excision with complete extirpation of the cyst lining is necessary to prevent recurrence.[142] Preoperative radiographic evaluation is recommended to determine the location of dental material and the number of cysts, and prior to anesthesia, the extent of the cyst should be defined by careful palpation. The preparation and surgical scrub should include the pinnae to allow manipulation during surgery. The sinus tract, which is identified using a malleable probe (Figure 29-5), is opened and the neck of the cyst is sealed with a ligature prior to dissection. The plane of dissection should be as close to the wall of the cyst as possible to prevent damage to the auriculopalpebral nerve and auricular muscles, as well as to minimize hemorrhage. Distention of the cyst is aids in its identification and removal. The auricular muscles are small and can be difficult to visualize because of the aberrant connective tissue associated with the dentigerous cyst. Opening the cyst should be avoided at all cost; spillage can result in contamination of the incision site and result in infection, dehiscence, and excessive scarring. The dental tissue can vary in size from a small and easily removable tooth to large areas of enamel-covered bone. Careful excision using a chisel or drill is required in such cases to prevent damage to the cranium and brain.[140] Failure to remove all the dental tissue may result in dehiscence and refistulization. Auricular muscles and fascial

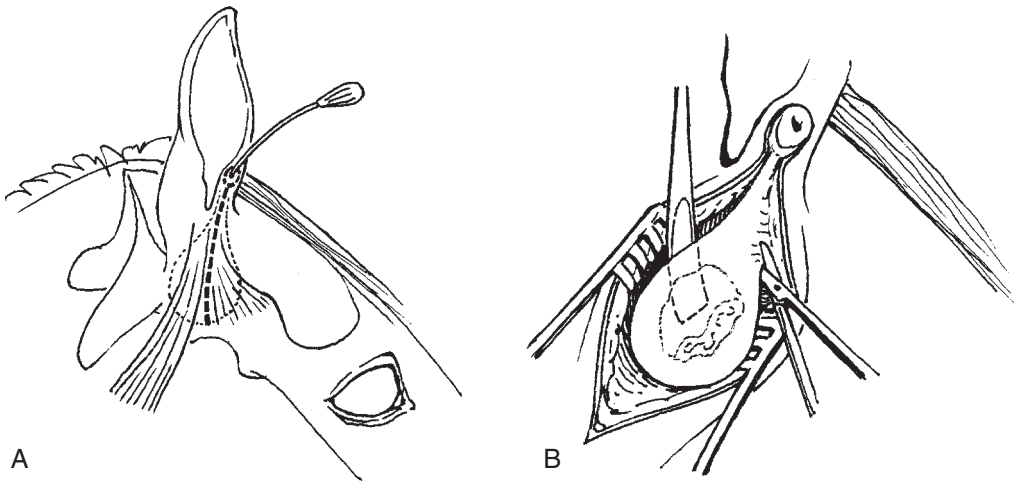

Figure 29-5. Dentigerous cyst. **A,** Identification of the sinus tract is made easier with a malleable probe inserted into the cloaca until it touches the dental material. **B,** After closure of the sinus tract, the cyst is removed intact. The dental material may need to be loosened with an elevator or an osteotome to complete cyst removal.

layers are apposed with 3-0 polydioxanone, and skin closure is routine. Postoperative antibiotics are not routinely indicated, although anti-inflammatory drugs are recommended, particularly when forceful extraction of dental tissue was required. In one report, an ectopic tooth was found compressing the cerebellum.[140]

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John A. Stick

CHAPTER
30

Oral Cavity and Salivary Glands

Padraic M. Dixon and Mathew P. Gerard

DENTISTRY

After almost a century of inactivity, equine dentistry has made rapid progress over the past 15 years, and equine dental disorders are now documented as one of the most common diseases recognized by U.S. practitioners.[1] Additionally, up to 10% of British equine practitioners' time is spent dealing with dental-related disorders.[2] This part of the chapter will concentrate on the many surgical aspects of this expanding field of practice, with other areas of equine dentistry adequately covered elsewhere. The modified Triadan system of dental nomenclature (Figure 30-1) is used in this chapter.

Examination of the Oral Cavity and Teeth

The equine incisors and canine teeth are readily examined in the unsedated horse. With the mouth closed, the rostral aspect of the mandible should be pushed sideways and the distance of lateral movement of the lower incisors in relation to their upper counterparts (e.g., 1 cm) before separation of the incisors (as the angled occlusal surfaces of opposing cheek teeth come into contact) can be measured. This measurement can be used to assess the angulation of the cheek tooth occlusal surfaces and can also detect the presence of major cheek tooth overgrowths

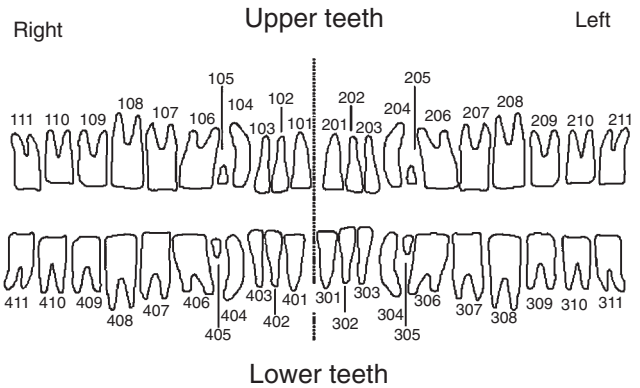

Figure 30-1. The modified Triadan system of equine dental nomenclature. To identify deciduous teeth, add 4 to the first number of its permanent successor. For example, the deciduous incisor 501 is replaced by permanent incisor 101.

that will suddenly block the lateral movement of the mandible.[3] This maneuver is easier to perform in sedated horses, as is examination of the oral cavity. There is great variation in the degree of separation of incisors that occurs during this maneuver in normal horses. However, some operators have arbitrarily determined that if a defined degree of incisor separation does not occur during the maneuver, it indicates the presence of overlong incisors. They then reduce the length of incisors until a normal degree of incisor separation occurs (i.e., "incisor bite alignment techniques"), sometimes exposing the incisor pulps during the reduction procedure. Because the cheek teeth have a surface area 10 to 15 times that of incisors and are composed of a harder type of enamel,[4] it is mechanically impossible for overlong incisors to keep the cheek teeth apart. However, after removal of large cheek tooth overgrowths, some horses may benefit from slight reduction of their incisors.

A combination of factors, including the limited angle of opening of the equine mouth (common to all herbivores), the rostral positioning of the lip commissures, and the length of the equine cheek teeth rows, make it very difficult to visually examine the cheek teeth, particularly in unsedated horses. Palpation through the cheeks may reveal food pocketing or major dental irregularities (such as a displaced or missing maxillary cheek tooth, or a large overgrowth of the rostral cheek teeth). Even if no such abnormalities are palpable, stimulation of a pain response during this palpation may indicate the presence of sharp overgrowths on the lateral aspect of the upper cheek teeth. A useful test for suspected cases of dental disease is to feed the (unsedated) horse a small amount of forage. Affected horses may not make the normal vigorous crunching sounds of mastication, may show restricted mandibular movements that may be confined to one side of the mouth, or may even quid (drop boluses of partially masticated forage) if a painful dental disease is present.

A detailed equine oral examination can only be performed using a full mouth speculum (gag), and every equine practitioner should have one (Figure 30-2). It is possible to perform a safe digital examination of the cheek teeth and oral cavity of most unsedated horses, but a more complete visual examination requires sedation, especially with painful disorders and in difficult horses. Food retained in the oral cavity should be removed by flushing the mouth using a large dental (dosing) syringe and water or a very dilute chlorhexidine solution. The

Figure 30-2. This horse is having a dental examination while being restrained in stocks equipped with an overhead extension to allow a dental head collar to be attached. Note the use of the Hausmann speculum and headlight for this examination.

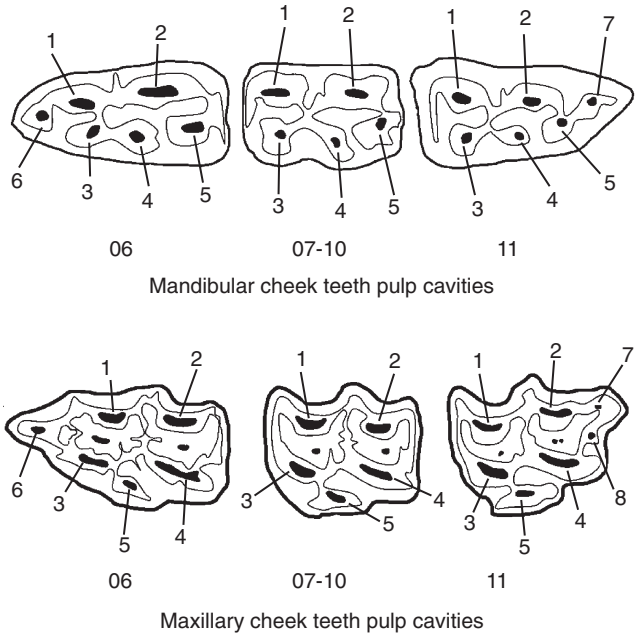

Figure 30-3. Identification of the individual pulp chambers of mature equine cheek teeth using a recently modified system of pulp nomenclature by du Toit et al.[3] The 06s *(left side of figure)* have six pulp horns. The 07s to 10s *(center of figure)* have five pulp horns. The 11s *(right side of figure)* have six or seven horns.

use of a strong headlight (a hand-held light is much less satisfactory), a dental probe or metal examination "basket" to displace the tongue, and an equine dental mirror (or a rigid dental endoscope, if available) allow full visual examination of the equine oral cavity.

Unless each tooth and the adjacent soft tissues (gums, cheeks, and tongue) are carefully visualized and palpated, major disorders, especially of the caudal mandibular cheek tooth and its periodontal membranes, can readily be missed. A long dental mirror or intraoral endoscope is essential to examine the gingival margins of the cheek teeth, especially on their buccal aspects, for the presence of periodontal disease and food impaction, and also to visualize the cheek teeth occlusal surfaces (e.g., to detect pulpar exposure and more fully assess dental fractures and infundibular caries). It is also useful to smell the oral cavity for halitosis, which usually indicates anaerobic infections, which are most commonly a result of periodontal food pocketing and infection, but halitosis is occasionally caused by advanced dental caries or cheek teeth fractures.

A very significant disorder, especially in the younger horse, is cheek tooth apical (the roots are often not yet even formed) abscessation. In most cases, little obvious change is visible on the clinical crown, except with some maxillary cheek tooth apical infections, where gross infundibular caries or sagittal fractures of the clinical crown may be present. However, close examination of the occlusal surface of the suspect cheek teeth with a dental mirror or endoscope, and probing the secondary dentine on the occlusal surface that overlies the five to seven pulps of the cheek tooth[5] (Figure 30-3) with a fine steel probe, will reveal pulpar exposure (i.e., dull, darker areas of the secondary dentine, with pitting and food pocketing into the pulp chamber) in 34% of infected mandibular[6] and 23% of infected maxillary teeth.[7] This finding can be of enormous value when other clinical findings and radiology are inconclusive in cases of apical infection. Occasionally, occlusal pulpar exposure in the absence of apical infection is found in older horses.[8]

Figure 30-4. This foal has a moderate degree of overjet, with the upper incisors protruding approximately 2 cm rostral to the lower incsors.

Disorders of the Incisors

Overjet and Overbite ("Parrot Mouth")

Many horses have some degree of what the older textbooks term mandibular brachygnathism ("parrot mouth," "overshot jaw"), referring to a rostral projection of the upper incisors beyond the lower incisors in a horizontal plane, which should correctly be termed "overjet" (Figure 30-4). However, in some of these cases,

Figure 30-5. This adult horse has a marked degree of overbite, with the upper incisors protruding rostral to the lower incisors and also lying rostral to them in the vertical plane.

there may not be shortness of the mandible. Instead, an overlong maxilla (maxillary prognathism) may be present. Both of these conditions induce cheek teeth wear disorders.[9] Consequently, a major significance of overjet is that affected horses commonly have overgrowths of the rostral aspects of 106 and 206 and of the caudal aspects of 311 and 411. In older horses with overjet, the reduced wear on upper incisors 101 and 201, in particular, may cause them to overgrow, and the upper incisors subsequently develop a convex occlusal surface, which has been termed a "smile." In more severe cases, the upper incisors, as well as projecting rostrally to the lower incisors in the horizontal plane, also grow down in front of the lower incisors in a vertical direction (i.e., overbite) (Figure 30-5), and this may prevent the small, normal amount of rostrocaudal mandibular movement that normally occurs in horses,[10] and it may even retard mandibular growth, thus exacerbating cheek teeth abnormalities.[11] Unless contact between opposing incisors is totally absent, overjet and overbite rarely cause the horse trouble in prehending food, as its mobile lips and tongue can compensate. However, this condition is esthetically undesirable, especially in show horses.

In affected foals, overjet can be corrected or partially corrected by use of an incisor orthodontic brace—that is, by placing steel wires (a tension band) around the upper incisors and fixing these wires around the 506 and 606 (or 507 and 607) to retard growth of the premaxilla and maxilla.[11,12] A newer technique involves passive extension of the rostral mandible following an osteotomy of both rami and implantation of a rachet fix device on each ramus (see Chapter 102). This surgery is best performed at around 3 months of age, but it can be of value in foals up to 8 months old. Under general anesthesia, a 1-cm horizontal stab incision is made in the skin of the cheeks, as dorsally as possible to avoid damaging branches of the dorsal buccal nerve, opposite the interdental spaces between the upper 06s and 07s, or between the 07s and 08s. A short Steinmann pin fitted to a Jacobs chuck (or a bone drill protected by a drill guide) can be pushed through the skin wound to puncture the cheeks and enter the oral cavity (Figure 30-6). Using a finger in the oral cavity for guidance, the point of the pin or drill is directed into the interdental (interproximal) space, as close to the gingival margin as possible, and it is subsequently forcibly

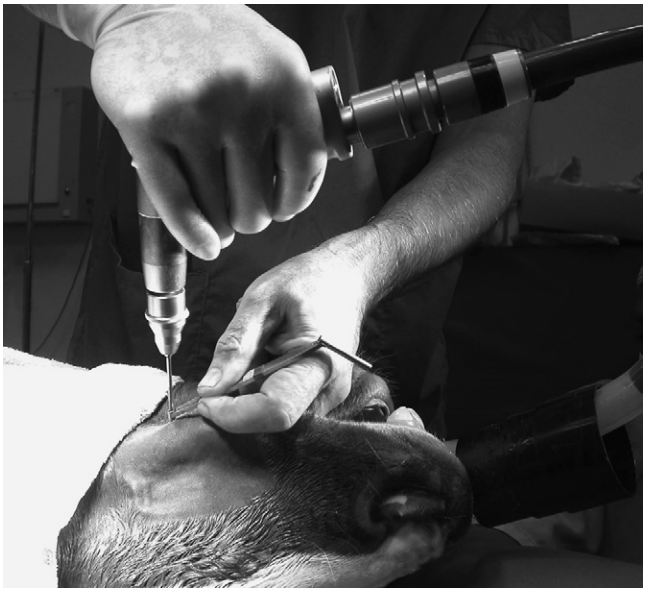

Figure 30-6. A bone drill and drill sleeve have been inserted through a horizontal stab incision in the skin and buccal muscles of this foal with overjet in order to drill between the deciduous cheek teeth for placement of a tension-band prosthesis.

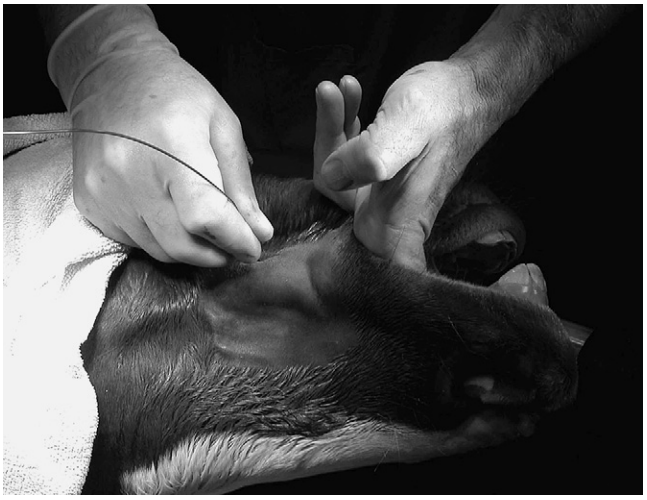

Figure 30-7. A length of 1.25-mm stainless steel wire is being inserted through the cheek incision into the interdental space between the first and second cheek teeth (606 and 607).

pushed (while twisting) through this tight space and directed to exit at the medial (palatal) interdental space, again close to gingiva (at the border of the hard palate). The Steinmann pin is withdrawn and a 14-gauge needle inserted along its path, followed by insertion of a 1.25-mm diameter stainless steel wire of 60-cm (24-inch) length through the needle into the interdental space and into the oral cavity (Figure 30-7). Most of the wire is drawn into the oral cavity and pulled rostrally to the incisors. The needle is withdrawn.

The external free end of the wire is directed beside the initial part of the wire through the buccal incision into the oral cavity, taking great care not to entrap any soft tissue (possibly the buccal nerve) and not to creat acute bends in the wire. The second wire end is palpated as it penetrates the buccal mucosa,

again as close as possible to the lateral (buccal) gingival margin of the interdental space. This part of the wire is subsequently drawn into the oral cavity along the lateral aspect of the cheek teeth. As the last of the wire is pulled through the cheek incision, a kink will occur in it, and this area of weak wire should later be discarded.

The two free ends of the wire are withdrawn from the mouth, on either side of the cheek teeth, while making them even in length. While pulling them rostrally, the free ends are twisted back to the rostral border of the upper 06s, as dorsally as possible. The twisted wires are then placed over the labial (rostral) aspect of the incisors. This procedure is then repeated on the other side of the mouth, and the free ends of both pairs of wires are twisted tightly together, just below the gingival borders of the upper incisors (or interwoven between some incisors at the gingival level) and their ends are trimmed (Figure 30-8). The wire knot should be embedded in polymethyl methylacrylate (PMMA/acrylic) to prevent soft tissue trauma.

If an overbite is present in young foals, the tension from this orthodontic brace may well cause further caudoventral deviation of the upper incisors and premaxilla toward the rostral aspect of the lower incisors, and this would simply worsen the overbite, rather than retard premaxillary and maxillary bone growth. In such foals, a "biteplate" can additionally be fitted along with the orthodontic brace to promote indirect occlusion between the upper and lower incisors.[11,12] The biteplate can be fashioned from a perforated aluminium plate (2 to 4 mm thick) that is cut to fit the shape of the rostral aspect of the hard palate, extending caudad about 4 to 5 cm (approximately 2 inches) from the incisors.

Having fitted the brace as just described, the underlying hard palate is covered in Vaseline, and the biteplate, covered with soft acrylic, is placed on the hard palate. Additional acrylic should be placed beneath the caudal aspect of the biteplate, so that the sloped plate will tend to push the lower incisors more rostrally during prehension. Acrylic is now also used to join the biteplate to the wires of the brace (Figure 30-9), ensuring that the acrylic does not extend too high on the gingiva above the incisors, where it can form a sharp rim that can traumatize the lip mucosa. After a biteplate is fitted, foals may have trouble suckling.

Another option involves the fitting of PMMA only, without the aluminum plate, to the rostral part of the hard palate and wires pressed at strategic locations into the soft PMMA before it hardens. Once the PMMA is hard, the wires can be used to attach the PMMA plate to the adjacent teeth.

The biteplate may also hurt the mare's udder during suckling, causing her to temporarily prevent the foal from suckling. In such cases, the mare should be milked for a few days and the foal bottle-fed with her milk. If the foal is uncomfortable because of the brace, it should be given low doses of nonsteroidal anti-inflammatory drugs (NSAIDs) and anti–gastric ulcer medication, such as omeprazole. Some clinicians recommend that foals be weaned before fitting orthodontic braces with biteplates. Any overgrowths on the rostral aspects of the upper 06s and on the caudal aspects of the lower 08s should be rasped off, to promote free rostrocaudal mandibular movement.

Wires may break unilaterally or bilaterally and thus cause the biteplate to loosen; owners must be prepared for this extra work and expense. Consequently, the foal's mouth needs to be regularly inspected, and broken wires need to be replaced, usually under general anesthesia. The brace can be removed when the incisors are aligned or nearly so. With severe overjet or overbite, complete resolution will not be possible, but esthetically pleasing results, and certainly much reduction in the concurrent cheek teeth overgrowths, can be achieved. The ethics of these orthodontic procedures, especially in animals that may be used for breeding, are debatable, and some breed societies do not allow registration of horses that have been treated with this technique. This aspect should be discussed with the owners in detail before orthodontic wires and biteplates are applied.

Orthodontic treatment is not feasible in adult horses. Large overjet/overbite incisor overgrowths in adult horses should be reduced using power or manual instruments, in stages of about 5 mm every 6 months, to prevent pulpar exposure. To partially remove incisor overgrowths, it is best to sedate the horse and place a plastic pipe gag in the interdental space (circular metal gags, such as Swale's gag, can fracture cheek teeth, and rubber wedge gags promote constant chewing movements). Once the overgrown incisors have been reasonably reduced (maximum 5 mm at a time), they should be rasped level biannually, as

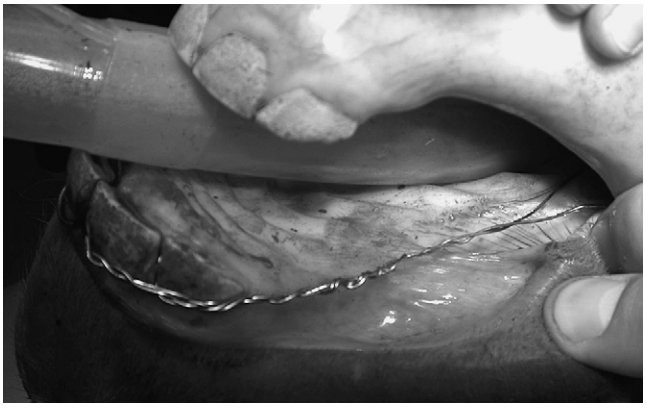

Figure 30-8. The twisted wires that are attached bilaterally to the cheek teeth have been interwoven between some incisors and then tightly joined together in front of the incisors, to retard premaxillary and maxillary growth.

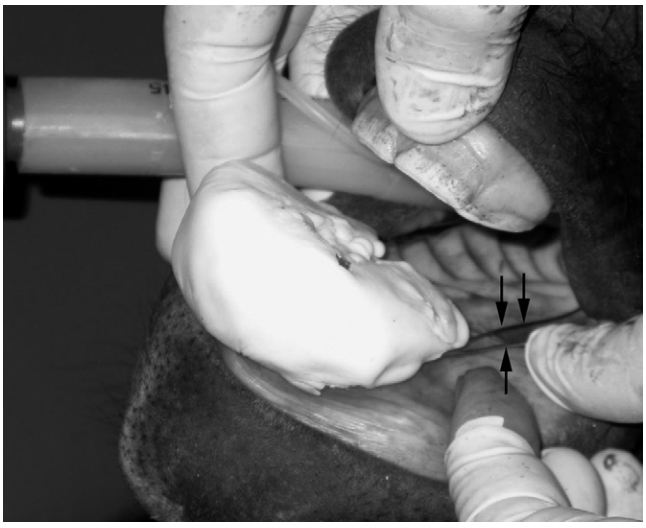

Figure 30-9. This foal had marked overbite as well as overjet, and it has had a perforated aluminum biteplate embedded in acrylic, attached to the orthodontic brace. The still soft polymethyl methacrylate is being molded around the prosthetic wires *(arrows)* (untwisted in this case).

should concurrent cheek teeth overgrowths (see "Disorders of the Cheek Teeth," later in this chapter).

Underbite ("Sow Mouth")

Prognathism ("undershot jaw," "underbite") is rare in the horse, except in miniature horses, and is usually clinically insignificant unless there is a total lack of occlusion between the upper and lower incisors. Severely affected horses eventually develop a concave upper incisor occlusal surface, which has been termed a "frown," and may develop lower 06 and upper 11 overgrowths. Correction may be achieved using the same orthodontic procedures as discussed for the maxilla, with the orthodontic device being applied to the mandible, or by extension of the premaxilla/maxilla (see Chapter 102).

Retained Deciduous Incisors

Deciduous incisors, which normally lie rostral (i.e., labial) to their permanent counterparts, are occasionally retained beyond their normal time of shedding, and they will cause the permanent incisor to be displaced caudally (lingually), occasionally leading to permanent changes in the incisor occlusal surface. If retained incisors are very loose, they can be removed using small-animal dental extraction forceps or equine "wolf teeth" forceps. If more firmly attached, they should be extracted using a small-animal dental elevator under sedation and local anesthesia (by injecting 2 to 5 mL lidocaine, directed 2 to 4 cm caudally, into the ostium of the mental or infraorbital canal). If a permanent incisor does not erupt beneath its deciduous predecessor, little resorption of the deciduous reserve crown may occur, and up to 6 cm of the deciduous tooth may remain in the alveolus. In such cases, much of the rostral aspect of the alveolar wall of the retained incisor will have to be removed with a bone chisel in order to extract the retained incisor (Figure 30-10). Occasionally, a permanent incisor erupts *rostral* to the deciduous incisor, and great care must be taken in removing the deciduous incisor that is now positioned on the caudal (lingual) aspect of the permanent incisor.[13]

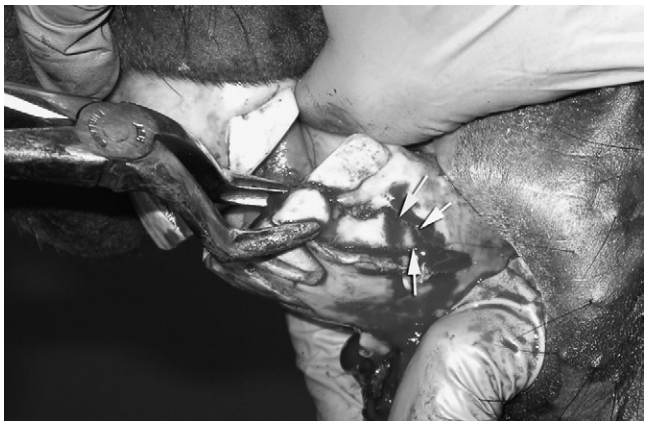

Figure 30-10. This horse has a large retained 702 (deciduous precursor of 302), and minimal resorption of its apex and reserve crown has occurred. A lingual (caudal) deviation of the permanent tooth is now present that would cause a permanent incisor malocclusion. About 5 cm of the rostral aspect of the alveolar wall of 702 was removed *(white arrows)*. The tooth was subsequently loosened with elevators and is now being extracted with incisor forceps.

Supernumerary Incisors

Supernumerary incisors are permanent teeth that are additional to the normal six permanent incisors on each arcade. They are usually morphologically identical to a normal permanent incisor, which helps differentiate them from retained deciduous incisors.[14] Supernumerary incisors may have long (more than 7 cm [3 inches]) reserve crowns that may be intimately related to the reserve crowns and roots of the normal permanent incisors, and consequently they are usually impossible to differentiate from normal incisors, both clinically and radiographically.[5,15] Consequently, extraction of supernumerary incisors (especially if multiple) is technically very difficult and also risks damaging the adjacent normal incisors. If they are grossly displaced rostrally (labially) and thus protrude submucosally, they are amenable to extraction, as described for a large retained deciduous incisor. As most supernumerary incisors cause few clinical problems, they are usually best left alone (except, perhaps, in show horses), with biannual rasping of any incisors that are not in occlusion to prevent overgrowths caused by their prolonged eruption.

Fractures of the Incisors

Fractures of the incisor teeth, and often of their supporting mandibular or premaxillary bones, can occur because of trauma (usually from kicks) and commonly result in exposure of the pulp (i.e., complicated dental fractures).[5,16] Idiopathic incisor fractures are rare, in contrast to the common presence of such fractures in equine cheek teeth. All young equine teeth (incisors and cheek teeth) have very wide apical foramina (root canal openings), along with a very large, vascular pulp, which can resist the infection, and more so the inflammation, that inevitably develops in orally exposed pulp.[17] Consequently, pulpar exposure, especially in younger horses, does not necessarily lead to the degree of pulpar inflammation that compresses its vascular supply (with subsequent pulpar ischemia and necrosis, and death of the tooth), as usually occurs with pulpar exposure in brachydont (e.g., human or canine) teeth. Horses with incisor fractures should receive tetanus prophylaxis and prolonged (7 to 10 days) antibiotic (e.g., trimethoprim/sulfadiazine) therapy.

A regional nerve block (mental or infraorbital nerve) followed by the removal of any loose dental fragments and débridement of any exposed pulp with a 16-gauge needle is of benefit as first aid, or as the main treatment if endodontic treatment is not possible. Pulpar bleeding can be controlled with pieces of compressed cotton wool held in a small hemostat (or by using dental paper points if available), and the exposed aspect of the pulp canal should be filled with a calcium hydroxide preparation that promotes reparative tertiary dentine formation.

If facilities are available, the preferred treatment involves endodontic management through the damaged occlusal aspect, which greatly increases the chances of saving the tooth. This usually consists of vital pulpotomy—that is, removal of devitalized pulp with a fine-pointed scalpel, control of hemorrhage from the underlying vital pulp with paper points or hemostatic agents, followed by sealing the healthy pulp with a calcium hydroxide preparation. Having sealed off the vital pulp, the remaining open pulp canal is prepared for filling by etching it for 1 minute with a phosphoric acid gel (to make the dental surface more porous to bond to dental restorative materials), which is then flushed away with water and air-dried. The pulp

canal (which may be undecut with a dental burr to help retain the restoration) is subsequently thinly coated with a bonding agent (Figure 30-11) and sealed, in layers if necessary, with a modern, composite restorative material (e.g., StarFill 2B dual-cure composite) (Figure 30-12).

In some young horses, the pulp becomes sealed off at the site of exposure by reparative (tertiary) dentine formation even without treatment, with the tooth remaining vital and continuing to erupt normally.[5] Treatment of premaxillary and mandibular fractures involving the incisors is discussed in Chapter 102.

Abnormalities of Incisor Wear

Incisor disorders include rectangular overgrowth of individual incisors ("steps") as a result of traumatic loss, or of maleruption (e.g., delayed eruption or displacement) of the opposing teeth, in addition to the previously noted incisor overgrowths occurring with overjet or underjet. Abnormal wear pattern of the occlusal and rostral aspects of the 01s and occasionally of the 02s occurs in crib biters. Another common abnormality of incisor wear is a tilted or slanted incisor occlusal surface, which is termed *slope mouth* or *slant mouth*. This defect is most commonly associated with a major unilateral abnormality of the cheek teeth that has caused a pronounced unilateral chewing action, thus causing uneven wear of the incisors.[5,18] The disorder of "wry nose" (campylorrhinus lateralis) can also cause this incisor defect as a result of incisor malocclusion (see Chapter 102). Such incisor overgrowths should be reduced in stages to prevent pulpar exposure, as previously described.

Figure 30-11. This horse is having endodontic treatment of its traumatically damaged incisors (402 and 403). Exposed necrotic pulp in 402 has been removed to the level of healthy pulp. The pulp cavitiy was sealed with calcium hydroxide paste, and the pulp canal above was acid-etched. A bonding agent is now being applied to the sealed pulp canal.

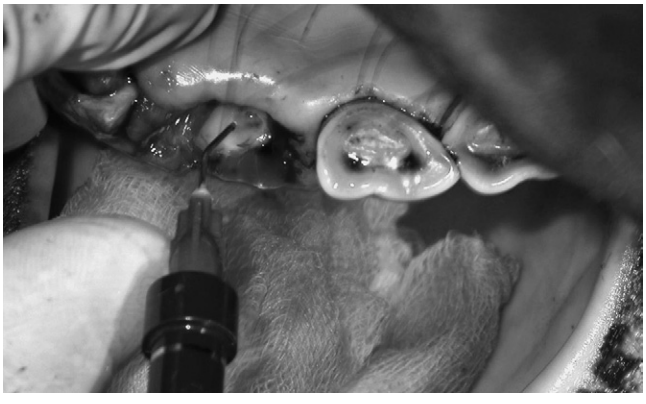

Figure 30-12. A modern, self-curing composite filler is being inserted into the prepared pulp cavity on top of the calcium hydroxide layer to further seal off the underlying vital pulp (same horse seen in Figure 30-11).

Disorders of the Canine Teeth (Triadan 04s)

Rarely, displaced canine teeth can interfere with the bit or injure oral soft tissues. The displaced aspects of such teeth should be partially ground down, taking care to avoid pulpar exposure that may cause pulpitis and possibly death of the tooth, which may not become evident until years later.[18] The sharp tips, or even most of the clinical crown of the canine teeth, are rasped off by some operators to allegedly prevent these teeth from lacerating the operator's hands during dental procedures and to prevent them from interfering with the bits. There is no objective evidence on the value of this procedure, and if pulpar injury occurs during this procedure, some horses develop painful pulpitis and later death of the tooth.

Rarely, canine teeth need to be extracted—for example, as a result of traumatic fracture or infection,[19] with the latter often being secondary to the recently described equine odontoclastic tooth resorption and hypercementosis (EOTRH) sydrome, which can also affect incisors.[20] Long-term unerupted canine teeth that are causing mucosal ulceration and bitting problems in horses older than 7 years can also be extracted if deep incision of the mucosa and underlying periosteum does not relieve the problem. Because of the great length of their unerupted crown (up to 7 cm long), extraction of uninfected canine teeth is difficult and should never be undertaken without good reason.[19] Following an appropriate nerve block in the standing sedated horse, extraction is performed by initially removing the vertical aspect of its lateral alveolar wall (Figures 30-13 and 30-14). A dental elevator is subsequently used to further loosen the canine tooth in the horizontal, more apical aspect of the alveolus, finally allowing complete extraction (Figure 30-15). It is a difficult procedure in firmly attached younger teeth, and the apical aspect of the canine tooth may fracture during extraction. In the absence of apical infection or EOTRH syndrome, up to one third of a healthy root (canine teeth are brachydont teeth) may be left in the alveolus without causing postoperative problems.

Disorders of "Wolf Teeth" (Triadan 05s)

Wolf teeth (Premolar 1, Triadan 05) are small vestigial teeth (usually 1 to 3 cm long) that are, without much critical evidence, blamed for many behavioral and bitting problems in horses, and therefore these teeth are frequently extracted. Wolf teeth most commonly develop in the maxilla, lying in front of the 06s and most are lost when the 06 deciduous cheek teeth is shed. If normally positioned and small, it is difficult to envisage how the upper wolf teeth can interfere with the bit. However, displaced, enlarged, fractured, or unerupted ("blind") wolf teeth with overlying painful or ulcerated mucosa may

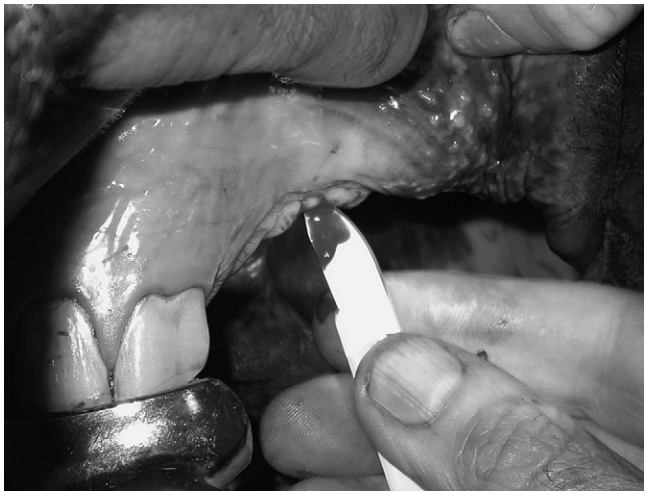

Figure 30-13. This horse had a long-term bitting problem associated with a partially erupted canine tooth (204) that caused chronic painful ulceration of the overlying mucosa. After sedation and infraorbital nerve block, an incision is being made over the ventrolateral aspect of the 204 alveolus.

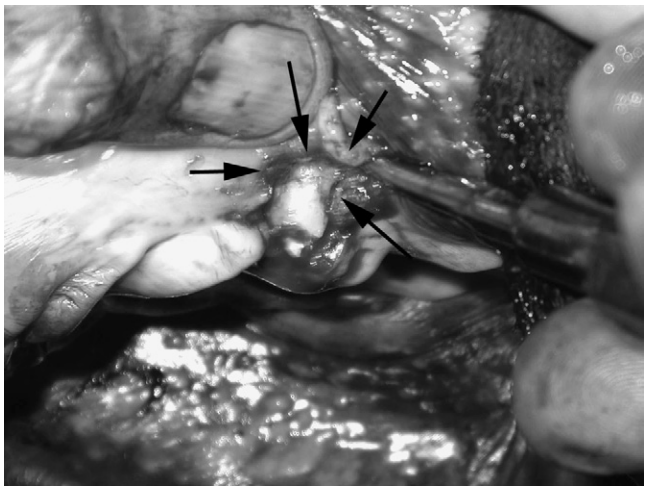

Figure 30-14. After retraction of the incised mucosa, the ventrolateral aspect of the alveolar wall of 204 has been removed with a dental elevator, revealing the vertical aspect of the crown of the problematic canine *(arrows)*.

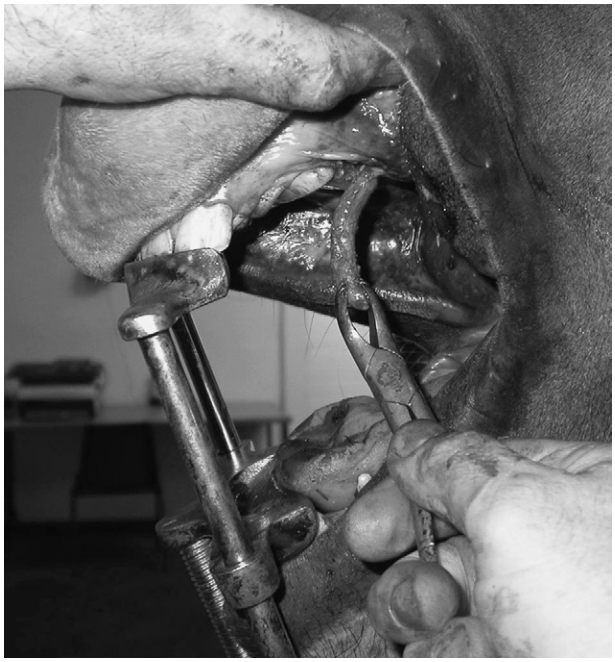

Figure 30-15. Note the great length and the curvature of the canine tooth, which has been extracted intact. A Butler gag is being used.

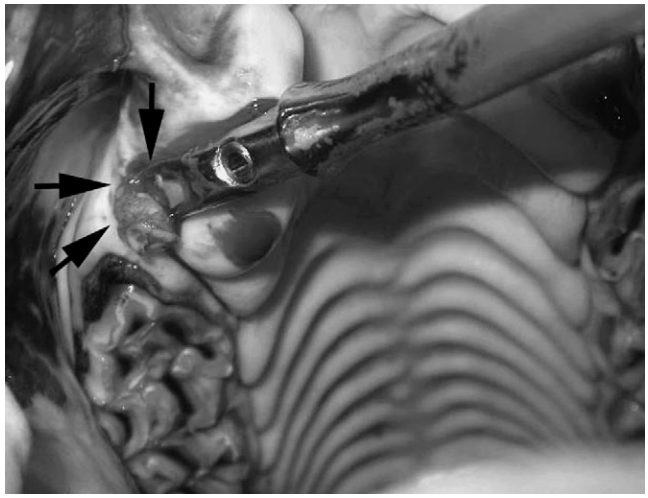

Figure 30-16. This large and rostromedially displaced "wolf tooth" (105) *(arrows)* has been loosened from its periodontal attachments with a Musgrave elevator. The intact, loosened tooth can now be readily extracted with forceps.

cause bitting problems. Such wolf teeth should be extracted, as should all mandibular wolf teeth, which invariably will contact a bit. Unless they are digitally loose (mainly in younger horses), wolf teeth should be extracted under sedation using subgingival local anesthesia or, preferably, infraorbital or mental nerve blocks.[19] The latter technique, which will anesthetize the more relevant sensory nerves deep in the periodontal ligament, is advised when extracting the more deeply embedded wolf teeth commonly found in older horses. These teeth often lie in a sclerotic alveolus.

A variety of specialized instruments (e.g., Burgess or Musgrave elevators) can be used to separate the gingiva around these teeth, and an elevator is subsequently inserted into different sites around the periodontal space to loosen the teeth (Figure 30-16), followed by forceps extraction when adequately loosened. Large (often unerupted) wolf teeth may have to be extracted through a mucosal incision, followed by loosening them from the underlying maxilla using an osteotome and mallet. In contrast, small mobile wolf teeth in younger horses may be readily extracted using forceps only. It is imperative to separate firmly attached wolf teeth from the alveolus and, most importantly, not to break them off above the alveolar level, where the protruding fragment often causes more oral discomfort than was present before the partial extraction. If the roots or adjacent apical parts of wolf teeth fracture below alveolar level during extraction, these alveoli usually heal over fully and so should be of little concern. The greater palatine artery can be lacerated during wolf tooth extraction, and local infection and tetanus can also develop after such procedures.

Disorders of the Cheek Teeth

Developmental Disorders of the Cheek Teeth

RETAINED DECIDUOUS CHEEK TEETH

Retention of the remnants of the deciduous cheek teeth ("caps") can occur in horses between 2 and 5 years of age. When loose, and especially partially retained by their gingival attachments, they may cause oral pain, and affected horses may show quidding, bitting problems, and occasionally, loss of appetite for a couple of days.[18] Such signs in this age group warrant a careful examination of the rostral three cheek teeth for evidence of loose deciduous cheek teeth. Loose caps should be removed using specialized cap extractors.

The prolonged retention of "caps" has been alleged to cause delayed eruption of permanent cheek teeth, and the development of large mandibular eruption cysts in 3- or 4-year-olds under the apices of the erupting permanent cheek teeth (07s and 08s),[21] but another study found no evidence of this relationship.[22] The presence of very enlarged eruption cysts, especially if unilateral, hot, or painful, should prompt a thorough oral and, if necessary, open-mouth radiographic examination for the presence of retained deciduous cheek teeth. Additionally, standard radiographic views to look for evidence of apical infection should be obtained. However, the practice of methodically removing deciduous teeth at set ages in horses results in the premature removal of some deciduous cheek teeth, causing pain to the horse and prematurely exposing the underdeveloped permanent counterparts lying below. In the upper cheek teeth, this leads to some loss of blood supply to the still-developing infundibular cementum, possibly leading to infundibular cemental hypoplasia (patent infundibulum) and thus predisposing the tooth to infundibular caries and possible apical infection or cheek teeth fracture later in life.

DIASTEMATA

The occlusal surfaces of all six cheek teeth are normally compressed closely together, and each cheek teeth row should function as a single grinding unit. This is achieved by the action of the angled rostral and caudal cheek teeth compressing the occlusal aspects of all six cheek teeth together.[17] Even with age, the progressively smaller reserve crowns usually remain tightly compressed at the occlusal surface (the cheek teeth taper in slightly from crown to apex). However, if a space, or diastema (plural diastemata), develop between the occlusal aspects of adjacent cheek teeth, clinical problems will occur.[22,23] Very severe clinical problems occur when these spaces are narrow (often only 2 mm wide) at the occlusal surface and wider (approximately 4 to 5 mm) at the gingival margin,[24] a condition termed *valve diastemata*.[25] In some cases, cheek teeth diastemata are caused by lack of sufficient angulation of the rostral (06s) and caudal cheek teeth (10s and 11s) to provide enough compression of the occlusal surface of the six cheek teeth. In other cases, diastemata occur with apparently normal cheek teeth angulation, suggesting that the dental buds have developed too far apart.[22,26,27] Individual diastemata can also be caused by displaced or supernumerary cheek teeth, or they may occur adjacent to overgrown cheek teeth (e.g., rostral to an overgrown lower 11 or caudal to an overgrown upper 06 that have been displaced caudally or rostrally, respectively, because of their focal overgrowths).

Food becomes impacted into these abnormal interdental spaces by the prolonged (up to 18 hours a day in some normal horses) grinding of tough forage (Figure 30-17), leading to progressively deeper and more painful packing of food into the sensitive periodontal spaces, which causes a secondary, malodorous periodontal infection. Diastemata can be recognized visually (with use of a dental mirror or intraoral endoscope) or by digitally detecting food fibers protruding between the affected teeth at the gingival level. In a longer-standing case, this food pocketing and periodontal disease may extend along the full width of affected teeth and extend deep into the mandible or maxilla, including into the maxillary sinuses.[28]

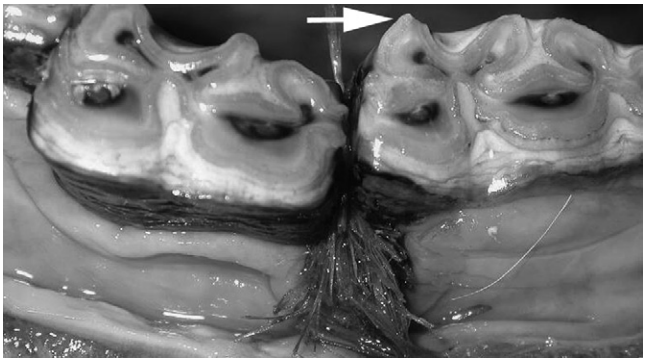

Figure 30-17. A diastema with deep interdental food pocketing is present between 410 and 411, both of which have slight medial displacement. A slight "steplike" overgrowth is also present on 411 *(arrow)*, which may have caused caudal displacement of this cheek tooth and so predisposed to the diastema.

Evaluation of the severity and numbers of diastemata can be difficult because the caudal mandibular cheek teeth are most commonly affected and the gingival margin of these teeth is hidden by the tongue. The use of oral endoscopy or intraoral mirrors, or obtaining 10- to 15-degree, latero-oblique radiographs with the horse's mouth open,[29,30] can be very helpful in assessing diastemata.

Treatment of cheek teeth diastemata can be difficult. Cleaning out the periodontal pockets manually (with specialised diastema forceps, dental picks, and long forceps) or with high-pressure pneumatic or water instruments, and filling the periodontal defects with antibiotics and plastic impression material is of value in some cases.[26] However in many cases, this treatment only gives temporary relief, unless the underlying mechanical predisposition to this food impaction is also treated. In younger horses with mild developmental diastemata where there is sufficient angulation of their cheek teeth (as judged radiographically), the abnormal spaces may close when further dental eruption occurs. Such horses should not have their diastemata mechanically widened. Many horses show severe clinical signs when fed hay and then improve greatly when out at grass in the summer. Likewise, feeding only a finely chopped (milled) diet, such as grass or alfalfa cubes (whose fiber length is too short to become entrapped in diastemata), often reduces or fully removes the clinical signs.

Currently, the best treatment for this disorder in mature horses appears to be to widen problematic diastemata—to about 4 to 6 mm wide on the occlusal surface—using a specialized diastema burr (such as, Powerfloat, D&B Enterprises, Inc., Calgary, Alberta, Canada).[27] Cases should be evaluated and selected for this treatment by careful oral examination, and also by use of open-mouth radiography in younger horses. Horses to be treated should be heavily sedated and the periodontal

pockets and diastemata cleared of food as described earlier, a procedure that usually causes much discomfort to horses. During diastemata widening, water should be sprayed over the cheek teeth being burred to prevent thermal pulpar damage and the site should be inspected frequently (e.g., at 5-second intervals) during the procedure to ensure the site of widening is correct and that adjacent pulp horns are not exposed. The pulp horns are closer to the caudal aspect of the cheek teeth, and therefore as much tooth as possible should be removed from the rostral aspect of the tooth positioned caudal to the diastema, to help prevent exposing dental pulp (Figure 30-18).[31] If painful diastemata are caused by a markedly displaced tooth (teeth), and diastema widening is found to be ineffective, extraction of the displaced tooth (teeth) will usually result in complete cessation of quidding, as food will not become trapped in a wider extraction space. Some young horses with severe, multiple diastemata and osteomyelitis of alveoli and supporting bones are very difficult to treat.

ROSTRAL POSITIONING OF THE MAXILLARY CHEEK TEETH ROWS

A common dental abnormality in horses is a rostral positioning of the maxillary cheek teeth rows relative to their mandibular counterparts, invariably occuring in conjunction with overjet/overbite ("parrot mouth"). This disorder eventually leads to the development of focal overgrowths of the rostral aspect of the upper 06s, which may cut the cheeks and interfere with the bit. If small, these overgrowths can be manually rasped level with a solid carbide float, but if they are large, a power tool is best to reduce them, in stages of about 5 mm every 3 to 6 months, to avoid pulpar exposure.

Similar overgrowths on the caudal aspect of 311 and 411 frequently go undetected and can lacerate the tongue, wear down the opposite cheek teeth (maxillary 11s) to gum level, and even penetrate the hard palate or lacerate the greater palatine artery. There is very little room between the occlusal surfaces of the caudal maxillary and mandibular cheek teeth, especially if large overgrowths are present on these teeth. Consequently, these overgrowths are best reduced using power tools, grinding them from their medial aspect if there is not enough room to reduce them from their occlusal surface, and then restoring the normal occlusal angle (i.e., have them sloping down in a lateral [buccal] direction). Molar cutters and percussion guillotines (which encircle this caudal hook) can fracture these teeth and cause pulpar exposure, especially in smaller breeds (e.g., Arabian horses and Welsh ponies) that have marked dorsal curvature (curve of Spee) of the caudal occlusal surface of their cheek teeth, which can be mistaken as dental overgrowths of the mandibular 11s. Such dental fracture and pulpar exposure may lead to apical infections or even to life-threatening cellulitis of the mandibular and pharyngeal areas.[32]

DISPLACEMENTS OF THE CHEEK TEETH

Most severe cases of equine (medial, lateral, rotatory) cheek teeth displacements, which usually involve the 09s and 10s, are developmental in nature and appear to be caused by overcrowding of the cheek teeth during eruption. This type of displacement is often marked and also may be present bilaterally.[22] Gross dental overgrowths later develop on areas of the displaced teeth and their opposite counterparts that are not in occlusal contact, causing soft tissue trauma. Additionally, painful food pocketing develops in diastemata that are invariably present on both sides of the displaced cheek teeth. This often causes severe and painful periodontal disease (Figure 30-19) and even osteomyelitis, sinusitis, or orosinus fistula formation. Acquired cheek teeth displacements (usually medial displacements of the lower 10s and 11s) can also develop in older horses and are usually associated with lesser degrees of cheek teeth displacement and overgrowth, but they still may cause painful diastemata.[22]

Abnormal protrusions or overgrowths on displaced cheek teeth should be removed, preferably with a power tool, and in stages to help prevent pulpar exposure. Painful diastemata can be widened with a diastema burr as described earlier, and this often alleviates periodontal pain and thus quidding. When a cheek tooth is very displaced and has advanced periodontal disease, the displaced tooth should be extracted, as noted earlier. This is readily performed *per os* in the sedated horse using cheek

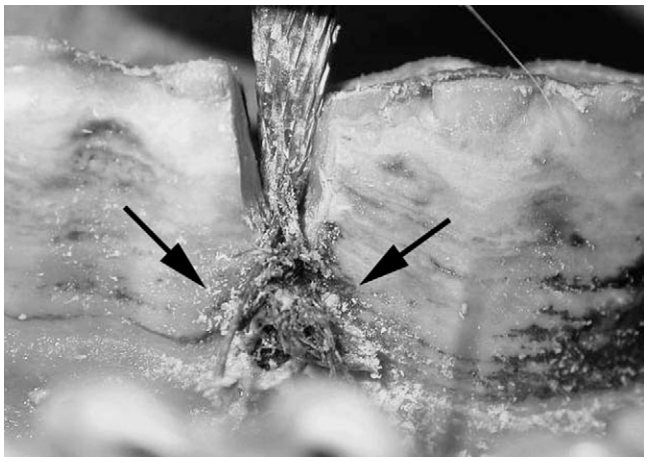

Figure 30-18. A diastema burr (Powerfloat, D&B Enterprises, Inc, Calgary, Alberta, Canada) is being used to widen the occlusal, interdental space in this specimen, which had diastema and food pocketing *(arrows)*. With valvelike diastemata, inserting the burr from the lateral and medial directions into the wider subocclusal space, followed by burring occlusally and caudally, is a more effective technique.

Figure 30-19. This mandible has a grossly laterally displaced cheek tooth that is probably developmental in origin. Because of its angulation, the displaced cheek tooth appears wider than its counterparts. Note the severe periodontal food pocketing in diastemata beside the displaced cheek tooth and to a lesser extent between other cheek tooth. Oral extraction of this displaced cheek tooth is indicated.

teeth extraction forceps (see later), and these horses usually cease quidding immediately following exodontia. Overgrowths on opposing cheek teeth should be reduced at 6-month intervals.

SUPERNUMERARY CHEEK TEETH

Supernumerary cheek teeth (i.e., the presence of more than six cheek teeth in a row) are common in horses, usually occurring at the caudal aspect of the maxillary, and less commonly the mandibular cheek teeth rows.[15,22,33] These may be large connated structures (i.e., formed from two to three fused, maldeveloped cheek teeth). Because they are sometimes large and irregularly shaped, or because of overcrowding of adjacent cheek teeth, abnormal spaces and subsequent periodontal food pocketing occurs between the supernumerary and adjacent teeth. This can result in pain and quidding and occasionally deeper infections.[15,22] Additionally, if the supernumerary teeth are present in just one row (i.e., are unopposed), they later form large overgrowths. If severe periodontal disease develops, particularly if associated with apical infection and sinusitis or an oro-sinus fistula,[28] the supernumerary cheek teeth should be extracted. Extraction should be performed *per os* if possible, as repulsion of the caudally positioned and also caudally angulated reserve crown and apex (apex likely to lie below the orbit) is technically very difficult. In the absence of periodontal disease, 6-monthly removal of overgrowths on supernumerary cheek teeth is required. Some of these caudal supernumerary cheek teeth (i.e., Triadan 12s) share an alveolus with the normal Triadan 11 or lie in a separate alveolus with poor periodontal attachments and can be orally extracted without too much difficulty.

Acquired Disorders of the Cheek Teeth

ACQUIRED OVERGROWTHS OF CHEEK TEETH

Feeding concentrates greatly alter the masticatory action of horses, causing them to chew with a more vertical rather than lateral mandibular action, and also for a much shorter time, than a horse that is only eating forage. This restricted lateral mandibular movement predisposes horses to develop overgrowths (initially of enamel) of their cheek teeth.[34] If these enamel overgrowths are neglected, the sharp enamel points eventually merge into a steeply angulated (e.g., 45 degrees versus the normal 10 to 30 degrees)[35] occlusal surface that is termed *shearmouth* or *scissor mouth*. A mechanical obstruction now additionally interferes with the normal side-to-side mandibular masticatory movements, rendering mastication even less efficient. Food stagnation and secondary periodontal disease also commonly develop, especially around the more caudal cheek teeth. In advanced cases of dental overgrowths, undulating irregularities of the occlusal surface of the cheek teeth rows in the rostrocaudal plane (i.e., "wavemouth") develop.

Some affected horses chew very slowly and make soft slurping sounds (instead of the normal vigorous crunching sounds) when chewing forage. Some affected horses use just one side of their mouth for chewing rather than alternating sides (and consequently develop a sloping incisor occlusal surface, called *slope mouth* or *slant mouth* as discussed earlier), or they may hold their head in an abnormal position during chewing. Affected horses may also have abnormal head carriage and bitting problems. The prime role in equine dental care is to prevent these major overgrowths from developing by routine oral examinations and effective teeth rasping.

TRAUMATIC DISORDERS OF THE CHEEK TEETH AND BARS OF THE MOUTH

In young horses, traumatic mandibular fractures often damage the long cheek teeth reserve crowns, which occupy much of this bone (see Chapter 102). In some cases of unilateral, nondisplaced mandibular fractures, conservative therapy (2 weeks of antibiotic administration and feeding a soft diet, such as soaked grass or alfalfa cubes for 2 to 3 months) is adequate, with the cheek teeth at the fracture site helping to stabilize the fracture, and the undamaged hemimandible acting as an effective splint. Traumatic fractures of maxillary cheek teeth are less common because of their anatomic position. Even if external sinus tracts develop after mandibular or maxillary fracture, it is worthwhile persevering with conservative therapy until radiographic changes (including the use of a metallic probe in any sinus tracts present) definitively confirm the presence of dental infection. Extraction of infected cheek teeth should be delayed for at least 3 months to allow mandibular or maxillary fracture healing to occur (see Chapter 102).

Bit-induced injuries to the dorsal aspect of the mandible at the interdental space ("bars of mouth") can occur as a result of very excessive bit force.[16,36] In most cases, a superficial periostitis or sequestration of the dorsal mandibular cortex will occur at this site. However, pathologic fractures of the mandible, mandibular osteomyelitis, and lower 06 cheek teeth infection can also occur.[16,36,37] These mandibular bone injuries should be assessed radiographically and sequestra removed under sedation (and mental nerve block if necessary). The horses should be ridden with a bitless bridle or hackamore for 4 to 6 weeks, and thereafter a wide rubber bit should be used with restraint. Exostoses (bone spurs) may develop at the sites of bitting injuries (especially on the caudal aspect of the lower bars of mouth).[38] However, some normal horses have a raised and variably sharp ridge at this site, and some also exhibit roughening of the mandibular cortex more laterally at the insertion of the buccinator muscles. Very large and irregular exostoses at this site may damage the overlying mucosa because of bit contact, and in doing so they cause permanent bitting problems. Large osseous protrusions can be removed under sedation and local nerve block or local infiltrative anesthesia, using an osteotome.[38]

IDIOPATHIC FRACTURES OF THE CHEEK TEETH

Idiopathic cheek teeth fractures (i.e., cheek teeth fractures in the absence of known trauma) are common in horses and mainly affect the upper cheek teeth.[37,39,40] Most commonly, "slab" fractures occur through the two lateral pulp cavities (pulp horns number 1 and 2) (Figure 30-20), usually of the upper 09s.[39] Less commonly, lower cheek teeth also suffer from such lateral sagittal fractures through pulp horns 1 and 2.[39] The maxillary cheek teeth slab fracture site usually becomes filled with fibrous food, thus laterally displacing the smaller lateral cheek teeth fragment into the cheeks, causing buccal lacerations with subsequent quidding and bitting problems. Spontaneous loss or extraction of the smaller, loose fragment with cheek teeth fragment forceps (Figure 30-21) usually resolves the clinical signs, even though radiographic and scintigraphic evidence of apical inflammation and alveolar remodeling often occur in these cases. Usually the two exposed pulp horns become effectively sealed off from the

Figure 30-20. Intraoral view of a horse with a lateral "slab" fracture of 208. The fracture site is filled with food that has slightly displaced the larger medial portion into the hard palate and markedly displaced the thin, lateral fragment *(arrows)* into the cheeks, where it has caused buccal ulceration *(arrowheads)*.

Figure 30-21. A lateral "slab" fracture of a maxillary cheek tooth that was readily removed with forceps. Gross caries on its medial aspect and destruction of its apical area *(arrowheads)* are evident.

fracture site with reparative dentine, and the remainder of the cheek teeth erupts normally.[40] Less commonly, apical infection and death of the tooth occurs and occlusal pulpar exposure can later be recognized in the remaining larger (medial) fragment. This remaining dead dental fragment will likely have to be removed within a year or so when it loosens or if it causes clinical signs of apical infection, such as sinusitis.[7]

Midline (sagittal) fractures of the maxillary cheek teeth occur less commonly than slab fractures, and the 09s are again most

Figure 30-22. This mandibular tooth had a sagittal fracture with subsequent food impaction and displacement. Both parts of the fractured (and now carious) cheek tooth were orally extracted with little difficulty (see extractor forceps marks on tooth).

commonly affected.[39,40] This type of fracture is believed to be secondary to advanced infundibular caries, with coalescence of two carious infundibula leading to mechanical weakening, followed by fracture of the cheek teeth. Infection of the underlying pulp and periapical aspect of the tooth commonly ensues. Maxillary sinusitis more commonly accompanies these midline sagittal cheek teeth fractures because the infection often spreads outside the alveolus. If clinical signs of apical infection occur, all parts of the fractured tooth should be extracted, *per os* if possible, even though the clinical crown is weakened.[41] This can best be performed by cleaning out the food in the fracture site and using cheek teeth extractors to close the two fragments together, followed by gently rocking the combined fragments until the tooth is loose. If much concurrent periodontal disease is present, extraction is usually easy (Figure 30-22).

Endoscopic assessment of the affected sinus for the presence of inspissated material, and its subsequent removal via sinoscopy or sinusotomy, is indicated.[42,43] Sinus lavage is adequate if sinusitis with liquid exudate is present. In the absence of clinical signs of apical infection, extraction of the looser fracture fragment (usually the lateral fragment) is sufficient to alleviate clinical signs such as quidding.[40] The remaining stable medial dental fragment should be reduced in height to prevent its medial displacement caused by normal masticatory forces. In the absence of apical infection, the retention of a large dental fragment is beneficial, because it prevents drifting of adjacent cheek teeth into its space, for a few years perhaps, and also decreases the development of major overgrowths (stepmouth) on the opposite cheek teeth.

FILLING OF CARIOUS INFUNDIBULA

An endodontic type of treatment has been used for treating deep caries of the infundibulum, in an attempt to prevent further infundibular decay and to structurally strengthen these cheek teeth. This treatment may prevent affected cheek teeth from fracturing and also prevent caries from extending into the pulp, which could lead to apical infection. After cleaning out

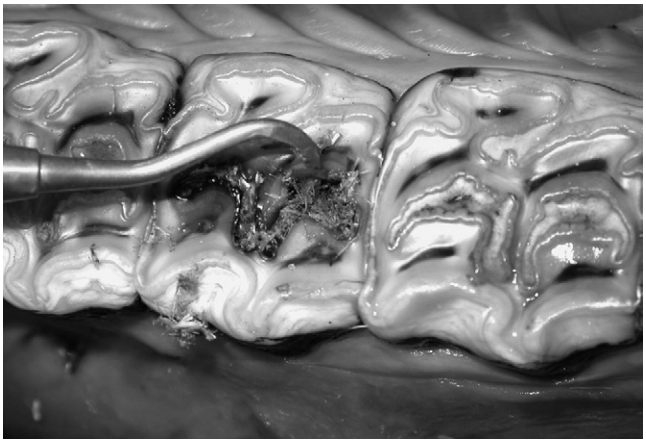

Figure 30-23. This maxillary cheek tooth specimen has impacted food in a carious (note dark infundibular enamel) infundibulum that is being removed with a dental pick.

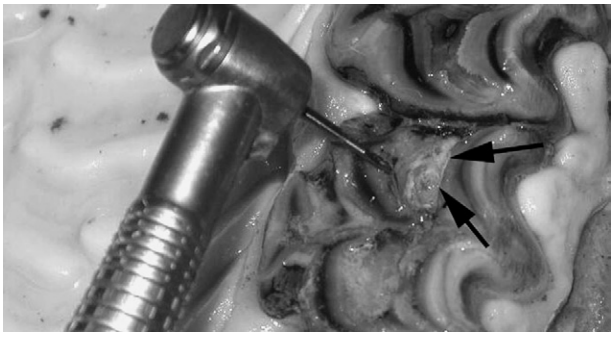

Figure 30-24. The carious infundibulum of Figure 30-23 has had all of the carious cementum and enamel removed with a high-speed dental drill *(arrows)*. The caudal infundibulum is filled with drill coolant water.

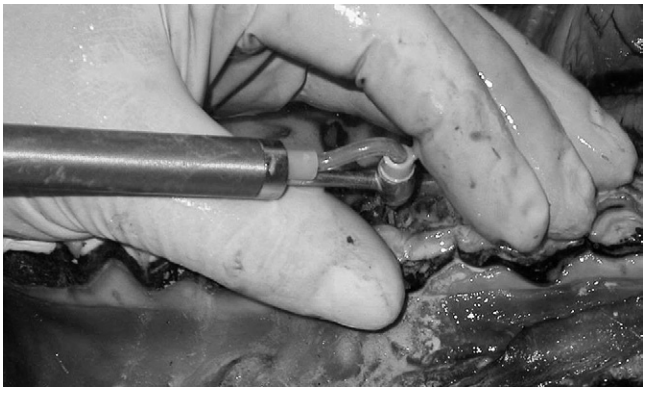

Figure 30-25. A high-pressure aerosol containing microfine aluminum powder (Equine Dental System, Pacific Equine Dental Institute, El Dorado Hills, CA) is being used to clean out a carious infundibulum in this specimen.

food material with a dental pick (Figure 30-23) or high-pressure aerosol or water lavage, the carious cementum in the infundibula is removed insofar as is technically possible using a dental drill (Figure 30-24) or by high-pressure aerosol abrasion with fine silica or aluminium powder (Figure 30-25).

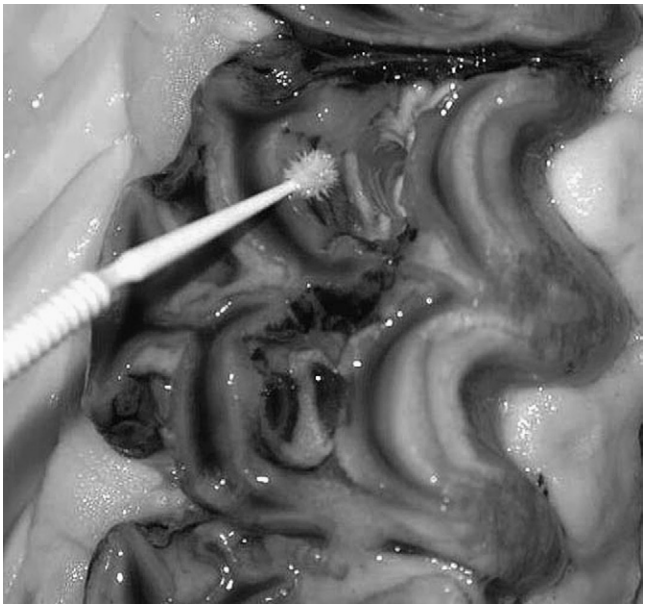

Figure 30-26. The cleaned infundibulum, which has also been acid-etched, washed, and dried, is now having bonding material applied with a flexible brush.

Figure 30-27. The prepared infundibulum has now been filled with a modern, self-curing, composite restorative material.

The cleaned infundibulum is flushed with sodium hypochlorite and acid-etched; then a bonding agent is applied (Figure 30-26) before it is filled in layers with a modern composite restorative material (Figure 30-27) as previously described (for incisor fractures). Because access to these carious infundibula (usually in the upper 09s) is difficult, it is not always possible to fully remove all carious cement (or enamel) and debris deep in the infundibula. Additionally, some infundibula have large dilated cemental defects deep (e.g., more than 5 cm) in the reserve crown that cannot by physically accessed from the occlusal surface.[44] Therefore it is unclear whether this therapy can always prevent extension of caries into the adjacent pulp or apex, but it should render the affected cheek teeth more mechanically sound and thus help prevent sagittal fractures.

Although encouraging anecdotal information is available, no objective data are yet available on the long-term value of this treatment.

DENTAL (ODONTOGENIC) TUMORS

Dental tumors are rare in horses, but when present they can mimic apical infections (i.e., with the presence of maxillary and mandibular swellings that later may become much larger than the swellings commonly associated with apical infections). Additionally, these swellings rarely develop sinus tracts, and they are usually firm and painless. Dental tumors include ameloblastomas, which are noncalcified epithelial tumors derived from the epithelium that forms enamel. A similar tumor, an ameloblastic odontoma, does induce calcification of adjacent mesenchymal tissues (and reciprocally of enamel epithelium) and so also contains dentine, cementum, and enamel. The appearance of these tumors can therefore vary from a noncalcified, polycystic, fibrous type of tumor to growths containing such tissues along with calcified dental tissues.

Dental tumors also include a variety of calcified tumors from dentinal tissues (odontoma) or cement (cementoma) or more commonly combinations of all three dental components (compound odontoma or, as noted earlier, ameloblastic odontoma).[45,46] Affected horses typically present with slowly growing, hard focal mandibular or maxillary masses that are usually very radiodense. The prognosis depends on their size and whether they are defined enough from the surrounding bones to allow complete surgical excision.[45-47]

CHEEK TEETH PERIAPICAL INFECTION

Apical (true roots may not even be formed at the time of infection) infections of cheek teeth may occur secondary to previously discussed abnormalities, including dental fractures or descending periodontal disease (e.g., beside a diastema). Additional proposed etiologies include bloodborne infection of pulp (or anachoresis), possibly predisposed to by vertical impaction of the rostral cheek teeth, which subsequently develops hyperemic, enlarged eruption cysts; and deep infundibular caries with extension of infection from the infundibulum into the pulp cavity (maxillary cheek teeth only).[48] Two studies have more clearly defined the causes of equine mandibular and maxillary cheek teeth apical infections.[5,6] The described equine cheek teeth pulp nomenclature[49] that has already been modified to improve its clarity[5] (see Figure 30-3) can be used to identify individual pulps in mature cheek teeth. Regardless of its etiology, if an apical infection (with death of pulp) has been present for many months, the secondary dentine normally worn away on the occlusal surface by mastication is not replaced and therefore occlusal pulpar exposure occurs over all pulp horns (Figure 30-28). Examination for pulpar exposure using a dental mirror or endoscope and a fine steel probe is simple and represents a currently underused but valuable clinical test for establishing dental viability (Figure 30-29).[50]

In the early stages, an anachoretic infection may remain confined to the apex, and all the pulp horns (or the common pulp in immature cheek teeth) may remain vital. At this early stage of infection, antibiotic treatment (e.g., 2 to 4 weeks of trimethoprim/sulfonamide) may be effective, but no factual data are available on the efficacy of such antimicrobial therapy. Surgical curettage of the affected apex has also been used with varying success, possibly having led to some failures that further compromised pulpar blood supply by damaging the apical vasculature.[48,51] In the later stages of apical infection, the common pulp or just some of the individual pulp horns may become necrotic. At this stage, dental extraction or possibly endodontic therapy is required.

Anachoretic apical infections occur mainly in younger horses, which have long reserve crowns and largely healthy periodontal membranes. Consequently, the infection cannot drain into the oral cavity via a periodontal route but instead spreads to the apical aspect of the alveolus and and from there to the adjacent supporting bones. Mandibular apical infections are invariably accompanied by unilateral, painful, ventral mandibular swellings that often develop external draining tracts. Infections of the

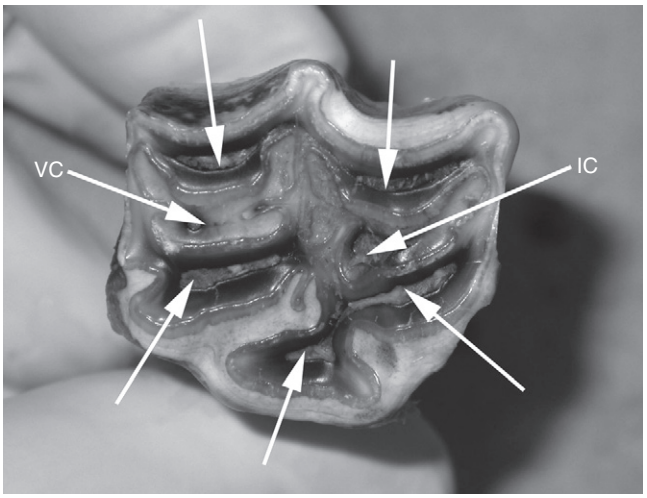

Figure 30-28. The occlusal surface of a cheek tooth extracted because of apical infection shows pulp exposure at all five pulps, all of which contain impacted food material *(arrows)*. One infundibulum contains a distinct vascular channel *(VC)*, and another one has localized clinically insignificant infundibular cemental caries *(IC)*.

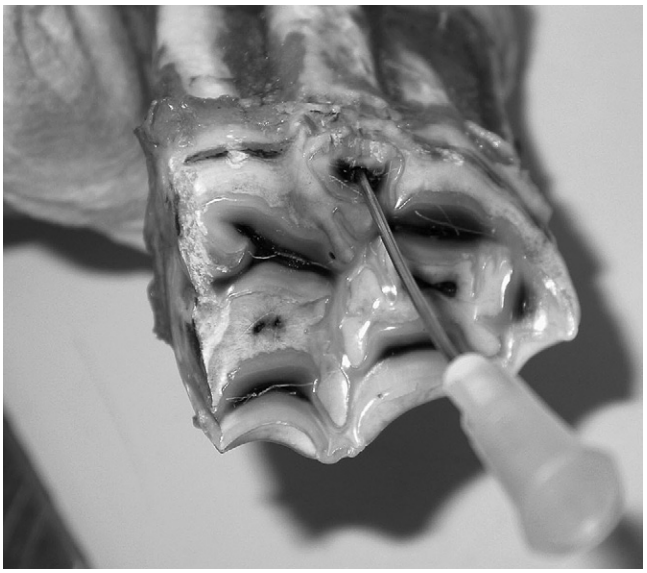

Figure 30-29. The occlusal surface of this cheek tooth (06s have six pulp horns) has intact secondary dentine over five pulp cavities but has a pulp exposure over one (pulp horn five, with needle inserted). Some minor discoloration is present in both infundibula.

upper 06 and 07 (occasionally the 08) cheek teeth cause focal swellings of the rostral maxilla (rostrodorsal to the rostral aspect of the facial crest), which are almost pathognomonic for such dental infections. Some of these horses also develop an external sinus tract or, less commonly, a tract drains into the nasal cavity. Apical infections of the caudal three or four maxillary cheek teeth (08s to 11s) usually result in a secondary maxillary sinusitis, with a chronic, malodorous, unilateral nasal discharge, and marked facial swelling. External draining tracts are rare with this type of sinusitis. In older horses, apical infections commonly drain through the shorter periodontal membrane into the mouth, and therefore swellings of the supporting bones or sinus tract formation seldom occur.

A thorough clinical examination of suspect sinusitis cases should be performed, including an intraoral examination for evidence of abnormalities, such as cheek teeth fractures, gross infundibular caries, diastemata, deep periodontal disease, or occlusal pulpar exposure (see Figures 30-28 and 30-29). Radiographic evaluation of apical infection should include 45-degree ventrolateral-lateral oblique projections for imaging mandibular cheek teeth apices and 30-degree dorsolateral-lateral oblique projections for imaging maxillary cheek teeth apices. The interpretation of equine dental radiographs is often difficult because of the frequent presence of overlying anatomic structures and because of combinations of lucent areas (from demineralization of apices and adjacent alveoli) and radiodense areas (from apical hypercementosis, alveolar sclerosis, and surrounding soft tissue inflammation). Interpretation is especially problematic in early apical infections in young horses where radiolucent normal eruption cysts have many radiographic similarities to apical infections.[32] If a sinus tract is present, as often occurs with mandibular or rostral (06s to 08s) maxillary cheek teeth infections, it is essential to obtain radiographs with a metallic probe *in situ* to confirm the presence of apical infection (Figure 30-30). This procedure also provides surgical landmarks (helpful if the infected tooth is to be extracted by repulsion). Similarly, metallic markers (e.g., skin staples) should be placed over areas of maximal facial swelling to establish possible anatomical relationships between facial swellings and the underlying cheek teeth apices. When available, computed tomography imaging is the optimal technnique to allow these complex three-dimensional structures to be more fully assessed. Endoscopic examination of the sinuses (sinoscopy) is helpful, as it sometimes demonstrates normal alveoli and so indicates that the sinusitis present is not of dental origin. The goal of these examinations is to definitively confirm whether any of the cheek teeth are diseased and need to be extracted.

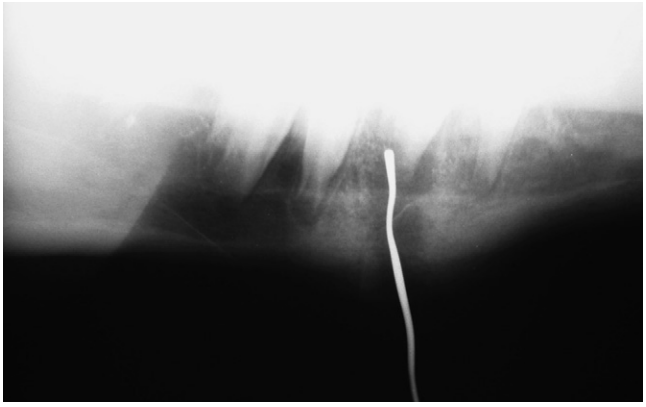

Figure 30-30. This radiograph shows an apically infected mandibular cheek tooth, with thickening of the underlying mandible, that also contains a lytic track leading to its caudal root. The caudal aspect of this root has been destroyed and is thickened more dorsally. The probe inserted up the sinus tract further confirms that infection is present in this cheek tooth.

When doubt exists about whether apical infection is present, scintigraphy may provide additional evidence. If any doubt still remains following all investigations, conservative therapy should be initiated—for example, a 2- to 4-week course of oral potentiated sulfonamides (possibly along with oral metronidazole therapy) for suspected mandibular infections and rostral (06 to 08) maxillary cheek teeth apical infections, and maxillary sinus lavage and similar antibiotic therapy for suspected caudal (09 to 11) maxillary cheek teeth apical infections. Failure to respond to this conservative therapy should prompt a further clinical and radiographic evaluation for the presence of apical infection. Serial radiographs of cases with dental sinusitis often later show definitive dental changes that were not present in earlier radiographs. Only when definite evidence of dental infection is present should dental extraction be considered.[19,48]

Treatment of Disorders of the Cheek Teeth

EXTRACTION OF CHEEK TEETH

Most cheek teeth apical infections occur in younger horses, and in these cases the infection is usually confined locally to the apical area, and great mechanical force is required to break down the remaining extensive, healthy periodontal membranes. Consequently, extraction of a long-crowned equine cheek tooth is a major surgical procedure with many possible immediate and delayed sequelae.[41] Oral extraction is the technique of choice for removal of most equine cheek teeth, as it can be performed in the standing horse, does not require surgery of the supporting bones, and greatly reduces postoperative complications compared to the repulsion or buccotomy technique.[41]

Oral extraction of cheek teeth

The availability of today's safe and effective sedatives and analgesics is a major reason for the revival of the oral extraction techniques. Most of the instrumentation has remained unchanged for over 100 years.[19,41,52,53]

A prerequisite for oral extraction of equine cheek teeth is excellent chemical restraint of the horse. This can be achieved by a combination of an α_2-adrenergic receptor and butorphanol (or morphine). A detomidine continuous-infusion technique has also been used successfully (loading doses of 4 mg IV of detomidine and 10 mg IV of butorphanol are given followed by 2 drops per second of a drip that contains 14 mg of detomidine in 250 mL of saline). Local anesthesia of the mandibular and infraorbital nerves can be used; for example, the upper 06s or 07s can be anesthetized by inserting a 5-cm, 21-gauge needle 3 to 4 cm caudad in the infraorbital canal and then slowly injecting 3 to 5 mL of lidocaine. The mandibular nerve, which is sensory to all mandibular teeth, can be anesthetized as it enters the mandibular canal on the medial aspect of the mandible. The mandibular foramen lies at the intersection of a vertical line at the caudal limit of the orbit with a line parallel to the occlusal surface of the cheek teeth. After subcutaneous local anesthetic infiltration and aseptic skin preparation, a 15-cm (6-inch), 18-gauge spinal needle is "walked" up the

periosteum of the medial aspect of the mandible and 20 to 30 mL of lidocaine is deposited at the site and 1 to 2 cm dorsocaudally to it.

With more difficulty, the maxillary branch of the trigeminal nerve can be anesthetized as it enters the caudal aspect of the infraorbital canal. After aseptic skin preparation, a 9-cm (3½-inch) spinal needle is inserted caudal to the highest point of the zygomatic arch and is then walked ventrorostromedially down the orbital aspect of the frontal bone to the caudoventral aspect of the orbit, where 20 to 30 mL of lidocaine is deposited. A simple, recently described technique to anesthetize the maxillary nerve (after aseptic skin preparation) is to insert a needle at right angles to the skin immediately ventral to the zygomatic process between the middle third and caudal third of the orbit. The needle is inserted 30 to 35 mm (3 to 3.5 cm) through the masseter muscle, and a change in consistency is detected as it enters the extraperiorbital fat body. The needle is advanced 15 to 20 mm (1.5 to 2 cm) further and 20 mL of local anesthetic is inserted.[54] It may take 20 minutes or so for the maxillary nerve to become anesthetized.

For oral cheek teeth extraction, the horse should be restrained in stocks with its head on a headstand or suspended in a dental head collar (see Figure 30-2). At least one assistant is needed to stabilize the head and help with the extraction. A good headlight is also required, especially when extracting caudal cheek teeth, to absolutely ensure that the correct cheek teeth are separated and that the correct tooth is subsequently correctly grasped with foceps and later extracted. In many horses, the medial (palatal) aspect of the maxillary cheek teeth contains very little exposed crown, with the gingival margin in some horses lying just a few millimeters below the occlusal surface. In such cases, a long-handled, pointed metal dental pick is used to detach the gingiva around the affected tooth to the level of the alveolar crest on the medial aspect of the tooth. This procedure normally exposes enough of the dental crown to allow cheek teeth extractors to be firmly applied on both the lateral and medial aspects of the tooth to be extracted.

A narrow-blade cheek teeth separator ("molar spreaders") can now be slowly inserted into the interdental spaces, first in front of and then caudal to the affected tooth (Figure 30-31). The separator should be kept in place for about 5 minutes to excessively stretch and therefore damage the periodontal ligaments. A series of cheek teeth separators with increasingly wider blades can then be used to further stretch the periodontal ligaments. When extracting an 07, separators should not be used between the 06 and 07 in case the (rostrally unsupported) 06 is excessively displaced rostrally and loosened. Separators must also be cautiously used when extracting caudal cheek teeth in horses with a marked curve of Spee, because the vertical blades of the separator will not fit into the nonvertical interdental spaces of such cheek teeth and may instead fracture them.[19,41]

A cheek teeth extractor is subsequently firmly attached to the crown of the diseased tooth and the cheek tooth is rocked in the horizontal plane, initially very gently (Figure 30-32). After a variable period (20 to 120 minutes), depending on the extent and health of the periodontal membranes, a "squelching" sound is heard. The tooth can now be more easily rocketed with the extractor, and foamy blood will appear at the gingival margin. The tooth usually becomes digitally loose as the squelching sounds increase. Only at this stage should a fulcrum be placed on the occlusal surface of the tooth rostral to the infected tooth. Vertical pressure is now exerted on the forceps, drawing the affected intact tooth from the alveolus. With caudal mandibular cheek teeth, it may be safer not to attempt elevation with a fulcrum, in case this vertical force fractures the obliquely positioned teeth. Instead, the tooth should be loosened digitally and extracted in a rostrodorsal direction using manual force (Figure 30-33). Unlike repulsed cheek teeth, the apices of extracted cheek teeth are virtually always intact (Figure 30-34).[41]

If no sinus tract or secondary sinusitis is present, the empty alveolus should be lavaged with saline, dried, and allowed to develop a blood clot (by gentle curettage if necessary) that will speed up alveolar healing. The alveolus can subsequently simply be filled with one or two cotton swabs (sponges) that contain metronidazole or another antibiotic (Figure 30-35), to prevent long fibrous food from being trapped in very deep alveoli before it granulates closed. In older horses with shallower alveoli (less than 5 cm deep), no alveolar packing is required. The alveolus should be checked 2 to 4 weeks later and the swab(s) removed if still present. The alveolus, if still patent, should now also be digitally palpated to detect roughened areas, which are usually

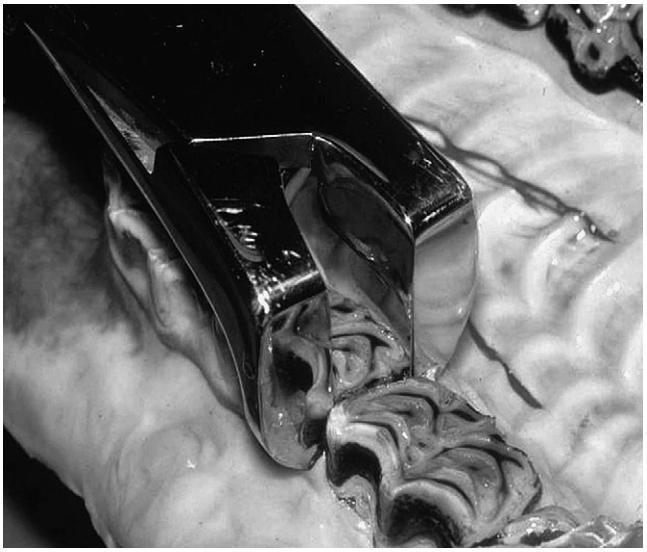

Figure 30-31. A narrow-blade, cheek tooth separator is being inserted between two cheek teeth in this specimen.

Figure 30-32. A Routeledge-type cheek tooth extractor applied to a mandibular cheek tooth (308), in the latter stages of dental extraction, when this cheek tooth has been elevated 2 to 3 cm into the oral cavity.

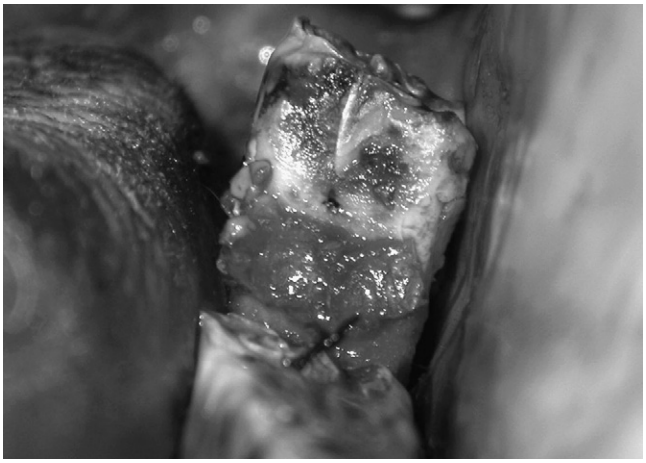

Figure 30-33. The final stages of oral cheek tooth extraction shows a mandibular cheek tooth that has been elevated into the oral cavity. It can be digitally removed at this stage.

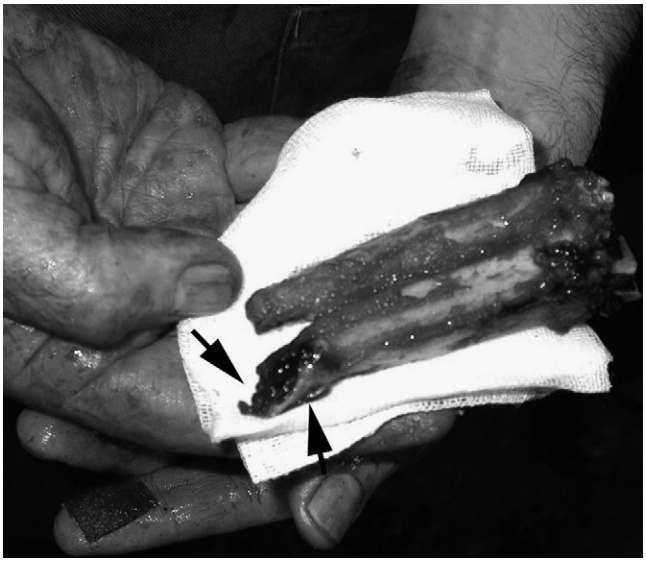

Figure 30-34. The extracted mandibular cheek tooth (shown in Figures 30-31 to 30-33) has extensive, healthy periodontal covering over all of the reserve crown and apex, except at the tip of the thickened, infected root *(arrows)*.

caused by sequestration of conical areas of alveolar cortex. If detected, such loose alveolar fragments should be digitally removed or curetted. Specialized long-handled, right-angled alveolar curettes are necessary to effectively curette the more caudal alveoli. Alveoli that still have a deep, large lumen may have a swab replaced for a further 2 weeks before being similarly rechecked.

If an external sinus tract was present before extraction, the bone at the base of the tract can be gently curetted percutaneously, and the tract will usually spontaneously heal within a few days. If the sinus tracts are wide or if gross alveolar infection is present, it is advisable to seal off the oral aspect of the alveolus with dental wax or an acrylic plug and to irrigate the sinus tract with a dilute povidine-iodine solution for a couple of days.

Oral extraction of the cheek teeth can also be performed on horses with infection of the caudal maxillary cheek teeth, which

Figure 30-35. Surgical gauze, impregnated with antibiotics, is being inserted into a mandibular cheek tooth alveolus. It will be left in place for a couple of weeks to prevent impaction of food deep within the alveolus.

have a secondary sinusitis. If the apical aspect of the alveolus digitally feels intact after cheek teeth extraction (as is almost always the case), it can simply be packed with an antibiotic-impregnated swab, as described earlier. If the alveolus appears disrupted (very rare), an acrylic plug should be inserted between the two adjoining cheek teeth (as described later) to prevent the development of an oromaxillary fistula. In all cases of maxillary sinusitis caused by dental infections, a separate small (e.g., 10-mm diameter) portal should be made in the ipsilateral frontal sinus to allow sinoscopic assessment for the presence of inspissated pus that should be removed by transendoscopic techniques if possible, using biopsy forceps, baskets, and high pressure lavage. This procedure will require fenestration of the ventral conchal bulla (see Chapter 43) if the 08 or 09 cheek teeth are infected and concurrent rostral maxillary and ventral conchal sinus infection are consequently present.[42,43] Alternatively (but more invasively) inspissated pus can be removed by sinusotomy, preferably in the standing horse to reduce intraoperative hemorrhage. The sinoscopy portal described earlier should be used for postoperative lavage of the infected maxillary sinus twice daily, with 5 L of very dilute povidine-iodine or saline solution for about 7 days or until the lavage fluid is odorless and runs clear.

After successful oral extraction of cheek teeth by experienced surgeons, postoperative complications are rare (they occur in about 10% of cases) and are usually of a minor nature—for example, nonhealing alveoli caused by alveolar sequestra, or localized osteitis. Most can be resolved by digital sequestrum removal or alveolar curettage, using long-handled equine dental curettes and antibiotic therapy.[41,55]

Repulsion

The standard method for extracting equine cheek teeth in the late 19th and most of the 20th century has been cheek teeth repulsion under general anesthesia.[19,52] This procedure has the expense and the inherent risks for morbidity and mortality of equine general anesthesia, and it is also associated with a high level of postoperative complications. A surgical window (e.g., 2 cm in diameter) is made using a bone saw, trephine, or chisel adjacent to the infected apex. For mandibular cheek teeth

repulsion, this site is beneath the ventral mandible (Figures 30-36 and 30-37), and for repulsion of maxillary 06, 07, and occasionally 08 cheek teeth, it is on the dorsorostral aspect of the rostral maxillary bone. For the latter trephination, care must be taken to avoid the infraorbital canal and nerve. Larger (e.g., 5 to 6 cm^2) caudal maxillary bone flaps (hinged in a dorsal direction) can be used to perform a sinusotomy (see Chapter 43), and this is preferable to making a small sinus trephine opening for repulsion of the caudal maxillary cheek teeth. Such larger windows allow a visual and digital inspection of the apices of the 09 to 11 cheek teeth and subsequently their repulsion, if apical infection is confirmed. Infected apices lose the smooth, rounded, soft tissue–covered appearance of normal apices; rarely, sharp edges of exposed apical enamel or the edges of alveoli are palpable, and more commonly, local granulation tissue and mucosal inflammation are evident.

Intraoperative radiographs should always be obtained if there is any doubt about the site and the direction of placement of the repulsion punch (Figures 30-38 and 30-39). Radiographs should also be obtained after the repulsion, unless the surgeon is certain that the tooth has been fully removed. As wide a punch as possible should be used during repulsion, with offset punches most suitable for upper cheek teeth repulsion. Hammering a thin metal blade down the periodontal space, especially on the (flat) rostral and caudal cheek teeth margins, can greatly decrease the time and trauma necessary to repulse a cheek tooth.

After cheek teeth repulsion, it should be determined that the alveolus is free of dental or alveolar remnants by digitally and visually examining the extracted cheek teeth and by obtaining postoperative radiographs. The oral (occlusal) aspect of the alveolus should be thoroughly dried and sealed with a dental wax plug (after mandibular or rostral maxillary cheek teeth repulsions), or with an acrylic plug attached to the two adjacent cheek teeth (for caudal maxillary cheek teeth repulsions). The plugs should lie below the occlusal level; otherwise they will loosen quickly with the high continual forces of mastication. They should also not extend more than 3 to 4 cm into the alveolus, or they will delay alveolar healing and can be very

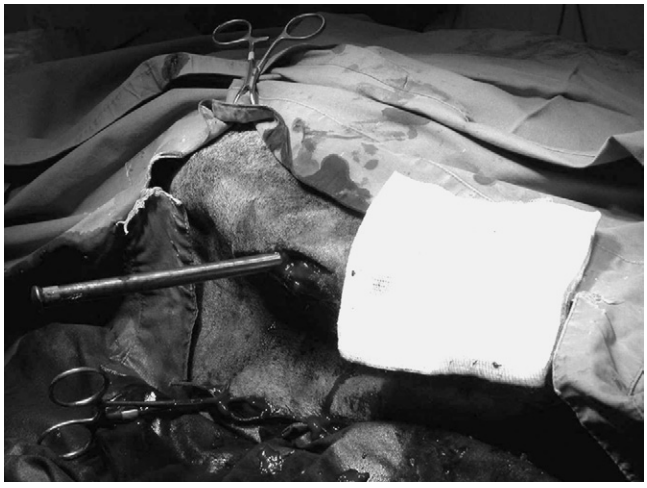

Figure 30-36. This horse is having a mandibular cheek tooth repulsed under general anesthesia. Note the steel punch in a ventral mandibular site.

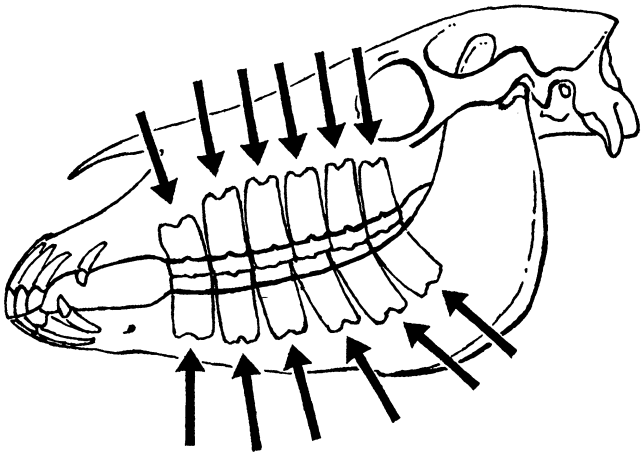

Figure 30-37. The direction of repulsion of the cheek tooth in a young horse.

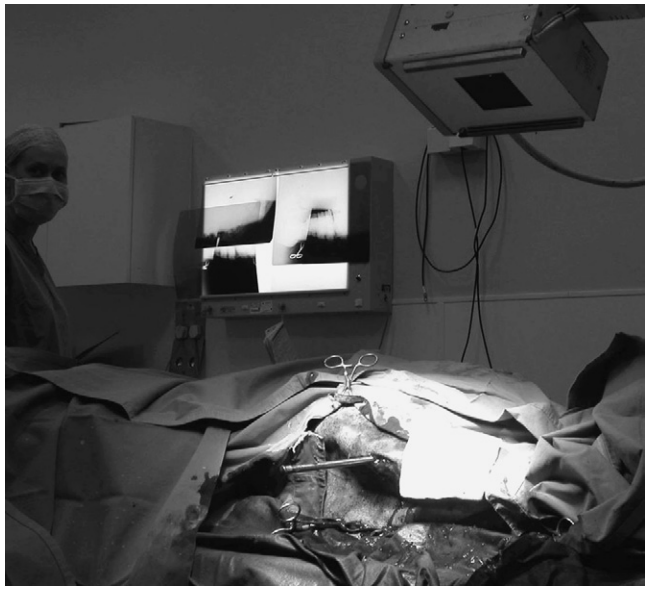

Figure 30-38. Intraoperative radiography of a horse having a mandibular cheek tooth repulsed. This radiograph is being obtained to confirm punch positioning and angulation to prevent iatrogenic damage to adjacent cheek teeth.

Figure 30-39. This intraoperative radiograph shows the punch correctly positioned over the affected apex and also shows that it is facing in a suitable direction to effectively remove this (09) cheek tooth.

difficult to remove. The surgical site used for repulsion of rostral maxillary or mandibular cheek teeth should be left open and irrigated (e.g., with saline or a very dilute povidone-iodine solution) for a few days, especially if they remain purulent or malodorous. Broad-spectrum antibiotics and NSAIDs should also be administered for at least 3 days after dental repulsions.

Complications commonly occur after repulsion of apically infected cheek teeth from younger horses, because much mechanical damage occurs to the alveolar and supporting mandibular or maxillary bones by the high forces and often prolonged nature of cheek teeth repulsion.[55] These problems include nonhealing alveoli as a result of residual dental fragments or alveolar sequestra; localized osteomyelitis; oronasal, orofacial, and oromaxillary fistulae; chronically draining facial tracts; damage to adjacent teeth; and chronic sinusitis. Some bone sequestration may be delayed and will not be detectable until many weeks following repulsion. A number of studies have shown that between 32% and 70% of cases of equine dental repulsion require additional surgical and nonsurgical treatments, with most complications following maxillary cheek teeth repulsions (Figure 30-40).[48,56-58] Careful radiographic assessment for the presence of dental or bone sequestra, osteomyelitis, adjacent damaged or infected cheek teeth, and chronic sinusitis helps (in cases where such problems are detected) in the selection of an appropriate treatment modality, such as alveolar curettage, replacement of loose alveolar plugs, sinus lavage, and antibiotic therapy.[55]

REMOVAL OF THE LATERAL ALVEOLAR PLATE (LATERAL BUCCOTOMY TECHNIQUE)

A further technique for equine cheek teeth extraction that has been used for over 100 years involves the removal of the supporting bone lateral to the alveolus. It is most suitable for extraction of the rostral upper three cheek teeth, but it has been adapted for extraction of the rostral three mandibular cheek teeth by the buccotomy technique and also for the more caudal mandibular cheek teeth by dissection through the masseter muscles.[19,58-60]

This is the most suitable technique for the rare cases of chronically (sometimes of many years' duration) apically infected cheek teeth, where progressive, dense cement deposition has occurred around the infected apex and adjacent reserve crown, making this area and its surrounding alveolus larger than the more occlusal aspect of the alveolus and thus mechanically preventing repulsion or oral extraction of the tooth. Under general anesthesia, a surgical approach is made directly through the skin and subcutaneous tissues to the lateral wall of the affected alveolus. A horizontal incision (to reduce the risk of cutting the buccal nerves) is made in the skin and soft tissues overlying the affected mandibular cheek teeth. The buccal nerve (and possibly the parotid duct) should be identified and isolated to prevent damage, if they cross the surgical field. A vertical incision is now made in the periosteum over the lateral aspect of the affected cheek teeth, and the periosteum is reflected. Through use of an oscillating bone saw or burr, the lateral wall of the alveolus is removed. The full length of the crown of the exposed diseased tooth is sectioned longitudinally with a diamond wheel or large solid carbide burr before the cheek tooth is extracted in sections.

The occlusal aspect of the alveolus is plugged with dental wax and the remaining alveolus is packed with an iodine-impregnated gauze bandage, which is gradually withdrawn through a small stab incision adjacent to the surgical site. A potential major disadvantage of this technique for extraction of the mandibular cheek teeth is that it can cause parotid duct rupture and buccal nerve damage with nasal paralysis.[61] In athletic animals, the former sequela is a disaster, as active dilation of the nostrils can no longer occur. Prolonged anesthesia is also required for this technique, which is a further disadvantage.

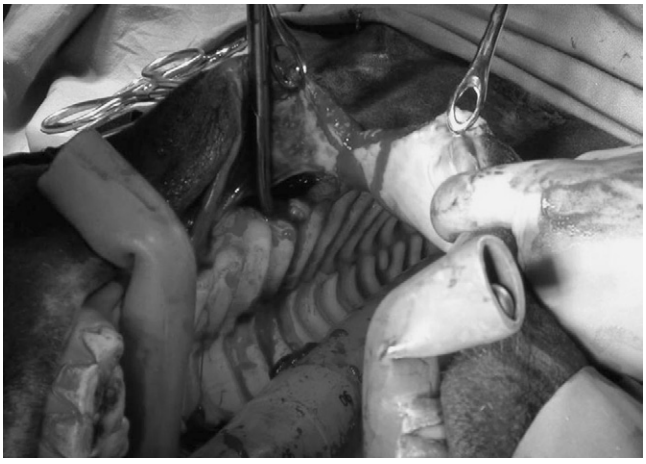

Figure 30-40. This horse had 206 and 207 repulsed some years previously, leaving it with a chronic, nonresponsive oro-nasal-facial fistula. After incision of the lips for access, a sliding hard-palate flap was used to close the large oral defect.

ENDODONTIC THERAPY

A number of workers have attempted to treat apically infected cheek teeth by use of endodontic (pulp canal) therapy. A major advantage of endodontics, if successful, is that the infected tooth is preserved and thus continues to erupt normally (at about 2 to 3 mm per year). This prevents the development of overgrowth ("stepmouth") of the opposing cheek teeth and drifting of the adjacent cheek teeth into the site of the extracted tooth. This drifting eventually causes focal overgrowths ("hooks") on the caudal and rostral aspects of the opposing cheek teeth row.

Equine cheek teeth endodontic treatment has been performed mainly through the apex of the affected cheek teeth, but it is also performed via an occlusal approach (as is used in brachydont teeth endodontics).[62-64] Endodontic treatment cannot be applied in cheek teeth with gross sepsis of the apex or where there is extensive periodontal disease (e.g., descending apical infections due to deep periodontal disease at a diastema). Apical endodontic therapy is performed via surgical approaches through the mandibular or maxillary bones (Figure 30-41). A consequence of this approach is the need for prolonged general anesthesia and surgical exposure of the overlying bone and affected apex for endodontic therapy. The exposed apex is visually assessed, and grossly infected or discolored calcified dental tissue is removed by high-speed, water-cooled burrs.

The large, common pulp (in young horses) or individual pulp horns (five pulp horns in all mature cheek teeth, except the 06s, which have six, and 11s, which have six or seven [see Figure 30-3]) are subsequently assessed for viability and removed with barbed broaches if found to be nonvital. Any impacted vegetable material (as a result of occlusal pulpar

Figure 30-41. Apicectomy and retrograde endodontic treatment of an apically infected 108 being performed under general anesthesia. Following a skin incision, the maxillary bone dorsal and lateral to the infected apex has been removed to allow exposure of apex. Note the row of needles inserted subcutaneously ventral to the surgical site and the row of skin staples dorsal to this site to provide radiographic landmarks for the procedure.

exposure) is removed and the canals are then filed using long endodontic files until normal-appearing dentine is reached (as adjudged by the color of the filings, i.e., changes from discolored to white or cream-colored dentine). The pulp canal can be sterilized by irrigating it with 2.5% sodium hypochlorite solution (household bleach) followed by lavaging with water and air-drying. The canal lining is subsequently etched with a phosphoric acid preparation and lavaged and dried again. The surface is coated with a dental bonding agent (some agents need to be cured by UV light) and the pulp canal filled, in layers, as completely as possible, with a modern composite restorative material, some of which also need to be cured by UV light in stages. Air pockets may preclude complete filling of the occlusal aspects of some long, narrow equine cheek teeth pulp canals in mature horses.

Baker described an 84% success rate with (apical) endodontic therapy for mandibular 08s and 09s but poor success with maxillary cheek teeth.[62] Other clinicians have had poor results in trying to treat infected teeth with apical endodontic therapy, with some horses requiring two or three courses of treatment, under general anesthesia, sometimes taking over 2 hours per treatment.[63] The most comprehensive report to date has shown a positive response in only 58% of cases and partial success in 17%.[64] Such repeated and prolonged surgery under general anesthesia is costly and has safety considerations for the horse. It is believed that in younger horses, the presence of a very large common pulp cavity is one reason for the failure of this technique in this age group. Success may increase if endodontic treatment is reserved for more mature teeth (e.g., cheek teeth that have been erupted for at least 2 years). Specialized training and equipment (including long drills and reamers) are required for endodontic therapy of the long equine pulp canals. Additionally, the debate continues over what is the most suitable material (e.g., modern two-part composites, self- or light-cured; amalgam or glass ionomers) for pulp canal filling in horses. Endodontic therapy offers much potential benefit in treating apical infections, but until larger studies have more fully evaluated case selection criteria, long-term efficacy, costs, and the safety of this technique, its wider role in equine dentistry remains unclear.

More recently, endodontic therapy via the oral cavity has been advocated in cases of pulpar exposure on the occlusal surface of cheek teeth. However, many such horses have deeply impacted vegetable matter down their pulp canals[5,6] that may be impossible to fully remove, even with use of high-pressure silicate or aluminium powder aerosols, and after completely drilling out the full occlusal surface of these canals. After cleaning, exposed pulp cavities are sealed with restorative material as previously described. If the affected canal still contains food material or if inaccessible areas of caries have not been removed, it is possible that endodontically sealing off its occlusal drainage may transform a nonclinical apical infection to one with clinical involvement of the supporting bones and sinuses. Notwithstanding these theoretical arguments against occlusal endodontic therapy, some clinicians have anecdotally reported success with this therapy.

ORAL CAVITY SOFT TISSUE TRAUMA

The soft tissues of the oral cavity are susceptible to traumatic injuries by harness bits or other oral tack, sharp external objects, blows to the head, injury during recovery from general anesthesia, and iatrogenic damage during intraoral procedures, such as administration of oral medications, dental extraction, or transoral epiglottic entrapment release.[65-69] The soft tissues of the face and oral cavity have a tremendous capacity for repair. Minor superficial lacerations of the mucosa, lips, and tongue can heal effectively by second intention, usually within 2 weeks, without leaving a scar. Management may entail flushing the oral cavity after meals with an antiseptic solution, warm saltwater, or clean water and the use of NSAIDs. Larger wounds should be considered for surgical repair, to maintain tissue function, and for cosmesis. For these repairs, antimicrobial therapy may be needed in selected cases.

Tongue

Lacerations of the tongue occur occasionally and can be severe, with transverse lacerations more frequent than longitudinal ones.[65,70,71] The free portion of the tongue is usually involved because of bit location and because this part has more exposure to the external environment. Clinical signs include oral hemorrhage, ptyalism, inappetence, anorexia, dysphagia, malodorous breath, pyrexia, and tongue protrusion from the mouth. Management of tongue lacerations is guided by the severity, duration, and location of the injury. Partial glossectomy, primary wound closure, or secondary wound healing are approaches to treatment. Surgical procedures are most easily performed on the anesthetized patient; however, the tongue can be operated on in the standing horse with effective sedation and infiltration of local anesthetic. Traction on the tongue for exposure can be achieved by placing towel clamps in the tongue caudal to the laceration or by using a gauze snare at this site, which also serves as a tourniquet.[70,72]

Partial glossectomy is reserved for cases in which the rostral tongue tissue is devitalized and minimal attachment is left between the severed section and the remaining body. Tissue color, temperature, and evidence of bleeding at an incision can

be used to assess viability. Observation of fluorescence after intravenous administration of sodium fluorescein allows a more objective assessment of tissue vascularization.[72] After amputation, the remaining stump is meticulously débrided of nonviable tissue. Mucosal-to-mucosal closure of the stump is not imperative but can be performed to aid hemostasis and hasten wound healing.[71] Correct dorsal to ventral apposition is assisted by removing a wedge of intervening musculature and closing the created space with multiple rows of interrupted absorbable 2-0 or 0 sutures (walking sutures, see Figure 16-9, *E*). The mucosal edges are subsequently closed with buried sutures of a similar size (Figure 30-42).[70] Involuntary loss of saliva from the mouth may be observed after amputation of a large part of the free portion of the tongue.[71]

Primary closure of severe tongue lacerations is encouraged whenever possible. The wound edges are débrided of necrotic and contaminated tissue and lavaged vigorously. A multilayer closure to eliminate dead space is recommended.[65,66,70,72] To relieve tension on the closure, vertical mattress sutures are preplaced deep in the muscular body of the tongue with absorbable or nonabsorbable size 0 or 1 monofilament suture material. Buried rows of simple interrupted 2-0 to 0 monofilament absorbable sutures are subsequently used to appose the muscles, obliterating dead space. The vertical mattress sutures are tied, and the lingual mucosa is apposed with simple continuous or interrupted vertical mattress sutures (Figure 30-43).

Second-intention wound healing for management of tongue lacerations is a viable option, particularly when economic constraints preclude surgical repair and for chronic and less-extensive lacerations. Oral lavage with a clean antiseptic solution two to three times a day and careful attention to the horse's ability to eat and drink are indicated. Lacerations that have healed by second intention but result in poor tongue functionality can be reconstructed using primary closure techniques after sharp débridement of scar tissue.[67,70]

After lingual surgery, most horses eat normally, and temporary feeding via a nasogastric tube is rarely required. Gruels of pelleted feeds mixed with water, bran mashes, and wetted hay can be given before introducing drier feeds.[65,69,70] NSAIDs are administered, and antimicrobial therapy is used according to the level of tissue devitalization. Nonabsorbable sutures are removed after 2 weeks. Postoperative complications include excessive swelling of the tongue and suture dehiscence. The cosmetic appearance is usually highly acceptable.

Lips

Lip trauma occurs from protruding rigid objects in the horse's environment, such as metal buckets, nails, bolts, and hooks, or from iatrogenic bit damage.[66,67] In cases with major disruption, surgery is indicated to preserve lip function and cosmetic appearance. Injuries with extensive tissue contusion and devitalization should be managed by delayed primary closure to optimize the amount of healthy tissue for suturing; otherwise, most lacerations can be repaired at presentation.[66] General

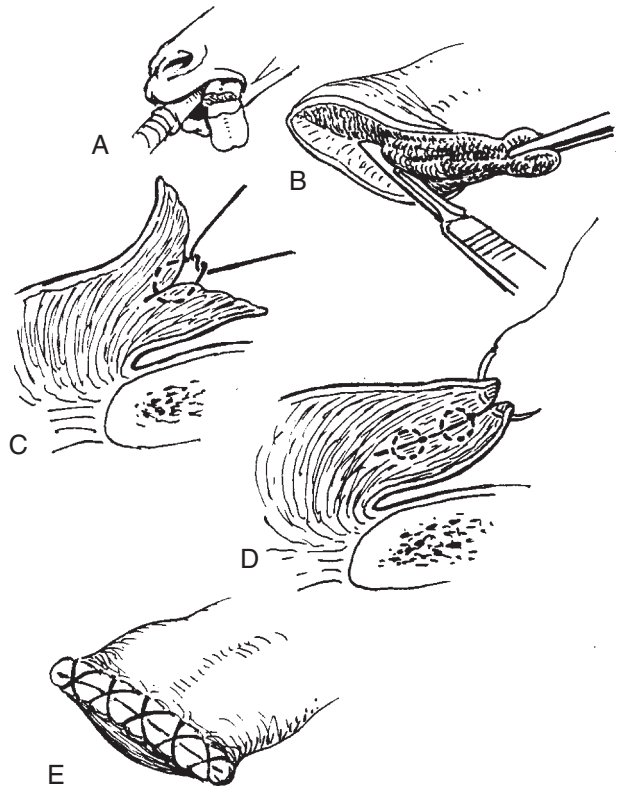

Figure 30-42. Closing the tongue stump after partial glossectomy. After amputation of the severely lacerated tongue **(A),** a wedge of intervening musculature is removed **(B).** The created space is closed with multiple interrupted rows of sutures **(C, D)** before closing the mucosa **(D, E).**

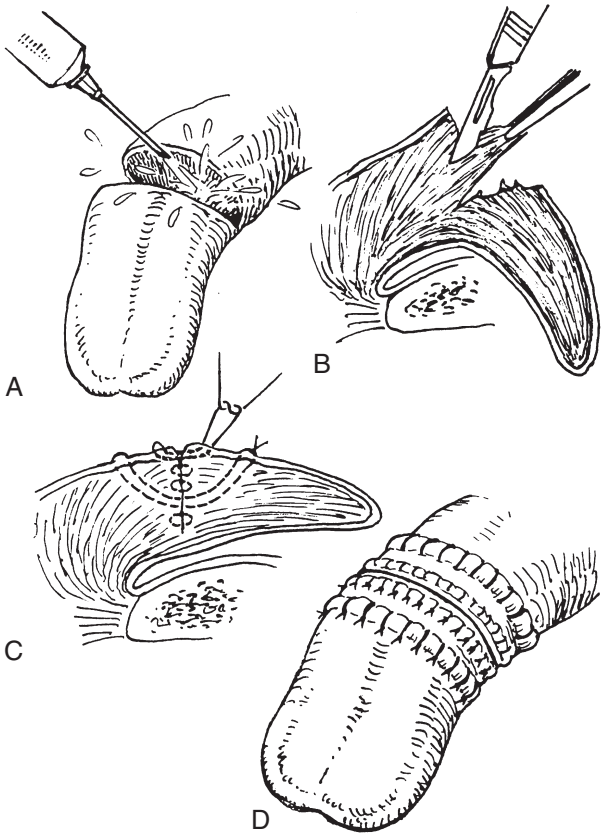

Figure 30-43. Tongue laceration repair. After vigorous lavage of the wound **(A)** and débridement of devitalized tissues **(B),** the laceration is closed with multiple layers of interrupted sutures **(C, D).** The large vertical mattress tension-relieving sutures are placed first, deep in the tongue musculature.

anesthesia facilitates a meticulous repair, but standing surgery is possible with regional anesthesia techniques. The wound edges are prepared routinely by sharp débridement and lavage. The lips are highly mobile tissues, and close adherence of the mucosa and skin to underlying musculature results in excessive motion at suture lines during prehension. This leads to a high incidence of dehiscence unless techniques are employed to stabilize the repair.[66,67,72]

To reduce motion on the suture lines, the margins of the skin and oral mucosa are sharply undermined for 1 to 1.5 cm from the edges of the wound. Vertical mattress 0 to 1 nonabsorbable sutures are subsequently preplaced from the extraoral side through the lip musculature and tied over quills of soft rubber tubing[66] or through buttons (see Figure 16-9, *C*).[67] The mucous membrane is closed with simple continuous or interrupted 2-0 monofilament absorbable suture. The skin is apposed with simple interrupted or vertical mattress 2-0 to 0 nonabsorbable monofilament sutures. At the mucocutaneous junction, a vertical mattress pattern is recommended for precise apposition.

Chronic lip lacerations are reconstructed using similar principles as for the repair of acute injuries. A three-layer closure is employed, with the layers being created by sharp dissection of the skin and mucous membrane from the intervening lip muscle and granulating scar tissue. When repairing lacerations involving the commissure of the lips, additional vertical mattress tension-relieving sutures are placed rostral to the primary repair for increased support in this highly mobile area (Figure 30-44).[66] For an extensive lower lip laceration with loss of tissue, a rotational flap may be used.[73] Avulsions of the lower lip should be supported with large mattress sutures passed through the mandibular symphysis. Wire or nonabsorbable suture material is threaded through holes drilled from the outside of the lip and chin to exit caudal to the incisors in the oral cavity, and stents of soft tubing or buttons are placed under the sutures on the oral and external sides.[66,67] Primary closure of the lip and gingival mucosa is performed if practical.

NSAIDs are administered, and the oral cavity is lavaged with a dilute antiseptic solution or water from a hose two to three times a day. Antibiotic administration is not required in most cases. A normal diet can be offered after surgery, and feeding by nasogastric tube or esophagotomy is unnecessary.[66] The stent sutures can be removed after 7 to 10 days and skin sutures after 2 weeks. Mattress sutures used for repair of avulsion injuries are removed 2 to 3 weeks postoperatively. The most common postoperative complication is dehiscence of the repair. Horses have a tendency to rub the repair site, which can be limited by muzzling or cross-tying.[67]

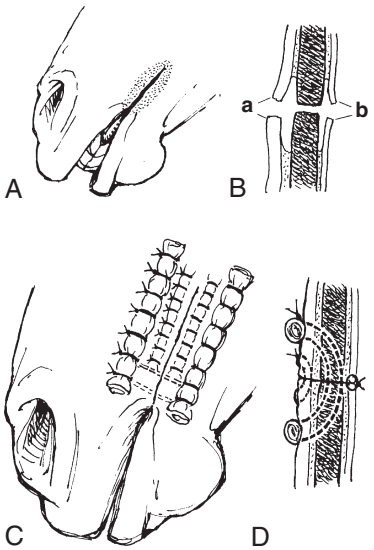

Figure 30-44. Repair of a laceration involving the commissure of the lips. **A** and **B,** After appropriate débridement and lavage of the laceration, the skin and mucous membrane margins of the laceration are undermined 1.0 to 1.5 cm (*stippled area* in **A**). **B** is cross-sectional view with *a*, skin; *b*, mucous membrane. Next, vertical mattress sutures tied over stent material are preplaced through the lip musculature before closing the mucous membrane and skin layers to reduce motion at the suture lines **(C, D).** Extra vertical mattress sutures can be placed rostral to the repair to further stabilize the site **(C).**

Cheek and Gums

Partial-thickness labial vestibule and buccal cavity lacerations are managed by second-intention wound healing with oral lavaging after meals and NSAIDs. Large, full-thickness injuries may be reconstructed to prevent orocutaneous fistula development. Repairing the oral aspect of the wound is difficult because of limited space. Suturing the wound from the external side, starting with the mucosal layer, is more practical. Tension-relieving vertical mattress sutures should be used for the musculature.

ORAL CAVITY FOREIGN BODIES

Various types of metallic, usually linear, foreign bodies can penetrate the soft tissues of the oral cavity after inadvertent ingestion, or penetration may occur iatrogenically during administration of oral medications.[69,74-76] Plant matter, such as grass awns or wood splinters, is also a frequent cause of foreign body reaction.[77] External clinical signs include focal or diffuse intermandibular, retropharyngeal, and facial swelling, depending on where the foreign body has lodged or what it is migrating through. Swellings typically have increased heat, and the horse has evidence of pain when they are palpated. Often, antibiotic therapy reduces the size of the swelling but it recurs when drugs are discontinued. A swollen, painful tongue; ptyalism; and partial to complete anorexia are the most common clinical signs of a tongue foreign body.[76] Other clinical signs may include halitosis, dysphagia, tongue paralysis, and fever. There may be a painful response and difficulty when attempts are made to open the jaw. Oral and oropharyngeal examinations (float the teeth before performing the latter to prevent lacerations of the operator's hands and arms) can reveal firm, painful swellings; the end of the foreign body; ulcerated mucosal surfaces where the foreign body has penetrated the tissue; or the site of spontaneous abscess rupture.

Diagnosis requires a combination of thorough history taking, external and oral examination, and imaging aids. Radiography is indispensable for detecting metallic foreign bodies, but care must be taken not to miss a fine, short structure. Open-mouth head radiographs can prevent tongue soft tissues from being superimposed over radiodense dental elements that may conceal a subtle metallic body (Figure 30-45). Ultrasonography is very useful to help pinpoint the exact location of a foreign body, and imaging via the intermandibular space and via oral direct

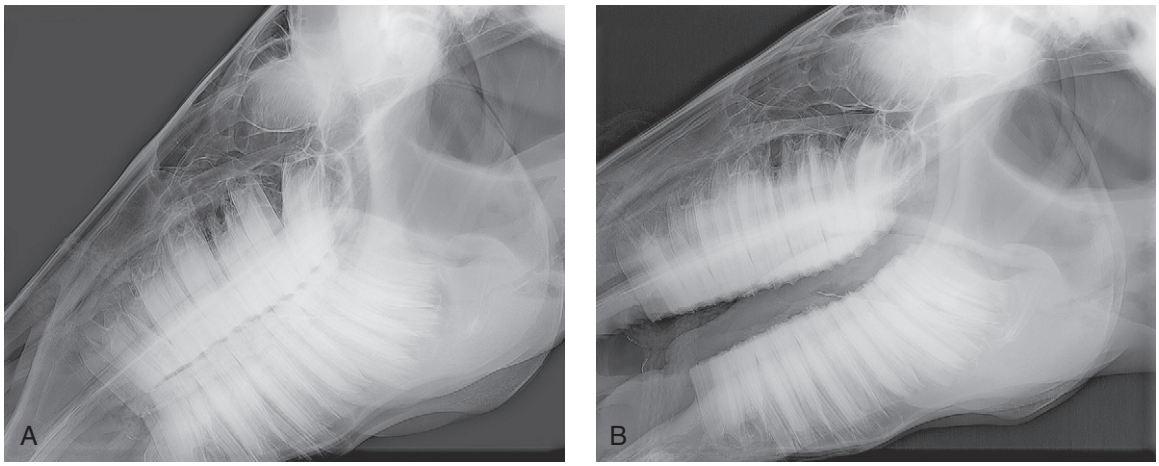

Figure 30-45. A, Closed-jaw, lateral radiographic view of a horse's skull. A barely discernable horizontal, linear opacity is visible superimposed under the mandibular cheek tooth (09s and 10s). **B,** Open jaw, radiographic lateral view of the same horse's skull. Separation of the dental arcades allows easy visualization of the opaque foreign body located in soft tissue (in this case, determined to be the tongue), surrounded by a lucent cavity.

contact with the tongue is recommended.[76,78] Direct tongue ultrasonography may be performed in the standing, sedated horse, with a speculum in place, or in a horse under general anesthesia. A 5 to 10 MHz rectal transducer provides excellent contact with the tongue surface. Ultrasonography is very useful for detecting the presence of lingual abscesses, and the combined imaging from two approaches identifies the best surgical access to the foreign body.[76] Nonmetallic foreign bodies such as wood splinters are best recognized with the help of ultrasonography.

Once a diagnosis is established, the treatment of choice is removal of the foreign body. In a few select cases, medical therapy alone may suffice if tongue function is normal and intralingual abscesses are minor or not present.[76] In other cases the foreign body can be removed manually if it is palpated during examination. Surgical approaches may have to be creative. An external approach to a foreign body that has migrated into the deep part of the masseter muscle requires care to avoid trauma to facial nerve branches, parotid duct, and blood vessels in that region. Foreign bodies associated with intraoral swelling may be approached by incising the mass on the oral side and draining exudate into the mouth. Digital or instrumental exploration and débridement of the cavity can then be performed. The cavity is lavaged and allowed to heal by second intention. Lingual foreign bodies can be very difficult to find and awkward to reach when they have implanted or migrated into the caudal body or base of the tongue.[75] Surgery can be associated with significant hemorrhage if large lingual vessels are invaded. An ulcerative defect on the tongue surface provides a useful starting point for exploring the necrotic tract that is associated with the path of the foreign body. A ventral approach between the hemimandibles is necessary in some cases and allows triangulation techniques to narrow down the field of exploration when a second instrument is also passed via the oral cavity into the tongue body. When necessary, the caudal body or base of the tongue can be exposed more effectively after a mandibular symphysiotomy.[75] An oral speculum, a bright light, and long-handled instruments are valuable aids. The mouth should not be fully opened with a speculum for prolonged periods of time (more than 30 to 45 minutes), to prevent muscle and temporomandibular joint soreness postoperatively. Intraoperative ultrasonographic, fluoroscopic, and radiographic guidance may be required to locate the foreign body. Oral endoscopy can aid visualization.

Surgical and necrotic tracts in the tongue are left to heal by second intention. Postoperative care may consist of a combination of oral lavaging, necrotic tract lavaging, and antibiotic and anti-inflammatory therapy. Ptyalism and tongue swelling can be a feature of the early recovery period. Soft feeds and gruels are fed after extensive tongue exploration, until the horse is more comfortable eating. The prognosis is excellent for complete recovery.

PERSISTENT LINGUAL FRENULUM

Ventral ankyloglossia (persistent lingual frenulum) is a very rare congenital condition in foals and may accompany other congenital craniofacial anomalies.[79] The tongue cannot be protruded normally because of a mucosal attachment between the ventral rostral free part of the tongue and the floor of the oral cavity. Difficulty with suckling may also occur. The condition is diagnosed by oral examination and is usually noted during the first routine neonatal assessment. Frenuloplasty is the treatment of choice, taking care not to incise the normal lingual frenulum. This may be performed under sedation and local anesthesia or under short-acting general anesthesia. The membrane is sharply transected with scissors. Electrocautery and laser division of the tissue are alternative approaches. With adequate release, the tongue should be easily extended from the mouth.

ORAL CAVITY SOFT TISSUE NEOPLASIA

Neoplasia of the oral cavity soft tissues is rarely encountered but is of considerable clinical significance when identified. The most common primary oral neoplasm is squamous cell carcinoma, affecting any of the mucosal surfaces.[80-82] Other primary or metastatic tumors include melanoma, fibrosarcoma, hemangiosarcoma, lymphosarcoma, rhabdomyoma, rhabdomyosarcoma, and chondrosarcoma of the tongue.[77,80,83-87] Clinical signs include ptyalism, halitosis, tongue protrusion, quidding, nasal

discharge with food material in it, dysphagia, inappetence, anorexia, and weight loss.[82,83,87,88] Tumor invasion of local bony and soft tissues is often advanced, and regional metastases may have already occurred before clinical signs become apparent and prompt veterinary attention. Manual examination and direct visualization (using a speculum and bright headlamp) or endoscopy of the oral cavity reveals most tumors. Caudal oropharyngeal tumors can be very difficult to see, even with oral endoscopy. They may also be difficult to palpate, and general anesthesia should be considered to facilitate thorough examination in select cases. Radiography can determine osseous involvement and soft tissue anomalies, and nasal endoscopy can indicate soft tissue masses on the tongue base displacing the soft palate dorsal to the epiglottis.[83] The definitive diagnosis requires biopsy and histopathologic examination. Often, submandibular lymphadenopathy in horses with oral neoplasia is caused by reactive inflammation rather than metastatic disease, so biopsy results of these lymph nodes are usually negative.

Successful treatment depends on the type and size of tumor present and its accessibility. Tumors of the lips and rostral tongue are most readily resolved, as they are often noticed early and can be adequately excised or are accessible for intralesional chemotherapy and radiotherapy. Invasive squamous cell carcinomas have a high recurrence rate after surgical excision and have often metastasized by the time they are diagnosed.[82,88]

Complementary radiotherapy after radical surgical excision of squamous cell carcinoma may prevent or prolong time to recurrence.[89] Fibrosarcomas also tend to recur after excision and are less responsive to radiation therapy. The prognosis for resolution of oral squamous cell carcinoma and fibrosarcoma is generally poor.[77] Lingual rhabdomyosarcoma and chondrosarcoma can be managed by surgical excision, with an unknown definitive long-term prognosis.[83,87]

Nonneoplastic conditions that should be differentiated from tumors are focal gingival hyperplasia, epulis, and exuberant granulation tissue.[77,86,90,91] These masses can appear neoplastic, but they carry a more favorable prognosis. Other rare conditions reported include vascular hamartoma of the tongue, which resulted in euthanasia of the horse,[92] and an atypical perineurial cell proliferative disorder identified in a horse with multiple focal and coalescing masses along the tongue.[93]

SALIVARY GLANDS

Anatomy

The major salivary glands in the horse are the paired parotid, mandibular (submaxillary), and polystomatic sublingual glands. There are also smaller buccal, labial, lingual, and palatine salivary glands.[94] The distribution of serous and mucous cells within the salivary glands determines the nature of the saliva secreted. The parotid salivary gland secretes mainly serous fluid, whereas the mandibular and sublingual glands produce a combination of serous and mucous fluids.

The largest of the salivary glands, the parotid, is located in the retromandibular fossa between the vertical ramus of the mandible and the wing of the atlas. The rostral border reaches and may partially overlap the temporomandibular joint and the masseter muscle along the caudal border of the mandible. The caudal border extends to the wing of the first cervical vertebra. Dorsally, the gland extends to the base of the ear, and ventrally it extends into the intermandibular space. The surfaces of the parotid gland are associated with important vascular and neural structures.[94] Glandular secretions are drained by multiple small ducts that converge at the rostroventral aspect of the gland and exit as the single parotid (Stensen) duct. The parotid duct initially passes along the medial surface of the mandible in close association with the facial artery and vein. The three structures then travel rostrolaterally around the ventral border of the mandible at the location of the easily palpable facial artery pulse. The duct ascends along the rostral edge of the masseter muscle and opens into the buccal cavity opposite the maxillary fourth premolar tooth (108, 208). A parotid papilla identifies the buccal ostium.

The mandibular salivary gland extends from the atlantal fossa to the basihyoid bone. Most of its lateral surface is covered by the parotid salivary gland and partly by the mandible, and its medial surface covers the larynx, common carotid, vagosympathetic trunk, and guttural pouch. Many small radicles unite to form a common duct that travels rostrally, ventral to the tongue. The duct opens a few centimeters rostrolateral to the lingual frenulum, at the sublingual caruncle. The small sublingual gland lies beneath the oral mucosa between the body of the tongue and the mandible. It extends from the level of the mandibular symphysis to approximately the first or second mandibular molar (09s and 10s). Sublingual ducts (about 30) open independently at small papillae on the sublingual fold.[94]

Disorders

Trauma

Lacerations or penetrating wounds to the salivary glands occur rarely. The superficially located parotid salivary gland and duct are subject to traumatic injury more often than the better protected mandibular and sublingual glands.

The hallmark of disruption of the gland or duct is the flow of saliva from the wound. This flow is accentuated when the horse is fed, and observation of the wound while offering feed to the horse provides a simple test to confirm that the discharge is saliva.

Diagnosis of parotid duct disruption may be facilitated by catheterizing the duct via the buccal ostium, with the horse under general anesthesia, and visualizing the catheter exiting the wound. Locating and accessing the parotid papilla for retrograde catheterization occasionally requires a buccotomy at the level of the 108 (or 208).[95] A second incision can be made a few centimeters rostral or caudal to the buccotomy to allow tunneling of the tubing subcutaneously to the buccotomy site. Contrast sialography can also be used to determine communication with a salivary gland and duct patency in the case of a suspected salivary fistula.[96,97] If there is no clear indication that a salivary duct or gland has been damaged, it is recommended that parotid wounds be left to heal by second intention.[98] When the duct has been disrupted but this was not recognized at the time of injury, horses may be presented later with a chronically draining tract.[99] Surgical management of salivary gland and duct trauma is fundamentally aimed at repairing the damaged structure or, alternatively, at eliminating saliva secretion. Most salivary fistulas spontaneously close within 1 to 3 weeks, so deferring treatment for a period of weeks is a worthwhile option.[99]

Fresh wounds of the parotid gland can be débrided and reconstructed with a multilayer closure, starting with the parotid

capsule. The potential for suture material that penetrates the glandular tissue to provide a nidus for calculus formation should be considered. Open wound management and healing by second intention is an alternative for older or heavily contaminated wounds and for economic reasons, and this often allows successful healing without the development of a salivary fistula.[98,99]

Treatment of lacerated salivary ducts may also be by primary surgical closure or secondary wound healing. Primary closure of an acutely lacerated duct or a nonhealing salivary fistula is facilitated by suturing it over an intraluminal tube or by placing three sutures to appose the two cut ends as a triangle and suturing between the apices.[95,96,98-100] Cannulation of the parotid papilla may not be practical in some cases.[100] Alternatively, size 2 nylon suture is threaded normograde through the distal lacerated duct end to the parotid papilla. Tubing is passed via a 14- to 16-gauge needle cannula inserted through the cheek tissue externally to internally to enter the oral cavity just rostral to the parotid papilla. The tubing is subsequently guided over the nylon suture into the duct, and the nylon and needle are removed.[95] When the tip of the tube emerges from the lacerated duct at the wound, it is redirected into the proximal part of the duct and passed to the ventral aspect of the parotid gland. The duct is closed with fine absorbable or nonabsorbable suture (4-0 to 7-0) using a simple interrupted pattern.[95,96,98,100] The external end of the tube is sutured to the side of the face, allowing ease of later removal and the ability to check for continued patency of the duct. The need to leave the stent tube in place while the duct is healing after anastomosis and how long to leave it are unclear.[99-101] The tubing may support the suture line and prevent stricture development while maintaining saliva flow.[95] For tears involving one side of the duct wall, suture repair without use of an *in situ* stent should be adequate.[96] A gauze mask can be placed over the horse's head to help prevent premature tube removal. If anastomosis of the duct is not possible because of loss of too much intervening tissue, an interposition polytetrafluoroethylene tube graft may be successful in restoring duct continuity.[102] Other options for salvaging salivary secretion capacity are to create a fistula from the duct to the buccal cavity proximal to the injury or alternatively perform duct translocation.[97-99] These techniques divert saliva from the distal wound site, allowing it to heal, but they are no longer favored because of inconsistent results.

Attempts to reduce salivation after duct or gland repair by withholding feed and water or feeding through a nasogastric tube for a few days have been recommended.[101] Others believe this management to be unnecessary and the continued flow of saliva to be important to prevent obstruction.[98-100] Perioperative NSAIDs and antibiotic therapy are administered.

The alternative approach to managing a chronic parotid salivary duct fistula is to attempt to eliminate saliva secretion. Horses can tolerate the loss of one parotid gland.[98,99] This may be performed by surgical removal of the gland, duct ligation, or chemical ablation of the gland. Parotid gland extirpation is difficult, as very careful avoidance of intimately associated large vessels and nerves is required, and removal of all glandular tissue may be impractical because of intervening vital structures.[103,104] Consequently extirpation has been largely replaced by chemical ablation.[99]

Chemical ablation and duct ligation are more economical and easier procedures to perform than major duct reconstruction. For ligation, the parotid duct is readily located where it crosses the tendon of insertion of the sternomandibularis muscle close to its origin from the gland. A catheter is passed retrograde through the duct from the salivary fistula site, toward the gland. The catheter can be palpated in the duct, and this allows definitive isolation of the duct during dissection. Two or three heavy-gauge nonabsorbable sutures should be used for duct ligation and should not be tied too tightly to prevent cutting through the duct wall.[105] The distal suture is tied first to distribute resulting back-pressure after ligation.

Successful chemical involution of the parotid gland was first described using Lugol iodine, with 1 to 2 mL injected transcutaneously into the gland at multiple sites.[105] However, patient discomfort, severe swelling, and the potential for nasopharyngeal collapse and consequent airway obstruction make this method unacceptable today. Other agents that have been examined critically are 10% formalin, 2% chlorhexidine, and 2% and 3% silver nitrate.[106] Of these chemicals, 10% formalin produces the least amount of necrosis and suppurative inflammation, so it is currently recommended.[98,106] Water-soluble iodinated contrast material is also effective in eliminating glandular secretions.[99,106] This must be considered when retrograde sialography is undertaken to investigate a draining fistula or duct atresia. Allowing the contrast material to drain out followed by lavage of the duct and gland with sterile saline solution before injecting the chemical agent is recommended. When using 10% formalin, the duct is cannulated and a ligature tied to prevent leakage. Thirty-five milliliters of formalin is injected through the cannula into the gland. It is left in for 90 seconds and then allowed to drain out. The cannula is left in place for 36 hours. Cessation of salivary secretions occurs by 3 weeks. NSAIDs are administered after treatment for pain relief. Postoperative complications after chemical ablation include periocular and facial swelling, transient facial nerve paralysis, anorexia, and dyspnea. Most of these complications have been associated with chlorhexidine and silver nitrate and not with formalin.[98,106]

Sialoliths

Sialoliths are hard concretions composed mostly of calcium carbonate and organic matter that develop within a salivary duct or less commonly a gland.[107-112] Plant material is often found as a nidus of the sialolith formation.[107-110] Sialoliths appear smooth or slightly spiculated, and gray, yellowish, or white.[113] They are rarely recognized but when they occur, they tend to affect older horses, and the parotid duct is most commonly involved. Typically, a nonpainful, movable, firm structure is palpable on the lateral aspect of the face near the rostral end of the facial crest (Figure 30-46), and it can be present for many years before additional clinical signs develop and lead to veterinary attention.[107,108] In some cases, the sialolith may be palpable orally.

Obstruction of the duct is often incomplete, and saliva may continue to pass around the sialolith. However, with acute or chronic complete obstruction, back pressure may cause duct and gland distention, which is noticed as a possibly painful swelling in the intermandibular and retromandibular space.[95,109] Other clinical signs, including the presence of purulent material in the mouth, reddening at the parotid papilla, mild icterus, fever, difficulty masticating, quidding, halitosis, decreased appetite, and acute development of a painful swelling that was previously nonpainful have also been reported in association with sialoliths.[95,107,109,110] Sialoliths usually occur singularly. If multiple

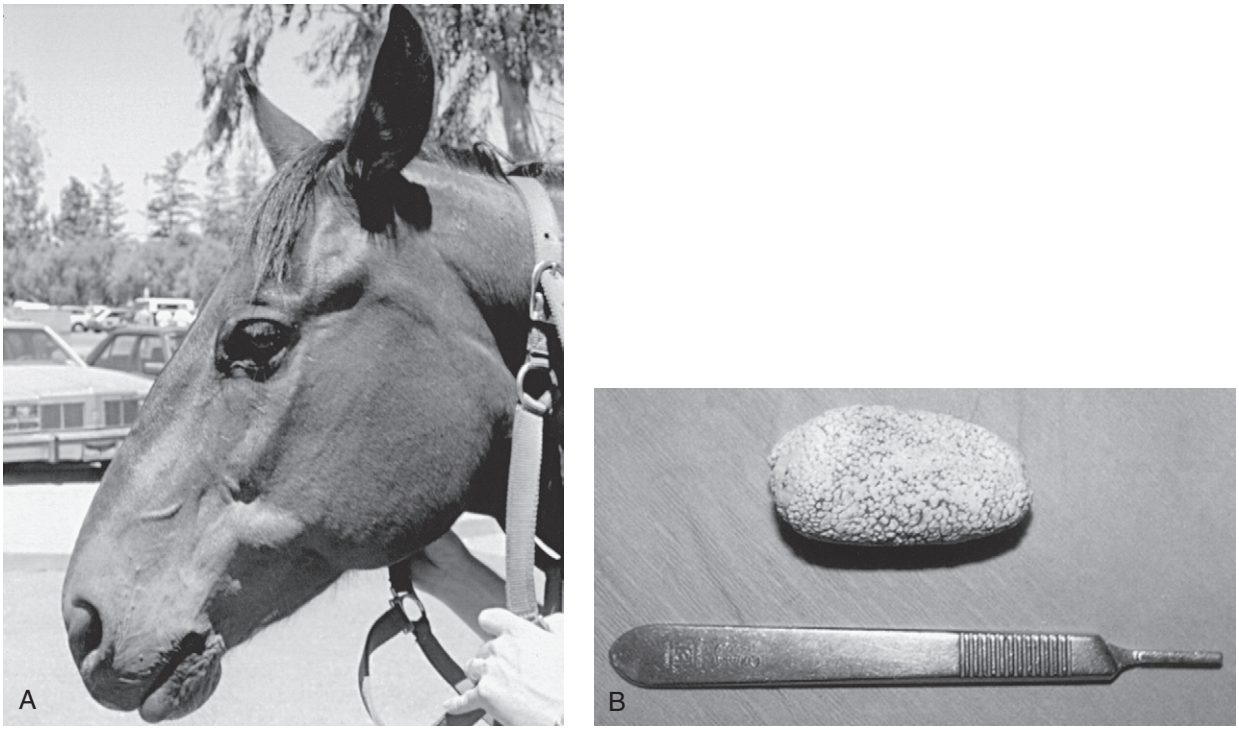

Figure 30-46. A, Photograph of a horse with a sialolith located in the left parotid duct near the rostral edge of the facial crest. **B,** Sialolith removed from a parotid salivary duct. (**B,** with permission, Equine Vet Educ 7:315, 1995.)

calculi are present in the same duct, a grating sound or sensation may be heard or felt on palpation.[112] Septic sialoadenitis is a rare occurrence in domestic animals but may precede or be a consequence of a developing sialolith.[101] Inflammation and infection of the duct (of ascending, hematogenous, or traumatic origin) results in obstruction by exudates, desquamated cells, and mucus. This material may provide the nidus necessary for a sialolith to form. Partial obstruction of the duct by a sialolith may decrease natural clearance of secretions, resulting in stasis and facilitating proximal movement of bacteria. Diagnosis of sialolithiasis is assisted by radiographs, but soft tissue swelling can impede identification of a concretion.[95,107-110]

Definitive treatment is by removal of the sialolith. Smaller calculi may be massaged out of the parotid papilla. If this is not possible, and the calculus is palpable orally in the buccal soft tissues, direct intraoral incision over the sialolith, leaving the wound to heal by second intention, is preferred.[97,98,105,107,108] This approach prevents external salivary fistula development. Calculi inaccessible by the intraoral route must be removed by external longitudinal incision of the duct. This can be performed as a standing surgery with local anesthesia. Cannulation of the parotid duct via the parotid papilla can be helpful to locate the exact calculus position and may act as a stent for suturing the duct if primary closure is performed.[109] The entire duct and gland should be lavaged with sterile polyionic solution. Closure of the duct is with a simple interrupted or continuous pattern of fine absorbable suture material. The incision can also be left to heal by second intention, recognizing the risk that a fistula may develop. Postoperative care is similar to repair of a duct laceration or fistula. Antibiotics and lavage of the duct and gland via catheterization of the oral papilla are recommended in infected cases.[109]

Atresia of the Parotid Salivary Duct

Congenital or acquired functional discontinuity of the parotid salivary duct is very rare in the horse.[103,114,115] Proximal to the obstruction, the dilated duct is characterized by a nonpainful, tortuous, subcutaneous, fluid-filled tube that extends caudad from a point rostral to the masseter (where the obstruction often is located) along the ventral surface of the mandible to the base of the ear on the affected side (Figure 30-47).[99,114,115] In congenital cases, swelling is present at birth and can increase with time. Swelling may also increase temporarily when the horse is presented with food. Aspirated fluid has the consistency of saliva with an alkaline pH and an acellular, proteinaceous background.[99,115] The swelling recurs after needle drainage, and occasionally saliva continues to drain into subcutaneous tissues after aspiration, setting up a severe inflammatory reaction, followed by persistent sialocele. Diagnosis is based on clinical appearance but can be confirmed with positive-contrast sialography and analysis of aspirated fluid. Dilated glandular radicles are seen coalescing into a single grossly distended parotid duct that terminates distally before reaching the buccal cavity.

Treatment options include surgically creating a new buccal ostium, duct excision and proximal ligation, gland extirpation, and chemical ablation of the salivary gland. Creation of a buccal fistula is not recommended because of its probable spontaneous closure and the potential for ascending contamination of the duct.[99] In one case, removing a large portion of the distended duct and ligating individual radicles was successful.[115] Duct ligation may not be appropriate in a chronic case, as severe dilation may not allow enough back pressure to be generated to cause atrophy of the gland and cessation of secretory activity.[116] Chemical ablation of the gland is an economical, effective, and practical solution and is recommended for most cases. Surgical

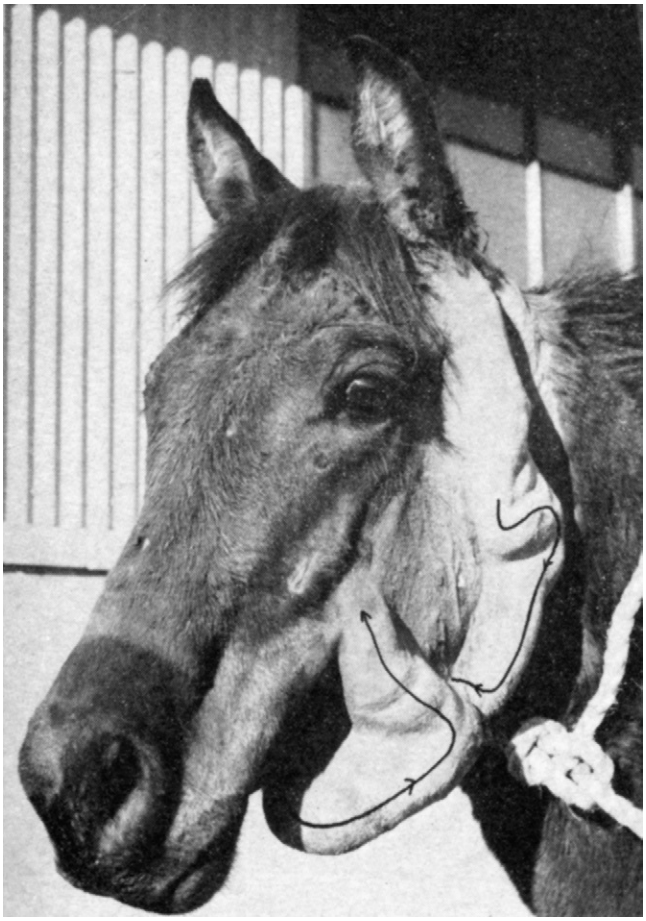

Figure 30-47. Severely dilated parotid salivary duct and gland of a 1-year-old Quarter Horse gelding with congenital atresia of the parotid duct. The duct ended blindly approximately 1.4 cm from the normal site for the buccal ostium. Line arrows mark the course of the duct. (With permission, J Am Vet Med Assoc 146:1404, 1965.)

removal of the duct can be performed for cosmetic reasons after ablation of the gland, but it is a difficult procedure.[99]

Salivary Mucocele and Ranula

On rare occasions accumulations of saliva occur in spaces adjacent the local gland or duct, possibly when an external wound has healed but the duct or gland continues to leak. A mucocele or sialocele refers to a pocket of saliva in a space not lined by epithelium. A ranula ("honey cyst") represents a mucocele of one of the sublingual salivary gland ducts and is seen as a bluish-tinged cyst on the floor of the mouth.[113] Fluid aspirated from a sialocele is generally brown and mucinous with a higher concentration of calcium and potassium than is seen in other accumulations of fluid.[101] Salivary fluid also contains amylase. Contrast sialography can determine any communication between the cavity and duct or gland. In some cases, complete surgical removal of the structure is appropriate. Ranulas respond well to marsupialization into the oral cavity.[99,101] A catheter can be sutured in place for 2 to 3 weeks to create a permanent fistula. Marsupialization of a mucocele into the buccal cavity is less likely to be successful because of a lack of epithelial lining.[99] Chemical ablation of the parotid gland would be effective and may be the simplest approach if this gland is known

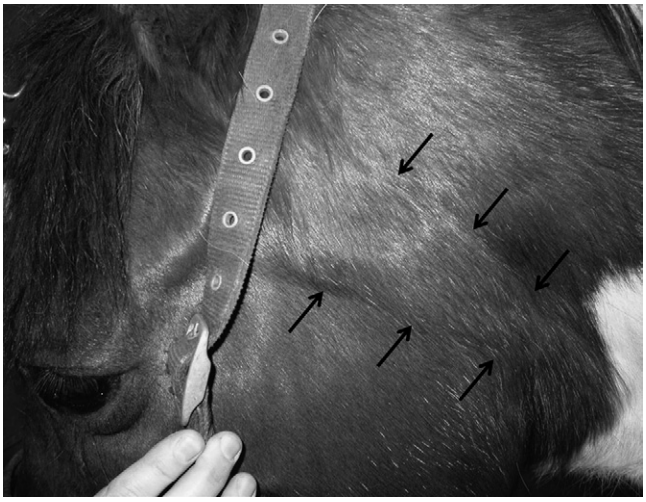

Figure 30-48. Parotid region of a pony showing a nonpainful swelling *(arrows)* that occurred when the pony was at pasture but resolved when it was kept stalled. (Courtesy Dr. P. Dixon, Edinburgh University.)

to communicate with the mucocele directly or via the duct.[98,99] Later, the mucocele can be drained without expected recurrence.

Heterotopic Salivary Tissue

Salivary tissue found in an abnormal location is a rarely diagnosed disorder and is described as heterotopic salivary tissue. It is possible that this condition goes unrecognized more frequently than it is confirmed; for example, unclassified subclinical lateral nasopharyngeal masses seen endoscopically may be heterotopic salivary tissue.[99] Heterotopic tissue associated with seromucoid draining tracts has been reported in the temporal region and the mid cervical region.[99,117] Drainage of saliva from heterotopic tissue does not increase when the horse is presented with feed. Definitive diagnosis is by histopathology, and complete surgical removal resolves the condition. Chemical ablation of the tissue may also be performed, if the nature of the tissue can be ascertained beforehand.[99]

Idiopathic Parotiditis, "Grass Glands"

Idiopathic parotiditis, or grass glands, refers to a syndrome of recurrent salivary gland swelling that occurs acutely in association with pasture turnout.[118,119] Typically, the parotid salivary glands become swollen during the day while the horse is on pasture, and the swelling resolves overnight when the horse is stalled. The condition is well recognized in Europe and Australia but is uncommon in the United States. Characteristically, there is a rapid development of bilateral swelling of the parotid gland. Glandular swelling is innocuous, firm, and nonpainful, occasionally causes edema at the caudal mandible, and can be mistaken for guttural pouch disease or enlarged lymph nodes (Figure 30-48). Once the horse is removed from pasture, the glandular swelling resolves. A pasture toxin is suspected but has not been identified. Sporadic or herd outbreaks are possible.

Neoplasia

The salivary glands are a rare site for neoplasia in the horse. Adenocarcinoma, acinar cell tumors, lymphomas, melanomas,

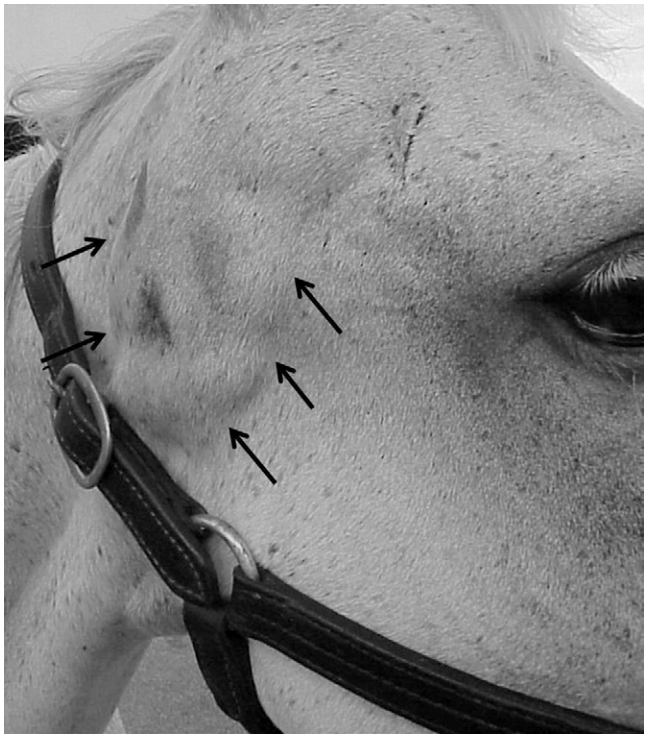

Figure 30-49. Parotid region of an aged Thoroughbred mare showing an irregular-surfaced, firm, nonpainful swelling *(arrows)* caused by melanoma development in the parotid salivary gland. Similar masses were present on the opposite side, and melanomas were visualized in this mare's guttural pouches.

and mixed cell tumors have been reported.[113,120,121] Melanomas are commonly seen in the parotid salivary gland of gray horses and may represent a primary or metastatic tumor.[84,121] Tumors in the parotid region can be identified by the presence of external swelling and are occasionally painful on palpation. Melanomas typically appear as nonpainful, firm, irregular- or smooth-surfaced masses (Figure 30-49), and endoscopy of the guttural pouches of affected horses will show focal black submucosal areas or the presence of melanomas, which will confirm the diagnosis. Diagnosis of other types of parotid region neoplasia may require endoscopy, radiology, ultrasonography, and cytology of a fine-needle aspirate. Biopsy and histopathology are indicated for a definitive diagnosis. Most parotid melanomas are slow growing and do not need to be treated. Treatment of other types of parotid tumors is often palliative, because wide surgical margins frequently fail to prevent benign mixed cell tumors or acinar tumors (and likewise, melanomas) from recurring. As previously mentioned, total parotidectomy is challenging and generally considered impractical in the horse.[99] Adenocarcinomas often metastasize.[120] Lymphomas can respond to radiation therapy.[122] Cimetidine may help with melanomas, but often this treatment is not justified because they are slow growing and most have low malignancy.[123-125]

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CHAPTER 31

Esophagus

John A. Stick

The clinical signs, diagnosis, and therapy of esophageal disease are specific and unlike those of the rest of the alimentary system. Clinical evaluation of the equine patient with esophageal disease includes physical, radiographic, and endoscopic examinations. Early and definitive diagnosis is paramount when dealing with esophageal injury, especially when signs of disease recur after initial treatment. Therapy of esophageal disorders often centers around conservative medical and manipulative management, with dietary alterations being a primary component of therapy. However, surgical management of esophageal disease in the equine patient has become commonplace and necessitates discussion of surgical anatomy, diagnostic and therapeutic considerations, surgical techniques, and complications.[1-3]

SURGICAL ANATOMY

The esophagus of adult horses varies in length from 125 to 200 cm (49 to 78 inches), depending on the size of the animal, and consists of cervical, thoracic, and abdominal parts. As it courses caudad, it deviates from a position dorsal to the trachea in the cranial one third of the neck to the left side of the medial plane in the middle one third of the neck. (In a small percentage of horses, the esophagus courses to the right side of the median plane.) It comes to lie ventral to the trachea at the thoracic inlet.[4] The cervical part of the equine esophagus is most accessible to surgery and makes up over 50% of the total length of the esophagus.

The wall of the esophagus is composed of four layers: a fibrous layer (tunica adventitia), muscular layers (tunicae muscularis), a submucosal layer (tela submucosa), and a mucous membrane (tunica mucosa). The muscular layers are striated from the pharynx to the base of the heart, where they gradually blend into smooth muscle. As the esophagus courses caudad, its muscular layers increase in thickness, whereas the lumen diminishes. Except at the upper esophageal sphincter, the two muscular layers are arranged spirally and elliptically.[5] On surgical incision, the esophageal wall separates easily into two distinct layers. The elastic inner layer, composed of mucosa and submucosa, is freely movable within the relatively inelastic outer muscular layer and adventitia (Figure 31-1). The mucosa, which provides the greatest tensile strength on closure of an esophageal incision, is covered with stratified squamous epithelium and lies in longitudinal folds that obliterate the lumen

except during deglutition. The mucosa is very heavily colonized by facultative and obligate anaerobic bacteria,[5,6] and therefore surgery on this organ necessitates the use of prophylactic antimicrobial agents (Figure 31-2).

The arterial supply to the cervical part of the esophagus originates from the carotid arteries. The thoracic and relatively short (2- to 3-cm [¾- to 1¼-inch]) abdominal esophagus is supplied by the bronchoesophageal and gastric arteries. The vascular pattern is arcuate but segmental, without generous collateral circulation, necessitating careful preservation of vessels during surgery.

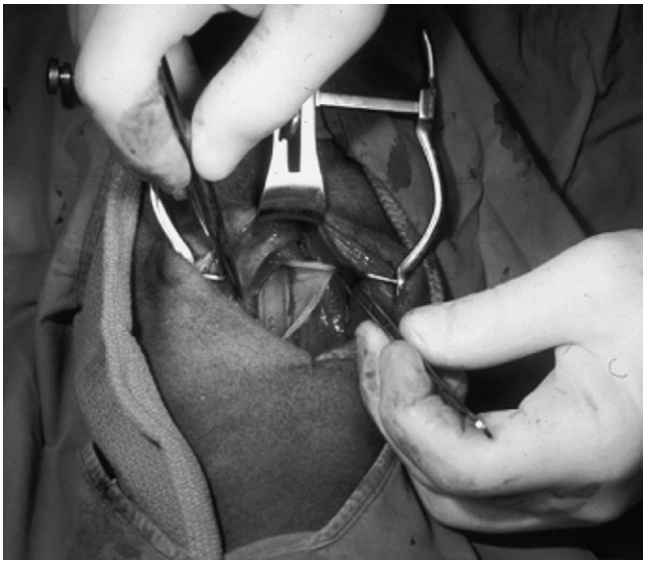

Figure 31-1. Traction on the incised esophagus, with a nasogastric tube in place, shows the elastic properties of the mucosa and submucosa (inner layer).

Innervation of the esophagus is derived from the ninth and tenth cranial nerves and the sympathetic trunk, as well as mesenteric ganglion cells within the muscle layers.

CLINICAL MANIFESTATIONS AND EVALUATION OF ESOPHAGEAL DISEASE

The obstructive disease of "choke" may be manifested by ptyalism, dysphagia, coughing, and regurgitation of food, water, and saliva from the mouth and nostrils. Attempts at ingestion are often followed by odynophagia (painful swallowing), repeated extension of the head and neck, and other signs of distress or agitation. The time interval from swallowing until these signs are shown by the patient depends on the location of the lesion within the esophagus. With obstruction of the distal esophagus, odynophagia and retching may occur 10 to 12 seconds after swallowing. With proximal esophageal obstruction, the signs may be evident immediately. This occurs because the propagation speed of the equine esophagus is about 9.4 cm/sec in the proximal two thirds but only 4.6 cm/sec in the distal one third. Therefore, over an average length of 116 cm (45¼ inches), a bolus of food would take about 16 seconds to traverse the body of the esophagus.[7,8] Intermittent signs of choke followed by periods of relief may indicate a disease other than simple feed impaction, and further diagnostic procedures are warranted. Anorexia, electrolyte imbalances, and dehydration accompany cases of long duration (see "Complications of Esophageal Surgery," later). Aspiration pneumonia frequently follows esophageal obstruction, and the clinical signs may be present as early as 1 day after the onset of choke.

Physical Examination

A thorough oral examination should be performed to rule out an oral foreign body, dental disease, cleft palate, or

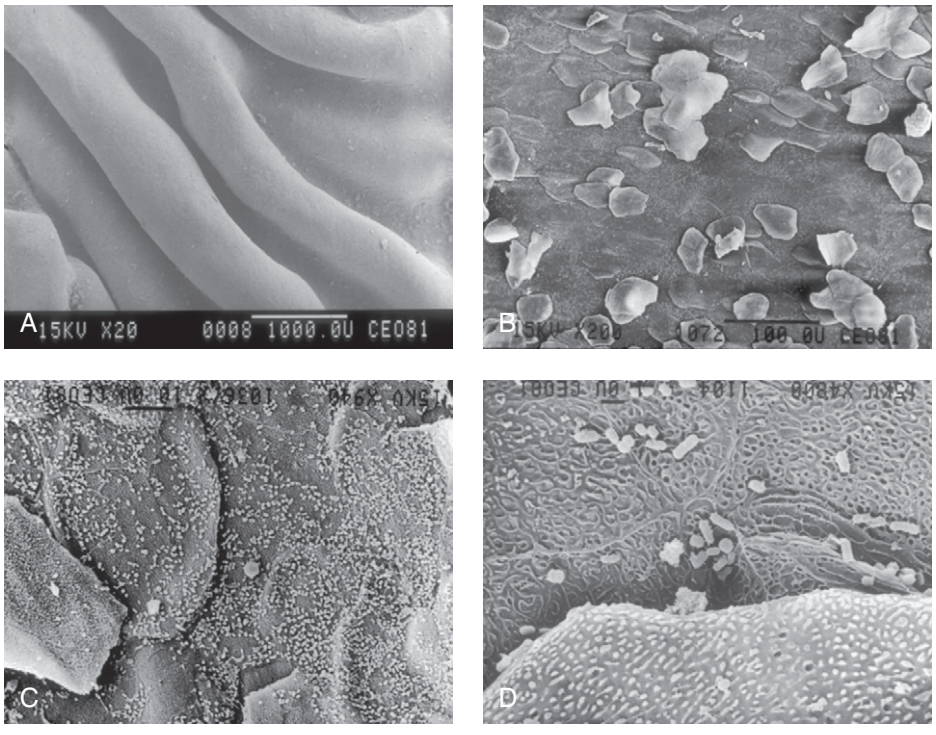

Figure 31-2. Scanning electron micrographic images of the esophageal lumen. **A,** Longitudinal mucosal folds. **B,** At a higher power, squamous epithelial cells. **C,** Colonization of bacteria on squamous cells. **D,** Close-up of squamous cells with mixed populations of bacteria. Because the lumen of the esophagus is heavily colonized under normal circumstances, prophylactic antibiotics are necessary any time this organ is incised.

oropharyngeal neoplasms. Observation and palpation of the neck in the area of the jugular furrows may reveal enlargement of the cervical esophagus. Simple food impaction of the cervical esophagus may be localized in this manner. Crepitation of a diffuse, firm enlargement may indicate loss of integrity of the esophageal wall. Passage of the nasogastric tube often confirms luminal obstruction and localizes the site of involvement. Gentle lavage of warm water through the tube may permit material to be flushed free of the obstruction if feed or consumption of bedding is the cause of the problem. At this time, sedation of the animal with xylazine (1.1 mg/kg IV) lowers the horse's head and prevents further aspiration. Lavage may be continued until the obstruction is relieved, and further diagnostic studies may be unnecessary. However, reobstruction indicates that other esophageal disease may be present, and the diagnosis should be pursued.

With any esophageal disease, auscultation and diagnostic imaging of the thorax are indicated to monitor development of aspiration pneumonia. This complication is common when an esophageal problem is encountered.

Diagnostic Imaging

Ultrasonographic examination of the cervical esophagus is easily performed and may aid in the determination of the etiology of the obstruction. Simple impactions can be localized and extramural masses may be identified. Rupture of the esophagus will be apparent as gas and free fluid outside the esophageal lumen. The extent of cellulitis and phlegmon development may also be determined ultrasonographically.[9]

Esophagography in horses provides diagnostic evidence of disease in most instances and should be considered a part of the complete esophageal examination in problems other than simple obstruction. A survey film is necessary to establish radiographic technique and the presence or absence of disease without contrast media (e.g., feed impaction or foreign body) (Figure 31-3). Barium liquid mixed in grain and fed results in a positive contrast esophagram, but preferably, barium paste (85% wt/vol with water, 120 mL) given by mouth outlines the longitudinal mucosal folds of the esophagus with the lumen undistended and localizes the obstruction or any disruption of the lumen (Figure 31-4). The feed bolus impacting the esophagus becomes coated with the barium, allowing it to be visualized on radiography; a complete obstruction halts barium flow orad to the bolus or lesion (Figure 31-5), and rupture of the esophagus permits barium to escape into surrounding soft tissues. If possible, the patient should not be sedated during this procedure because sedation suppresses the swallowing reflex and causes barium to be held in the mouth, reducing the amount available to coat the esophagus.

Liquid barium (72% wt/vol with water, 480 mL) may be administered under pressure by a dose syringe through a cuffed nasogastric tube to prevent reflux into the pharynx (Figure 31-6). This technique demonstrates strictures and associated prestenotic dilation of the esophagus, as well as space-occupying masses that displace the esophagus. Liquid barium (480 mL) followed by air (480 mL), delivered by dose syringe under pressure, provides a double-contrast study (Figure 31-7), permitting examination of mucosal folds with the esophagus distended. This latter technique gives the best definition of mucosal lesions,

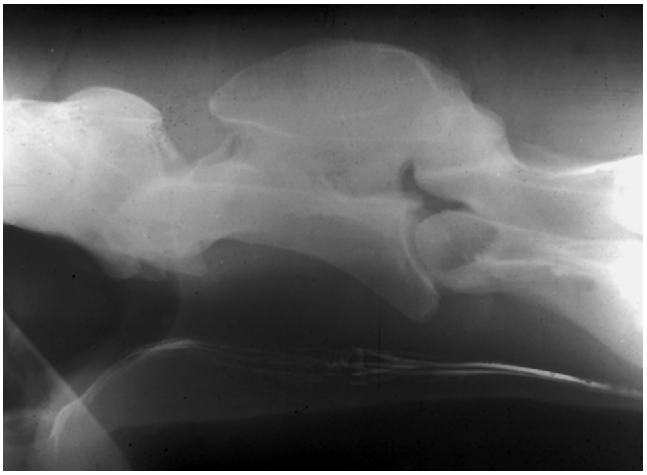

Figure 31-4. Barium paste (120 mL) given by mouth outlines the normal longitudinal folds of the mucosa in the undistended lumen of the esophagus.

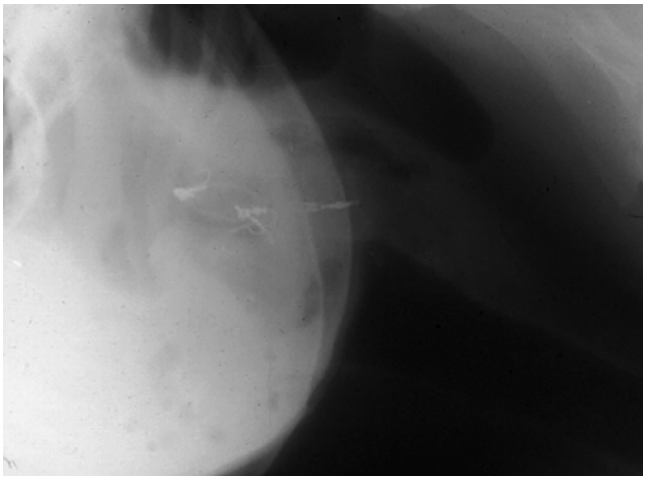

Figure 31-3. Lateral cervical radiograph of a 9-year-old gelding with anorexia and odynophagia. A metallic foreign body (fishing lure) is lodged in the cranial esophageal sphincter.

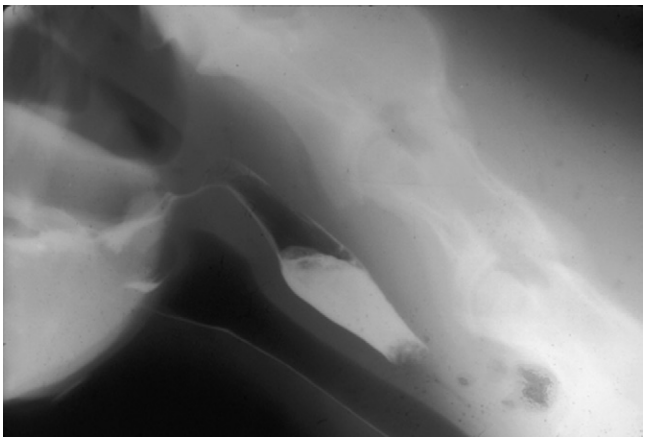

Figure 31-5. Complete obstruction of the esophagus is localized on esophagography after barium paste swallow. This adult horse had an esophageal stricture. Note the prestenotic dilation.

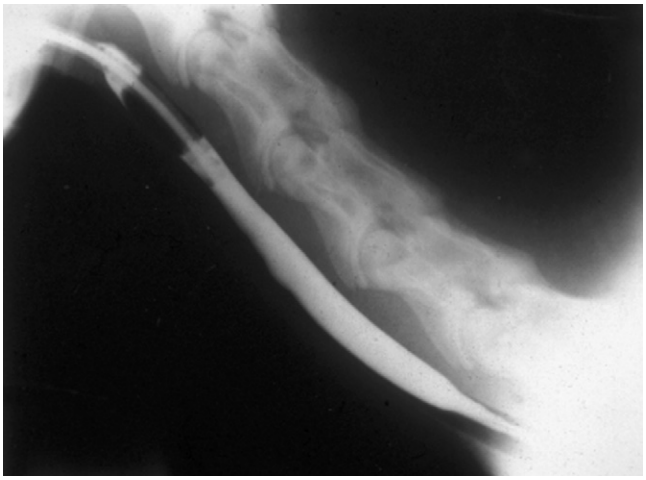

Figure 31-6. Positive-contrast esophagogram (using liquid barium administered under pressure through a nasogastric tube fitted with an inflatable cuff) shows the distended lumen of the normal esophagus. The cuff prevents reflux of barium into the pharynx and aspiration into the trachea.

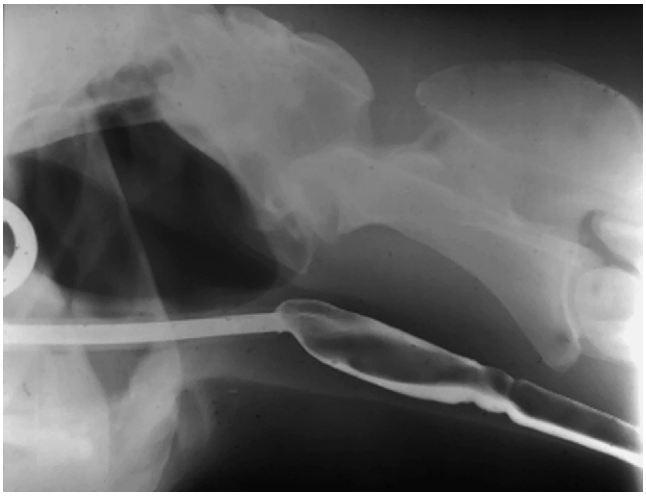

Figure 31-7. Double-contrast esophagogram (using liquid barium followed by a bolus of air) shows the esophagus with the lumen distended. This technique outlines any abnormal transverse mucosal folds and identifies a circumferential ulcer in this horse.

such as circumferential mucosal ulcers after feed impaction. Although a diagnosis can often be made without using all three techniques, each demonstrates lesions not seen with the other two.

In the cranial cervical area, where the esophagus lies dorsal to the trachea, lesions that restrict distention of the esophageal lumen can be demonstrated with negative-contrast radiography (Figure 31-8). A flexible endoscope can be used to localize the lesion and permits insufflation of the esophagus during radiography. Alternatively, air (480 mL) can be delivered by dose syringe under pressure through a cuffed nasogastric tube to achieve the same results. However, negative-contrast radiography alone does not yield much information about the caudal cervical and thoracic portions of the esophagus because of superimposition of the air density of the trachea and lungs.

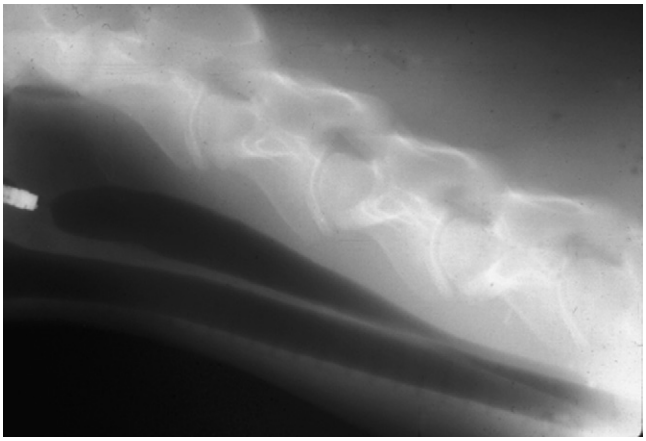

Figure 31-8. Negative-contrast esophagogram (using air insufflation introduced through the flexible endoscope) permits visual localization of the lesion with the endoscope and demonstrates a stricture.

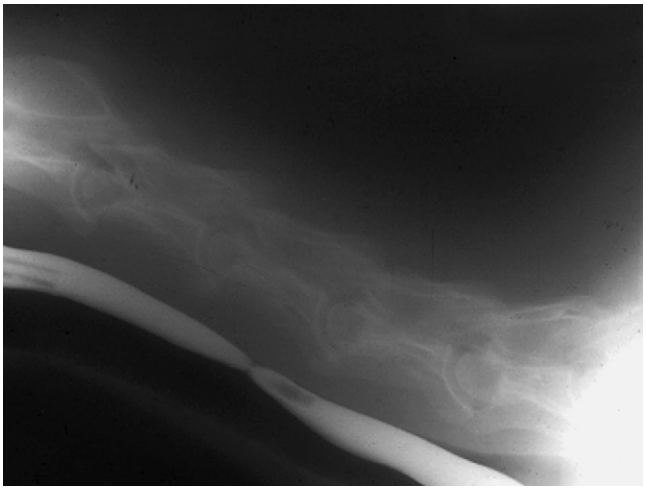

Figure 31-9. Barium esophagogram shows false signs of a stricture when barium is administered under pressure and the radiograph is made during swallowing. This swallow artifact can be avoided if xylazine is administered 5 minutes before the study is begun.

Swallowing during contrast studies, when the lumen is being distended, produces false signs of esophageal stricture (Figure 31-9). Xylazine (1.1 mg/kg IV), 5 minutes before the barium-under-pressure, double-contrast, or negative-contrast esophagogram, helps eliminate this swallow artifact by decreasing the reflex "secondary swallows" that follow luminal distention. However, if detomidine is used, false signs of megaesophagus can persist for more than 30 minutes.[10]

Endoscopic Evaluation

Esophagoscopy may better define the severity and extent of esophageal lesions diagnosed on radiography and can be used as an ancillary diagnostic aid. Additionally, endoscopic examination should always be performed when radiographic findings are not diagnostic. If the endoscope is 200 cm (78 inches) or longer, the entire esophagus may be examined, and esophageal lesions in the thorax of the adult horse undetected on radiographic examination can be diagnosed. A flexible endoscope

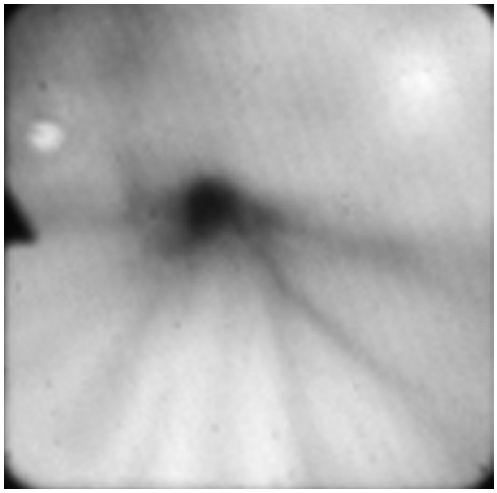

Figure 31-10. Appearance of the normal longitudinal mucosal folds of the undistended esophagus when the endoscope is pulled craniad.

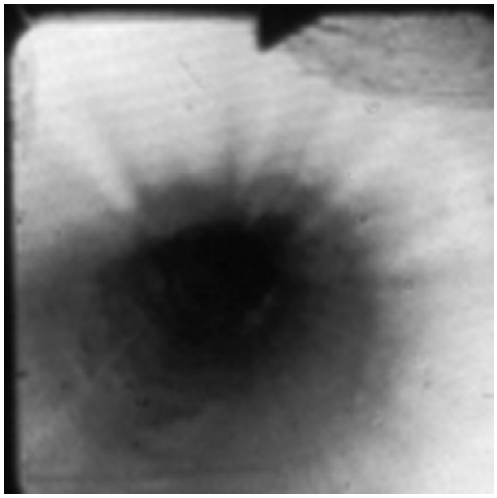

Figure 31-11. Appearance of the normal esophageal lumen when it is insufflated and the endoscope is pulled craniad.

that allows irrigation and insufflation is necessary to provide good observation of mucosal lesions and changes in luminal size.

Endoscopic examination may be performed safely on the restrained standing animal in most instances. Diagnostic observations are best made by starting with the endoscope fully inserted and the esophageal lumen insufflated, followed by slowly withdrawing the endoscope tip toward the horse's head. After each swallow, the endoscope should be cleared by irrigation and the esophagus dilated before further withdrawal. Several passes should be made over any area of suspected disease.

Longitudinal mucosal folds in the esophagus are normally seen when the endoscope tip is moved proximad and the esophagus is in the relaxed position (Figure 31-10). Insufflation flattens these folds and permits observation of luminal size (Figure 31-11). Inability to insufflate the esophagus and flatten the longitudinal mucosal folds usually indicates disease. This is noted cranial and caudal to a stricture. Transverse folds can be produced iatrogenically by moving the endoscope tip toward the stomach. When the cervical esophagus is insufflated, the outline of the trachea can often be seen through the esophageal wall. Swallowing produces changes in the lumen that give the appearance of diverticula or strictures to the untrained observer. The normal mucosa is white to light pink; reddened discolorations are evidence of mucosal disease.

The cranial aspect of the cervical esophageal sphincter is difficult to examine because the swallowing reflex is stimulated repeatedly and the larynx directs the endoscope tip dorsad. Radiographic assessment of this area may be more diagnostic. Additionally, the longitudinal mucosal folds along the rest of the esophagus are absent in this area.

Frequently, the endoscopic appearance of an esophageal obstruction is obscured by saliva mixed with ingesta that collects proximal to the obstruction. This should be removed by suction through a nasogastric tube, and the endoscope should be reinserted immediately to observe the area of obstruction.

Manometric Evaluation

Esophageal dysfunction in humans is routinely evaluated using intraluminal pressure manometry. Manometric techniques have been developed, and reference esophageal pressure profiles have been established in healthy horses. The four functionally distinct regions of the esophagus demonstrated were cranial esophageal sphincter, caudal esophageal sphincter, and "fast" (cranial two thirds) and "slow" (caudal one third) regions in the body of the esophagus (Figure 31-12). In some physiologic disorders of the equine esophagus, manometry better defines problems in which more conventional methods have not yielded a diagnosis. Manometry has yielded new information on clinical manifestations of esophageal obstruction and the effect of drugs used to treat it.[11] However, the availability and technical difficulty of manometry have limited its use as a diagnostic tool in equine practice.

Figure 31-13 outlines a systematic scheme of examination using physical, radiographic, and endoscopic findings to diagnose esophageal disorders. Alternative pathways may be used, depending on the disease, clinical signs, available diagnostic aids, and experience of the clinician. Physical examination, including passage of a nasogastric tube and esophageal lavage, and diagnostic imaging yield a diagnosis for most common esophageal problems. Endoscopy allows definitive diagnosis of some anatomic disorders not observed with radiography. Manometry, cineradiography, and electromyography are seldom necessary and have not been commonly used in diagnosis of esophageal diseases.

SURGICAL APPROACHES

Three surgical approaches to the equine esophagus can be used. Each approach is dictated by anatomic location of the lesion and purpose of the surgery. The ventral cervical approach is best used for esophagotomy, esophagomyotomy, and resections involving the proximal third of the cervical esophagus. A ventrolateral approach is recommended for placing a feeding tube in the midcervical esophagus (esophagostomy) or for approaching the distal one fourth of the cervical esophagus, especially near the thoracic inlet. Thoracotomy is necessary to approach the distal half of the esophagus; the choice of intercostal space is dictated by the aim of the surgery and location of the lesion.

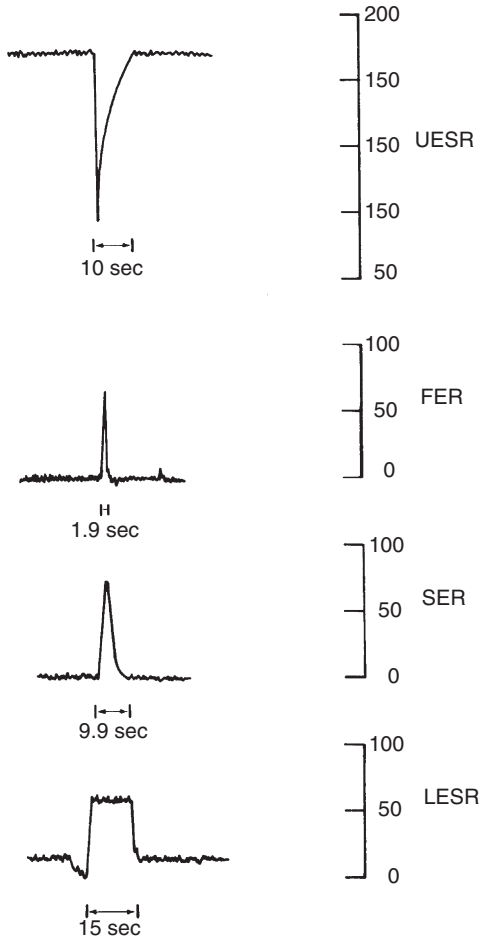

Figure 31-12. Manometric recording of a swallow as it passes through the four functionally distinct regions of the esophagus. *FER,* Fast esophageal region; *LESR,* caudal, or lower, esophageal sphincter region; *SER,* slow esophageal region; *UESR,* cranial, or upper, esophageal sphincter region; units in mm Hg.

If the surgeon expects to invade the esophageal lumen, broad-spectrum antibiotics are indicated and should be chosen on the basis of sound surgical principles, expected complications, and recognized bacterial colonization of this organ.[6,12] Before induction of anesthesia, a nasogastric tube should be passed as far into the esophagus as the surgical site (or beyond if possible) to facilitate identification of the esophagus at surgery. It also is necessary to avoid damage to the recurrent laryngeal nerve and vagosympathetic trunk, which are easily traumatized when retracting the carotid artery away from the esophagus. Owners should be advised that laryngeal hemiplegia is a common complication of esophageal surgery.

Ventral Approach

Surgical procedures are conducted with the animal under general anesthesia and placed in dorsal recumbency. A 10-cm (4-inch) skin incision permits exposure of about 6 cm (2½ inches) of the esophagus. The skin and subcutaneous fascia are divided sharply using a scalpel. The paired sternothyroid, sternohyoid, and omohyoid muscles are separated along the midline to expose the trachea (Figure 31-14). Blunt separation of fascia along the left side of the trachea permits identification of the esophagus containing the nasogastric tube. Retraction of the trachea to the right of the median plane and gentle sharp dissection of overlying loose adventitia expose the ventral wall of the esophagus.

Ventrolateral Approach

Placement of a feeding tube using the ventrolateral approach facilitates firm anchorage of the tube to the skin and permits it to lie in a comfortable position on the patient's neck while preventing impingement of the feeding tube on the trachea (Figure 31-15). This approach also affords better access to the middle and distal cervical esophagus, where the ventral cervical musculature becomes more heavily developed, making the ventral approach less desirable. This surgical approach may be performed with the horse standing, using local anesthesia, or with the horse in dorsal or right lateral recumbency under general anesthesia.[12,13]

A 5-cm (2-inch) skin incision (for feeding tube placement) is made just ventral to the jugular vein. The sternocephalicus and brachiocephalicus muscles are separated, and the deep cervical fascia is incised to expose the esophagus. It may be necessary to incise the cutaneous colli muscle in the distal cervical area.

Approach to the Thoracic Esophagus

An approach through the thoracic esophagus can be used for vascular ring anomalies (in my experience, the left fourth or right fifth interspace was used in two patients) or when the suspected lesion can be resolved surgically without entering the esophageal lumen. A 5-month-old foal was successfully treated for an intrathoracic esophageal stricture with esophagomyotomy through the eighth intercostal space, and an intrathoracic esophageal pulsion diverticulum was resected in a 7-month-old foal by resecting the left eighth rib.[14,15]

The patient is placed in right lateral recumbency under general anesthesia, and positive-pressure ventilation is used. The skin, subcutaneous tissue, cutaneous trunci, serratus ventralis, and latissimus dorsi muscles are sharply divided. A subperiosteal rib resection has been described. However, in my experience, this is not necessary in foals, because rib retractors provide adequate exposure.

MANAGEMENT OF ESOPHAGEAL DISORDERS

Impaction

The most common type of obstructive esophageal disease is impaction with ingesta or bedding.[9] It can occur in the normal esophagus of a gluttonous animal and has a typical radiographic appearance (Figure 31-16). Nasogastric tube passage and gentle warm water lavage are usually successful in relieving the obstruction. Xylazine sedation to lower the patient's head during this procedure greatly reduces the hazard of aspiration of ingesta that is flushed free and passes retrograde through the esophagus into the nasopharynx.

Several alternative techniques may be necessary if gentle lavage is unsuccessful. A cuffed nasogastric tube may be placed, the animal sedated, and water lavaged under pressure with a dose syringe or stomach pump. The tube helps prevent reflux or ingesta from entering the pharynx while permitting pressure of the water to push the obstruction distad. External massage and to-and-fro movement of the water resolve most impactions.

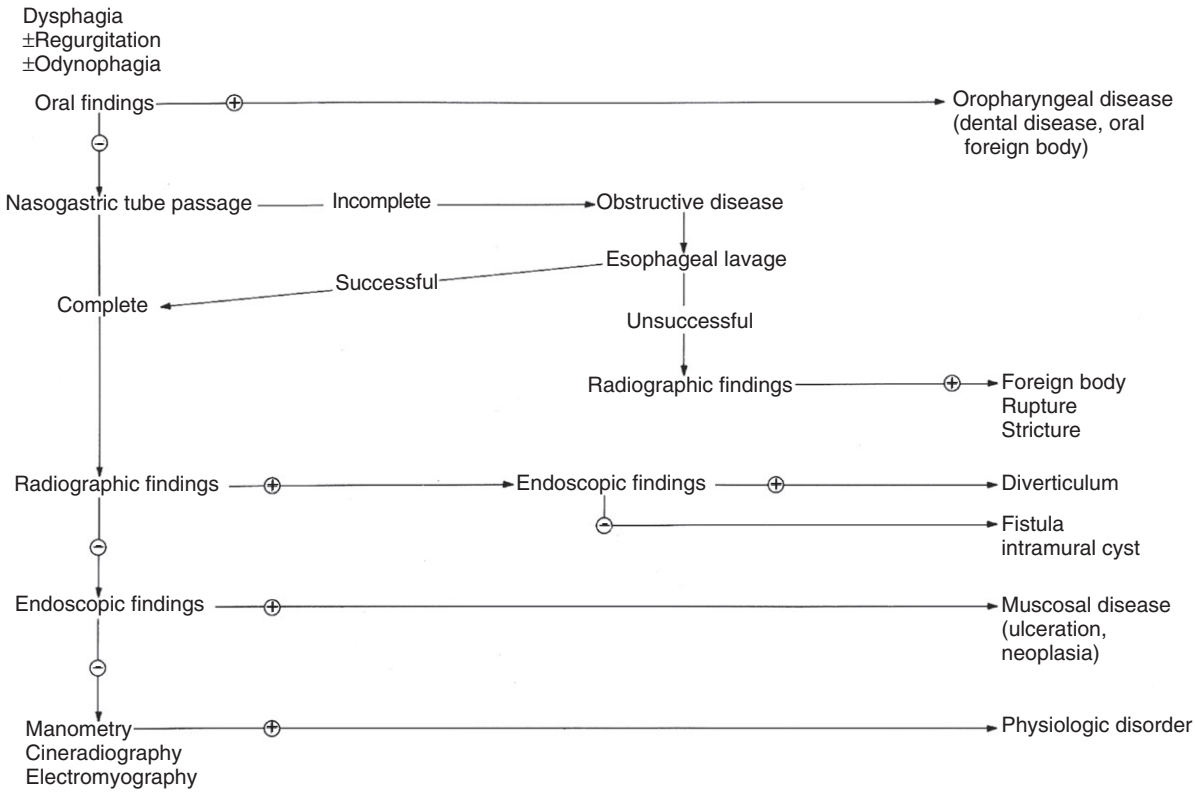

Figure 31-13. Scheme of examination of esophageal disorders. *Arrows* outline pathways that are usually diagnostic for the conditions shown at the right.

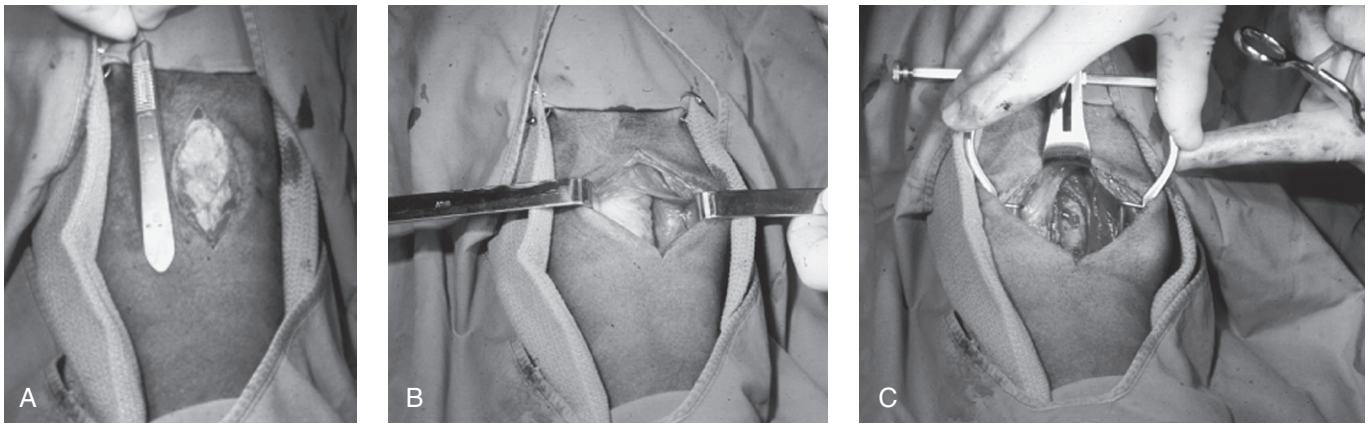

Figure 31-14. A, A 10-cm (4-inch) incision through the skin and subcutaneous fascia, which is sharply divided using the scalpel. **B,** The paired muscles of the sternothyroid, sternohyoid, and omohyoid are separated along the midline to expose the trachea. **C,** A baby Balfour retractor placed through the incision allows easy access to the esophagus.

If this technique is not successful, the animal should be muzzled to prevent food and water intake and left alone for 8 to 12 hours, and the treatment should then be repeated. Frequently, the initial treatment softens the impaction and it becomes dislodged by swallowing or is easily relieved by a second treatment. Some clinicians claim that atropinization (0.02 mg/kg) for a horse with esophageal impaction aids dissolution of the impaction by promoting esophageal relaxation and reduction of salivary secretions that might otherwise be aspirated. The effect of acepromazine on esophageal relaxation is supported in one experimental study.[16] However, oxytocin administered intravenously (at 0.11 and 0.2 IU/kg) results in short-term significant relaxation of esophageal musculature in experiments, and it has become popular as an adjunct treatment for esophageal impaction.[11] Because xylazine has similar effects, these drugs could be used together to reduce the tone of the esophageal musculature, allowing passage of the obstructions with reduced risk of esophageal injury.

Refractory cases or intractable horses may benefit from general anesthesia and water lavage under pressure. This method has the advantages of providing some relaxation of the esophageal musculature, reducing the chances of aspiration (because

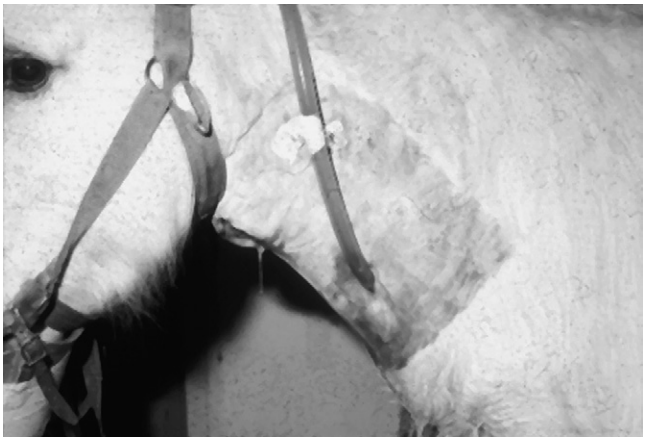

Figure 31-15. Placement of a feeding tube ventral to the jugular vein permits it to lie in a comfortable position on the neck. Note the butterfly tape bandage sutured to the skin to firmly anchor the tube, along with saliva loss around the tube.

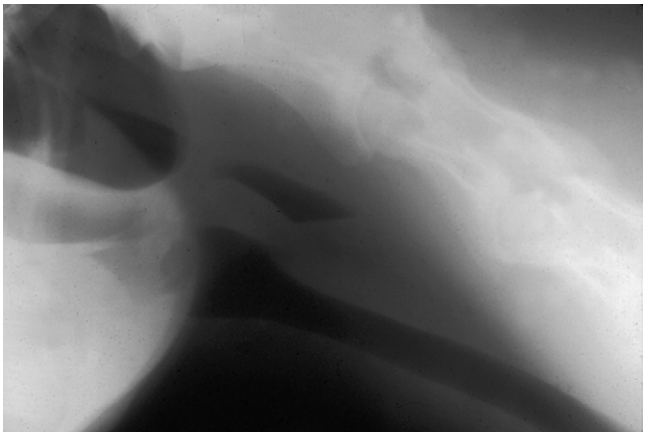

Figure 31-17. Lateral cervical radiograph of a horse after impaction of the esophagus has been relieved shows a fluid line in an area of dilation that extends from the distal point of obstruction proximad to the upper esophageal sphincter. The fluid line is produced by saliva that has collected in the dilated area.

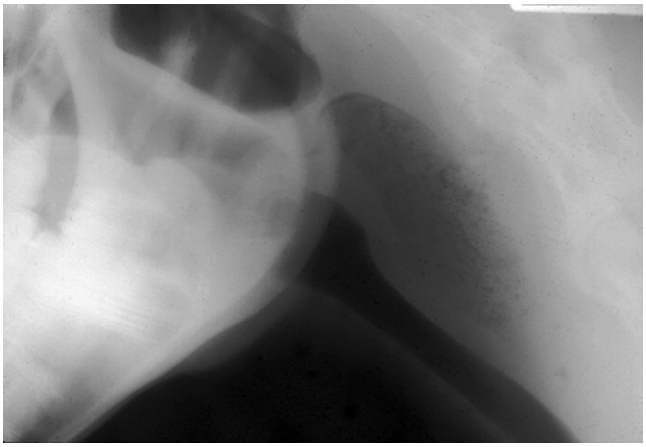

Figure 31-16. Lateral cervical radiograph shows typical appearance of impaction of ingesta in the cranial cervical esophagus.

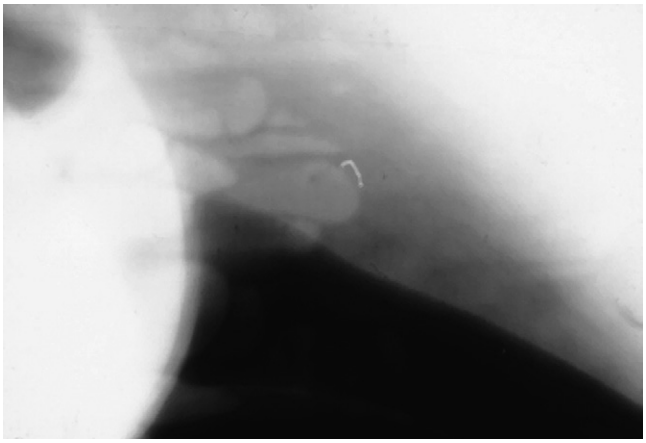

Figure 31-18. Lateral cervical radiograph of a 5-year-old Quarter Horse gelding with cervical swelling, dysphagia, and odynophagia. A metallic foreign body (wire) is lodged in the lateral wall of the cranial esophagus. A nasogastric tube passed easily. Note the gas in the tissues.

the horse's head is lowered), and decreasing the risk of esophageal perforation with the tube in an intractable horse. Gentle manipulation is mandatory with this technique to avoid rupture of the esophagus. Impactions that do not respond to conservative therapy should be definitively identified by radiographic and endoscopic examination and, if amenable, relieved by longitudinal esophagotomy (see "Foreign Body," next). Surgery is preferable to repeated trauma of the esophagus through attempts to relieve the obstruction with a nasogastric tube. Use of the nasogastric tube as a probang is not recommended and can result in severe trauma to the larynx and esophagus.[17] Cervical esophagotomy can be performed with the horse standing or under general anesthesia, and obstructions can be lavaged through the incision if necessary.

One aftermath of simple impaction is fusiform dilation of the esophagus that predisposes to reobstruction (Figure 31-17). This condition resolves in 24 to 48 hours, provided the dilation is kept free of ingesta. Food should be withheld or only small quantities of a soft diet fed for 2 days after an episode of choke to permit the lumen to resume its normal diameter. Glucose–electrolyte solutions for drinking should be provided in addition to fresh water so the electrolyte abnormalities secondary to salivary loss can be compensated.

Broad-spectrum antimicrobial therapy is indicated for 5 to 7 days because the risk of aspiration pneumonia is high after choke. If pneumonia is not the major limiting factor in this disease, simple obstruction has a favorable prognosis.

When an obstruction has been present for several days or is refractory to initial treatment, examination using radiography or endoscopy is warranted after the obstruction has been relieved. Circumferential mucosal ulceration occurs occasionally in these cases and can result in esophageal stricture.

Foreign Body

Small pieces of wood, wire, fishing tackle, and medication boluses can become esophageal foreign bodies in horses.[18,19] They often perforate the esophageal wall, resulting in phlegmon or abscessation (Figure 31-18). The swelling that accompanies

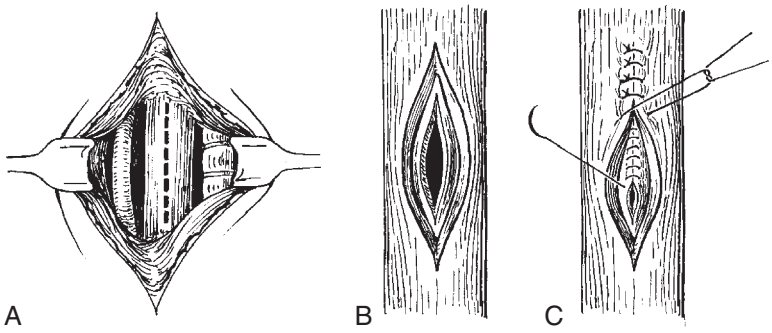

Figure 31-19. A, Longitudinal skin incision exposes the carotid artery *(left)*, esophagus *(center)*, and trachea *(right)*. **B,** In this longitudinal esophagotomy, a scalpel is used to incise both the outer layer (muscularis and adventitia) and the inner layer (mucosa and submucosa). **C,** The esophagotomy is closed with simple-continuous sutures in the inner layer (mucosa and submucosa) and simple-interrupted sutures in the outer layer (muscularis and adventitia).

these conditions usually obstructs the esophageal lumen and results in impaction. Diagnosis is made by radiography or esophagoscopy.

Retrieval of small sharp foreign bodies under endoscopic guidance is possible but difficult; general anesthesia is recommended to prevent the swallowing movements during manipulation that may produce pain and further esophageal trauma. It is necessary to relieve the impaction before attempting this method of treatment. Blunt or round foreign bodies may be treated similar to feed impactions. Although the nasogastric tube may be used as a probe to push the object into the stomach, the risk of perforation is great. Gentle manipulation is mandatory to prevent rupture. Additionally, this technique has the risk of moving the foreign body from the cervical region only to have it lodge in the thorax, a less-accessible site. Longitudinal esophagotomy with primary closure results in minimal complications when performed in a region of normal esophagus and has become the accepted method of removing a foreign body.[20,21]

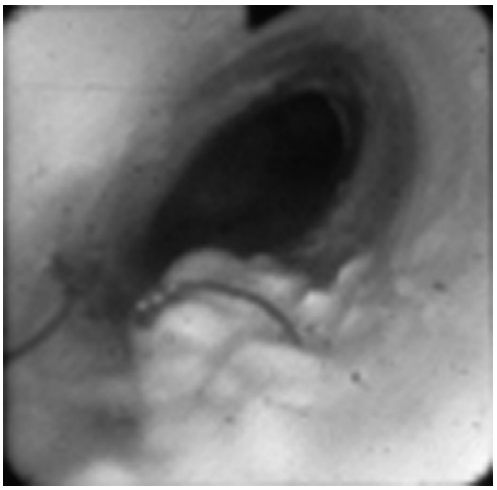

Figure 31-20. Endoscopic appearance of a longitudinal esophagostomy that healed by first intention 48 days after surgery. One loop of the suture remains in the healed esophagotomy site.

Esophagotomy

Passage of a nasogastric tube as far as the foreign body (or beyond, if possible) facilitates identification of the esophagus during surgery. General anesthesia is preferred if closure is to be attempted. The patient is placed in dorsal recumbency, and the skin of the ventral surface of the neck is prepared and draped for aseptic surgery. A 10-cm (4-inch) skin incision is made and the esophagus is approached (see earlier in this chapter). Care should be taken to preserve the small vessels that supply the esophagus. Elevation of the esophagus from its bed of adventitia should be avoided. The left carotid sheath, containing the carotid artery and vagus and recurrent laryngeal nerves, should be retracted laterally. Pediatric-size Balfour abdominal retractors aid exposure of the esophagus, which then can be sharply incised through the muscle, submucosa, and mucosa cranial to, caudal to, or directly over the foreign body (Figure 31-19). The exact location of the incision into the esophagus depends on the mobility of the foreign body within the lumen and the amount of swelling in and compromise to the esophageal wall.

After removal of the foreign body, if the esophagus has a normal appearance in the area of the incision, closure should be completed using a simple-continuous suture of 3-0 polypropylene suture material with the knots tied in the lumen (see Figure 31-19). Esophageal musculature may be apposed with simple-interrupted sutures of 3-0 absorbable or monofilament nonabsorbable suture material, at the surgeon's preference. Muscular layers, subcutaneous tissue, and skin may be apposed with 0 suture material in a simple-interrupted pattern. A polyethylene drain with an outer diameter of 0.63 cm (¼ inch) is placed beside the esophagus and exited ventral to the skin incision through a small stab wound. This drain is maintained under constant suction for 48 hours to remove serum and blood from the surgical site and to provide early detection of salivary leakage should dehiscence occur.

Postoperatively, feed should be withheld for 48 hours. Parenteral administration of electrolyte solution, the composition of which depends on the horse's acid-base and hydration status, may be used to maintain hydration. Small quantities of pelleted feed in a slurry should be fed over the next 8 days before normal feeding can be resumed. Most esophagostomy incisions heal by first intention, and the intraluminal suture will slough into the lumen within 60 days (Figure 31-20).

If removal of the foreign body is necessary through an obviously diseased segment of the esophagus, the incision may be closed and an esophagostomy tube placed through a separate incision closer to the stomach or directly into the esophagotomy incision (see "Esophagostomy," later). Feeding of a complete pelleted diet through the esophagostomy tube can begin immediately after surgery. Particular attention to the patient's water and electrolyte balance is necessary when closure of the esophagus is not possible (see "Acid-Base and Electrolyte Alterations," later, and Chapter 3).[22]

Ulceration and Esophagitis

Mucosal ulceration and esophagitis commonly occur secondary to long-standing impactions. Longitudinal mucosal ulcers can be produced from impactions; more frequently, circumferential ulcerations occur. Other causes of esophageal ulceration in foals include phenylbutazone toxicity, in which generalized gastrointestinal mucosal disease is a feature, and severe gastroduodenal ulcer disease that produces secondary reflux esophagitis. Diagnosis of esophageal ulceration is best made by endoscopy (Figure 31-21), although contrast radiography frequently defines the margins of the ulcer (see Figure 31-7).

Conservative management should be instituted to minimize trauma to the mucosa, reduce inflammation, and control infection. A low-bulk, minimally abrasive diet (mash), nonsteroidal anti-inflammatory drugs (NSAIDs) (only if they are not implicated as causative agents), and broad-spectrum antimicrobial therapy are indicated. Because this diet results in hunger, the patient should be muzzled between feedings, or all bedding should be removed from the stall to avoid ingestion of straw or wood chips. Reexamination is recommended every 10 to 14 days. Stricture (see later) may occur within 30 days of the original insult, when the circumferential ulcer is more than 2.5 cm (1 inch) long.[23] Longitudinal mucosal ulcers (especially if they are not extensive and are localized to one area of the esophagus) and circumferential ulcers less than 2 cm (¾ inch) long usually heal without stricture.

Rupture, Perforations, and Lacerations

Rupture of the esophagus can occur secondary to long-standing obturation, repeated or aggressive nasogastric tube passage, foreign body perforation, external trauma to the cervical area (usually a kick), or extension of infection from surrounding strictures.[24-27] Cervical swelling usually prevents successful passage of a nasogastric tube, even after irrigation of the esophagus with water. Swallowed air escapes from the rupture and causes subcutaneous emphysema; this can be recognized ultrasonographically or radiographically (see Figure 31-18). Positive-contrast techniques demonstrate escape of barium into surrounding tissues (Figure 31-22).

Ruptures that cannot drain to the outside lead to leakage of saliva and ingesta into tissues of the neck, resulting in severe infection and phlegmon development. Ruptures or perforations that allow escape of saliva and ingesta through the skin are less likely to cause systemic illness and extension of infection into the thorax. Early establishment of drainage, preferably on the ventral midline, is necessary with all ruptures of the esophagus to avoid mediastinitis, pleuritis, and even septicemia (Figure 31-23). This should be followed with daily lavage.

Perforation or lacerations of the esophagus accompanied by minimal escape of saliva and ingesta can be repaired using the technique described for esophagotomy. Drainage and the feeding regimen used after esophagotomy should be employed. Secure closure of a ruptured or perforated defect is usually possible only in patients operated on shortly after the perforation has occurred (within 12 hours). In early cases, when esophageal tissues are too damaged to hold sutures or when infection or contamination with ingesta has already occurred, some means of draining the esophageal contents to the outside must be provided. The patient should receive systemic antibiotics and water; electrolyte and nutritional requirements should be met by tube feeding. In some cases, the feeding tube can be placed through the site of rupture into the stomach (Figure 31-24). An alternative method of feeding allowing spontaneous healing of the rupture or successful repair of the rupture when edema and infection have been controlled is an esophagostomy performed distal to the rupture (closer to the stomach).

Esophagostomy

Cervical esophagostomy is an excellent method of extraoral alimentation that prevents the discomfort and irritation of the indwelling nasogastric tube. An additional advantage is prevention of deleterious influences on healing of an esophageal wound by an intraluminal tube located in the immediate region.[28] To use this advantage, the esophagostomy should be placed distal to the area of esophageal injury. The surgery can be performed with the horse in lateral recumbency under general anesthesia or standing under mild tranquilization and

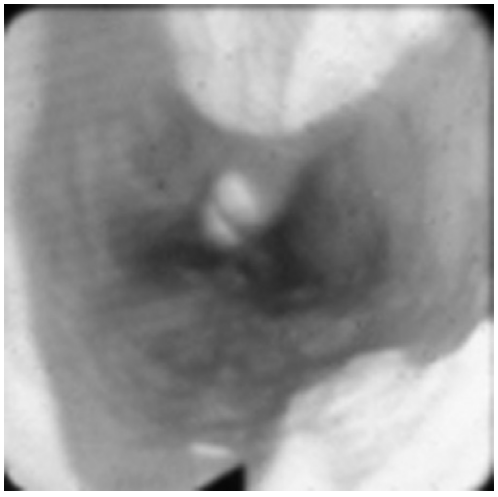

Figure 31-21. Endoscopic appearance of esophagitis with circumferential ulceration 24 hours after feed impaction. A stricture subsequently formed but responded to medical management.

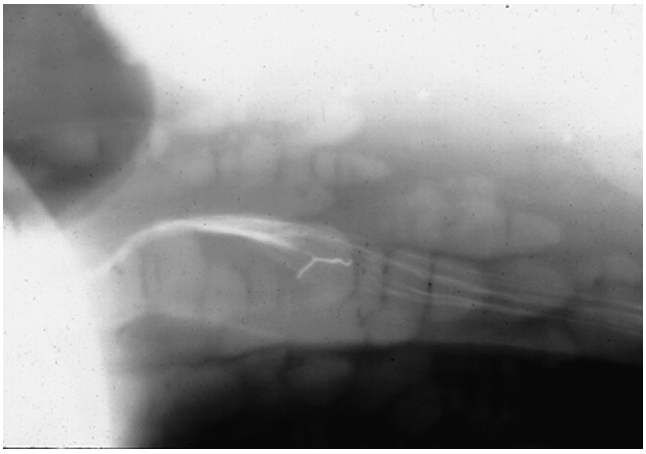

Figure 31-22. Barium swallow in a horse with a penetrating foreign body (wire) shows swallowed air and barium that has escaped into the periesophageal tissue (same horse as in Figure 31-18).

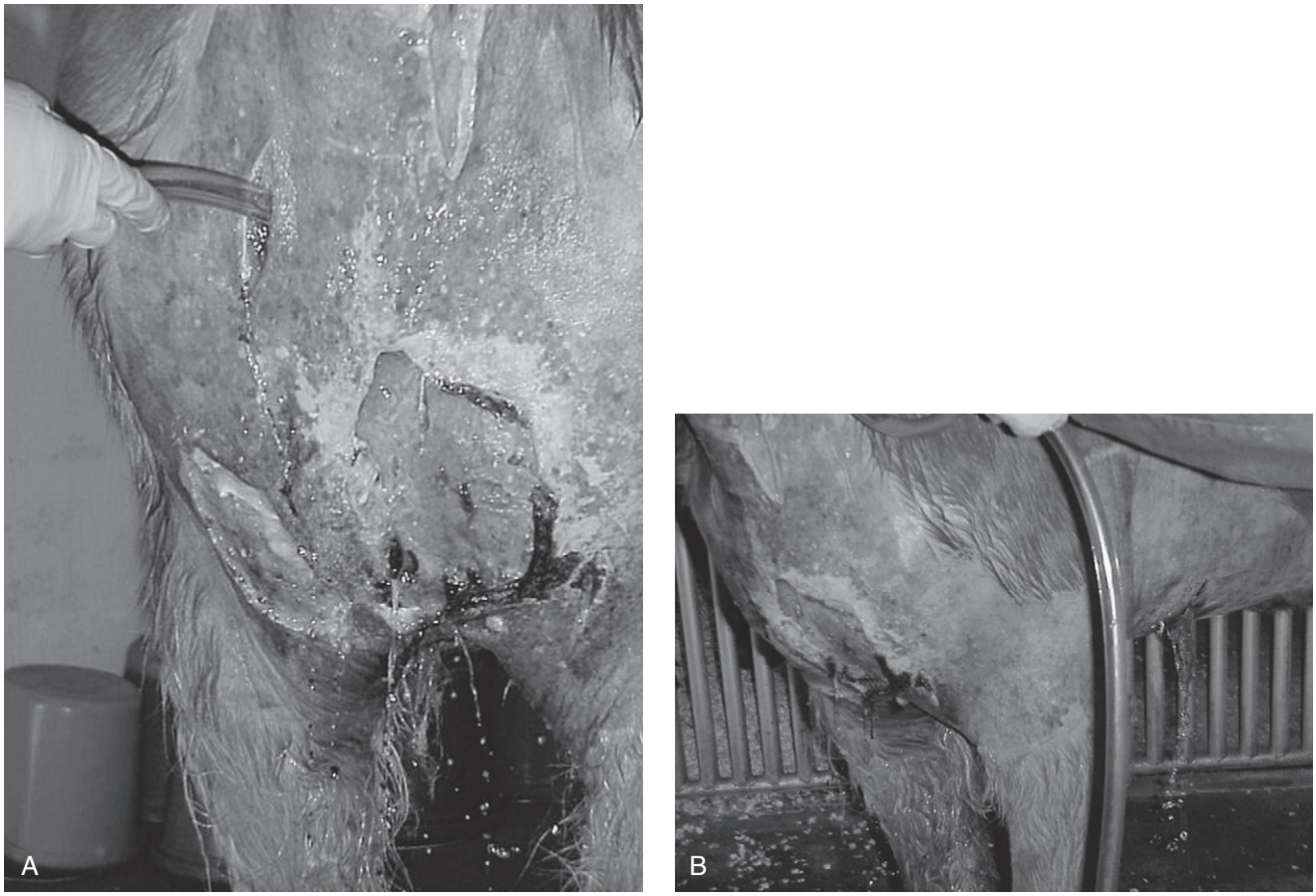

Figure 31-23. A, Establishment of ventral drainage following esophageal rupture in which multiple incisions needed to be made to drain food material from the cranial pectoral area. **B,** Daily lavage with copious amounts of water through the incisions shows the communication between the caudal cervical esophageal incisions and incisions in the pectoral area and caudal chest area behind the elbow.

Figure 31-24. Slurry made of complete pelleted feed is used to meet the nutritional needs of the patient.

local anesthesia. Passage of a nasogastric tube facilitates identification of the esophagus at surgery.

The skin over the left jugular furrow is prepared for surgery in the desired area. The esophagus is occasionally located on the right side of the trachea and should be approached over the right jugular furrow in those horses. A 5-cm (2-inch) skin incision is made ventral to the vein. The esophagus is sharply incised longitudinally for 3 cm (1¼ inches) down to the indwelling nasogastric tube. The nasogastric tube is removed, and a polyethylene nasogastric tube (with an outer diameter of 14 to 24 mm [¼ to 1 inch]) is placed into the stomach through the esophagostomy. Failure to place the tube into the stomach allows easy dislodgement. Care should be taken to ensure that the end of the tube is placed into both the elastic inner layer and the inelastic outer muscle layer of the esophagus. Difficulty in tube placement is usually an indication that the incision in the muscle layer is inadequate to accommodate the diameter of the tube. Sutures can be placed in the mucosa to form a seal around the tube but probably are unnecessary because they do not prevent leakage of saliva. The tube should be secured firmly, first with butterfly tape bandages sutured to the skin (see Figure 31-15) and then with elastic tape bandages. Tubes of large diameter are preferred to prevent plugging with ingesta during feeding. They should be capped between feedings and flushed with water at the end of each feeding to maintain patency.

Figure 31-25. Esophagostomy tube should remain in place for a minimum of 7 to 10 days to permit granulation tissue to form a true stoma.

Esophagostomy tubes should remain in place for a minimum of 7 to 10 days to permit granulation tissue to form a true stoma (Figure 31-25). A longer period is necessary if the tube is placed in the area of a rupture or perforation. When the tube is removed, normal feeding may be resumed. A large portion of swallowed feed may be lost through the stoma when the patient is fed from the ground (Figure 31-26). When feed is placed at the height of the withers, however, less of the bolus is lost through the stoma with each swallow. The stoma heals spontaneously after the tube is removed, and fistula formation is rare (Figure 31-27).

However, complications of this form of alimentation are well documented and can result in death. Early detection and ventral drainage of infections that dissect along the trachea and esophagus are vital for a successful outcome. Patients should be maintained on antimicrobial therapy until a mature stoma develops (7 to 10 days).

Resection of the esophagus after rupture is warranted if the muscular layer is obviously necrotic and does not act as a tube along which the mucosa may regenerate and if the proximal and distal segments of the esophagus can be anastomosed without undue tension (see "Partial Resection" and "Resection and Anastomosis," later). Few ruptures of the esophagus requiring resection occur without necrotizing cellulitis. Usually a delay in repair is necessary to permit acute inflammation to subside before surgery. During this period, the patient may be accommodated with a change in diet and tube feeding.

Stricture

Narrowing of the esophageal lumen due to stricture formation is usually an annular lesion and can be classified into the following three types, depending on the anatomic location of induration and fibrosis: (1) mural lesions that involve only the adventitia and muscularis, (2) esophageal rings or webs that involve only the mucosa and submucosa, and (3) annular stenosis that involves all layers of the esophageal wall. Stricture of the esophagus may be acquired as a result of external or internal trauma to the esophageal walls (especially after impactions that produce circumferential ulceration), leakage of saliva or dehiscence after surgery, or external compression by or attachment to adjacent structures. In most instances, strictures are less likely

Figure 31-26. After removal of the esophagostomy tube, a large portion of masticated feed and saliva may be lost through the stoma when the patient is fed from the ground. Placing the feed at the height of the withers allows less of the bolus to be lost through the stoma with each swallow. The horse may electively eat the masticated feed, and this should be encouraged because the salt content from the saliva will be reswallowed and eventually ingested.

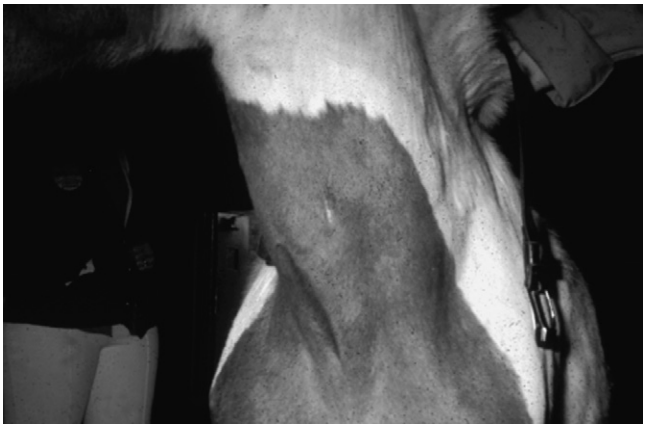

Figure 31-27. The stoma heals spontaneously after the tube is removed, and fistula formation is rare. (Three weeks after tube removal from the animal seen in Figure 31-25.)

to occur when traumatic insults involve only a portion of the circumference of the esophagus. For this reason, mobilization of the esophagus from its fascial attachments during surgery should be avoided when possible.

Strictures usually impede complete passage of the nasogastric tube and are best demonstrated by positive-pressure contrast esophagograms (Figure 31-28). Conservative management of a stricture is aimed at dilation of the stenotic segment. Bougienage or pneumatic or hydrostatic dilators have limited practical value in adult horses because of the inaccessibility of special equipment and the chronicity of the problem. There is a report describing resolution of an esophageal stricture in a 1-month-old colt using a balloon dilation procedure,[29] but in my experience, repetition of the procedure is often necessary and is rarely successful. However, early lesions, such as postsurgical strictures or those following circumferential ulceration, can be dilated with the frequent feeding of small quantities of soft food over a period of several months.

In seven horses that developed stricture after esophageal impaction, observation over 2 months revealed that the esophageal lumen was maximally reduced (strictured) 30 days after circumferential ulceration was observed (Figure 31-29), after which lumen diameter increased to normal by 60 days.[23] This also has been documented after experimental resection and anastomosis and has been observed in a series of cases involving foals.[30] Therefore a low-bulk diet and anti-inflammatory and antimicrobial therapy should be used, and surgical intervention should be delayed for 60 days after the original insult. It is important to impress on horse owners that several episodes of choke may occur up to 40 days after the original obstruction.

Strictures more that 60 days old have usually matured to the point where the cicatrix is too firm to yield to dilation by this method and therefore may be classified as chronic (Figure 31-30). Chronic strictures of the esophagus may be corrected by esophagomyotomy, partial or complete resection and anastomosis, or patch grafting. Complications may occur with any of these surgical treatments, and the surgeon should take care to pick the most conservative therapy that will meet the aim of treatment. Leakage of luminal contents and reformation of the stricture requiring prolonged medical management are to be expected after resection and patch grafting. Strictures that are mural in origin respond to myotomy and have the best

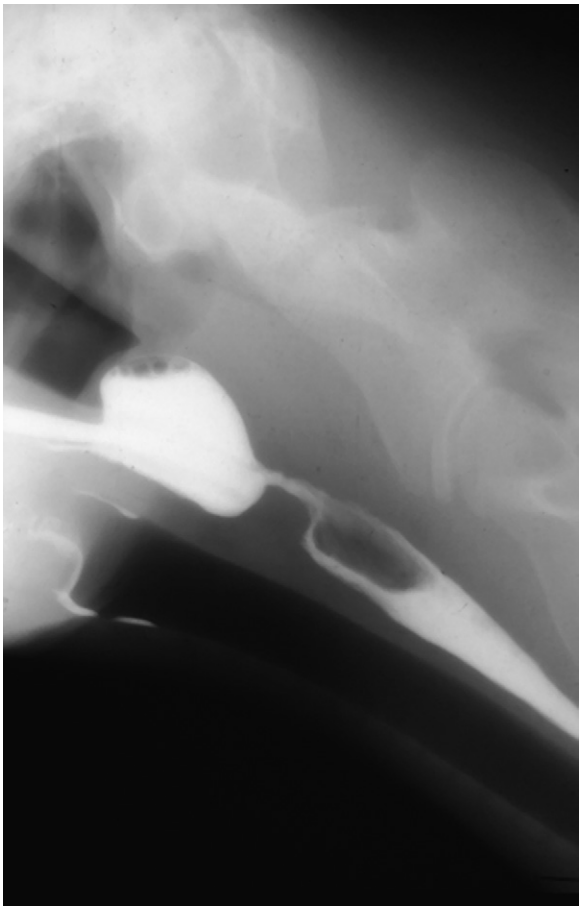

Figure 31-28. Positive-pressure esophagogram showing a stricture in a foal. Note the prestenotic dilation. The lesion was resolved by partial resection and anastomosis.

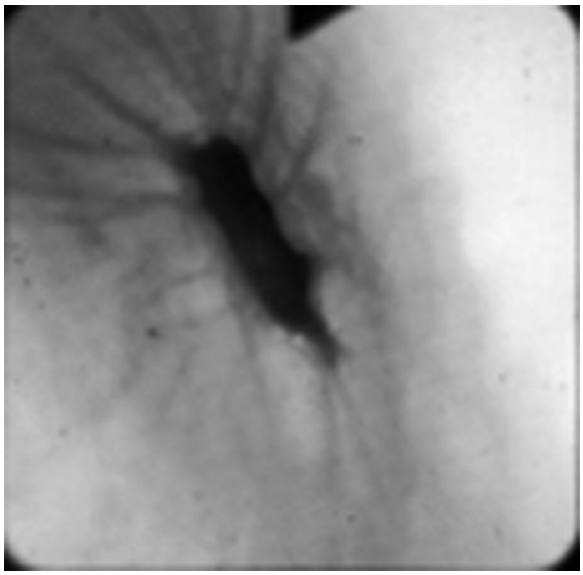

Figure 31-29. Endoscopic appearance of a stricture 30 days after circumferential ulceration.

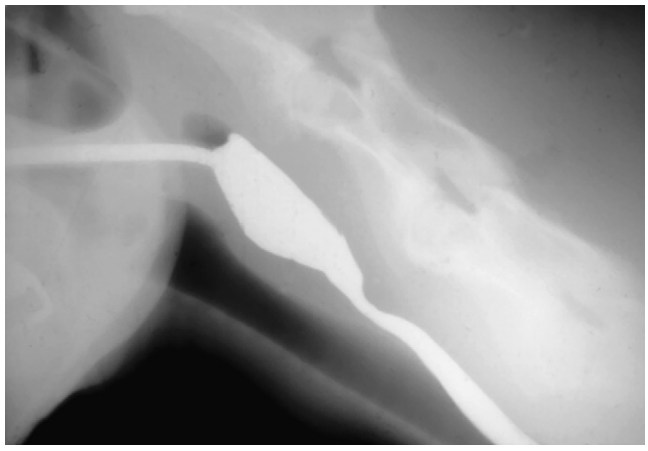

Figure 31-30. Positive-contrast esophagogram using liquid barium administered under pressure through a cuffed nasogastric tube shows stricture with prestenotic dilation. The stenosis was subsequently resolved by esophagomyotomy.

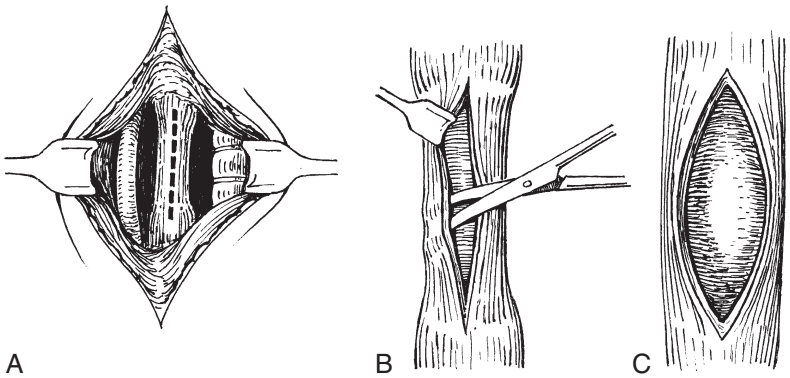

Figure 31-31. A, Esophagomyotomy: longitudinal incision of the outer layer of the esophageal wall. **B,** Elevation and separation of the outer (muscularis and adventitia) and inner (mucosa and submucosa) layers of the esophageal wall to complete the esophagomyotomy. **C,** The muscularis and adventitia are not sutured.

prognosis for recovery without restricture. Three successful reports appear in the literature; I have had similar experience with five other horses.[31,32] The surgery should be performed with the horse under general anesthesia, with a nasogastric tube passed to the level of the stricture to permit easy identification of the involved area.

Esophagomyotomy

The esophagus is incised longitudinally to the level of the mucosa, through the stricture, and 1 cm (½ inch) distal and proximal to it (Figure 31-31, *A*). The nasogastric tube may be passed through the stenotic area at this point. From this single incision, the muscularis is separated by sharp dissection from the mucosa around the entire circumference of the esophagus (see Figure 31-31, *B*). When the mucosa is freed in this manner, removal of a portion of the muscularis or multiple myotomy incisions are seldom necessary. The myotomy is not sutured (see Figure 31-31, *C*), and the approach incision is closed and drained in a routine manner. If the mucosa is opened inadvertently, it should be closed immediately with 3-0 polypropylene sutures in a simple-continuous pattern. Postoperatively, frequent feeding of small quantities of soft feed may be necessary if a prestenotic dilation was present before surgery. When this dilation is no longer evident radiographically, normal feeding may be resumed.

Recurrence of postsurgical cicatricial stricture is slow to develop, with clinical signs occurring weeks or months after the operation. Conservative treatment (changing from hay to a complete pelleted diet) may be all that is necessary to resolve recurrent obstructions. A postsurgical stricture seen long after the original operation usually is the result of a mature nonresilient cicatrix and may not respond to dilation. If surgery is necessary, the surgeon should not hesitate to perform a second esophagomyotomy; however, performing another surgery in a stenotic area of the esophagus before allowing the acute inflammation of the previous surgery to subside greatly increases the propensity for restricture. If such is the case, a more hazardous procedure may have to be selected eventually to try to correct the problem.

Partial Resection

Longitudinal esophagomyotomy combined with mucosal resection provides relief of stricture caused by esophageal rings or webs or annular stenosis of all muscle layers.[33,34] Performed under general anesthesia, the procedure is indicated when the cicatrix involves the mucosa and prevents passage of a nasogastric tube after myotomy.

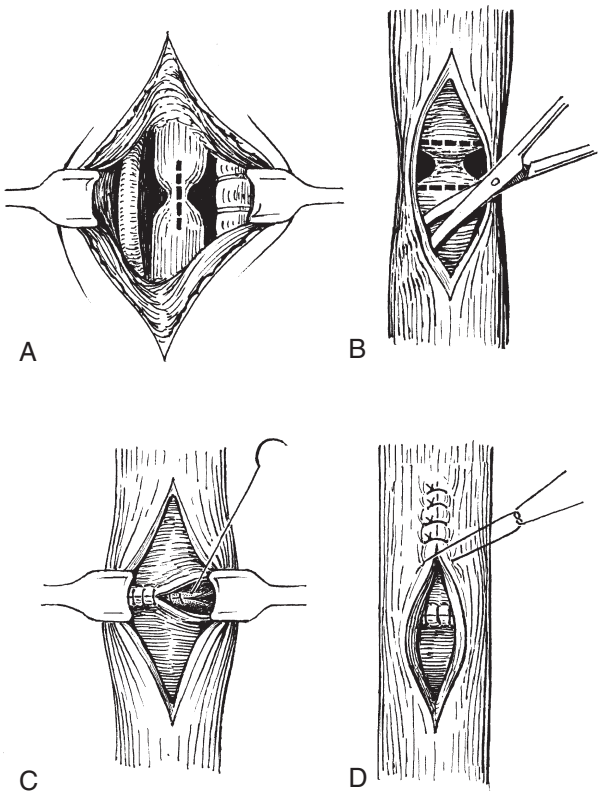

Figure 31-32. Partial resection and anastomosis. After longitudinal incision of the outer layer **(A),** the inner layer is resected **(B)** and, when possible, closed transversely using several simple-continuous sutures **(C).** The outer layer is then closed with interrupted sutures **(D).**

The esophagus is exposed and incised as described previously for esophagomyotomy. A longitudinal incision is made through the mucosa long enough to permit identification of the diseased segment (Figure 31-32, *A*). The mucosal scar is separated by sharp dissection from normal or diseased muscle layer. Circumferential incisions are made at the proximal and distal edges of the mucosal cicatrix and it is removed, leaving the muscular tube intact (see Figure 31-32, *B*).

If cut edges of the mucosa can be brought into apposition without undue tension, they are apposed by three equally

spaced 3-0 polypropylene simple-continuous sutures with the knots tied in the lumen (see Figure 31-32, *C*). When mucosal rings or webs are the cause of stenosis, the normal esophageal muscle should be apposed over the mucosal anastomosis (see Figure 31-32, *D*), but in the case of an annular stenosis that involves the entire esophageal wall, the muscularis should not be sutured. A drain is placed next to the esophagus, and the approach incision is closed. If space permits, tube feeding through an esophagostomy placed through a separate incision distal to the stricture is ideal. When this is not possible, frequent feeding of small quantities of soft food may begin 48 hours after surgery and should be continued for 10 days before normal roughage is offered to the patient.

When the stricture is extensive and the mucosa cannot be sutured, regeneration of the mucosa within the muscle tube occurs readily. The muscularis may be sutured if it is healthy, or it may be left open if only scar tissue remains. Spontaneous healing is aided if esophagostomy tube feeding (located closer to the stomach than the operative site) is used. When the stricture is located too close to the thoracic inlet to permit placement of a separate esophagostomy incision, the tube may be inserted directly through the stricture site. Recurrence of stricture after this procedure may be an indication for esophagomyotomy or an esophagostomy tube placed through the stricture.

Resection and Anastomosis

Complete resection and anastomosis should be reserved for rupture of the esophagus in which the muscularis is not viable. The esophagus is exposed as described previously. The area to be resected and several centimeters of normal esophagus distal and proximal to it are mobilized. Umbilical tape or rubber drain tubing (0.63 cm [¼ inch]) is placed around the esophagus and held in place with hemostatic forceps to occlude the lumen a convenient distance from the area to be resected. Crushing clamps of any type should not be used on the esophagus. A point is selected proximal to the diseased segment, where the esophagus is sharply transected, leaving healthy tissue for closure. This procedure is repeated distad, and the diseased portion is removed. The mucosal-submucosal layer is apposed with 3-0 simple-interrupted monofilament nonabsorbable polypropylene sutures placed about 3 cm (¼ inch) from the cut edge, 2 to 3 mm (1/12 to ¼ inch) apart, with the knots tied in the lumen. Tension of the sutures is adequate to form a tight seal without interference of the blood supply.

The esophageal muscle is apposed with interrupted horizontal mattress sutures of 2-0 polydioxanone or monofilament nonabsorbable suture material. The muscle layer has limited elasticity and, if necessary, a relief incision in the form of a circular myotomy 4 to 5 cm (1½ to 2 inches) proximal or distal to the anastomosis can decrease tension on the repair. The approach incision is closed as described previously.

Alternative successful methods of closure have been described, but postoperative management is a major determinant of the outcome of this procedure, regardless of closure technique.[35,36] Prevention of undue tension on the anastomosis by use of a standing martingale and special feeding regimen is necessary (Figure 31-33). If oral alimentation is elected, soft foods should be given only after 48 hours and until endoscopic or radiographic evaluation of the surgery shows primary healing has occurred. Esophagostomy is the preferred method of feeding, if possible. An extensive cervical esophageal stricture produced by annular stenosis of the entire wall may preclude successful repair by techniques described previously. The diameter of the equine esophageal lumen can be increased by using a patch graft of the sternocephalicus muscle or by esophgopexy to create a diverticulum.[37,38]

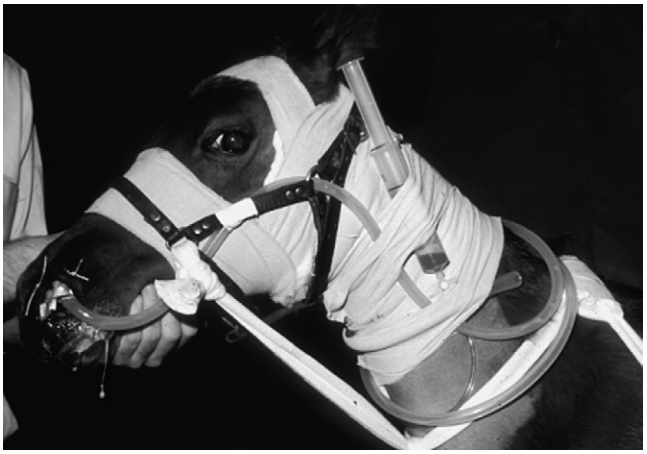

Figure 31-33. When complete resection anastomosis is performed, undue tension frequently results in dehiscence. Undue tension on the anastomosis can be prevented by using a standing martingale, as shown, which prevents extension of the head and neck.

Patch Grafting

Antibiotics should be given preoperatively and maintained for 6 to 10 days after surgery. With the horse under general anesthesia, a ventral midline or lateral approach to the esophagus is made and, depending on location of the defect and the approach used, the brachiocephalicus and sternocephalicus muscles serve as donors for the graft. With a nasogastric tube passed to the level of the stenosis, a longitudinal incision is made through the muscularis from a point 3 cm (1¼ inch) distal to and extending 3 cm proximal to the stricture. The mucosa and submucosa are sharply incised as the nasogastric tube is passed into the stomach. A caudal portion of the muscle belly of the brachiocephalicus or sternocephalicus is mobilized by blind separation of muscle fibers. The strip of muscle should maintain its proximal and distal attachments and should be freely movable so as not to exert tension on the closure when the patient's head and neck are moved. The graft should be wide enough to appreciably increase the lumen of the esophagus.

The edges of the mucosa and submucosa are sutured to the muscle graft using 3-0 polypropylene interrupted through-and-through mattress sutures, and the edges of the muscularis are sutured to the graft with 3-0 simple-interrupted sutures of monofilament polypropylene. Preplacement of mattress sutures and closure of both layers on one side of the esophageal defect at a time facilitate repair. The nasogastric tube should be removed before the second edge is closed. Suction drains are placed next to the esophagus, and the approach incision is closed.

Postoperatively, extraoral alimentation is preferred for 10 days (by esophagostomy, if space allows, or intravenous feeding), but soft foods may be given *per os* as early as 48 hours

if salivary leaking has not occurred. An indwelling nasogastric tube provides a stimulus for salivation and increases the incidence of fistula formation.

Fistula

Fistulas of the esophagus result from causes previously described for ruptures and may be treated similarly. However, small fistulas may be a diagnostic challenge. Occasionally, they are observed endoscopically (Figure 31-34). More frequently, findings of endoscopic examinations and barium swallow esophagograms are normal. Esophageal fistulas should be included in the differential diagnosis when cervical swelling, fever, and dysphagia are present; a nasogastric tube can be passed to the stomach; and endoscopic findings are normal. Contrast radiography, using liquid barium administered under pressure, best demonstrates the lesion (Figure 31-35). When ventral drainage has been established, fistulas almost always heal spontaneously. During this time, food and water may be expelled through the cloaca (Figure 31-36). Large fistulas can extrude copious amounts of masticated food and saliva. Elevating the food and water sources will decrease the amount lost. If healing does not occur, resection of the sinus tract and closure of the esophageal stoma may be necessary (Figure 31-37). Closure of the esophageal defect should be performed as for esophagotomy.

Diverticulum

In horses, diverticula of the esophagus only occasionally cause esophageal dysfunction. Usually they are acquired lesions that result from contraction of periesophageal fibrous scar tissue, causing outward traction and tenting of all layers of the esophageal wall (traction, or true, diverticulum). They can also occur from protrusion of mucosa and submucosa through a defect in the esophageal muscularis (pulsion, or false, diverticulum) (Figure 31-38).

A traction diverticulum commonly develops at the site of a healed esophagostomy, at the site of a postsurgical or posttraumatic wound of the esophagus that is allowed to heal by second intention as a fistula, or after penetration of the esophageal lumen (traumatic or surgical) in which leakage of saliva has caused inflammation or abscess formation. Pulsion diverticula are caused by fluctuations in esophageal intraluminal pressure and overstretch damage to esophageal muscle fibers by impacted feedstuffs. A more probable cause is external trauma to the cervical area.[39-41]

Diverticula in the cervical esophagus should be considered when an enlargement in the neck results in dysphagia and yet a nasogastric tube can be passed. On barium swallow

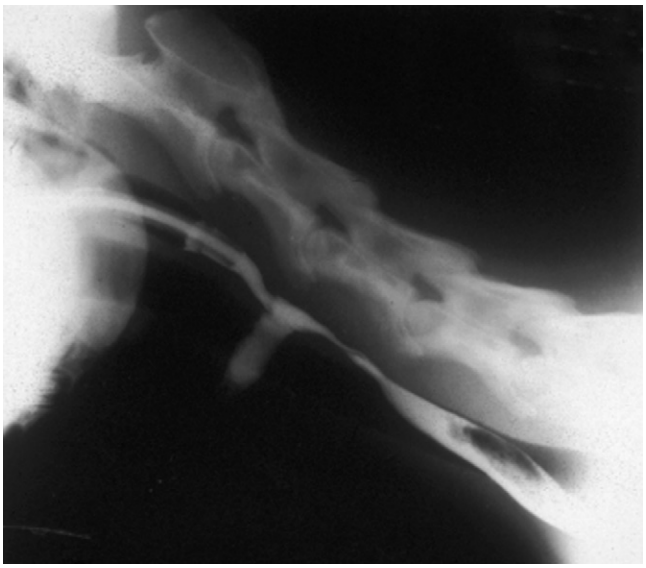

Figure 31-35. Positive-contrast esophagogram shows a large fistula remaining after removal of the esophagotomy tube. The cuff on the nasogastric tube prevents reflux of barium into the pharynx.

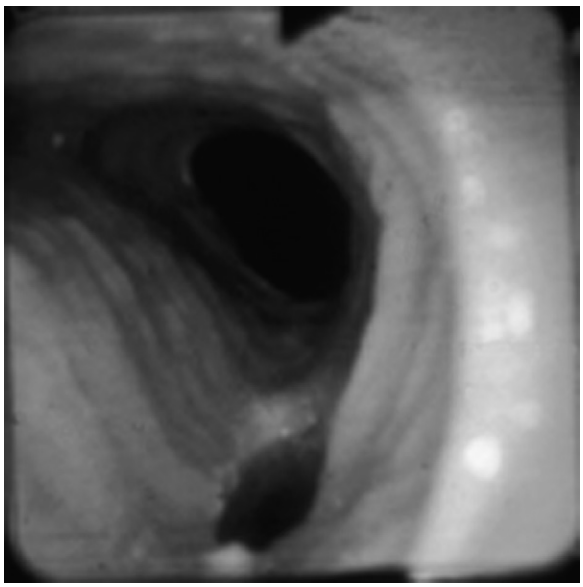

Figure 31-34. Endoscopic appearance of a large esophageal fistula. Smaller fistulas are often difficult to locate endoscopically.

Figure 31-36. A horse drinking, with a large esophageal fistula. When the water source is below the head, almost all of the water is lost. Therefore food and water should be elevated to decrease the amount lost through the fistula.

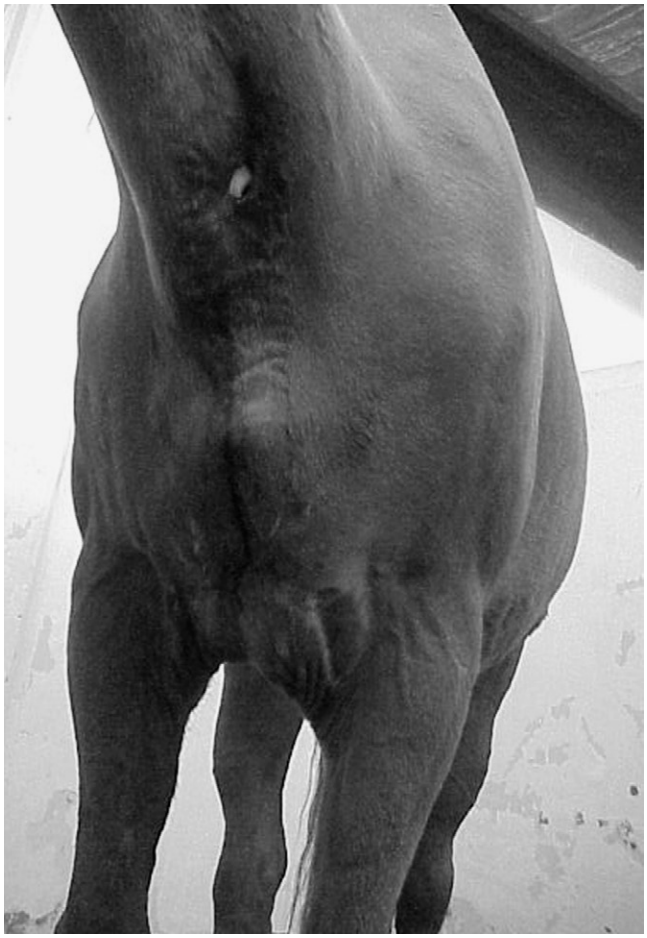

Figure 31-37. An esophageal fistula resulting from placement of the esophagostomy tube through a ruptured esophagus. Redundant mucosa prevented this fistula from healing spontaneously, and it was closed using the techniques described for esophagotomy.

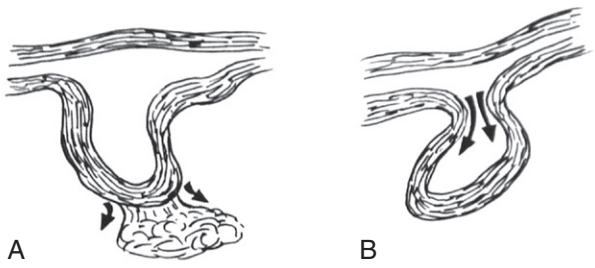

Figure 31-38. A, Traction diverticulum or true diverticulum develops at the site of a postsurgical or posttraumatic wound of the esophagus that is allowed to heal by second intention (e.g., a fistula or penetration of the esophageal wound). This type of diverticulum has a wide neck that seldom needs surgical correction. **B,** Pulsion diverticulum is caused by external trauma to the cervical area that ruptures the muscular layer. This type of diverticulum has a narrow neck and may continue to enlarge or ferment feed within the lumen, requiring surgical correction.

esophagograms, traction diverticula are spherical and have a wide neck (Figure 31-39), whereas pulsion diverticula are flask-like in configuration, with a narrow neck. Differentiation of these two types of diverticula can be aided by additional contrast techniques that distend the esophageal lumen and outline

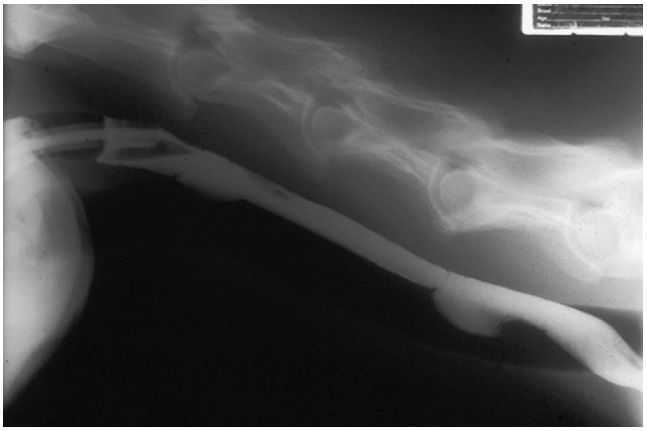

Figure 31-39. Barium swallow procedure in a horse with an esophagotomy that healed by second intention shows a traction diverticulum. Note the wide neck of the diverticulum.

the opening into the evagination. Esophagoscopy also defines the relative size and configuration of the opening of a diverticulum (Figure 31-40).

A traction diverticulum, even when quite large, produces few clinical signs and seldom requires treatment. A pulsion diverticulum, however, has a tendency to enlarge progressively so that risk of obstruction and rupture increase with time. Surgical repair is indicated. A pulsion diverticulum can be repaired by diverticulectomy with resection of the mucosal-submucosal sac, followed by reconstruction of the mucosa, submucosa, and muscularis, or by inversion of the mucosal-submucosal sac with reconstruction of the muscular layer. Diverticulectomy should be used when the mucosal sac is very large and the neck of the diverticulum very narrow. There are reports of repair of apparent congenital esophageal diverticula using this technique.[42]

However, mucosal inversion is the preferred technique because it decreases the chance of postoperative leakage, infection, or fistula formation and does not appear to predispose to postoperative obstruction complications.

Mucosal Inversion

The esophagus is exposed, and the diverticulum and defect in the muscularis are identified by careful dissection (Figure 31-41). Inadvertent perforation of the mucosa and submucosa should be repaired immediately. The edges of the muscular defect should be débrided back to healthy-appearing tissue. The sac is inverted, and the edges of the muscularis are apposed with simple-interrupted sutures of 3-0 polypropylene in a manner to avoid undue tension on the closure and prevent stenosis of the esophageal diameter. Postoperatively, feeding should consist of soft foods for 4 to 6 weeks.

Intramural Cyst

Esophageal cysts are rare in all species, but several equine cases have been documented.[43-45] I have seen three additional horses with esophageal cysts: a 3-year-old Morgan mare, in which the cyst lining had radiographic signs of calcification, and two yearling Quarter Horse colts, both admitted for dysphagia. In all horses, an epithelial inclusion cyst (lined by stratified squamous

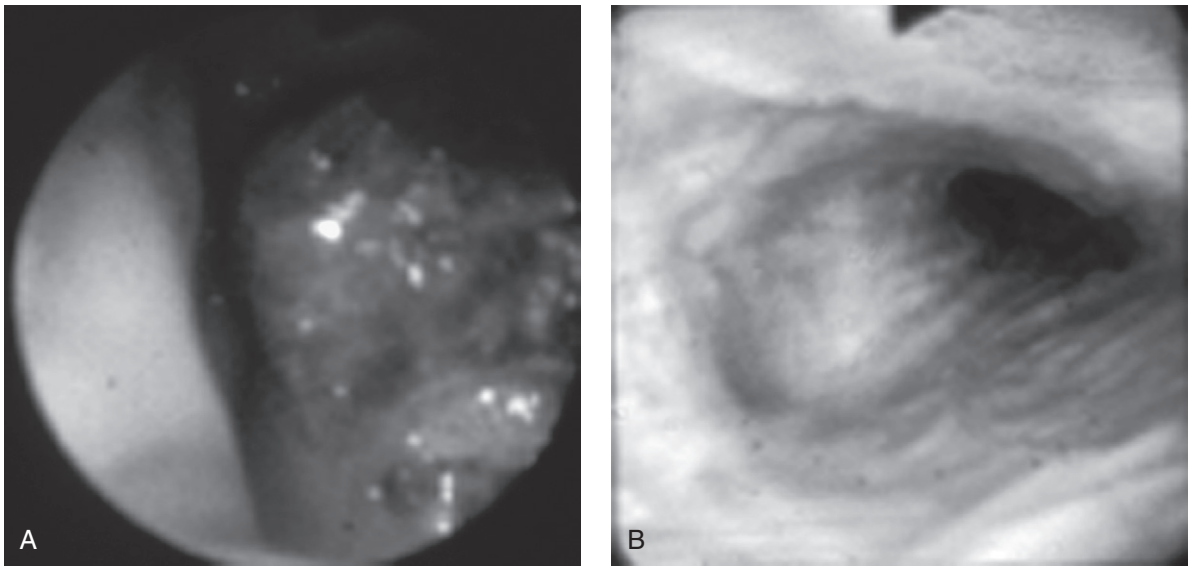

Figure 31-40. Endoscopic appearance of an opening into a traction or true esophageal diverticulum. Note feed in the diverticulum **(A)** and the normal-appearing mucosa **(B)** after lavage via drinking.

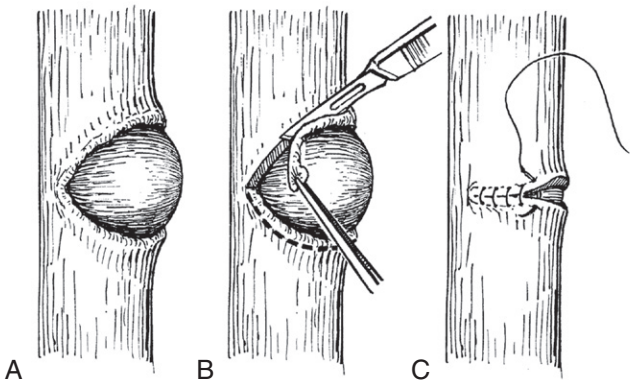

Figure 31-41. A, A pulsion diverticulum repaired by inversion of the mucosal/submucosal sac with reconstruction of the muscular layer. **B,** The edge of the muscular defect should be débrided back to healthy tissue. **C,** The sac is inverted, and the edges of the muscularis are apposed with simple-interrupted sutures of 3-0 polypropylene in a manner that avoids undue tension on the closure and prevents stenosis of the esophageal diameter.

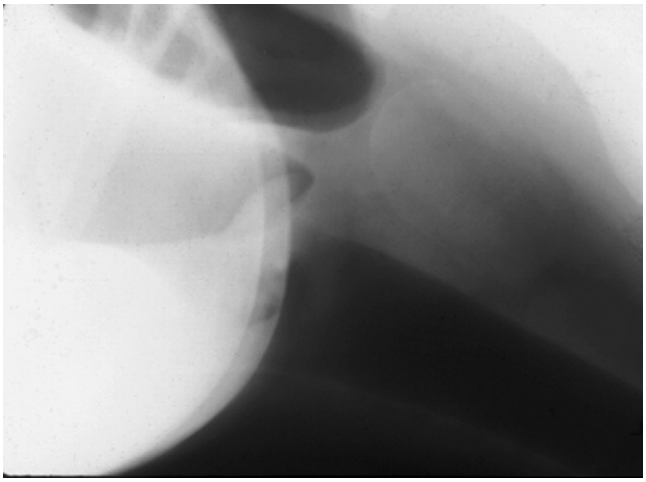

Figure 31-42. Radiographic appearance of an inner mural inclusion cyst of the esophagus in a 3-year-old Morgan mare in which the cyst lining had radiographic signs of calcification. This is an unusual finding, as survey films are usually nondiagnostic.

epithelium and filled with keratinaceous debris) was found in the esophageal wall. Mural cysts are probably a developmental anomaly.

Clinical findings include dysphagia, regurgitation, a palpable soft tissue mass in the neck, and resistance to passage of a nasogastric tube at the site of the cyst. The diagnosis can be made on survey films if the cyst has started to calcify (Figure 31-42). However, it is better confirmed on contrast radiography by the classic appearance of a filling defect caused by a mural lesion (Figure 31-43). Endoscopically, the esophageal lumen may appear partially occluded, but significant gross changes are usually not observed because the mucosa appears normal. Intramural esophageal cysts may be removed through enucleation with inversion or resection of redundant mucosa. The esophagus is exposed, and the cyst is identified by manipulation of a nasogastric tube within the esophageal lumen. A longitudinal incision is made through the muscularis over the cyst. By careful dissection, the cyst is separated from its position between the mucosa/submucosa and the muscularis and is removed intact (Figure 31-44). The cyst can be mistaken for an abscess if it is inadvertently incised by the inexperienced surgeon. Positioning the nasogastric tube caudal to the cyst helps prevent inadvertent perforation of the mucosa. After the cyst is removed, it leaves an area of redundant mucosa, or a sac, which may be treated as a pulsion diverticulum (i.e., by inversion, preferably, or by diverticulectomy). The muscularis is closed with simple-interrupted 3-0 sutures of polydioxanone or polypropylene at the surgeon's preference.

Megaesophagus

Megaesophagus refers to dilation and muscular hypertrophy of the esophagus oral (proximal) to a constricted distal segment. Although idiopathic muscular hypertrophy does occur in the horse (without dilation), it is an asymptomatic and an incidental finding at necropsy.[46] Therefore the term megaesophagus, as used here, includes congenital ectasia (dilation of unknown origin), achalasia (failure of the distal esophagus to relax because of neural dysfunction, resulting in proximal dilation), and megaesophagus secondary to vascular ring anomalies. One case of vascular ring anomaly causing megaesophagus in a foal has been reported.[47] The anomalous defect was a result of a persistent right aortic arch and was suspected on radiographic examination and confirmed on necropsy. Another case of megaesophagus was reported secondary to chronic gastroesophageal reflux.[48]

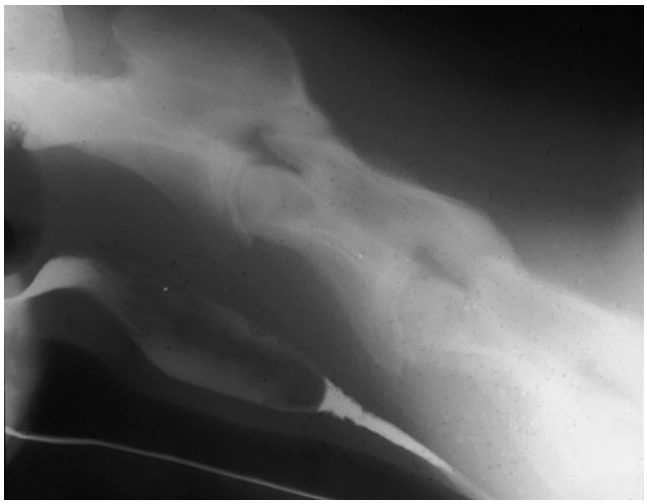

Figure 31-43. Barium esophagogram shows the classic appearance of an intramural lesion of the esophagus—in this case, an epithelial inclusion cyst. The esophageal lumen is narrowed caudad and is dilated but not filled with barium craniad because the mural mass produces a filling defect.

Although hypomotility of the esophagus has been documented in one foal and neural defects of the esophagus were found in another, achalasia as defined in dogs and humans has not been reported.[49,50] Treatment of these horses was not successful, even with a modified Heller myotomy in one foal. I have treated congenital ectasia conservatively with good results. A 5-month-old foal was admitted because of recurrent choke that began at the time of weaning. Esophageal lavage produced 8 L of feed regurgitated through the nasogastric tube. On radiography and endoscopy, generalized ectasia was found from the midcervical to the midthoracic esophagus (Figure 31-45), with an annular ring noted at the point where the esophagus passed through the thoracic inlet (Figure 31-46). The foal was fed a mash diet (pellets mixed with warm water) for 6 months, with the feed trough elevated above the animal's withers. The mash diet was changed to complete pelleted feed for an additional 6 months before the horse was fed normal feed. Although fluid did not pool in the lumen of the esophagus after 6 months of the mash diet and the size of the dilated

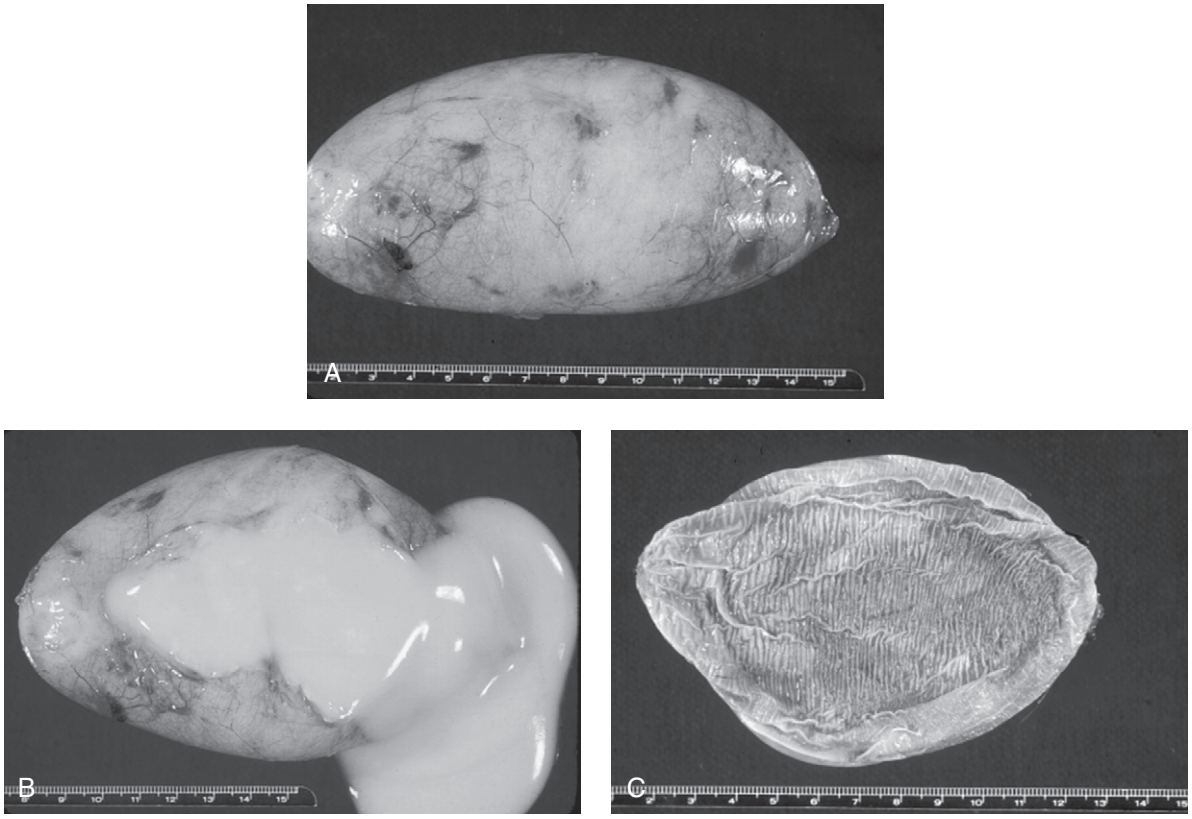

Figure 31-44. An excised intramural inclusion cyst **(A)** following puncture **(B).** The epithelial cell contents could be mistaken for an abscess ruptured during surgery. The cyst lining is normal esophageal mucosa **(C).**

esophagus did not change over the next 24 months during several radiographic examinations, the horse could eat normally and is being used for show and pleasure riding. Conservative management of this problem should be considered if the owners are willing to provide the extensive nursing care (no access to roughage, including bedding in the stall, and total confinement, with exercise in-hand only) and are willing to face the possibility of several episodes of choke requiring treatment.

Reduplication

It is not clear whether reduplication of the esophagus is a congenital or an acquired condition, but it is included here for the sake of completeness. This condition has been reported twice.[51,52] Clinical signs were similar to those of other forms of esophageal obstruction. Diagnosis may be difficult, but the problem does appear to be amenable to surgery. This condition closely resembles one of the complications of esophagostomy tube feeding, in which a dissecting tract develops parallel to the esophageal wall or within it, causing signs of obstruction.

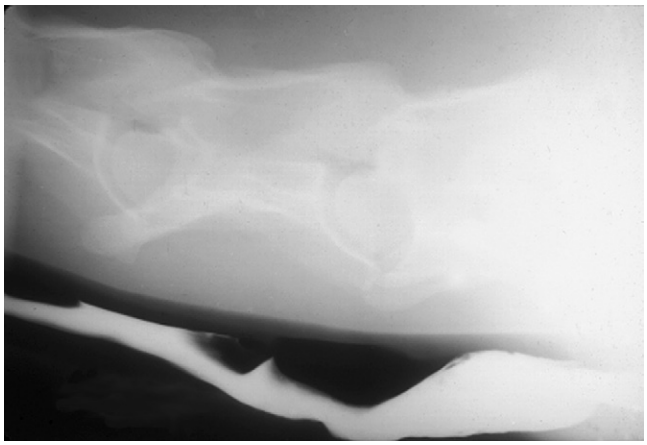

Figure 31-45. Positive-contrast esophagogram shows megaesophagus at the mid-cervical level. The foal was managed with a mash diet and recovered esophageal function over a 1-year period.

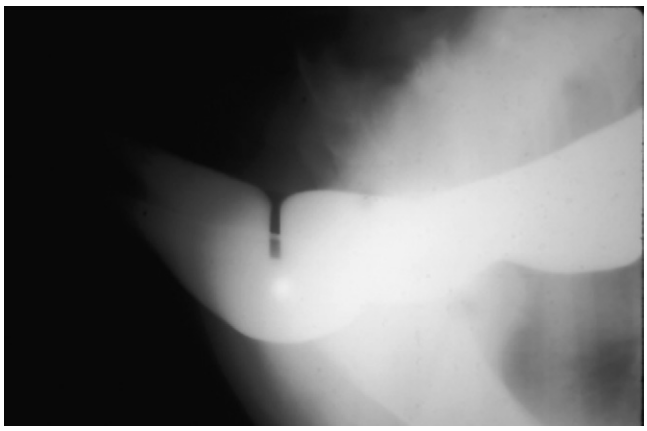

Figure 31-46. Barium pressure esophagogram shows megaesophagus in the foal seen in Figure 31-45. Note the narrowing of the esophageal lumen as it passes through the thoracic inlet.

Neoplasia

Neoplasms causing signs of esophageal obstruction are rare.[1] Squamous cell carcinoma is the most common neoplasm and has been reported in detail in four cases. One was an extension of gastric carcinoma, one was located in the tracheal bifurcation, and two were located in the distal cervical esophagus.[53-56] Biopsy and cytologic examination of brush samples obtained through the endoscope may provide an early diagnosis before stenosis of the esophagus develops, but advanced cases may be detected on radiography. The value of resection is questionable.

COMPLICATIONS OF ESOPHAGEAL SURGERY

The surgeon has the following three clinical objectives: (1) to obtain leakproof healing of a primary anastomosis or incision, (2) to dilate a restricted aperture, and (3) to return the enlarged or disrupted esophagus to near-normal size and function. Despite meticulous technique, complications can occur. If managed properly, however, they can be resolved with a favorable outcome for the patient.

Dehiscence and Stricture

The keys to handling breakdown of a sutured esophageal incision are early recognition and treatment. For this reason, a suction drain placed next to the esophagus at the time of surgery

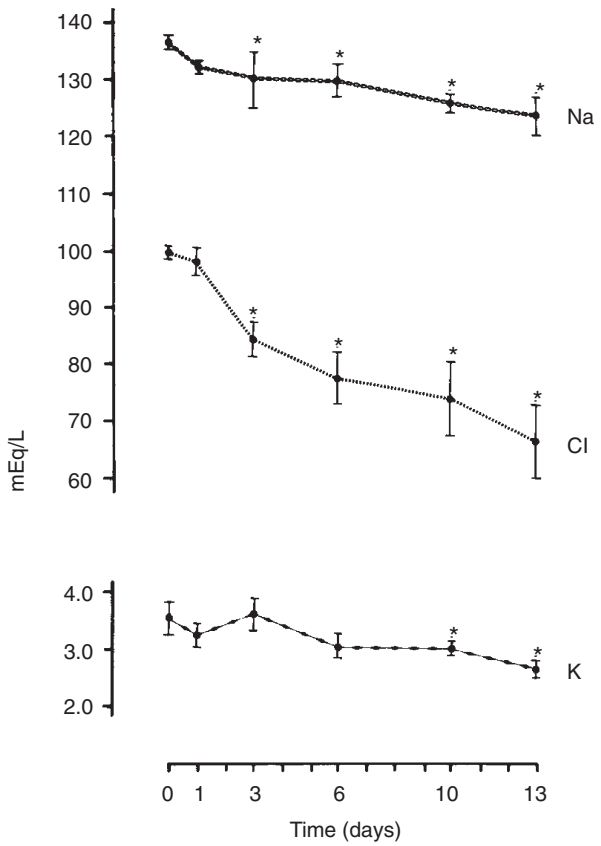

Figure 31-47. Influence of salivary depletion on serum electrolyte levels (Na, K, Cl) before (day 0) and after the esophageal fistulation. *Bars* indicate ± 1 SE ($N = 6$). *Stars* indicate days on which serum electrolyte levels were significantly different from baseline values (day 0).

serves a second function in addition to drainage of the serum and blood from the surgical site. It provides a method to detect salivary leakage. Not all incisions that leak saliva dehisce, especially if the saliva is removed from periesophageal tissues. However, if extreme amounts of saliva are leaking through the incision or complete dehiscence occurs, the original approach incision should be completely opened and lavaged daily to remove any ingesta or saliva. Dissection planes ventrally along the trachea should be drained to the outside to prevent eventual mediastinitis or pleuritis. Dissecting infections can occur after esophagostomy as well, and inadvertent placement of a dislodged feeding tube into a dissection plane or the thorax would clearly have an unfavorable outcome.

The patient may be permitted to drink, which in many cases lavages the wound. Nutritional requirements may be met by oral feeding or placement of a feeding tube into the esophagus at the point of dehiscence or distad through a separate esophagostomy. The outcome of this complication from this point depends on management of water and electrolyte balance. The most common complication after treatment of an annular lesion is stricture (see Figure 31-30). This complication is the bane of the esophageal surgeon, and its successful treatment is limited by the owner's financial commitment.

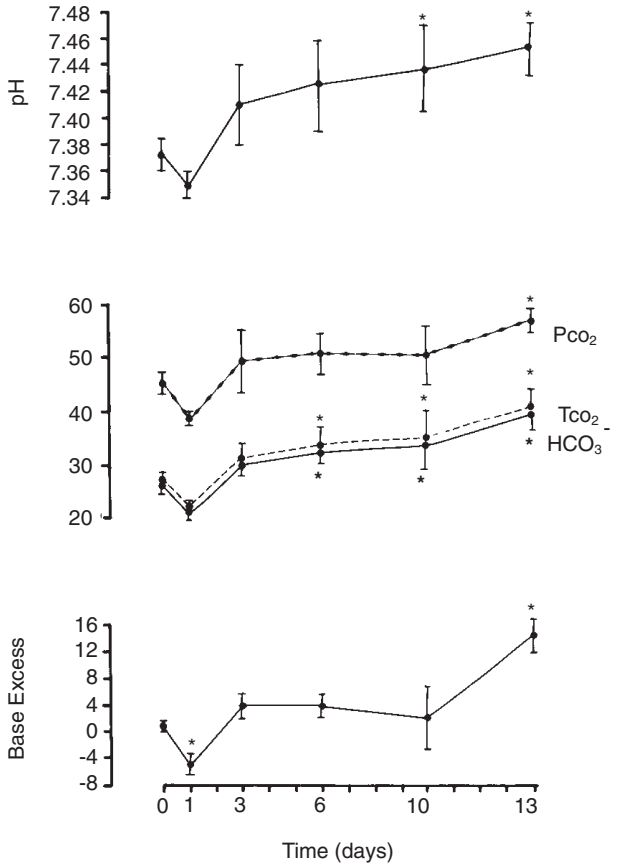

Figure 31-48. Effect of salivary depletion and serum electrolyte alterations on acid-base values (pH, P_{CO_2} [mm Hg], T_{CO_2} = total CO_2 [mEq/L], HCO_3^- [mEq/L], and base excess [mEq/L]) before (day 0) and after esophageal fistulation. *Bars* indicate ± 1 SE ($N = 6$). *Stars* indicate days on which each value was significantly different from baseline values (day 0).

Acid-Base and Electrolyte Alterations

In the face of adequate nutrition, daily losses of large amounts of saliva result in hyponatremia, hypochloremia (Figure 31-47), and transient metabolic acidosis followed by progressive metabolic alkalosis (Figure 31-48).[57] The alkalosis probably results from renal compensation for electrolyte imbalances. Oral administration of sodium chloride daily reverses the electrolyte imbalance (potassium requirements are adequately met in feed); alkalosis is corrected through renal mechanisms.

Laryngeal Hemiplegia

Manipulative procedures or disease of the esophagus in the cervical area can easily result in laryngeal hemiplegia because of the proximity of the recurrent laryngeal and vagus nerves. The surgeon should be aware that apparently minor manipulations of these nerves can have deleterious effects on their function.

Carotid Artery Rupture

Chronic feeding tubes placed in the distal third of the cervical esophagus can lead to jugular vein or carotid artery ulceration. Bleeding episodes should be an indication for exploratory and vessel ligation to prevent exsanguination.

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CHAPTER
32 Stomach and Spleen

David A. Wilson and Anthony T. Blikslager

STOMACH

Anatomy

The equine stomach is small relative to the body size of the horse, having a capacity of approximately 5 to 15 L.[1] Even when the stomach is filled to capacity, it does not reach the ventral body wall. It is located predominantly on the left side of the abdomen under the cover of the ribs, with only the pyloric region of the stomach to the right side of the midline. Its most caudal component is the fundus, which lies adjacent to the 14th and 15th rib spaces (Figure 32-1).[2] The stomach can be divided into several regions, including the cardia at the opening of the esophagus, the fundus (which forms a blind sac), the body, and

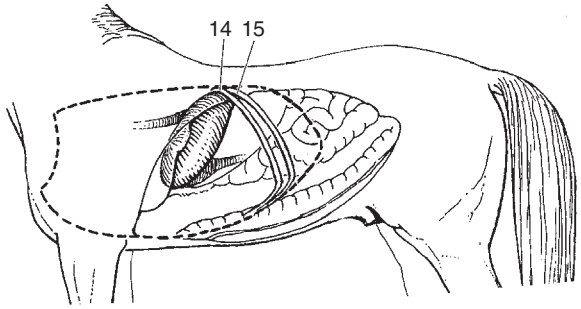

Figure 32-1. Diagram of the anatomic location of the equine stomach in the abdomen.

the pyloric region. The stomach is sharply curved at its lesser curvature so that the cardia and pyloric regions lie adjacent to each other.[2] The cardia is attached to the diaphragm by the gastrophrenic ligament. This ligament is a continuation of the phrenicosplenic ligament and the gastrosplenic ligament on the left side of the abdomen. The greater omentum attaches along the greater curvature of the stomach, and it blends into the gastrophrenic ligament.[1,2] The greater omentum forms a blind potential space called the *omental bursa*. The entrance to this bursa is the epiploic foramen, which is located between the caudate process of the liver and the right lobe of the pancreas. The epiploic foramen is also bordered dorsally and ventrally by the caudal vena cava and portal vein, respectively. The lesser omentum, which connects the stomach and duodenum to the liver, consists of the hepatogastric and hepatoduodenal ligaments.[2] The stomach receives its blood supply from the celiac artery. Venous drainage from the stomach is via gastric veins to the portal vein.[1]

Physiology

During digestion, the stomach is capable of dramatically lowering the pH of its contents and of secreting pepsinogen to begin breaking down protein. However, both HCl and pepsinogen (the latter being converted to pepsin) are inherently damaging to mucosa, which possesses a number of mechanisms to prevent injury, depending on the region of the stomach. There are four regions of the stomach based on the type of mucosal lining (in an orad-to-aborad order): nonglandular stratified squamous epithelium, cardiac epithelium, proper gastric mucosa (glandular mucosa), and pyloric mucosa.[3] The equine stomach has a very extensive region of stratified squamous mucosa that occupies approximately half of the stomach mucosal surface area, and this changes abruptly to cardiac glandular mucosa at a line of demarcation called the *margo plicatus*.[4] The function of the stratified squamous mucosa in the horse is unknown, since this region of the stomach does not have any secretory ability, although the squamous mucosa may protect underlying tissues from abrasion by roughage.[3] However, the squamous mucosa does not frequently come into contact with stomach contents, as the gastric fill line is typically the margo plicatus. The cardiac epithelium forms a belt of tissue adjacent to the margo plicatus. Relatively little is known about this mucosal region, although it has the ability to secrete bicarbonate in other species and has been shown to contain cells capable of secreting somatostatin in equine mucosa, which serves as a negative feedback mechanism for gastric acid secretion. The proper gastric mucosa contains secretory glands containing HCl-secreting parietal cells and pepsinogen-secreting zymogen cells. In addition, this segment of mucosa contains enterochromaffin-like (ECL) cells that secrete histamine in response to various stimuli, which in turn amplifies HCl secretion by the parietal cells. The pyloric mucosa contains both G-cells, which secrete gastrin, and D-cells, which secrete somatostatin. These hormones enhance or reduce gastric acid secretion, respectively.[4]

Gastric Barrier Function

Mechanisms by which the stratified squamous mucosa resists injury are critical in the horse, where ulceration is prevalent.[5-7] When mounted in Ussing chambers for the purpose of measuring epithelial transport function, equine stratified squamous epithelium has baseline transepithelial resistance measurements of approximately 2000 to 3000 Ω/cm^2, which is an order of magnitude higher than that of the adjacent cardiac mucosa.[5] Since transepithelial resistance is essentially a measure of ion permeability, the stratified squamous mucosa is exceptionally impermeable. This is the only mechanism of defense against injury in this type of mucosa. The stratified squamous epithelium consists of four layers: the outer stratum corneum, the stratum transitionale, the stratum spinosum, and the basal stratum germinativum. However, not all of these layers contribute equally to barrier function. Interepithelial tight junctions in the stratum corneum and muco-substances secreted by the stratum spinosum are mainly responsible for barrier function.[5,8] The stratified squamous mucosa in other species is relatively impermeable to HCl, suggesting that although most of the literature on equine ulceration pertains to the effects of HCl and inhibitors of HCl secretion, other factors may be critical to the development of gastric ulcer disease.[9-13] Nonetheless, exposure of equine stratified mucosa to acid (pH 1.7) results in significant reductions in measurements of transepithelial electrical resistance, regardless of whether other injurious compounds are added, indicating that acid alone can damage the mucosa.[14] It is not clear whether less-acidic solutions can also cause damage in the presence of other injurious factors, such as bile or pepsin.

The site of HCl secretion (proper gastric mucosa) is protected from back diffusion of H^+ ions by a relatively high transepithelial electrical resistance (compared to cardiac mucosa), but there are also a number of other critical mechanisms to prevent acid injury. The gastric mucosa secretes both mucus and bicarbonate, which together form an HCO_3^-–containing gel that titrates acid before it reaches the lumen.[15,16] However, the mucus layer does not form an absolute barrier to back diffusion of acid. Thus, for acid that does diffuse back into the gastric mucosa, epithelial Na^+/H^+ exchangers are capable of expelling H^+ once the cell reaches a critical pH.[16]

Gastric Mucosal Pathophysiology

Mechanisms of Gastric Ulceration

Ulceration in horses most commonly involves the stratified squamous epithelium and is somewhat analogous to gastroesophageal reflux disease in people.[5] This may occur because the glandular portion of the stomach has a number of protective mechanisms, including mucus and bicarbonate secretion, whereas the squamous mucosa has none of these protective mechanisms and is periodically exposed to acid contents.[4] Although stratified squamous epithelium is relatively impermeable to HCl, a number of factors can dramatically enhance the damaging effects of HCl in this epithelium. In particular, bile salts and short-chain fatty acids are capable of breaking down the squamous epithelial barrier at an acid pH, thereby exposing deep layers to HCl, with subsequent development of ulceration. Relatively high concentrations of short-chain fatty acids normally exist in the equine stomach as a result of microbial fermentation.[5] These weak acids penetrate squamous mucosa, and they appear to damage Na^+ transport activity, principally in the stratum germinativum. Bile salts may also be present in the proximal equine stomach as a result of reflux from the duodenum. Although such reflux has a relatively high pH, it appears that bile salts adhere to stratified squamous epithelium, becoming lipid soluble and triggering damage once the pH falls below

4.[17] Diet and management (e.g., periods of fasting) also play crucial roles in the development of conditions conducive to gastric ulceration. Typically, there is a pH gradation in horses from proximal to distal compartments of the stomach, with the lowest pH values in the distal stomach.[18] However, during periods of fasting, this stratification is disrupted, so that low pH values may be recorded in the proximal stomach.[19] Fasting conditions also increase the concentration of duodenal contents within the proximal stomach, particularly bile.[17] The type of diet also influences the stratification of stomach contents.

Mechanisms inducing injury to proper gastric mucosa may be entirely different from those inducing injury to stratified squamous mucosa. In people, the majority of gastric mucosal ulcers is induced by infection with *Helicobacter pylori*, which has the effect of raising gastric pH because of disruption of gastric glands. Such an infection also induces an inflammatory response that causes damage.[20] In particular, *H. pylori* containing the *cagA* gene is most pathogenic. However, there is very little evidence that this organism is involved in gastric ulcers in domestic animals. In the absence of a known role for infectious agents in equine gastric ulcer disease syndrome, ulceration most likely develops from an imbalance between protective mechanisms and injurious factors, which include gastric acid, bile, and nonsteroidal anti-inflammatory drugs (NSAIDs). However, some factors that are important to inducting squamous epithelial ulceration may not be important in development of proper gastric mucosal ulceration. For example, feed deprivation and intensive training reproducibly induce squamous epithelial ulceration in horses, but they have little effect on proper gastric mucosa in horses.[21] Gastric acid very likely plays a key role, whereas other factors such as NSAIDs serve to reduce gastric defense mechanisms. In particular, inhibition of prostaglandin production reduces mucus and bicarbonate secretion, while also reducing gastric mucosal blood flow, and would be expected to induce ulceration of proper gastric mucosa.[22] In addition, some of the NSAIDs also have a topical irritant effect, but this appears to be of minor significance, since the route of administration (oral or parenteral) seems to have little influence on development of ulceration.[23] Other risk factors for the development of gastric ulcer syndrome in the horse include stress, transportation, high-protein diet, stall confinement, intermittent feeding, racing, illness, and management changes.[24,25]

Gastric Reparative Mechanisms

Mechanisms of gastric repair are highly dependent on the extent of injury. For instance, superficial erosions can be rapidly covered by migration of epithelium adjacent to the wound—a process termed *epithelial restitution*. However, ulceration (full-thickness disruption of mucosa and penetration of the muscularis mucosae) requires repair of submucosal vasculature and matrix. This is initiated by formation of granulation tissue, which supplies connective tissue elements and microvasculature necessary for mucosal reconstruction. Connective tissue elements include proliferating fibroblasts that accompany newly produced capillaries that form from proliferating endothelium. Studies indicate that nitric oxide is critical to both of these processes, which probably explains the reparative properties of NO in the stomach.[26-28] Once an adequate granulation bed has been formed, newly proliferated epithelium at the edge of the wound begins to migrate across the wound. In addition, gastric glands at the base of the ulcer begin to bud and migrate across the granulation bed in a tubular fashion.[29] Epidermal growth factor is expressed by repairing epithelium, and it appears to facilitate these processes.[30] In addition, these events are facilitated by a mucoid cap, which retains reparative factors and serum adjacent to the wound bed. Importantly, this cap maintains a neutral pH to facilitate healing.[22] Once the ulcer crater has been filled with granulation tissue and the wound has been reepithelialized, the subepithelial tissue remodels by altering the type and amount of collagen.[31]

Diagnostic Techniques

Endoscopy

Before gastric endoscopy, foals up to 20 days of age should be held off feed for 3 hours, whereas older foals require up to 10 hours to sufficiently empty the stomach. Adult horses should be held off feed for 24 hours to allow complete visualization of the stomach.[1] A 3-m endoscope is required to adequately visualize the stomach, including the pylorus, in adult horses, whereas a 1-m endoscope may be sufficient for foals.[1] Although endoscopy is the principal diagnostic aid for detecting gastric ulceration, a study comparing endoscopic to histologic scoring of the severity of ulceration indicated that endoscopy tends to underestimate the number and severity of gastric ulcers of the nonglandular equine stomach. Furthermore, endoscopy may not allow detection of glandular gastric ulcers. Therefore clinicians should realize that ulceration is frequently more severe and extensive than that observed via the endoscope.[32]

Ultrasonography

Ultrasonography can be used to image the wall of the stomach, and it may be particularly useful in foals with suspected gastric outflow obstruction. The stomach is best imaged from the left side of the abdomen between rib spaces 8 and 14. If gastric outflow obstruction is present, a distended stomach with a gas–fluid interface may be detected. The duodenum can also be imaged at this site, and it may show evidence of thickening and reduced motility. The duodenum can additionally be imaged from the right paralumbar fossa, cranial to the right kidney, where there may be additional evidence of thickening or obstruction.[33,34]

Radiography

Contrast radiography can be performed to allow visualization of the stomach. In a series of foals with gastroduodenal obstruction, barium sulfate solution was administered.[35] In addition, functional gastric emptying can be determined by the administration of barium sulfate after a 12- to 18-hour fasting period. In normal horses, barium appears in the small intestine by 10 minutes, and none remains within the stomach by 35 minutes. With pyloric outflow obstruction, barium may remain in the stomach for up to 8 hours.[1] Barium sulfate solutions should be diluted 1:1 with water, and a volume of approximately 1 L should be administered into the thoracic portion of the esophagus by nasogastric tube.[36] Double-contrast radiography with insufflation of air, followed by administration of a barium sulfate suspension, can enhance visualization of the stomach. This technique has been useful in the diagnosis of gastric outflow obstruction and gastric neoplasia.[1,37]

Measurement of Gastric Emptying

Assessment of gastric emptying can be performed by nuclear scintigraphy, acetaminophen absorption, or breath testing after the administration of ^{13}C-octanoic acid.[38-40] For scintigraphic determination of gastric emptying, one study involved administration of 370 megabecquerels (MBq) of technetium-99m (^{99m}Tc) sulfur colloid incorporated into egg albumin (to determine solid phase gastric emptying) and 37 MBq of diethylenetriamine pentaacetic acid (DTPA) labeled with indium-111 in 120 mL of water (to determine liquid phase gastric emptying). These labeled products were administered via a nasogastric tube after a 12-hour fasting period. Scintigraphic images obtained from the left and right flanks were subsequently used to assess gastric emptying. Liquid phase and solid phase 50% gastric emptying times are 30 and 90 minutes, respectively, in normal horses.[41] In experimental trials assessing efficacy of breath testing, ponies were fasted for 14 hours, after which they ingested a test meal with ^{13}C-octanoic acid. Breath samples were analyzed by mass spectrometry for the appearance of the isotope.[40] This method correlated closely with gastric emptying documented with nuclear scintigraphy after administration of sulfur colloid labeled with technetium-99m.[39] Similarly, acetaminophen absorption has been shown to closely correlate with scintigraphic measurements of gastric emptying. For example, in one research trial, acetaminophen (20 mg/kg) and ^{99m}Tc-pentetate (10 mCi) were administered in 200 mL water, and their clearance from the stomach was shown to be similar, based on serum acetaminophen and scintigraphic analyses, respectively.[42]

Disorders

Gastric Ulcer Clinical Syndromes

The clinical syndromes of gastric ulceration are age dependent; the age categories being neonates, weanling foals, and horses older than 1 year.[43] In neonates, ulceration of the glandular mucosa is the most clinically important. Clinical signs include poor appetite, colic, and diarrhea, but foals may have ulcers and not demonstrate overt clinical signs of gastric pain or failure to thrive.[44] The pH of neonatal gastric contents is not typically as low as that of older foals or adults, suggesting that other factors may be important in the pathogenesis of ulceration.[13]

In suckling and weanling foals, ulceration frequently occurs in the squamous mucosa adjacent to the margo plicatus. Severe ulceration of the squamous portion of the stomach in foals of this age group is frequently associated with diarrhea, but foals may also appear unthrifty, with a rough hair coat and a pot-bellied appearance.[43] Diffuse ulceration is also typically associated with teeth grinding and colic. This age group of foals may also be affected with gastroduodenal ulceration, which may result in gastric outflow obstruction (see later).

In yearlings and adult horses, lesions occur predominantly in the squamous portion of the stomach adjacent to the margo plicatus. These horses may have very subtle signs of disease, such as poor performance, reduced appetite, and failure to thrive, but they do not typically suffer from diarrhea. The diagnosis of gastric ulcers is confirmed with gastroscopy.[7] When evaluating squamous lesions in foals, it is important to differentiate ulceration from desquamation.[8] The latter involves shedding of sheets of epithelium without ulceration, and it is unlikely to be clinically important.[45] Treatment is aimed at elevating the pH of the gastric contents, which may be achieved with a number of histamine receptor (H_2) antagonists, such as ranitidine (6.6 mg/kg, PO every 8 hours, or 1.5 to 2 mg/kg IV every 6 to 8 hours) or proton pump inhibitors such as omeprazole (2 to 4 mg/kg PO every 24 hours).[24,25,44,46]

Gastric Impaction

Impaction of the stomach typically consists of excessive dry, fibrous ingesta, but it may also consist of ingested materials that form a mass, such as persimmon seeds or mesquite beans.[47-50] Other feeds that tend to swell after ingestion, such as wheat, barley, and sugar beet pulp, may also cause impaction. Furthermore, dental disease may increase the likelihood of gastric impaction because of improper chewing of feed. Clinical signs include colic that ranges from acute and severe to chronic and mild. For example, in one report on four horses with gastric impaction, colic was moderate or severe and of 8 to 12 hours' duration,[51] whereas in another report on a pony with gastric impaction, colic was chronic (7 days' duration) and associated with prolonged recumbency, anorexia, and lethargy.[49] Additional signs may include dysphagia, dropping of feed, and bruxism.[49,52]

The diagnosis is frequently made at the time of surgery, although endoscopy reveals gastric impaction and may provide information on the specific nature of the impaction. Medical treatment includes nasogastric intubation and frequent attempts at softening the ingesta with water, followed by refluxing the fluid contents, or by using back-and-forth agitating movements of water with a 16-ounce dose syringe attached to the nasogastric tube. Nasogastric lavage with a carbonated cola soft drink was successfully used in a pony with persimmon seed impaction of the stomach.[53]

At surgery, the impaction can be massaged and infused, most commonly via insertion of a needle adjacent to the greater curvature, followed by infusion of a balanced polyionic fluid such as saline. There is also a report including the details of a pony and a horse in which the impacted stomach was packed off from the abdomen with towels, and an incision was made parallel and caudal to the attachment of the omentum on the greater curvature of the stomach. The stomach contents were evacuated, followed by a double-layer inverting closure. In the horse in this report, the ventral midline incision was extended craniad to the xiphoid process.[52] However, this aggressive approach is rarely necessary, as the impaction can usually be resolved with more conservative management.

Chronic Gastric Impaction and Dilation

Typically, gastric impaction develops relatively quickly and the diagnosis is often made at surgery. In contrast, chronic impaction and dilation of the stomach develops slowly over weeks or months with minimal clinical signs of abdominal pain. Clinical signs are often mild, with weight loss, reduced performance, bruxism, and salivation reported. Gastric endoscopy may reveal fibrous ingesta, which does not change after 24-hour starvation. A large mass may be palpable *per rectum*. Abdominal radiographs or ultrasonography may demonstrate a large distended and impacted stomach. Gastrotomy to remove ingesta and partial gastrectomy to remove flaccid stomach wall have been unsucessful. Postmortem findings include marked transmural hypertrophy of the nonglandular region of the stomach near the cardia and large chronic ulcers along the margo plicatus.[54-56]

Gastric Rupture

Rupture of the stomach appears to have two general causes: primary, as a result of excessive accumulation of ingesta, or no identifiable cause; and secondary, in association with another causative condition, such as obstruction of the small intestine.[57] The site of the rupture is most commonly the greater curvature of the stomach, although other sites of rupture have been identified, including the lesser curvature of the stomach.[58] The condition is almost universally fatal, because release of stomach contents into the abdomen through the gastric tear causes septic shock that cannot be adequately reversed with abdominal lavage and repair of the defect. However, there are some case reports of partial gastric tearing that have been repaired. In one report, a subserosal hematoma associated with a focal serosal perforation was repaired by use of a two-layer inverting closure, and the horse survived.[59] In another report, a seromuscular tear with intact mucosa bulging from the tear was noted, and it was oversewn with an inverting suture pattern. However, in neither case was there any evidence of abdominal contamination with gastric contents.[60]

In a report on 54 horses with colic taken to surgery, 11% of deaths were attributable to gastric rupture. Interestingly, at least 6 of the 54 horses had indwelling stomach tubes, indicating that nasogastric intubation does not negate the possibility of gastric rupture.[57] In a separate report on gastric rupture, it was determined that 5.4% of all colic accessions had gastric rupture. One of the striking clinical findings was that the abdominal fluid cell counts were frequently inaccurate, most likely because the acid stomach contents lysed cells infiltrating the abdomen. However, a number of abdominal fluid samples were not analyzed because they had the appearance of ingesta. This report also determined a number of risk factors for gastric rupture, including a diet exclusively of grass hay or of grass and alfalfa hay and drinking water from a bucket, stream, or pond. Alternatively, horses fed grain had a reduced risk.[58] The cases in this study were principally primary gastric ruptures (60%). Predisposing factors may include the opportunity to consume excessive water or roughage, leading to expansion and subsequent rupture of the stomach.

Gastric Neoplasia

Neoplasia of the stomach is rare. The most common form of gastric neoplasia is squamous cell carcinoma, which typically forms in the cardia of the stomach. In one report, a tumor encircled the esophagus at the cardia of the stomach, where it caused recurrent esophageal obstruction.[61] Clinical signs may include anorexia, weight loss, abdominal distention, abnormal chewing behavior, lethargy, coughing, hypersalivation, colic, dysphagia, fever, and ventral abdominal edema.[62-66] The diagnosis can be based antemortem on results of gastric endoscopy and biopsy or on thoracoscopy and biopsy.[67,68] In addition, approximately 50% of horses have had neoplastic cells evident in abdominal fluid or in thoracic fluid.[63,69,70] Other nonspecific laboratory findings have included anemia, neutrophilia, and hypoalbuminemia.[63] Additionally, one horse had a clinical diagnosis of pseudohyperparathyroidism in association with gastric carcinoma, based partially on evidence of hypercalcemia and hypophosphatemia.[70] In a case series on gastric squamous cell carcinoma, horses have had multiple metastases, including masses throughout the abdomen. Therefore the prognosis is grave, and the condition has been universally fatal. The reported median time from onset of clinical signs to death is 4 weeks.[63,64,66,69]

Other neoplasms that have been identified in the stomach include leiomyosarcoma (leiomyoma), mesothelioma, and adenocarcinoma. A leiomyosarcoma was diagnosed in a 12-year-old Thoroughbred gelding that was evaluated because of anorexia, weight loss, and intermittent fever. At surgery, an inoperable mass was discovered in the cranial abdomen, which was found to be a leiomyosarcoma after histopathologic analysis. The mass was associated with the distal portion of the esophagus and the cranial two thirds of the stomach.[71] Adenocarcinomas have been documented in the glandular portion of the equine stomach.[1,72]

Gastric Outflow Obstruction

Gastric outflow obstruction may be the result of pyloric stenosis, which can be caused by congenital muscular hypertrophy, or by development of a mass at the pylorus that reduces gastric outflow.[73,74] A mass may develop at the pylorus associated with gastroduodenal ulceration or neoplasia.[75-80] Clinical signs include weight loss, reduced appetite, abdominal pain, teeth grinding, ptyalism, frequent recumbency, and poor performance. Foals with gastric outflow obstruction are typically 2 to 6 months of age, with signs of gastric ulceration and an unthrifty appearance. However, foals may also have a history of enteritis and an absence of clinical signs typical of foals with gastric ulcers.

Gastric outflow obstruction can be diagnosed using endoscopy, radiography, ultrasonography, and gastric emptying tests, as described previously. Medical treatment for foals includes decompression of the stomach, antiulcer medications, broad-spectrum antibiotics, prokinetics, and intravenous fluids. However, surgery is indicated if medical treatment does not reverse clinical signs within a short period.[33] The principle of surgery for treatment of gastric outflow obstruction is bypass of the pylorus, typically by performing a gastrojejunostomy. Pyloric stenosis has been relieved in a 2-month-old Thoroughbred by a modification of the Heineke-Mikulicz technique, in which a full-thickness longitudinal incision through the pylorus was closed transversely.[74] The pylorus was mobilized by severing the hepatoduodenal ligament.

Alternatively, a series of bypass techniques can be performed, depending on the location of the obstruction.[35,81,82] In one series evaluating surgical results in 13 foals with gastroduodenal obstruction, bypass procedures included gastroduodenostomy, duodenojejunostomy, or gastrojejunostomy (Figures 32-2 to 32-4).[35] A jejunojejunostomy was also performed with the latter two surgical procedures to allow outflow of proximal small intestinal contents.[35,82] Aligning the jejunum from left to right (oral to aboral portions of the jejunum) along a relatively avascular region of the caudal ventral aspect of the stomach for a gastrojejunostomy has been reported to substantially improve long-term outcome in 40 foals (Figure 32-5).[82] These anastomoses can be hand-sewn or performed with automated stapling equipment. However, hand-sewn anastomoses may be simpler to perform because of the limited space within a foal's abdomen, which reduces maneuverability of larger stapling instruments.[36] Although a three-layer hand-sewn technique has been described (seromuscular, muscular, and mucosal layers), one case report suggested that a two-layer hand-sewn anastomosis (seromuscular and mucosal layers) was sufficient and more rapidly performed in a foal in which a gastroduodenostomy was created.[36]

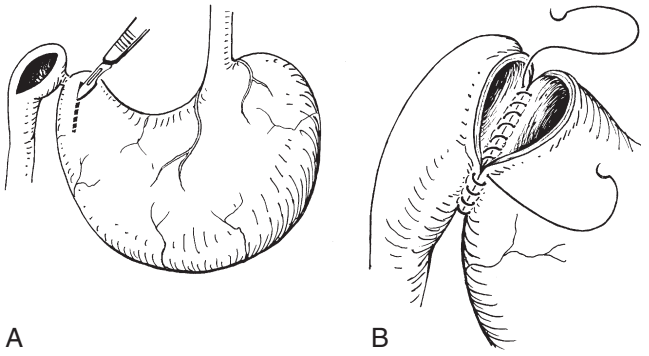

Figure 32-2. A, Locations for incisions for a gastroduodenostomy to bypass a pyloric stenosis. **B,** A three-tiered hand-sutured anastomosis to complete the gastroduodenostomy. (Redrawn from Orsini JA, Donawick WJ: Surgical treatment of gastroduodenal obstruction in foals. Vet Surg 15:205, 1986.)

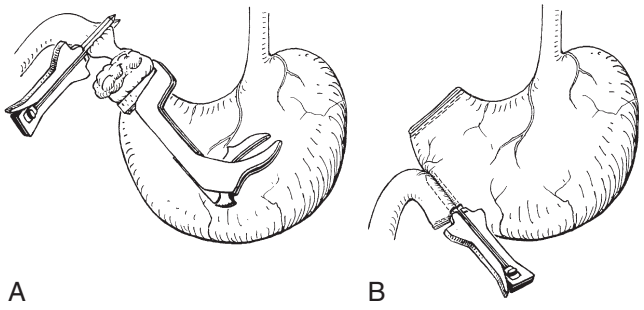

Figure 32-3. Partial gastrectomy **(A)** and gastroduodenostomy **(B)** to relieve pyloric obstruction. Stapling instruments have been applied proximal and distal to the obstruction, allowing resection of the distal stomach. Care is taken to avoid the hepatic duct in the proximal duodenum by stapling proximal to it. The blind-ending duodenum is subsequently anastomosed to the distal stomach using an automated stapling device. (Redrawn from Orsini JA, Donawick WJ: Surgical treatment of gastroduodenal obstruction in foals. Vet Surg 15:205, 1986.)

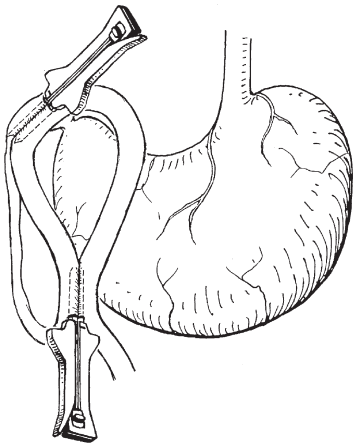

Figure 32-4. Duodenojejunostomy. The duodenojejunostomy and jejunojejunostomy are performed using an automated stapling device or a three-tiered suturing technique. The duodenojejunostomy is performed with the duodenal component proximal to the hepatic duct, and the jejunojejunostomy is performed with a segment of jejunum distal to the duodenocolic ligament. (Redrawn from Orsini JA, Donawick WJ: Surgical treatment of gastroduodenal obstruction in foals. Vet Surg 15:205, 1986.)

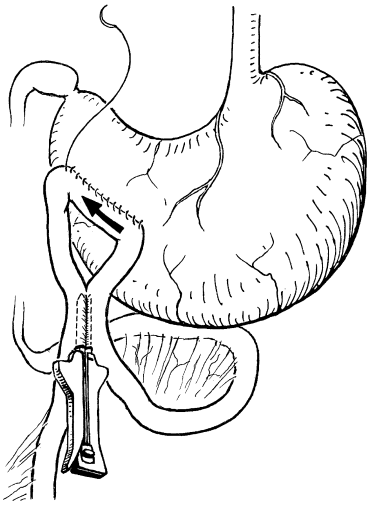

Figure 32-5. Completed left to right gastrojejunostomy with jejunojejunostomy. *Black arrow* indicates oral to aboral direction of the jejunum. (Redrawn from Zedler ST, Embertson RM, Bernard WV, et al: Surgical treatment of gastric outflow obstruction in 40 foals. Vet Surg 38(5):623, 2009.)

The prognosis for foals with gastric outflow obstruction is fair to guarded.[33,82] In one report on results of surgical intervention in foals with gastroduodenal obstruction, 6 of 13 foals (46%) survived.[35] One report indicated that foals with a localized pyloric obstruction should have a more favorable prognosis than foals with pyloric and duodenal obstructions, because the surgery for the former (gastroduodenostomy) is simpler than the surgery for the latter (gastrojejunostomy and jejunojejunostomy).[36] In a more recent report, 25 of 36 foals (69%) for which follow-up information was available were alive more than 2 years after surgery.[82] Obstruction of the duodenum, adhesions to the duodenum, and postoperative ileus were significantly associated with decreased long-term survival.

SPLEEN

Anatomy

The spleen is a falciform or sickle-shaped organ located in the left mid-dorsal part of the peritoneal cavity (Figure 32-6). The exact position of the spleen varies, depending on, for example, the amount of the stomach filling, intestinal distention or displacement, and splenic disease. The cranial border of the spleen is concave and the caudal border is convex. The parietal or lateral surface is slightly convex and is in intimate contact with the diaphragm.[83] The visceral or medial surface is generally concave and contains a longitudinal ridge, the hilus, in which the vessels and nerves are located. The area cranial to the hilus is in contact with the left side of the greater curvature of the stomach. The area caudal to the ridge is more extensive and is

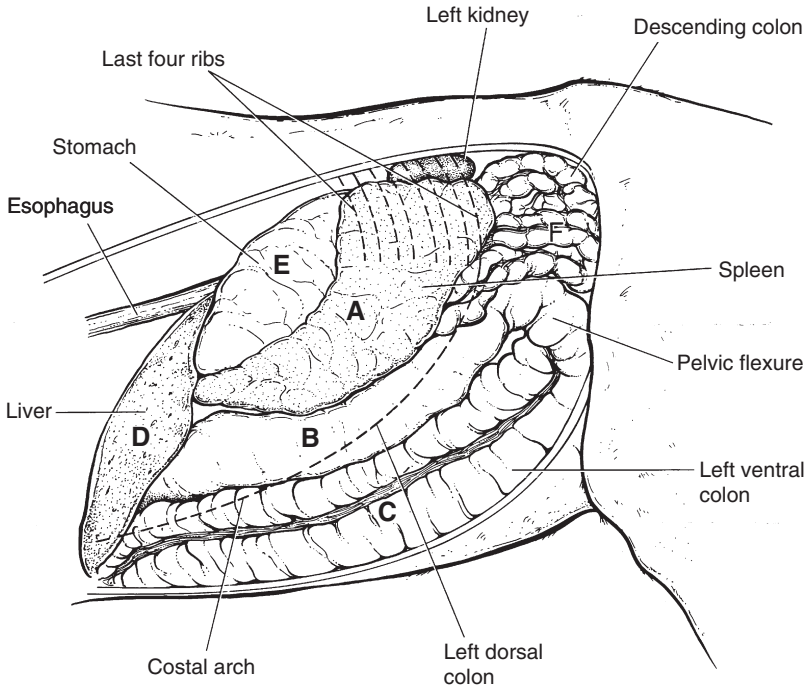

Figure 32-6. Relative topography of the abdominal viscera as viewed from the left side. The lateral and ventral walls are removed. The proximal one third of the last four ribs *(short dotted lines)* shows the relative position of the spleen protected by the rib cage (*long dotted line* defines the ventral and caudal border of the rib cage). *A,* Spleen; *B,* left dorsal colon; *C,* left ventral colon; *D,* liver; *E,* stomach; *F,* small bowel.

variably in contact with the descending colon, the left parts of the ascending colon, and the small intestine. The dorsal extremity or base of the spleen is located between the left crus of the diaphragm and sublumbar muscles dorsally and on its medial surface by the pancreas and left kidney. The base generally corresponds to the last three or four ribs, exceeding the 18th rib caudad in the paralumbar fossa by 2 to 3 cm. Only the caudodorsal angle (between the base and the caudal border) can be palpated *per rectum*. The ventral extremity or apex is small and usually found opposite the 9th, 10th, or 11th rib, proximal to the costal arch. The spleen is suspended within the peritoneal cavity by means of the phrenicosplenic and the renosplenic (or nephrosplenic) ligaments, and attached to the stomach by the gastrosplenic ligament. The latter continues with the superficial wall of the greater omentum.[84]

The size and weight of the spleen vary greatly, both among and within animals, depending on the amount of blood it contains. Its weight varies from about 0.5 to 3.5 kg. The spleen is approximately 50 cm (20 inches) long and about 20 to 25 cm (8 to 10 inches) wide. It is usually bluish red to purple.[83,85]

The hilus protects the vessels and nerves of the spleen. The splenic artery, a branch of the celiac artery, runs within the groove of the hilus and branches to supply the spleen and short gastric arteries that nourish the greater curvature of the stomach (Figure 32-7). The splenic vein is an affluent of the portal vein and lies caudal to the artery in the hilus. The splenic lymph nodes are scattered along the splenic artery and drain lymph to the celiac lymphocenter. The splenic plexus, branching from the celiac plexus, supplies the spleen with both sympathetic fibers from the major splanchnic nerve and parasympathetic fibers from the vagus nerve.

The splenic tissue is intimately covered by a fibrous capsule, which sends numerous trabeculae into the substance of the spleen. The capsule consists of collagen and elastin fibers and contains many smooth muscle cells.[84,86] The trabeculae form a spongy framework, which is the support of the splenic pulp. The splenic pulp is mostly red or dark red and is known as the *red pulp*.[86] The red pulp consists of arterial capillaries, small venules, and a reticulum filled with macrophages and blood. Throughout the red pulp are scattered gray foci just visible to the naked eye, which are known as the *white pulp*. The white pulp represents the lymphatic tissue of the spleen organized as lymphatic nodules.[85,87] Lymphocytes and macrophages supported by a scaffozlding of branched connective cells called *reticular cells* make up the lymphatic tissue. The reticulum is made up of a meshwork of supporting cells and extracellular fibers. The white pulp is distributed along the course of the arterial vessels, and it is almost as abundant as the red pulp.[85,88]

Physiology

The spleen has several important functions, including storage of erythrocytes and platelets, removal of aged or damaged erythrocytes and platelets, hematopoiesis during fetal development, iron recycling, and immunologic functions.[88] Unlike human spleens, animal spleens have the ability to store red blood cells. The equine spleen is particularly well adapted and can regulate the red cell storage by relaxation and contraction. The equine spleen can store up to 50% of circulating red blood cells.[89] The contractile ability is under sympathetic control and, in times of stress or strenuous exercise, is stimulated to release the stored red blood cells into the circulation, increasing the red cell concentration of the blood. At least one report has questioned the role of splenic emptying in exercise-induced changes in red cell indices.[90] Phenylephrine (an α_1-adrenergic receptor agonist) administration has been reported to cause a transient, dose-dependent splenic contraction of up to 83% of the original splenic mass and may be useful for certain medical or surgical procedures in the horse, such as during splenectomy or as an adjunct to nonsurgical correction of nephrosplenic entrapment of the ascending colon.[91] Epinephrine has also been reported to

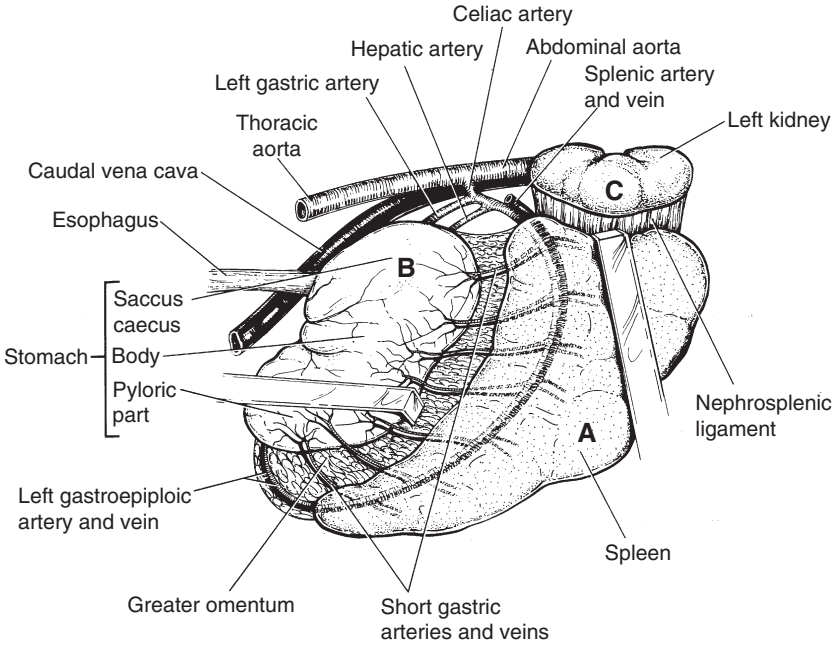

Figure 32-7. Left view of the spleen *(A)*, stomach *(B)*, and left kidney *(C)* with pertinent vascular supply. Retractors are placed along the greater curvature of the stomach to reveal the short gastric arteries and veins and along the dorsal extremity of the spleen to show the left kidney.

transiently reduce splenic length approximately 68% when administered as a bolus.[92]

The removal of aged or damaged erythrocytes occurs in the spleen owing to its unique structure. Blood is circulated through the splenic red pulp, terminating in small, highly porous capillaries. The blood cells pass out of the capillaries into the cords of the red pulp and eventually return to circulation through the endothelial wall of the venous sinuses. The red pulp and venous sinuses are loaded with macrophages (reticuloendothelial cells), which phagocytize unwanted debris and old or abnormal red blood cells. Also removed by the phagocytic cells are abnormal platelets, bloodborne parasites, and bacteria.[86]

In addition to its filtering and phagocytic activity, the spleen has another role in the immune-mediated defense against disease. It has a direct effect on the pattern and level of antibody response of the body. The spleen is the site of production of IgM, the homing hub for B cells, and the resevoir for immunocompetent lymphocytes.[88,93]

The resistance of the horse to disease caused by blood protozoa such as *Babesia* or *Theileria* species is significantly decreased after splenectomy.[94-97] Subclinical infections by protozoa may become patent infections after removal of the spleen.

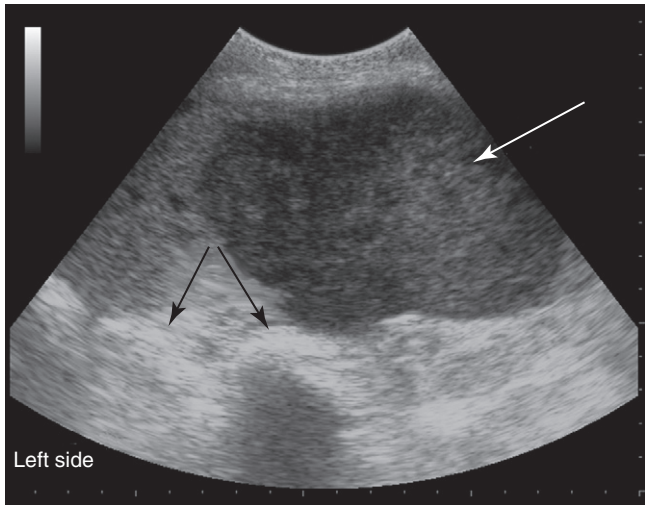

Figure 32-8. Ultrasonogram of a splenic mass with a 3 MHz curvilinear probe. The mass, later determined to be lymphosarcoma, involves most of the right half of the image *(white arrow)*. The relatively normal appearing splenic parenchyma is on the left. Ventral colon sacculi are in the background *(black arrows)*. (Courtesy Dr. Shannon Reed, University of Missouri, Columbia, MO.)

Diagnostic Procedures

Surgical disorders of the spleen may be relatively difficult to diagnose because of their rare occurrence and nonspecific clinical signs. Rectal palpation usually reveals thickening and rounded margins of an enlarged spleen. Ultrasonography either *per rectum* or through the left abdominal wall may also reveal an enlarged spleen. Ultrasonography is useful to help determine the cause of the splenic abnormality (Figure 32-8).[98] A splenic biopsy may be performed for histologic evaluation of splenomegaly. Normal coagulation function should be determined before a percutaneous biopsy is performed. Laparoscopy may also be used to aid in the diagnosis of splenic abnormalities and to help with the decision between medical therapy and splenectomy.[99-101] Laparoscopic ultrasonography allows video-guided ultrasound examination of targeted organs and tissues within the abdomen. Laparoscopy can also be used to guide biopsy and needle aspiration to minimize the risk of bleeding, intestinal puncture, and exudate leakage.[101]

To examine the spleen, a long (30 cm [12 inch]) laparoscope is introduced into the dorsal aspect of the left paralumbar fossa facing craniad. The head of the spleen, left kidney, and renosplenic ligament can be readily identified in the left dorsal quadrant of the abdomen. The caudal edge and lateral wall of the spleen can be visualized as the laparoscope is swept down

toward the left ventral quadrant, and abnormalities over most of the body of the spleen can be identified. Lesions in this region can be biopsied with additional instruments placed through separate portals in the left paralumbar fossa. Biopsies can be obtained with laparoscopic scissors as wedge biopsies or with an instrument similar to a uterine biopsy forceps. Hemorrhage should be expected and may be controlled with bipolar cautery forceps or a vessel sealing device (LigaSure; EnSeal—see Appendix B).[100,102] Additional diagnostic procedures used to differentiate disorders of the spleen include a complete history and physical examination; complete blood cell count, including total plasma protein and plasma fibrinogen; coagulation profile; abdominocentesis; the Coggins test (agar gel immunodiffusion test for equine infectious anemia); Coombs test; and aerobic and anaerobic bacterial cultures.

Indications for Surgery

Splenectomies are performed primarily for research purposes, to aid in the evaluation of the cardiovascular system during exercise and to alter the reticuloendothelial system, which controls to a great extent the infections caused by blood protozoa such as *Babesia* spp. or *Theileria* spp.[94-97,103-108] Splenomegaly with or without splenic infarction and rupture have been the only reported disease processes requiring splenectomy.[109-114] Other possible indications for surgery include neoplasia, trauma, infarction, and possibly an autoimmune disease in which the spleen would play a role in erythrocyte destruction.[115]

Splenomegaly

Splenomegaly in the horse may be primary or idiopathic, caused by congestion or hyperactivity, or it may be secondary.[112] Secondary splenomegaly has been attributed to subcapsular hematoma, equine infectious anemia, lymphoma-leukemia (lymphosarcoma),[116] metastatic melanoma, isoimmune hemolytic anemia, salmonellosis, babesiosis or theileriosis, ehrlichiosis and borreliosis, and anthrax.[109,112,117,118] Infarctions have also been reported as causes or consequences of splenomegaly.[110,111,119] Splenectomy is indicated for splenomegaly for chronic congestion, thrombosis, or primary tumors of the spleen with no evidence of metastases. Clinical signs of horses with splenomegaly include tachycardia, anemia, colic, arching of the back, and standing with the limbs drawn together beneath the body.[110,117,119]

Neoplasia

Lymphosarcoma (malignant lymphoma), metastatic melanoma, and hemangiosarcoma are the only primary neoplastic diseases of the equine spleen.[116,120-124] Lymphosarcoma is a neoplastic disease of lymphoid cells. Splenic lymphosarcoma is classified as an alimentary form of the disease. The cells metastasize via the lymphatics (i.e., in the spleen, the white pulp is first infiltrated). In later stages of the disease, the spleen's architecture may become so distorted that the pathway of spread is not apparent. Leukopenia and anemia are often present.

Melanomas may occur as a primary tumor of the spleen of horses, but the spleen is more likely a site of metastases.[125-128] Hemangiosarcoma or hemangioendothelioma is a frequent primary tumor of other domestic animals and humans but has been only rarely reported as a primary tumor in the horse.[124,129] This tumor is more commonly reported in the spleen of the horse as a site of metastasis.[124,130-132] All of these tumors may cause splenomegaly and may manifest with clinical signs of an acute abdominal disease owing to an increased weight of the spleen and stretching of the splenic ligaments.[124,125,133]

Splenic Infarction and Splenic Abscesses

Splenic infarction occurs concurrently with splenomegaly.[110,111] Venous thrombosis and infarction may occur any time the spleen is enlarged. Splenic arterial or venous thrombosis, or both, is seen with autoimmune hemolytic anemia, purpura, hemorrhagic pancreatitis, and splenic abscesses. The last have been reported to be caused by *Habronema*, *Strongylus*, *Bacteroides*, *Clostridium*, *Streptococcus*, and *Mycobacterium* spp.[98,134,135]

Splenic Rupture

Splenic rupture is relatively rare in horses.[113,114,136-140] It may be caused by direct, severe trauma, or it may occur secondary to anthrax, lymphosarcoma, echinococcus infestation, splenic hematoma, or hemorrhage. In most reported cases, horses have been found dead, died during the course of the illness, or were euthanatized shortly thereafter. The cause of rupture is usually unknown, but in one report, it was thought to be as a result of a previous splenomegaly.[139] Most of the ruptures occur on the visceral surface of the spleen. Very few cases of rupture to the parietal surface have been reported.[138,139] Three cases of nonfatal subcapsular hematoma on the parietal surface have been reported.[99,117,141] The clinical signs associated with splenic rupture include serosanguineous or sanguineous peritoneal fluid, pale mucous membranes, anemia, tachycardia, dyspnea, profuse sweating, anorexia, and colic. Immediate surgery is indicated to control hemorrhage. The clinical signs reported for splenic hematoma include pancytopenia, anorexia, depression, and abdominal distention.[141]

Preoperative Considerations

Preoperative considerations for splenectomy include assessment of hydration, acid-base status, and clotting function. The presentation of horses requiring a splenectomy is usually on an emergency basis (i.e., acute abdominal disease, acute blood loss), and therefore the horses may be dehydrated and in shock. Careful assessment should differentiate those with pain because of stretching of the splenic ligaments from those with pain as a result of acute blood loss from splenic rupture.

Surgical Techniques

Splenectomy

APPROACHES

Several reports have described the technique for splenectomy.[94,109,110,115,142-148] All the methods describe an approach from the left side, with the animal standing or in right lateral recumbency. The variations in the techniques center on the exact position of the skin incision. Originally, the procedure was described as a paralumbar approach (i.e., caudal to the last or 18th rib).[142] Because of the difficulty in accessing the base of the spleen, and therefore its primary vessels, various techniques

have been proposed to gain better access. Techniques have been described for access to the spleen between the last two ribs or by resection of the 18th rib, the 17th rib, or the 16th rib.[94,110,144-148] One report also describes removal of the distal aspect of the last three ribs.[143] Another report describes removal of the 17th and transecting of the 16th and 18th ribs for removal of an enlarged spleen.[109]

Laparoscopic splenectomy has been reported in humans, but its potential use in the horse at this time is only speculative.[149-152] Laparoscopic-assisted splenectomy has been reported in one horse.[112] Hand-assisted techniques may soon be developed similar to those reported for removal of diseased kidneys and ovarian tumors in horses.[153,154] The friability of the diseased spleen and its vascular supply may complicate potential laparoscopic procedures, although the visualization afforded by laparoscopy may well offset the potential complications. The caudal reflection of the pleural cavity should be considered when determining the technique to be used. The lateral thoracic wall attaches to the diaphragm along a line called the *diaphragmatic line of pleural reflection*. This line extends from the eighth and ninth costal cartilages dorsocaudally in a gentle increasing curve, so that its most caudal aspect is at about the middle of the cranial border of the last rib forming the caudal border of the pleural cavity.[155] Therefore, resection of either the 16th or 17th rib is almost always associated with opening the pleural cavity, resulting in a pneumothorax. This is not necessarily a significant problem, because many horses have an intact mediastinum. However, if the mediastinum is incomplete, assisted ventilation is required to maintain respiration.

17TH RIB RESECTION TECHNIQUE

The horse is placed under general anesthesia in right lateral recumbency and prepared for aseptic surgery. A vertical incision is made over the 17th rib from the lumbar muscles (iliocostalis thoracis and longissimus muscles) proximally to the costochondral junction distally. The dissection continues through the subcutaneous tissues and the cutaneous trunci, serratus dorsalis caudalis, and external abdominal oblique muscles to expose the lateral periosteum of the 17th rib. The periosteum is incised and elevated from the lateral aspect of the rib with a periosteal elevator starting at the costochondral junction and continuing proximad. Circumferential elevation of the periosteum over the full length of the exposed rib is achieved using a large Doyen, Alexander, or other similar periosteal elevator. An obstetrical cable (Gigli wire), bone-cutting forceps, or oscillating bone saw is used to transect the rib as far proximad as possible. The body of the rib is disarticulated at the costochondral junction through lateral and ventral pull and is removed. The medial periosteum and underlying peritoneum are incised. Proximally, the pleural cavity may be entered. The cavity should be closed by suturing the diaphragm to the intercostal muscles using an absorbable suture material in a continuous pattern. While the pleural cavity is open, the horse's respiration may need to be assisted with mechanical ventilation or intermittent manual compression of the rebreathing bag.

SURGICAL PROCEDURE

The edges of the incision should be draped with saline-moistened laparotomy sponges and separated with a Finochietto rib spreader. The dorsal part of the spleen may be elevated into the incision (Figure 32-9). The nephrosplenic ligament is transected to expose the splenic vein. The vein is located more superficial or lateral than the artery and may be isolated for separate ligation. Care should be taken not to tear the vein. Intravenous infusion of phenylephrine will result in significant, transient splenic contraction to reduce the weight of the spleen and allow better access to its visceral portion. Some specialists recommend separate ligation of the vessels, with the artery being ligated first followed by massage of the spleen to remove any excess blood from the organ. A variation involves injecting 10 to 15 mL of 1:1000 epinephrine at several sites in the spleen to expedite its contraction. The vessels should be triple-ligated and transected between the distal ligatures, leaving two ligatures on each (if ligated separately) of the vessels (Figure 32-10). The remainders of the nephrosplenic, phrenicosplenic, and gastrosplenic ligaments are transected near the hilus. Ligatures or stainless steel hemostatic clips are applied to the short gastric vessels as needed. As the spleen is separated from its attachments, the free cut edge of the greater omentum may be sutured to the free cut edge of the gastrosplenic ligament. This is most easily performed while the spleen is still attached (Figure 32-11).

Abdominal wall closure is accomplished in three layers as per the surgeon's preference. The initial layer includes the peritoneum and periosteum, the second layer consists of the

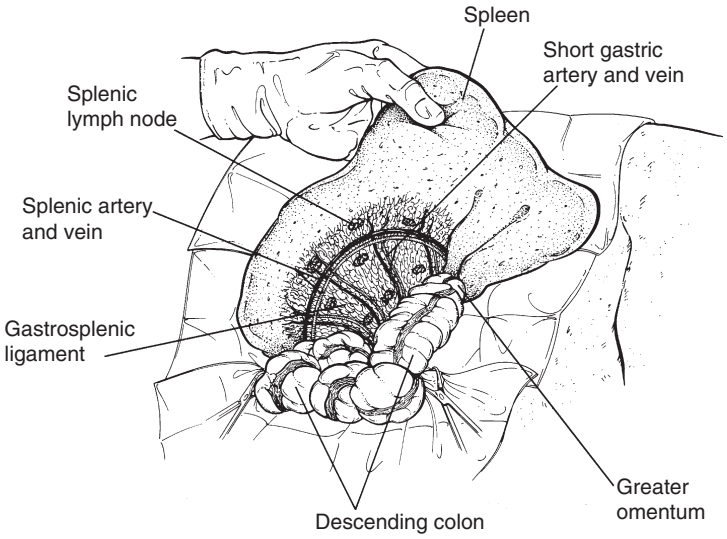

Figure 32-9. Intraoperative view showing elevation of the dorsal part of the spleen into the incision. The renosplenic ligament prevents complete elevation of the spleen.

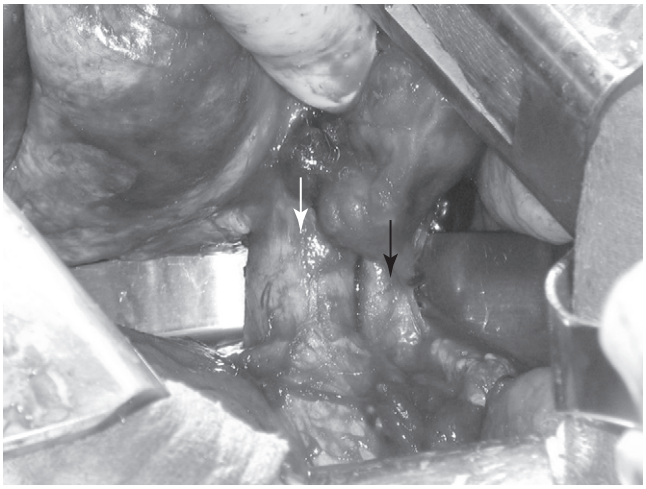

Figure 32-10. Splenic artery *(black arrow)* and splenic vein *(white arrow)* isolated during splenectomy surgery. (Courtesy Dr. Shannon Reed, University of Missouri, Columbia, MO)

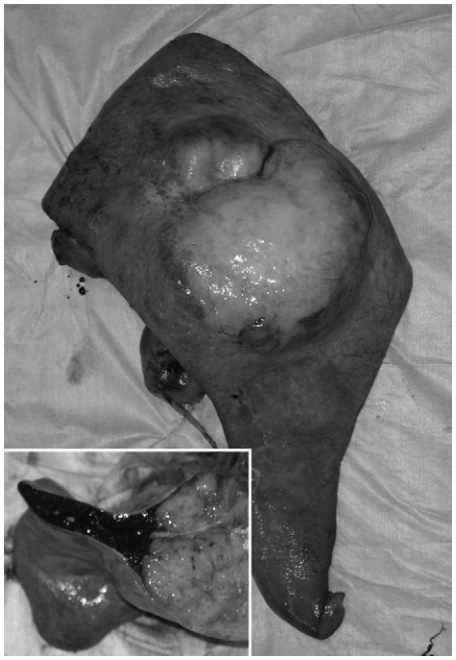

Figure 32-11. Spleen specimen after removal with a large mass later determined to be a lymphosarcoma involving the majority of the midbody of the spleen in a 10-month-old Arabian colt. The inset at lower left demonstrates the cut surface of the splenic mass on right and relatively normal splenic parenchyma on the left. (Courtesy Dr. Shannon Reed, University of Missouri, Columbia, MO.)

subcutaneous tissues, and the skin represents the final layer. Either active or passive drains are recommended between muscle layers to minimize serum accumulation and subsequent wound dehiscence.

Splenorrhaphy and Partial Splenectomy

Lacerations of the spleen, such as may occur during abdominocentesis or minor ruptures of the splenic capsule, may be corrected by splenorrhaphy. Absorbable sutures placed across the

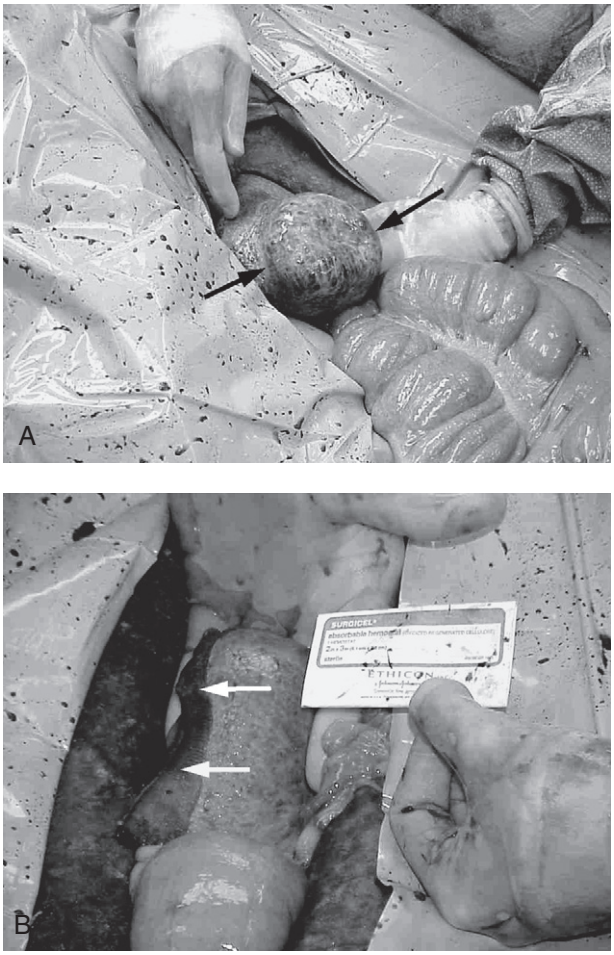

Figure 32-12. Intraoperative view of a partial splenectomy. **A,** A splenic mass confined to the distal third of the spleen was identified via a midline laparotomy *(black arrows)*. The patient was admitted with a history of chronic colic. **B,** An automated stapling device was used to resect the distal third of the spleen. The resection margin was covered with an oxidized cellulose product *(white arrows)* to reduce minor hemorrhage from the spleen.

defect are usually sufficient to control hemorrhage. Large vessels may need to be individually ligated. Partial splenectomy or splenorrhaphy has not been reported in the horse. Various techniques have been described in humans and canines, including the use of mattress sutures, stapling devices, hemostatic (hemostyptic) agents, ultrasonic cutting devices, CO_2 lasers, a microwave coagulator, an argon-beam coagulator, and absorbable mesh consisting of polyglycolic acid or polyglactin 910.[156-167] The authors have applied a TA-90 stapling device, followed by application of an oxidized cellulose hemostatic agent (Surgicel), to accomplish partial splenectomy without complication in a horse with a splenic mass (Figure 32-12).

Closure of the Renosplenic Space

Closure of the renosplenic space has been advocated to prevent recurrence of left dorsal displacement of the large colon.[168] More recently, laparoscopic techniques have been described for closing the renosplenic space (see Chapter 37).[169-172] The techniques involve suturing the most dorsal visceral surface of the spleen to the renosplenic ligament to eliminate the area between

the spleen and the left kidney to which the large colon frequently becomes displaced.[168-172] The use of polypropylene mesh has been described to obliterate the space in five horses.[171] Etilefrine (Effortil), a sympathomimetic drug, has been reported to aid in the procedure by inducing splenic contracture.[172] There have been no reported cases of left dorsal displacement after closure of the renosplenic space. The only reported complication of the procedure was adhesion formation of the descending colon to the polypropylene mesh.[171]

Aftercare

After recovery from anesthesia, the animal should be placed in a stall and hand-walked for the first 7 to 10 days. Body temperature, pulse and respiratory rates, and appetite should be monitored regularly. The incision should also be monitored for signs of heat, swelling, drainage, seroma formation, or infection. Antibiotics may be administered as indicated by clinical signs and the results of bacterial culture and sensitivity testing. Analgesics such as flunixin meglumine, ketoprofen, or phenylbutazone should be administered during recovery and for at least 24 hours after surgery to control pain. Drains placed at the time of surgery should be removed after 2 to 3 days. Drainage and lavage of the incision site should occur if evidence of seroma formation or infection is noted.

Complications

The most serious complication after splenectomy is intra-abdominal hemorrhage.[115] Incisional complications include drainage, seroma, edema, infection, and dehiscence.[94,115,143] Additional complications related to the transthoracic approach include pyothorax, pneumothorax, and pleuritis.[115] Complications in humans after splenectomy include atelectasis, pleural effusion, pneumonia, hemorrhage, subphrenic abscess formation, pancreatic fistula and pancreatitis, gastric fistulation, thrombocytosis and thrombosis, and sepsis.[173] Overwhelming postsplenectomy infection syndrome is a rare but fatal complication recognized in humans.[174] Therefore, splenectomy should not be considered an innocuous procedure. The spleen is an important component of the immune system and its removal may predispose the horse to subsequent infections.[93] Therefore, conservative therapy and partial splenectomy should be attempted as alternatives to splenectomy whenever possible, particularly in young horses.

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CHAPTER
33

Colic: Diagnosis, Surgical Decision, and Preoperative Management

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The veterinarian presented with an equine colic faces a true emergency and a diagnostic challenge. Through the combination of history, physical examination, and diagnostic procedures, the veterinarian must determine and communicate to the owner the source of the abdominal pain, the prognosis for recovery, the correct treatment strategy, and the estimated costs. Although a great deal of valuable information can be gained from an accurate signalment, history, and physical examination, a range of further diagnostic procedures are necessary to aid in this process. It is therefore essential to understand the indications, significance, and limitations of these techniques. This information may then be interpreted, and a decision to continue medical treatment or perform an exploratory celiotomy can be made.

DIAGNOSIS

History and Signalment

The signalment of the colic patient is important when determining the specific information that should be obtained during history taking, and which physical examination and diagnostic procedures are indicated. The signalment itself can often lead to an early differential diagnosis that may be investigated during the subsequent history and physical examination. The information obtained from the owner should include both the medical history and management practices. Details of the medical history related to the current and previous episodes of colic, other illnesses or surgery, and current and previous medications administered are essential. A history of recent nonsteroidal anti-inflammatory drug (NSAID) use may suggest a possible impaction of the ascending colon or right dorsal colitis.[1] Knowledge of all analgesics and sedatives administered before presentation is crucial when interpreting signs of pain and physical examination findings, as they may alter clinical signs. A description of the current management of the horse and any changes to diet, exercise, stabling, and anthelmintic regimen is important in identifying potential risk factors for certain conditions. These include the association between the feeding of coastal Bermuda hay and the risk of ileal impaction,[2] and the association between the behavior of crib biting and epiploic foramen entrapment.[3]

Physical Examination

The physical examination of the colic patient should be conducted in a thorough, logical order and should not be limited to the abdomen. The measurement and recording of the temperature and the heart and respiratory rates on initial examination allows the response to medication and therapy to be quantified. The heart rate is an indicator of the physiologic response to pain, dehydration, and endotoxemia and is useful in determining prognosis in both large and small intestinal disease.[4,5] Conditions associated with pyrexia include anterior enteritis, colitis, and pleuropneumonia and do not generally require immediate surgical intervention. Therefore, complete auscultation of the thorax to rule out conditions of the respiratory tract should be performed. Examination of the oral mucus membranes, including measurement of capillary refill time, aids in the determination of hydration status and the diagnosis of endotoxemia. In the endotoxemic horse, the capillary refill time is prolonged and the mucous membranes develop a brick red or purple color. A dark "toxic line" may be apparent along the gum line of the horse.

Auscultation of abdominal borborygmi allows the subjective assessment of large intestinal motility. Cecal motility may be auscultated over the right flank, whereas the pelvic flexure and ascending colon are auscultated over the left flank. Audible movements of the cecum and ventral colon include propulsive, retropulsive, and mixing contractions.[6] Propulsive contractions of the cecum and colon occur approximately every 3 to 4 minutes but are decreased in frequency by conditions including anorexia and sedation (e.g., α_2-adrenergic receptor agonists).[7] Ileus of the large or small intestine will result in the absence of intestinal borborygmi and is therefore a significant physical examination finding. Intestinal borborygmi can also be increased in certain conditions, including the early stages of distention and inflammation.

A critical aspect of the examination of any horse presented for colic is the assessment of the degree and persistence of signs

of pain.[8] It is often easiest to observe a horse in a box stall where it may display signs of pain that are not apparent during handling or restraint in stocks. Obvious signs of pain include pawing at bedding, looking at the flank, kicking at the abdomen, repeated lying down and standing, and rolling. Abdominal pain may also manifest itself by more subtle behavior including a dull appearance, lowered head position, and reluctance to move.[9] The severity of pain is often related to the degree of intestinal injury, which is in turn related to the need for surgical intervention. A large colon volvulus resulting in large colon distention and ischemic injury will cause severe pain that is refractory to treatment with analgesics and sedatives. In contrast, a nonstrangulating obstruction results in lower grade pain that responds to analgesia. Observation of the response to treatment with NSAIDs or sedatives is important when characterizing the type of pain.[8] Mild pain typically responds to treatment with an NSAID alone for a period of 8 to 12 hours. In contrast, moderate pain will respond to analgesia for a limited period and requires repeated administration. Severe pain is manifest by violent behavior and may not respond to analgesia, which is frequently an indicator of the need for immediate abdominal surgery.

During the physical examination, when indicated, it may be prudent to place an intravenous jugular catheter and begin fluid therapy while further diagnostic procedures are performed.

Rectal Examination

Rectal examination should be performed using a suitable combination of physical and chemical restraint to allow palpation of the cecum, left dorsal and ventral colon, pelvic flexure, descending colon, and reproductive tract. If sedation does not provide adequate relaxation to allow a safe examination, the instillation of lidocaine into the rectum or administration of *N*-butylscopolammonium bromide can reduce straining, improve the quality of the rectal examination, and reduce the risk of rectal tearing.[10,11] Careful palpation should be performed to prevent rectal tears and allow diagnosis of any existing tear. The position and size of each palpable organ may be assessed as well as the content, which may be ingesta, fluid, or gas. Entrapment of the large colon in the nephrosplenic space may be palpable in the upper left abdominal quadrant. The small intestine is not normally palpable *per rectum* and is therefore an abnormal finding on rectal examination. The small colon is normally distinguished by the presence of fecal balls and a broad antimesenteric band. If these features are not palpable it suggests impaction of the small colon. In the pregnant mare, the broad ligaments should be palpated to diagnose a possible uterine torsion. If a tight broad ligament is palpated, the direction of the torsion should be determined to allow correction.

Nasogastric Intubation

The passage of a nasogastric tube should be performed during all colic examinations to allow decompression if necessary and prevent gastric rupture. Water is flushed through the tube to begin a siphoning action, and should be measured to allow the net volume of fluid recovered to be determined. The color and smell of the fluid should be assessed. It is normal to recover up to 2 L of green, nonodorous fluid. Excessive fluid indicates either gastric outflow obstruction or decreased small intestinal motility resulting in an accumulation of fluid in the stomach. Anterior enteritis cases often yield a large volume of malodorous orange or yellow fluid. Large amounts of feed in the gastric fluid may indicate gastric impaction. Gastric outflow obstruction may also be caused by gastroduodenal ulceration or neoplasia. The cause of gastric outflow obstruction may be further investigated by endoscopy. Following decompression of the stomach, the nasogastric tube may be left in place during surgery to prevent fluid aspiration and allow intraoperative decompression.

Clinical Pathology

The use of clinical pathology has become a crucial part of the assessment and treatment of the colic patient. The measurement of the blood packed cell volume (PCV) and total protein (TP) can be performed using a centrifuge and refractometer to quickly assess the patient's hydration status. A high PCV has been shown to be associated with a poor prognosis in horses with both small and large intestinal disease.[4,12,13] In contrast, a low TP has been associated with a poor prognosis in horses undergoing surgery for the treatment of small intestinal disease.[5,12] The introduction of point-of-care analyzers has created widespread availability of blood electrolyte and acid-base balance information. When using a point-of-care analyzer it is important to store and prepare the cartridges for use according to the manufacturer's instructions to ensure accuracy.[14] Although the majority of changes seen on hematology are nonspecific, it is useful in diagnosing inflammation, endotoxemia, or sepsis. These changes may be evident as leukopenia, neutropenia with appearance of immature and toxic neutrophils, lymphopenia, and thrombocytopenia.

The blood electrolyte profile of horses losing fluids through gastric reflux or diarrhea often reveals abnormalities, including low sodium, potassium, calcium, and bicarbonate levels that may be addressed during fluid therapy. Because lactate is a product of anaerobic glycolysis, its measurement may reflect ischemic injury and may aid in determining the prognosis.[15] Among horses with large colon volvulus, a serum lactate concentration greater than 6 mmol/L has been associated with a poor prognosis for survival.[16] Measurement of the anion gap allows the indirect measurement of blood lactate and is of value when determining prognosis.[17] A study of horses with large colon displacement revealed an increased serum γ-glutamyltransferase (GGT) in 49% of right dorsal displacement cases but in only 2% of horses with a left dorsal displacement.[18] This increase in GGT in horses with right dorsal displacement of the large colon is due to obstruction of the bile duct. Additional information on acid-base derangements and their management can be found in Chapter 3.

Abdominocentesis

Peritoneal fluid can be examined as both a diagnostic and prognostic aid. Peritoneal fluid can be collected by clipping and aseptically preparing the most dependant part of the abdomen, on or slightly right of midline to avoid the spleen, and inserting an 18-gauge needle. Alternatively, following local anesthesia, a small incision can be made using a No. 15 scalpel blade and inserting a teat cannula. Care must be taken during the collection of fluid to avoid enterocentesis, particularly in horses with distended viscera, or amniocentesis in the pregnant mare.

Where fluid is not easily obtained, abdominal ultrasonography can be performed to identify an area of fluid accumulation for collection. The abdominal fluid should be collected in a plain tube for the measurement of protein concentration and in an EDTA tube for a cell count and hematology.

Immediately following collection, the gross appearance of the fluid should be visually assessed. Normal peritoneal fluid has a clear, colorless to light yellow appearance. When a strangulating lesion is present, there is movement of protein followed by red blood cells and finally leukocytes into the peritoneal cavity. This results in the peritoneal fluid becoming turbid and red to brown. The presence of ingesta in the peritoneal fluid suggests a ruptured viscus and a hopeless prognosis. In this situation, care must be taken to ensure that the sample was not obtained by enterocentesis. The normal total protein concentration of abdominal fluid is less than 2 g/dL, but this will increase with intestinal disease. The appearance of red blood cells in the abdominocentesis sample may be the result of an intestinal strangulation or an iatrogenic source. A small amount of blood contamination may occur if a vessel in the abdominal wall is punctured. If the peritoneal fluid is normal, on centrifugation a small pellet of red blood cells will collect, leaving fluid with a normal appearance.[19] It is also possible to insert the needle into the spleen, resulting in the collection of a sample with a PCV similar to blood.

Clinical biochemistry may be performed on peritoneal fluid to determine other factors, including fibrinogen, lactate, phosphate, glucose, and pH. A high peritoneal lactate has been shown to be a more sensitive indicator of a strangulating obstruction of the intestine than plasma lactate.[15] In those horses with suspected septic peritonitis, the serum and peritoneal fluid glucose levels can be compared. A difference of greater than 50 mg/dL between the serum and peritoneal fluid glucose level, a low peritoneal fluid glucose level (less than 30 mg/dL), and pH of less than 7.3 are indicators of septic peritonitis.[20]

Ultrasonography

Ultrasonography has become an important part of the diagnosis, treatment and management of the colic patient.[21] Abdominal ultrasonography is generally performed using a percutaneous approach following preparation of the skin by clipping and application of alcohol or coupling gel. A low-frequency (2.5 to 5 MHz) linear, curvilinear, or sector transducer will produce a diagnostic quality image while providing sufficient penetration to identify deeper structures. It is possible to identify the stomach, small intestine, cecum, and large colon and determine their size, position, wall thickness, and motility.[21] The stomach may normally be imaged cranially on the left of the abdomen between the 11th and 13th intercostal spaces. The gastric volume can be estimated and stomach decompression confirmed by measurement of the gastric wall height at the 12th intercostal space.[22]

The small intestine can be identified in the cranial ventral abdomen and can be examined for wall thickness, diameter, and motility. The normal wall thickness of the small intestine is less than 3 mm, and an increase may indicate enteritis or strangulating obstruction (Figure 33-1). Obstruction results in distention of small intestinal loops, which can be identified and measured ultrasonographically (Figure 33-2). The motility of these loops should be assessed, because ileus may be diagnosed as hypomotile small intestine on ultrasonographic examination.

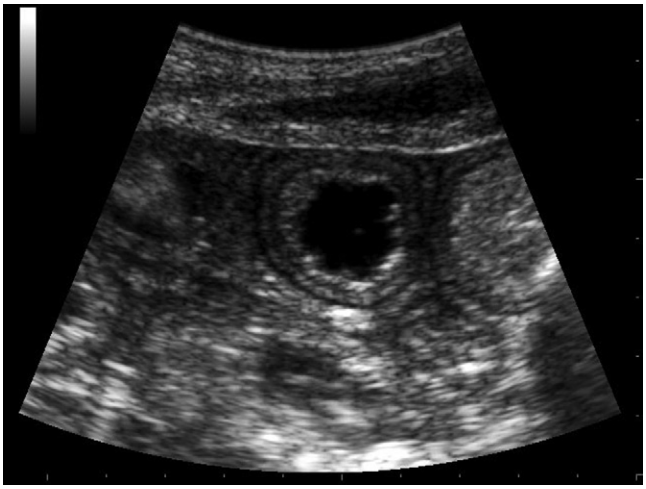

Figure 33-1. Ultrasonographic examination of the ventral abdomen of a horse using a 3.5 MHz curvilinear probe. A cross section of the jejunum can be identified, and measurement reveals a thickened wall (1.01 cm) characteristic of enteritis. (Courtesy M.K. Sheats, North Carolina State University, Raleigh, NC.)

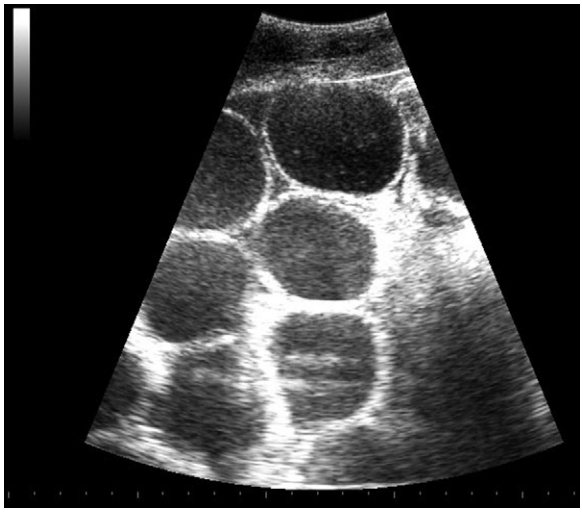

Figure 33-2. Ultrasonographic examination of the ventral abdomen of a horse using a 3.5 MHz curvilinear probe. Several cross-sectional loops of distended jejunum can be identified with a normal intestinal wall thickness. These findings are characteristic of a functional or obstructive ileus. (Courtesy M.K. Sheats, North Carolina State University, Raleigh, NC.)

Ultrasonography has been demonstrated to be extremely useful for the diagnosis of large colon volvulus in the horse.[23,24] The colon is identified on the ventral abdominal midline, and the appearance and thickness of the wall and motility are assessed. Normally, the sacculated ventral colon with a wall thickness of less than 5 mm is identified. If a large colon volvulus of 180 or 540 degrees is present, the nonsacculated dorsal colon can be identified on the ventral abdomen.[24] Measurement of the colonic wall thickness has been shown to be useful in the diagnosis of large colon volvulus (Figure 33-3). A colonic wall thickness measurement of greater than 9 mm had a sensitivity of 67% and a specificity of 100% in diagnosing large colon

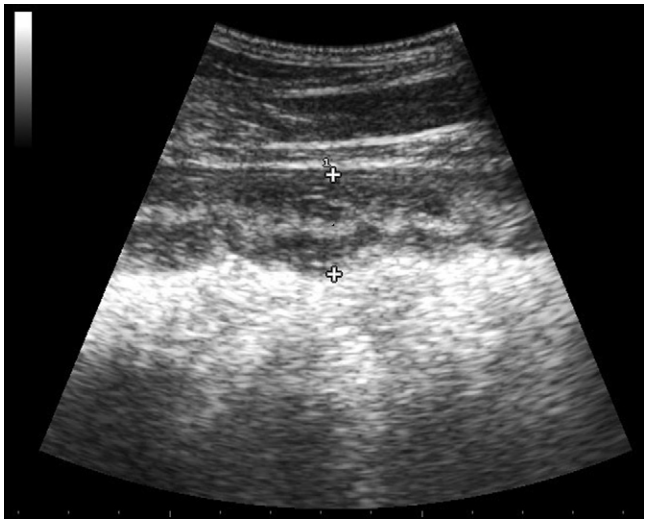

Figure 33-3. Ultrasonographic examination of the ventral abdomen 2 cm caudal to the xyphoid of a horse using a 3.5 MHz curvilinear probe. A longitudinal image of the ventral colon reveals significant thickening (1.97 cm) diagnostic of a large colon volvulus. (Courtesy M.K. Sheats, North Carolina State University, Raleigh, NC.)

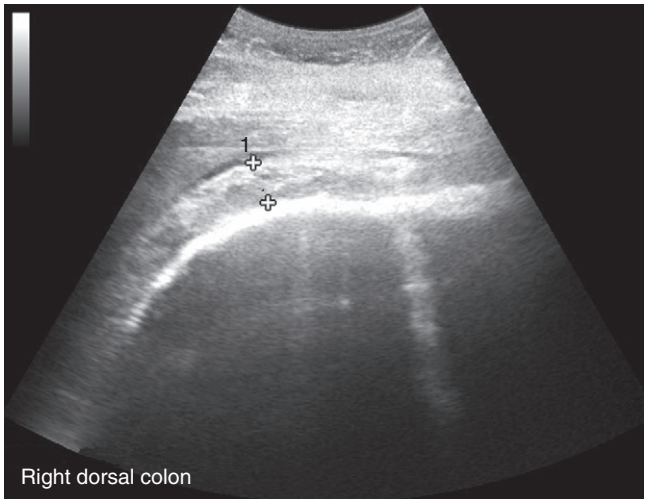

Figure 33-4. Ultrasonographic examination of the 11th intercostal space on the right side of a horse using a 5 MHz curvilinear probe. The right dorsal colon is thickened (1.46 cm), and a hypoechoic layer of edema, cellular infiltrate, and granulation tissue is visible. These changes are consistent with right dorsal colitis. (Courtesy M.K. Sheats, North Carolina State University, Raleigh, NC.)

volvulus.[23] In addition to the diagnosis of large colon volvulus, ultrasonography can be used to monitor postoperative recovery of the colon.[25]

The horses in which right dorsal colitis is suspected, a diagnosis can be confirmed using ultrasonography performed between the 10th and 14th right intercostal spaces.[26] Ultrasonographic changes associated with right dorsal colitis include a thickened colon wall and a hypoechoic layer of submucosal edema and inflammatory infiltrate (Figure 33-4).[26] Left dorsal displacement of the large colon can be diagnosed ultrasonographically through identification of the large colon lateral

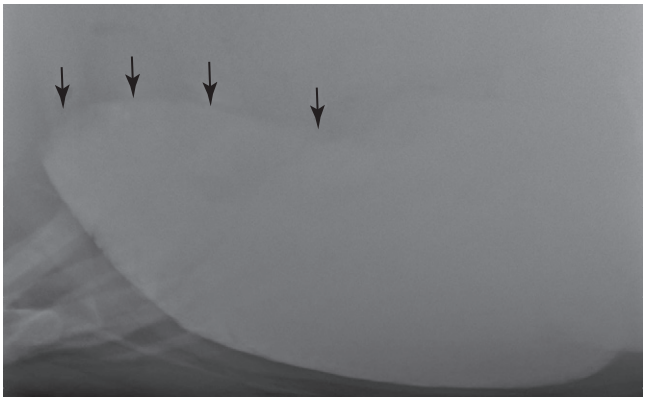

Figure 33-5. Lateral radiograph of the ventral abdomen of a horse. Accumulation of sand is apparent in the ventral colon *(arrows)*.

or dorsal to the spleen.[27] The displaced colon prevents visualization of the right kidney. If nonsurgical management is performed, ultrasonography can be used to confirm correction of the displacement.[27] Although radiography remains the gold standard for diagnosing sand impaction of the colon, ultrasonography can identify sand, which produces a hyperechoic signal and acoustic shadowing of deeper structures.[28] The presence of a sand impaction can also reduce colonic motility.

Radiography

In the examination of the adult horse presented for colic, radiography is useful when the presence of radiopaque material is suspected. Therefore, abdominal radiographs are particularly useful for the diagnosis of sand accumulation and enterolithiasis (Figure 33-5).[29,30] When performing abdominal radiography, adequate exposure is critical to maximize diagnostic quality and reduce the incidence of false negative examinations. The sensitivity and specificity of radiography for the diagnosis of enterolithiasis have been described as 76.9% and 94.4%, respectively.[30]

The improved image quality of abdominal radiography in the foal and small horse allows examination of the stomach and small and large intestines. Radiography can be performed using both plain film and contrast techniques to allow diagnosis of obstruction, intussusception, and radiopaque foreign bodies. Contrast radiography can be performed using 30% wt/vol barium sulfate suspension administered orally or rectally.[31] This technique is useful for identifying delayed gastric outflow obstruction and obstruction of the small, transverse, and large colons.

ANCILLARY DIAGNOSTIC AIDS

Endoscopy

Examination of the esophagus, stomach, and duodenum can be performed in the adult horse using a 3-m flexible endoscope. During a colic examination, endoscopy can be used to confirm gastric decompression and diagnose gastric ulcer disease, gastric impaction, and gastric squamous cell carcinoma. Endoscopic examination of the rectum allows the minimally invasive investigation of rectal tears.

Laparoscopy

The use of laparoscopy has been described for investigation of both the acute and chronic colic patient.[32] Laparoscopy is an option in those patients with controlled abdominal pain. Its use is limited in the horse by the inability to completely visualize the abdominal contents and by the difficulties associated with manipulating the large viscera of the horse. The sensitivity and specificity of laparoscopy as a diagnostic technique are greater in horses with acute colic than chronic colic.[32] When performing laparoscopy in the acute colic patient, care must be taken to prevent penetration of gas-distended abdominal viscera. Although laparoscopy is generally a diagnostic technique in the acute equine colic patient, its use has been described for correcting left dorsal displacement of the large colon.[33]

Laparoscopy is suitable for diagnosis of a range of abdominal conditions, including mesenteric tears, uterine rupture, intestinal adhesions, small intestinal strangulating lesions, large colon displacement (Figure 33-6), and visceral rupture.[32]

DECISION FOR SURGERY

Following the examination and diagnostic procedures, there are three general options available for the management of the colic patient. In patients with a poor prognosis, the owner may elect to have the horse euthanized. If that is not the case, the veterinarian must decide whether an immediate exploratory celiotomy is required or if medical management and further observation should be instituted. The decision to perform an exploratory celiotomy is largely based upon the ability to control pain and the abnormalities identified by physical examination and diagnostic procedures. Prompt surgical intervention is critical in maximizing the probability of a successful outcome, whereas a delayed exploratory celiotomy may result in visceral rupture or deterioration in the patient's condition. Therefore surgery is often performed before a definitive diagnosis of the cause of the colic has been reached.

The response to immediate medical therapy and analgesia is often a strong indicator of the need for an exploratory celiotomy. An exploratory celiotomy is often necessary in those horses with uncontrolled abdominal pain, even if other diagnostic procedures find no abnormalities. The presence of distended, hypomotile small intestine on rectal and ultrasonographic examination is commonly associated with the need for surgical intervention. Serosanguineous peritoneal fluid with an increased total protein and white blood cell count is an indicator of significant pathology and requires an exploratory celiotomy. The continued production of gastric reflux or deterioration in physical parameters including hydration status may suggest an exploratory celiotomy is necessary for both diagnostic and therapeutic purposes.

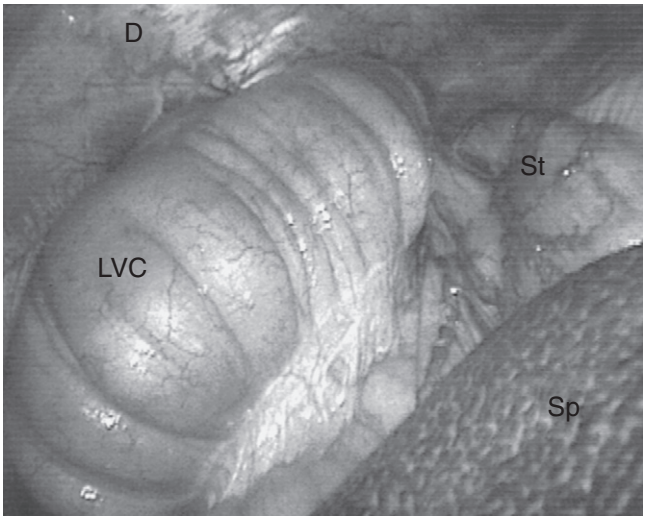

Figure 33-6. Left-sided laparoscopic view of a large colon displacement (i.e., retroflexion of the pelvic flexure). *D*, Diaphragm; *LVC*, left ventral colon; *Sp*, spleen; *St*, stomach.

PREOPERATIVE MANAGEMENT

The preoperative management of the colic patient requires preparation to undergo anesthesia and administration of prophylactic antimicrobial and anti-inflammatory agents. Although an exploratory celiotomy is frequently an emergency surgery, it is important to attempt fluid therapy to address hydration status and acid-base and electrolyte abnormalities before anesthesia. Because the duration of an exploratory celiotomy may be long and the gastrointestinal tract is often entered, prophylactic antimicrobials should be administered 30 to 60 minutes before surgery and repeated during surgery if necessary. If the colic patient has not already received an NSAID, flunixin meglumine (0.25 to 1.1 mg/kg) may be administered preoperatively to reduce inflammation and treat endotoxemia.

Before surgery, a nasogastric tube should be placed to allow stomach decompression during surgery as necessary. The horse's mouth could be rinsed to prevent aspiration of feed material during intubation. Any bedding or other material in the horse's hair coat should be removed with a dry brush to reduce contamination of the operating room.

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CHAPTER 34

Surgical Approaches to the Abdomen

Martin R. Kummer

Surgical approaches to the abdomen are laparotomies of different sizes and locations. A laparotomy is a surgical procedure involving an incision through the abdominal wall to access the abdominal cavity, also known as celiotomy. Using this definition, even the portals created for laparoscopic approaches can be considered small laparotomies.

VENTRAL MIDLINE APPROACH

The most common approach applied in equine abdominal surgery is performed through the ventral midline, specifically through the linea alba (Figure 34-1) because it allows exteriorization of 75% of the intestinal tract (Figure 34-2). The stomach, duodenum, distal ileum, dorsal body and base of the cecum, distal right dorsal colon, transverse colon, and terminal descending colon are the only segments that cannot be exteriorized. The ventral midline approach creates minimal hemorrhage, is easy to perform, can be extended if needed, and contains strong fibrous tissues for closure.

After positioning the patient in dorsal recumbency and clipping the hair of the surgical field, an indwelling urinary catheter should be placed in male horses to prevent the possibility of urine contamination during surgery. The penis is carefully snared with a gauze bandage, properly cleaned, and positioned in the prepuce after prior cleaning. The preputial cavity is subsequently sutured closed, allowing only the catheter to exit the suture line. Routine aseptic preparation of the surgical field is the next step.

The linea alba extends from the xiphoid process to the prepubic tendon and contains the median fibrous raphe of the external oblique and the transverse abdominal muscle aponeuroses. It consists of dense connective tissue composed of sheets of cross-linked collagen bundles and fibroblasts. The thickness of the linea alba gradually increases from craniad, where it measures about 3 mm, to caudad, where it reaches a thickness of approximately 10 mm.[1] The midline incision should be large enough to allow exteriorization of viscera without applying excessive pressure on the intestines, which would increase the risk of iatrogenic tears. The time saved during the procedure and the increased safety of a larger incision more than compensate for the extra time required to close a longer incision. Additionally, a smaller incision will be traumatized more during the manipulations of a surgical procedure than a larger one, which may delay its healing and lead to postoperative infection and herniation, especially after colic surgery. After incising the skin and subcutaneous tissue, the linea alba is incised beginning in the umbilical region where it is the widest and thickest. The incision is subsequently continued craniad to its desired length,

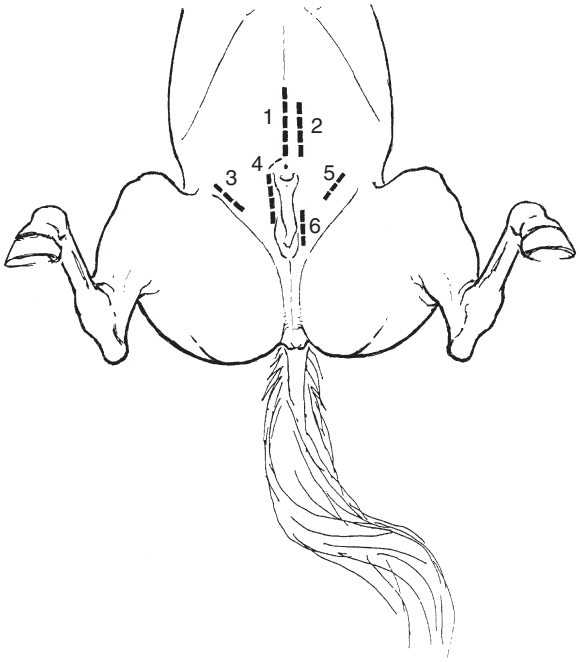

Figure 34-1. Abdominal approaches through the ventral abdominal wall: ventral midline *(1)*, paramedian *(2)*, inguinal *(3)*, combination of a ventral midline and ventral paramedian *(4)*, parainguinal *(5)*, suprapubic paramedian *(6)*.

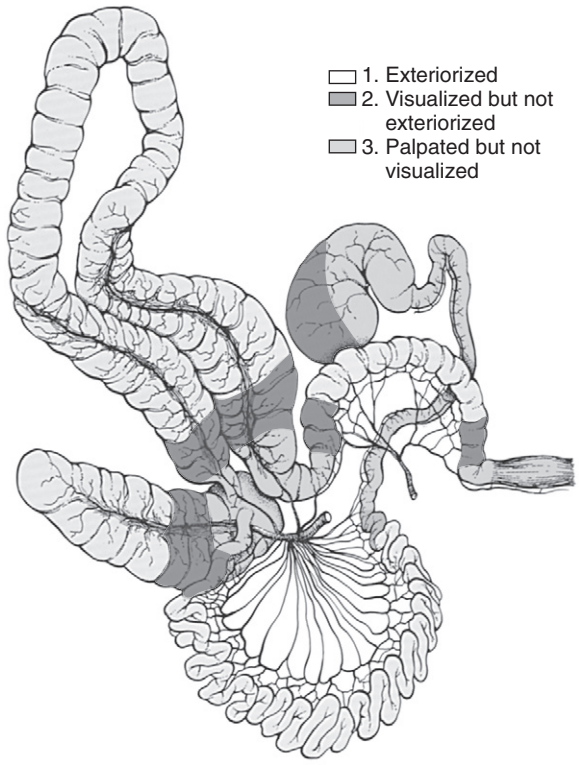

Figure 34-2. Anatomic drawing of the equine intestinal tract. Differential shading indicates the portions that may be *(1)* exteriorized, *(2)* visualized and palpated but not exteriorized, and *(3)* palpated only via a standard ventral midline approach. (Redrawn from Sack WO: Guide to the Dissection of the Horse. Edwards Brothers, Ann Arbor, MI, 1977.)

avoiding penetration of the rectus abdominus muscle. The latter structure can easily be palpated as a local thickening at the internal side of the ventral abdominal wall. Incisional hemorrhage is only encountered in the skin and the subcutaneous tissues.

Although there are different suture patterns that can be used to close the celiotomy, a continuous suture pattern with loops positioned 1.2 to 1.5 cm from the incisional edge of the linea alba provides the most strength.[1,2] Likewise, various suture materials can be used for incisional closure, but in adult horses I prefer polydioxanone (USP size No. 7, metric size No. 9) or polyglactin 910 (USP size No. 6, metric size No. 8).[3] To increase the bursting strength in heavy horses or pregnant mares, two to four single cruciate sutures can be placed 2 to 3 cm from the incisional edge after the continuous suture has been placed. In my experience a full abdominal bandage with an adhesive bandage applied before recovery decreases the strain on the incisional closure and covers the sutures during recovery phase. This reduces the risk of postoperative infections and incisional hernias (personal experience).

VENTRAL PARAMEDIAN APPROACH

The ventral paramedian incision, the second most common approach used in colic surgery, is performed 8 to 12 cm lateral to the midline (see Figure 34-1). Some surgeons use this approach for cystotomy, cesarean sections, ovariectomy, cryptorchidectomy, and repair of ruptured bladders in foals.[4,5] The incision may be performed on either side of the midline through the rectus abdominis muscle. Surgical exposure of the abdominal cavity is not significantly reduced, but the border of the incision is thicker than when it is made in the linea alba. Care must be taken not to injure the superficial and deep epigastric vessels, when encountered. With this approach, hemorrhage is more extensive than with the linea alba incision, but that does not compromise wound healing. The main indication for a ventral paramedian incision is to avoid a previous linea alba incision if there are signs of infection, excessive inflammation, or adhesions.

Closure of the paramedian incision involves suturing the facia of the rectus abdominis sheath. Suturing the muscle does not appear to contribute to the strength of the closure. For bladder surgeries in adult male horses I prefer a combination of a ventral midline and ventral paramedian approach (see Figure 34-1). The skin incision starts caudad just lateral to the prepuce and continues in a slightly curved line around the prepuce to join the midline and continues along that plane craniad. The prepuce is undermined along the fascial plane and reflected to extend the ventral midline incision through the linea alba caudad as needed.

INGUINAL APPROACH

The inguinal approach (see Figure 34-1) is used in conjunction with a ventral midline incision when performing surgeries on stallions with inguinal or scrotal hernias. The inguinal approach usually does not allow a thorough exploration and decompression of the prestenotic and poststenotic bowel. The inguinal herniorrhaphy is often combined with unilateral castration and closure of the external inguinal ring with USB 2 or 3 (Metric 5 or 6) absorbable suture material in a simple-continuous or simple-interrupted pattern with sutures placed 1.5 cm apart. An

abdominal testis can also be removed by a lengthened inguinal incision. Generally, after skin incision over the superficial inguinal ring, blunt dissection through the inguinal soft tissue is performed to prevent damage to the large veins around the inguinal ring. At this point, the gubernaculum can be used to retrieve the testis from the abdomen. If this is not successful, the dissection is carried down to the annulus vaginalis, which is perforated with a finger to enter the abdominal cavity. After bluntly dilating the peritoneal opening, the whole hand can be inserted into the abdomen. Closure of this approach is completed by suturing the superficial inguinal ring described in Chapter 59, followed by suturing two to three inguinal fascia layers and an intradermal skin suture pattern.

FLANK APPROACH

Nowadays, the indications for a flank approach are rare. A longer incision in the flank may be necessary to remove normal or neoplastic ovaries, with or without combined laparoscopy. A standing flank approach can also be used for surgical correction of uterine torsions.[6] With the exception of surgeries of the small colon, intestinal problems cannot be solved through a flank approach.

The grid technique is most commonly used. After aseptic preparation and local infiltration of the skin and muscle layers of the flank region the skin incision is centered between the tuber coxae and last rib, just proximal to the palpable dorsal edge of the internal abdominal oblique muscle. The external abdominal oblique muscle is subsequently sharply divided vertically, whereas the internal abdominal oblique and transverse abdominal muscles are bluntly divided parallel to their fiber directions, usually with just the surgeon's hand. The peritoneum is perforated by a short thrust with the fingers. Closure of the incision is performed by apposition of the different muscle layers with absorbable sutures and stapling or suturing the skin.

OTHER OPEN APPROACHES TO THE ABDOMEN

The parainguinal or suprapubic paramedian approach (see Figure 34-1) can be used for cryptorchidectomy (for more details see Chapter 59). A caudal ventral midline approach, performed in the same plane as the ventral midline incision but beginning 10 cm caudal to the umbilicus is the most commonly used approach for cesarean section (see more details in Chapter 62).[7] The modified low flank approach (Marcenac) (Figure 34-3) is performed under general anesthesia with the horse positioned in lateral recumbency. The incision is carried out in the ventral flank region, preferably in an oblique craniodorsal to caudoventral direction. The muscle layers are separated bluntly to reduce hemorrhage.[8] For nephrectomy or surgeries at the base of the cecum, a transcostal approach with removal of the 16th or the 17th rib or an intercostal approach at the 15th or 16th intercostal space (see Figure 34-3) is necessary (for more details see Chapter 64).

LAPAROSCOPIC APPROACHES TO THE ABDOMEN

Laparoscopic surgical procedures are distinguished between those performed in dorsal recumbency under general anesthesia and those conducted on standing sedated horses.

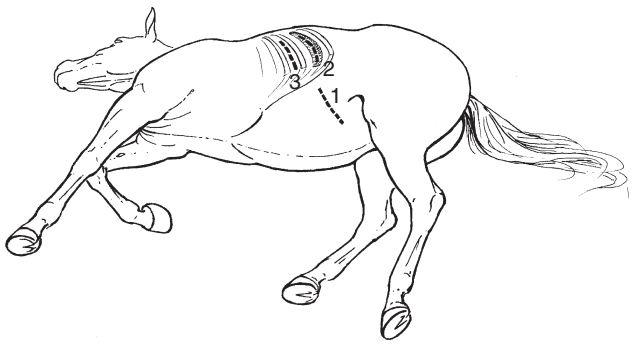

Figure 34-3. Abdominal approaches in lateral recumbency: modified low flank (Marcenac) *(1)*, transcostal *(2)*, intercostal *(3)*.

Laparoscopic Approaches to the Abdomen in Standing Horses

To safely perform a standing laparoscopic surgery in a horse, an optimal protocol for sedation is needed. My preference is to sedate the patient with a bolus of 0.005 mg/kg (0.002 to 0.01 mg/kg)[9] IV detomidine hydrocloride (Dormosedan) and 0.1 mg/kg (0.02 mg/kg) IV butorphanol tartrate (Torbugesic).[9] This is followed by a continuous infusion of 12 mg detomidine in 250 mL 0.9% saline administered at a rate of 2 drops per second. The infusion rate is adapted to the depth of sedation. The horse is placed in stocks with its head supported either with a padded stand or by a well-padded special halter that is tied to a solid contraption above its head (see Figure 30-2). Also, it is prudent to tie the tail to a crossbar of the stocks. Before starting surgery, all the feces accumulated in the rectum are manually removed and the positions of abdominal organs are checked to ensure that none are in direct contact with the abdominal wall of the flank region. After aseptic preparation of the whole flank region, each of the planed insertion sites for the trocars is locally desensitized with 10 to 15 mL of 2% mepivacaine, 2% lidocaine, or carbocaine. Linear cutaneous infiltration (line blocks) or local anesthesia also can be used for standing laparoscopic procedures in the paralumbar fossa. However, an epidural analgesia technique (combination of xylazine at 0.17 mg/kg and lidocaine at 0.2 mg/kg)[10] only partially anesthetizes the paralumbar fossa and has the potential risk of inducing ataxia and falls.

It is beneficial to place the initial laparoscopic portal in the left flank without prior insufflation, because presence of the base of the cecum on the right side presents an increased risk for injury to the viscera. A 1.5-cm incision is made through the skin and the superficial fascia at the level of the proximal border of the internal abdominal oblique muscle, equidistant between the last rib and the tuber coxae (Figure 34-4).[9,11,12] The laparoscopic cannula with a blunt obturator is carefully inserted horizontally and in a slightly caudad to distad direction through the skin and muscle layers. A mild resistance is felt when reaching the peritoneum, which is perforated with a quick, short thrust. After entering the abdominal cavity, a rush of room air will enter the trocar because of the negative pressure normally found within the abdominal cavity. To reduce the risk of inadvertent traumatization of intra-abdominal organs during insertion of the first cannula, an initial pneumoperitoneum can be induced by insufflation via a Veress cannula. An open approach or the use of an Endopath Optiview surgical trocar are other

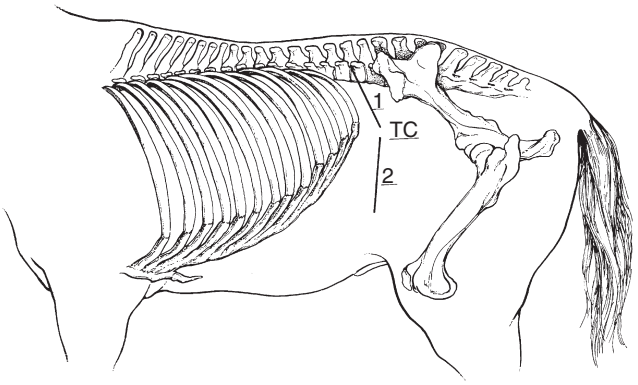

Figure 34-4. Portals for standing laparoscopic procedures: standard laparoscopic portal *(1),* two instrumental portals *(2* and *3),* and optional laparoscopic portal for closure of the nephrosplenic space *(4).*

options. For additional information on this subject (please review Chapter 13).

Secondary portals are always established under direct visualization. The vessels of the abdominal wall are protected by fat and can usually be avoided when inserting the trochar for the instrument portal. Standard instrument portals are created 3 to 10 cm dorsal and ventral to the initial portal containing the laparoscope to provide a triangulation approach.[9] Modifications of the instrumental portals are described for laparoscopic ovariectomy.[11,12] For closure of the nephrosplenic space, the portal for the laparoscope is selected between the 17th and 18th ribs. The two instrument portals are located caudal to the 18th rib.[13]

Laparoscopic Approaches to the Abdomen in Recumbent Horses

Under general anesthesia, the horse should be securely supported on the table in the Trendelenburg position (see Figure 13-16). Mechanical ventilation is mandatory in horses anesthetized for laparoscopy. Positive inspiratory pressure ventilation is used to assure adequate ventilation despite the increased pressure exerted upon the diaphragm by the cranially shifted intestines. Also, higher peak inspiratory pressures (25 to 35 cm H_2O) are often necessary to maintain appropriate ventilation and oxygenation.[9] Placement of a urinary catheter facilitates decompression of the bladder.

Generally, a 1.5-cm incision is created in the midline at the level of the umbilicus. The abdomen is insufflated with a teat cannula to a pressure of 15 to 20 mm Hg. The abdomen should be well insufflated before inserting the sharp trocar in the umbilical region. In foals, the teat cannula and the portal for the endoscope is placed about 2 cm lateral of the umbilicus to avoid penetration of umbilical structures. For surgeries in the region of the testis (cryptorchidectomy) and the inguinal region (herniorrhaphy) two instrument portals are created 10 cm lateral to the midline and 10 to 15 cm cranial to the external inguinal ring. For ovariectomy and removal of granulosa cell tumors, working with four instrument portals is beneficial. The first instrument portal is created midway between the umbilicus and the mammary gland, about 10 cm lateral to the midline. A second portal is established between the first instrument portal and the mammary gland. If the instrument portals are positioned at the right locations, identical portals are created on the opposite side of the midline. A very useful technique for removing uroliths is a laparoscopic-assisted cystotomy with the scope at the umbilicus and the instrument portal 2 to 3 cm medial to the left external inguinal ring, followed by enlarging the instrument portal to exteriorize the apex of the bladder.[14] Instrument portals cranial to the umbilicus are described for a laparoscopic colopexy.[15]

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CHAPTER 35

Principles of Intestinal Injury and Determination of Intestinal Viability

Anthony T. Blikslager and John F. Marshall

Intestinal injury, although typically associated with ischemic lesions, occurs during any obstructive intestinal disease to varying degrees depending on the type of obstruction and the extent of vascular compromise. In addition, in some instances the intestinal lumen is patent, but there is vascular compromise—for example, in nonstrangulating infarctions. Intestinal obstructive lesions are classified as either simple or strangulating obstructions. A great deal of work has been done to assess the level of injury encountered with these lesions at surgery and during the postsurgical phase following correction and reperfusion of these lesions. Although more is known about mucosal injury than about injury encountered in other intestinal layers, it is clear that substantial injury also occurs at the levels of the serosa and the muscularis, which very likely contributes to postoperative complications such as adhesion formation and ileus. Understanding the pathophysiology of these lesions allows the surgeon to more adequately perform surgical and postoperative procedures to optimize patient survival. For example, attention has been focused on the development of intestinal injury during reperfusion and the possibility of inhibiting these lesions with various treatments. Furthermore, to combat the postoperative complication of serosal inflammation and secondary adhesion formation, surgeons have adopted a number of new treatments.

TYPES OF OBSTRUCTION

Simple Obstruction

Although simple obstruction, most commonly caused by an intraluminal impaction, does not initially result in intestinal injury, studies show that as distention progresses, mucosal lesions similar to those encountered during ischemic injury occur.[1,2] More specifically, there are critical intraluminal pressure levels beyond which intramural vascular compression takes place, reducing tissue perfusion.[3,4] Furthermore, direct compression of the intestinal wall by obstructive masses may lead to a lesion that has the appearance of an infarction as a result of occlusion of the local arterial supply. Causes of these compressive lesions tend to be hard masses such as enteroliths or fecaliths, which, if not recognized during early lesion formation, may result in intestinal rupture.[5] Injury caused by progressive intestinal distention, particularly in the case of fluid accumulation proximal to a small intestinal obstruction, is more difficult to recognize clinically than compressive infarctions, but nonetheless it is important to be aware of it to minimize postsurgical morbidity.

Strangulating Obstruction

Strangulating obstruction results from simultaneous occlusion of the intestinal lumen and its blood supply. The clearest examples of such lesions include large colon volvulus, and strangulation of the small intestine within an internal hernia or via an abnormal band of tissue such as the stalk of a pedunculated lipoma. The degree of injury attributable to occlusion of the blood supply depends on the nature of vascular occlusion. In most instances, the veins are occluded earlier in the course of strangulating obstruction than the arteries because of their thinner and more compliant walls. This results in a disparity in blood flow during the early phases of strangulating obstruction, with continued pumping of arterial blood into the intestinal wall, which, in the absence of patent outflow, causes a hemorrhagic lesion termed *hemorrhagic strangulating obstruction*. This results in not only ischemic injury but also tremendous congestion of the tissues. Alternatively, the strangulation may exert sufficient pressure on the veins and arterial supply that both are occluded simultaneously, resulting in so-called ischemic strangulating obstruction. This results in rapid degeneration of the mucosa.[6]

Clinically, hemorrhagic or ischemic strangulating obstruction may be noted in horses with large colon volvulus. In horses with a 360-degree large colon volvulus, hemorrhagic strangulating obstruction is most frequently noted, particularly if there is ample ingesta within the lumen to reduce how tightly the intestine twists around its mesenteric axis. However, there are also cases of ischemic strangulating obstruction, particularly in horses with a volvulus that exceeds 360 degrees or in horses with little ingesta in the lumen.[7]

Nonstrangulating Infarction

Nonstrangulating infarction most commonly occurs secondary to cranial mesenteric arteritis caused by migration of *Strongylus vulgaris*, which has become a relatively rare disorder since the advent of broad-spectrum anthelmintics.[8] Although thromboemboli have been implicated in the pathogenesis of this disease, careful dissection of naturally occurring lesions has not revealed the presence of thrombi at the site of intestinal infarctions in most cases. These findings suggest that vasospasm plays an important role in this disease.[8] Any segment of intestine supplied by the cranial mesenteric artery or one of its major branches may be affected, but the distal small intestine and large colon are more commonly involved.[8] These lesions have no obvious cause of obstruction, but they have clearly demarcated regions of degeneration or necrosis, depending on the extent of vascular occlusion.

MECHANISMS OF INJURY

Luminal Distention

Investigators have examined the role of small intestinal intraluminal distention and decompression on the microvasculature. In one study, distention of the jejunum to an intraluminal hydrostatic pressure of 25 cm H_2O for 120 minutes resulted in a significant reduction in the number of perfused vessels in the seromuscular and mucosal layers, and vascular perfusion remained abnormal after decompression.[2] This in turn led to epithelial sloughing, similar to that noted in ischemic lesions.[9]

Earlier studies indicated that there was no histologic evidence of epithelial sloughing with pressures up to 18 cm H_2O for 4 hours, although the authors did note mucosal and submucosal edema.[3] Thus it is clear that the nature of mucosal lesions depends on the degree of intraluminal pressure. In experimental studies on the effects of distention, the seromuscular layer appeared to be more severely affected, and it had evidence of mesothelial cell loss, neutrophil infiltration, and edema that progressed after decompression, suggesting reperfusion injury in this tissue.[10]

This type of inflammatory seromuscular lesion has also been noted in studies of the proximal resection margins of naturally occurring small intestinal strangulating obstructions, indicating that distention leads to seromuscular injury proximal to obstructive lesions, despite the fact that this intestine may appear grossly normal.[11] Experimentally, this seromuscular injury and inflammation leads to adhesion formation. Thus foals subjected to intraluminal distention of a segment of jejunum to 25 cm H_2O for 2 hours in one study developed bowel-to-bowel and bowel-to-mesentery adhesions within 10 days of the surgical procedure.[12] Similar experiments in the large colon have revealed that the colon is far more resistant to seromuscular injury than the small intestine.[10]

Mucosal Ischemic Injury

To understand how the mucosa becomes injured during ischemia, critical anatomic features of the mucosa have to be considered. In the equine small intestine, the villus tip is the region most susceptible to ischemia, largely because of the countercurrent exchange mechanism of blood flow in the small intestinal villus.[13] This countercurrent exchange is attributable to the vascular architecture, which consists of a central arteriole that courses up the core of the villus, arborizes at the tip, and is drained by venules coursing down the periphery of the villus.[14] As oxygenated blood enters the central arteriole, oxygen diffuses across the wall of the arteriole, through the interstitial tissues, and into the peripheral venules, which are flowing in the opposite direction. Although this enhances intestinal absorption, it short-circuits the oxygen supply, resulting in a villus tip that is relatively hypoxic even under normal conditions. Countercurrent exchange is exacerbated when the rate of the arterial blood flow is reduced, essentially providing more time for the oxygen to diffuse out of the arteriole across to the venules. When the arterial flow is reduced to a critically low level, the tip of the villus becomes absolutely hypoxic, with attendant epithelial injury.[13,15] This mechanism may explain why the small intestinal mucosa is more susceptible to ischemic injury than the colon, which has no villi. For example, the time required to produce severe morphologic damage to the equine colon is approximately 25% longer than that for the small intestine.[16]

Mucosal epithelium is particularly susceptible to hypoxic injury because of the relatively high level of energy required to fuel the Na^+/K^+–ATPase mechanism that regulates epithelial ion and nutrient transport. The first biochemical event to occur during hypoxia is a loss of oxidative phosphorylation. The resulting diminished ATP concentration causes failure of the energy-dependent Na^+/K^+–ATPase mechanism, resulting in intracellular accumulation of sodium and water. The pH of the cytosol drops as lactic acid and inorganic phosphates accumulate from anaerobic glycolysis, which damages cell membranes and results in their detachment from the basement membrane.[17]

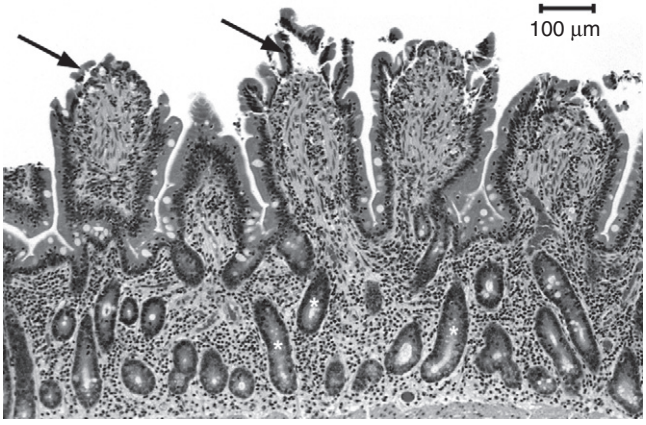

Figure 35-1. Photomicrograph of jejunal mucosa from a horse subjected to complete ischemia, similar to that seen in natural cases of ischemic strangulating obstruction. Note sloughing of epithelium into the lumen *(arrows)*. This sloughing progresses from the tips of the villi toward the crypts *(asterisks)* as the duration of ischemia progresses.

As epithelium separates from the underlying basement membrane in the small intestine, a fluid-filled space termed *Grüenhagen's space* forms at the tip of the villus.[15] The fluid accumulation exacerbates epithelial separation from the basement membrane. Subsequently, epithelium progressively sloughs from the tip of the villus (Figure 35-1) toward the crypts, which are the last component of the intestinal mucosa to become injured.[18-20] The relative resistance of the crypts to injury probably relates to their vascular architecture, since crypts receive a blood supply that is separate from the vasculature involved in the villus countercurrent exchange mechanism. The early morphologic changes observed in the equine large colon during ischemia are similar to those described in the equine small intestine, with initial loss of the more superficially located surface cells, followed by cellular injury and sloughing within the crypts.[16,21]

Reperfusion Injury

Events that culminate in reperfusion injury in the small intestine are initiated during ischemia when the enzyme xanthine dehydrogenase is converted to xanthine oxidase, and its substrate, hypoxanthine, accumulates as a result of ATP use.[22,23] However, there is little xanthine oxidase activity during ischemia, because oxygen is required as an electron acceptor. During reperfusion, xanthine oxidase rapidly degrades hypoxanthine in the presence of oxygen, which acquires a single additional electron, producing superoxide.[22] This reactive oxygen metabolite contributes to oxidative tissue damage, but it is relatively lipid insoluble, limiting the level of injury.

However, the more important role for superoxide is in the generation of neutrophil chemoattractants.[24,25] Superoxide interacts with lipid membranes, triggering arachidonic acid metabolism and generation of lipid neutrophilic chemoattractants such as leukotriene B_4. Because of the critical role of xanthine oxidase, inhibition of this enzyme in feline studies of intestinal ischemia and reperfusion injury prevented infiltration of neutrophils and subsequent mucosal injury.[25,26] Studies went on to show that reperfusion injury could be inhibited at several levels of the reperfusion cascade, including scavenging

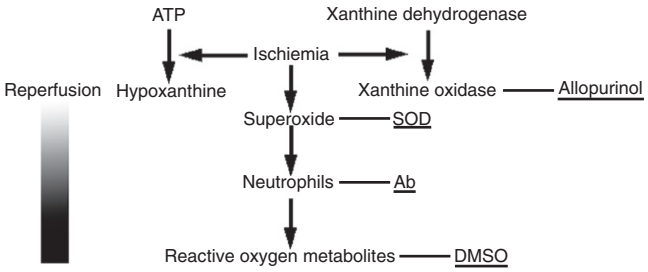

Figure 35-2. Diagram of the events that lead to reperfusion injury. Note that as reperfusion progresses *(shaded bar)*, the concentration of reactive oxygen metabolites amplifies as neutrophils are attracted to the site of injury. Points of experimental therapeutic intervention to block critical stages of reperfusion injury are *underlined*. *Ab,* Antibodies; *ATP,* adenosine triphosphate; *DMSO,* dimethyl sulfoxide; *SOD,* superoxide dismutase.

superoxide with superoxide dismutase, inhibiting neutrophil infiltration with monoclonal antibodies directed against neutrophil adhesion molecules, and scavenging neutrophil-released reactive oxygen metabolites.[22,25] Researchers were initially optimistic that this would provide practical therapeutic interventions, because many of the treatments tested, including the xanthine oxidase inhibitor allopurinol, could potentially be administered before reperfusing an ischemic lesion at surgery (Figure 35-2).

Unfortunately, treatment of mucosal reperfusion injury has not proved to be highly effective in most cases of strangulating obstruction. One important reason for this is that strangulating obstruction induces maximal mucosal injury during the ischemic phase, as compared with studies in laboratory animals in which "low-flow" ischemia was used.[27] The latter involves reduction but not cessation of arterial flow, which induces relatively minor levels of injury while priming tissues for injury during subsequent reperfusion.[28,29] Studies in horses using low-flow ischemia indicate that equine tissues are susceptible to reperfusion injury after this type of ischemia.[9,30-32] For example, studies of equine jejunum have shown that increased capillary permeability is associated with neutrophil infiltration during reperfusion.[30] Although a direct link to xanthine oxidase has not been shown, equine small intestine expresses substantial levels of xanthine dehydrogenase, which is converted to xanthine oxidase during ischemia.[33] Low-flow ischemia studies in equine colon have shown continued mucosal degeneration during reperfusion associated with marked neutrophil infiltration, despite the fact that equine colon does not express appreciable levels of xanthine oxidase. Alternative oxidant enzyme sources such as aldehyde oxidase have been offered as potential sources of reactive oxygen metabolites.[31]

Once the capacity of equine tissues to develop reperfusion injury was shown, additional studies were performed to determine whether reperfusion injury occurs after clinically applicable models of ischemia that simulate strangulating obstruction. For example, in one study assessing either arteriovenous or venous occlusion in equine jejunum, a small degree of reperfusion injury was documented after 3 hours of ischemia. However, the level of ischemic injury was nearly maximal after 3 hours, and the additional injury that developed during reperfusion was not sensitive to allopurinol, a xanthine oxidase inhibitor, or dimethyl sulfoxide, a reactive oxygen metabolite scavenger.[34] The likely cause of reperfusion injury in this instance was initiation of epithelial injury during ischemia that could not be reversed during reperfusion.[17] More recent studies in equine small intestine and studies in equine colon using ischemic models relevant to strangulating obstruction have failed to detect any level of reperfusion injury.[18,35] Studies in laboratory animals on strangulating obstruction have also shown that this type of ischemic insult is less likely to develop into reperfusion injury.[36,37]

In a veterinary review of the pathogenesis of intestinal reperfusion injury in the horse, the concept of a therapeutic window wherein treatment of reperfusion injury would be beneficial was suggested.[22] The basis of this concept is that there are certain conditions under which ischemic injury is minimal, and that tissues are severely damaged during reperfusion.[36] Thus, under conditions of low-flow ischemia, very little injury is demonstrated during 3 hours of ischemia, but remarkable injury occurs during 1 hour of reperfusion.[24-26] However, a very narrow therapeutic window may exist under conditions of strangulating obstruction, where severe injury occurs rapidly during ischemia, leaving relatively little potential for further injury that can be potentially offset with treatments such as antioxidants during reperfusion.

Nonetheless, this does not mean that treatments directed against reperfusion injury have no potential place for treatment of horses with strangulating obstruction, particularly as these treatments become more effective. For example, multimodal therapies involving intravascular or intraluminal infusion of solutions containing antioxidants, intestinal nutrients, and vasodilators have proven very effective *in vitro* and *in vivo*, although these treatment modalities have been used predominantly in low-flow ischemia models.[38-41] The recent study of lidocaine for the treatment of intestinal ischemic injury, rather than its more recognized use as a treatment for postoperative ileus, has has been of considerable interest based on its ability to improve the clinical outcome in colic patients.[42] Using a model of strangulating obstruction and recovery of the equine jejunum, the effect of a continuous rate infusion of lidocaine on the function of the mucosal barrier has been examined.[43] The administration of flunixin meglumine alone inhibits the recovery of the mucosal barrier following injury and increases permeability to lipopolysaccharide (LPS).[43] In contrast, when a continuous-rate infusion of lidocaine is combined with flunixin meglumine, this reduced barrier function and increased permeability to LPS is prevented. Furthermore, the combination of lidocaine with flunixin meglumine prevented the mucosal infiltration by neutrophils associated with flunixin meglumine treatment alone.[44] However, an *in vitro* study did not find an inhibitory effect of lidocaine on the migration or adhesion of equine neutrophils.[45] Therefore the anti-inflammatory mechanism of lidocaine in the horse is currently unclear.

It is also becoming apparent that the consequences of reperfusion injury are widespread and involve tissues other than the intestinal mucosa. For instance, low-flow ischemia models have demonstrated neutrophil infiltration into the seromuscular layers that very likely contributes to important complications such as adhesions and postoperative ileus.[12,46] This neutrophil infiltration appears to be relevant to strangulating obstruction, because similar neutrophil infiltrates were noted in the resection margins of resected strangulated small intestine.[11] Thus, although antioxidant-based therapies may be unable to reduce mucosal injury because of its rapid onset during ischemia, they may be able to reduce neutrophil infiltration in other layers of the intestine.

DETERMINATION OF INTESTINAL VIABILITY

Clinical Assessment of Bowel Viability

The viability of intestinal tissue is most commonly assessed by clinical observation—observing the color of the serosa and assessing the color of the mucosa, which may in some instances be observed before resection. For example, when a pelvic flexure enterotomy is performed in horses with large colon volvulus, the mucosa can be directly observed before making any decisions on recovering the horse or performing a resection. Changes in coloration are not uniform between the mucosa and the serosa, suggesting that much information can be gained by observing both of these layers of the intestine. For example, in one study on naturally occurring cases of large colon volvulus, the serosa was noted to turn from various shades of purple to pink in some instances as the colon was reperfused, whereas the mucosa consistently remained black. This probably relates to the metabolic demand of mucosa, which requires tremendous energy reserves to fuel the Na^+/K^+–ATPase mechanism that is responsible for driving mucosal transport function. Although there has been some concern about the utility of assessing mucosal color at the pelvic flexure and about whether this accurately reflects the level of injury in the more proximal regions of the colon, studies suggest that injury is uniform throughout the strangulated portion of the large colon.[47]

Clinical determination of viability has the obvious advantage of being noninvasive and rapid to perform, but it has the disadvantage of being inaccurate for fully determining the level of injury. For example, in a study assessing the resection margins after small intestinal resection, it was noted that there was marked serosal inflammation at the proximal resection margin, a region where surgeons would have had the choice to resect additional intestine if needed.[11] In other instances, when the resection margins are forced by anatomic considerations, surgeons can often discern substantial intestinal injury, but they have to decide whether the horse can survive if injured intestine is left in place. This is a particular problem with ileal resection and with large colon resection. Generally, if the intestine appears completely nonviable (on the basis of dark coloration, a dull appearance to the serosa, and an inability of the surgeon to rehydrate the surface of the intestine with lavage solutions), owners are advised that euthanasia is the most reasonable option.[7]

However, bowel that appears nonviable sometimes does survive. For example, in a study of ponies subjected to small intestinal venous strangulation obstruction, in which assessment of viability was based on color, wall thickness, peristalsis, and palpable mesenteric arterial pulsation, clinical assessment was only 54% accurate in predicting viability. In particular, clinicians were misled by intramural hemorrhage and edema, which resulted in the faulty judgment of nonviable tissue in some instances.[48] Ultimately, histopathology is the gold standard for determining viability, but when this service is unavailable because the situation is an emergency, any change in coloration during reperfusion or evidence of bleeding from the vasculature during resection may be used as evidence that there is some likelihood of survival. On the other hand, hemorrhage and edema within the tissues must be interpreted with caution, because some apparently devitalized bowel may survive.

Ancillary Methods of Determining Intestinal Viability

Fluorescein Dye

Fluorescein dye may be administered intravenously (6.6 to 15 mg/kg), after which it is rapidly distributed throughout the body, including the intestinal tract. Perfusion can be detected by illumination of the bowel with an ultraviolet light within 60 seconds of administration. Although there are distinct patterns of fluorescence in different segments of the intestinal tract, this technique has not gained wide clinical use, most likely because of inconsistencies of the technique in enhancing accuracy of determining viability. In particular, a complete lack of fluorescence may be noted in clinical cases of ischemic small intestine that has subsequently been documented to survive.[49] This may be caused by the degree of intestinal distention or by the compromised systemic cardiovascular status, both of which are common problems in horses with colic. One way to overcome this problem is to carefully compare a segment of suspected ischemic intestine with adjacent normal-appearing intestine. A generalized lack of fluorescence over the entire region would indicate that the technique should not be used to make definitive decisions in that particular case, whereas fluorescence in adjacent normal intestine would at least suggest that fluorescein is adequately perfusing the intestinal segment of interest.

In a study comparing the accuracy of fluorescein administration with clinical judgment and Doppler ultrasound in determining viability in strangulated small intestine, fluorescein administration uniformly predicted viability correctly in cases of ischemic strangulating obstruction, but it was very poor in predicting viability in cases of hemorrhagic strangulating obstruction. The latter may result from reduction in fluorescence as a result of the presence of hemorrhage and edema, leading to an overly pessimistic interpretation of viability.[48] As opposed to viability of the small intestine, large intestinal viability may be more adequately assessed using the fluorescein technique. However, great caution has to be exercised when interpreting the results. For example, a patchy pattern of fluorescence is not necessarily an indication of nonviability, but if the fluorescent patches constitute a minor portion of the colonic surface, nonviability is likely. On the other hand, a diffuse granular pattern of fluorescence usually indicates that tissue is viable.[49]

Surface Oximetry

An alternative technique for assessing viability is the measurement of intestinal serosal surface oximetry, which is an indirect measure of tissue oxygenation. As with other techniques for assessing viability, other circulatory factors have to be considered, including the overall cardiovascular status of the patient. A contact probe may be placed on the intestinal serosal surface, which measures surface oxygen partial pressure (PSO_2). Normal PSO_2 values for the jejunum, ileum, and pelvic flexure of healthy anesthetized horses are 71 ± 20 mm Hg, 61 ± 8 mm Hg, and 55 ± 13 mm Hg, respectively.[50] This information has been used to assess the viability of the colon after correction of large colon volvulus. Overall, the accuracy of surface oximetry in predicting colonic viability was 88%, using a set point of 20 mm Hg surface oxygen tension.

Additional analyses revealed that a horse with a PSO_2 value of 20 mm Hg or less was 7.4 times more likely to die than a

horse with a higher PSO_2 value. However, PSO_2 values have to be interpreted cautiously. In particular, the sensitivity of the technique was only 53% (calculated as the number of horses with an oximetry value of 20 mm Hg or less divided by the total number of horses that died), whereas the specificity was 100% (calculated as the number of horses that survived with an oximetry value of greater than 20 mm Hg divided by the total number of horses that survived). Thus the technique is very useful for detecting horses that will survive, but predicting nonsurvival in horses with low PSO_2 values is very inaccurate.[51] The other major disadvantage of this technique is that it measures surface oxygen tension in focal areas of application and cannot be used to indicate the viability of an entire region of intestine.[48]

Doppler Ultrasonography

Doppler ultrasonography has been used to discern viability on the basis of detection of flow through the microvasculature. A study described earlier (see "Fluorescein Dye") compared Doppler ultrasonography to use of clinical judgment and intravenous fluorescein.[48] Doppler ultrasonography was shown to be the most accurate technique for detecting viability in poststrangulated small intestine (accuracy values of 88%, 53%, and 53% for Doppler, clinical judgment, and fluorescein, respectively). The increased accuracy for Doppler ultrasonography was particularly evident in horses with experimental small intestinal hemorrhagic strangulating obstruction, in which viability was far more accurately determined with Doppler than other techniques. However, clinical judgment and fluorescein were slightly more accurate than Doppler for determining viability in experimental small intestinal ischemic strangulating obstruction.[48] In general, this lesion-dependent variability in diagnostic accuracy has resulted in limited clinical application of Doppler ultrasound and fluorescein for determination of intestinal viability.

Luminal Pressure

Increased intraluminal pressure is correlated with nonsurvival in horses with small intestinal obstruction and is probably related to the degree of mucosal injury generated by compression of the intramural blood supply. For example, in one study, survivors and nonsurvivors with small intestinal lesions had mean pressures of 6 cm H_2O and 15 cm H_2O, respectively.[4] Although 15 cm H_2O is lower than the level that induced epithelial sloughing in experimental studies, the duration of the pressure changes may be far greater in horses with naturally occurring lesions.[3] Thus a single pressure measurement may not be predictive of the severity of mucosal lesions without factoring in the duration of the disease. In horses with large colon volvulus, an increasing pressure is also correlated with nonsurvival.[52] Using a set value of 38 cm H_2O, survivors were separated from nonsurvivors (with a sensitivity and specificity of 89% and 91%, respectively), indicating that this test is highly accurate for predicting survival of horses with large colon volvulus.

Histopathology

The gross and histopathologic appearances of tissues were carefully evaluated in horses with naturally occurring large colon volvulus,[16] and a number of parameters described in this study were subsequently used to predict survival in horses with large colon volvulus. Biopsies were taken from the pelvic flexure (previously shown to accurately reflect mucosal changes along the length of the colon) and histologically examined.[47,53] The study used biopsies fixed in 10% neutral buffered formalin or frozen in liquid nitrogen. Although the latter technique resulted in some loss of tissue detail, the authors did not feel that this impaired the use of these biopsies for determination of survival. Furthermore, frozen tissues would be required if this technique were to be used intraoperatively. However, postoperative evaluation of formalin-fixed specimens may provide useful information on horses that have survived surgery but that are experiencing major problems in the postoperative period. Owners may elect euthanasia in some cases, if the lack of postoperative progress is reinforced by histopathology that suggests nonviability of bowel remaining in the horse.

Specific parameters recorded on histologic evaluation in these studies included the percentage loss of surface epithelium, the percentage loss of glandular epithelium, and the widths of the crypts and of the interstitial space between the crypts. The latter measurements were expressed as an interstitium-to-crypt width (I:C) ratio. Normal values for this ratio are 1 or less. Additional scored parameters included the degree of hemorrhage and the degree of edema (0, no edema or hemorrhage; 4, marked edema or hemorrhage), but these scores were not used in overall survival analyses. Colonic tissue was considered to be nonviable when the I:C ratio was greater than 3 and the loss of glandular epithelium was greater than 50%. Using these parameters, survival was correctly predicted in 94% of horses.[53]

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CHAPTER
36

Small Intestine

David E. Freeman

ANATOMY

The equine small intestine varies from 10 to 30 m in length, with an average length of approximately 25 m.[1] The duodenum, with an average length of 1 m, extends from the pylorus to the right of the midline, along the right dorsal abdomen, to a point caudal to the root of the mesentery. The duodenal ampulla and cranial flexure bend the first segment into the S-shaped sigmoid ansa. The major duodenal papilla (opening of the bile and pancreatic ducts) and the minor duodenal papilla (opening of the accessory pancreatic duct) are located in the cranial flexure. Caudal to the right kidney, the duodenum is attached to the

transverse colon by the duodenocolic fold. It forms the duodenojejunal flexure at its junction with the jejunum.

The jejunum measures 17 to 28 m in adult horses and is situated mainly in the left dorsal quadrant of the abdomen, between loops of small colon. The arterial supply from the cranial mesenteric artery is carried in arcades in the long mesojejunum, which allows the jejunum much mobility. Each vascular arcade is composed of a major jejunal vessel, an arcuate vessel that forms a loop with the next jejunal vessel, and several vasa recta that pass from the arcuate vessels to the intestinal wall. Veins run parallel to the arterial supply and enter the portal vein. The ileum is 0.7 m long, and its length is marked by the distinct ileocecal fold that attaches its antimesenteric side to the dorsal band of the cecum.[1] The arterial supply is provided by the ileocecal artery, a branch from the cranial mesenteric artery that travels along the ileum to anastomose with the jejunal arteries.[2]

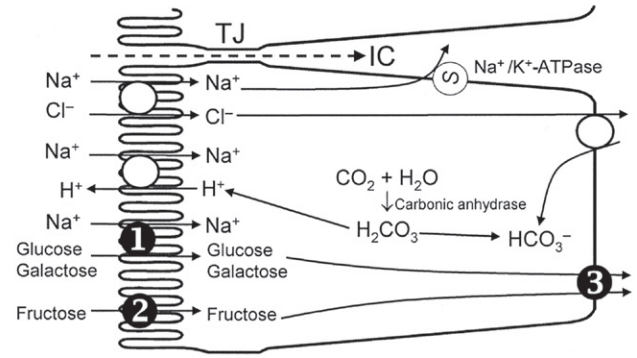
The terminal ileum forms a papilla that projects into the cecum. The ileal orifice is located in the center of the papilla, surrounded by the cecal musculature, an annular fold of mucous membrane, and a venous network.[2-4] The muscle of the ileocecal papilla is composed of three layers: an inner circular layer, a central longitudinal muscle layer from the ileum, and an outer layer formed from the circular muscle of the cecum and arranged into two semicircular lips.[3,4] The papilla lacks a true sphincter, although the lumen of the ileum appears reduced at the ileocecal junction.[5] The proposal that the venous network and annular fold contribute to the sphincter mechanism is rendered less tenable by the observation that the veins are most engorged when the ileum is discharging its contents into the cecum.[3,4] A functional sphincter does exist and appears to contract in synchrony with contractions of the cecal base.[5,6] Endoscopic studies of the cecal base of the horse have demonstrated that the ileal papilla is very prominent and that it becomes more prominent when the cecum is active.[4]

Digestive Anatomy

Digestion and absorption of nutrients take place predominantly in the upper half of the small intestine.[7] Unique to the small intestine in adults are fingerlike projections of the epithelial surface called villi, and each villus is surrounded by approximately six to nine crypts of Lieberkühn.[8] The equine small intestine lacks mucosal folds and/or plicae circulares.[9]

Columnar absorptive cells or enterocytes constitute about 90% of the small intestinal epithelial cells, and the remainder are mucous (goblet) cells, enteroendocrine cells, Paneth cells, and undifferentiated columnar cells.[10] After at least two divisions within the crypt, the columnar cells migrate onto the villus as mature absorptive cells, and they are finally extruded at the villous tip, usually with a turnover time of 2 to 3 days.[8,11] Cells in the upper third of the villi are capable of surface digestion of nutrients and active transport of digestion products.

The apical surface of a mature enterocyte is arranged into microvilli that form a brush border membrane. Enterocytes are connected to each other by tight junctions that restrict the transmucosal flux of large molecules, although they are permeable to water and many low-molecular-weight substances (Figure 36-1).[12,13] The intercellular space and the tight junctional complexes form the paracellular channel, and the intercellular space expands during water absorption.[13,14] Enzymes that complete digestion of carbohydrates (disaccharidases) and proteins (peptidases) as well as various specific transport systems for absorbing digestive end products are located within the microvilli (Figure 36-2).



Lumen	Cell interior	Blood
[Na+] = 150 mM	[Na+] = 15 mM	[Na+] = 150 mM
[Cl−] = 150 mM	[Cl−] = 50 mM	[Cl−] = 150 mM
0 mV	− 40 mV	+ 5 mV

Figure 36-1. Role of sodium in providing energy for different transport systems in a surface epithelial cell of the small intestine. Examples are cotransport with chloride and exchange with hydrogen generated within the cell, as well as cotransport with glucose by the SGLT1 transporter *(1)*. Fructose movement from the lumen into the cell by facilitated diffusion through the GLUT5 transport system *(2)* does not require sodium. All absorbed monosaccharides diffuse down their concentration gradient into plasma through a GLUT2 passive transport mechanism *(3)*. The *bottom panel* gives approximate concentrations of sodium and chloride and potential differences across cell membranes. This illustrates that sodium enters the cell down its electrical gradient (–40 mV) and chemical gradient (15 mM, interior concentration) but exits against its electrochemical gradient, hence the need for the sodium pump, or Na^+/K^+–ATPase. *Arrow with a broken line* designates diffusion through the paracellular route, composed of the tight junction *(TJ)* and intercellular space *(IC)*.

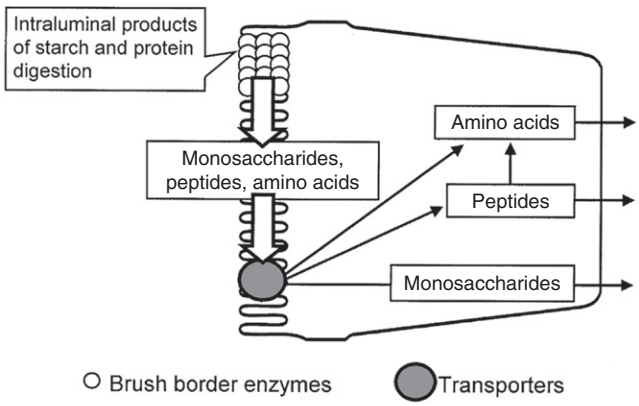


Figure 36-2. Role of brush border membrane in both digestion and absorption of products of intraluminal digestion of carbohydrates and proteins. Enzymes and transporters are simplified, but several oligosaccharidases, disaccharidases, and peptidases exist in the membrane, as well as highly selective carriers for their breakdown products. The largest peptides that can be transported into the cell are tripeptides.

PHYSIOLOGY

Intraluminal Digestion

Dietary carbohydrates, fats, and proteins are broken down by pancreatic enzymes in the small intestine, and their breakdown

products are hydrolyzed further by brush border enzymes.[8,15] Hydrogen ions in the duodenum cause the release of secretin from S-cells, which stimulates the pancreas and liver to secrete HCO_3^- and water.[11,16] Bicarbonate neutralizes H^+ ions in the proximal small intestine and thereby prevents acid-pepsin damage to duodenal mucosa, provides a functional pH for pancreatic and brush border enzymes, and increases solubility of bile acids and fatty acids.[7] Protein and fat in the duodenum stimulate the release of cholecystokinin (CCK) from I-cells, and CCK causes the pancreas to secrete enzymes for the digestion of carbohydrates (amylase), fat (lipase), and protein (trypsin, chymotrypsin, carboxypeptidase, and elastase), as well as cofactors (colipase) that aid in enzymatic digestion.[7,16] Trypsinogen, the precursor form (or zymogen) of trypsin, must reach the small intestine to be activated by the brush border enzyme enterokinase.[7] Other enzymes that are secreted as zymogens can be activated only by trypsin.

Resting pancreatic secretions in the horse are profuse, apparently continuous (10 to 12 L/100 kg body weight per day), and increase to very high rates by eating and by neural or hormonal stimulation.[17] Hormonal stimulation can be mediated through CCK and secretin, as in other animals, but the horse is unique in the marked secretory response elicited from the pancreas by gastrin.[17] The concentration of HCO_3^- is low, so that Cl^- remains the predominant inorganic anion in the horse's pancreatic secretion at all rates of flow.[17,18] Therefore the pancreatic secretion may provide a source of anions (Cl^-) for exchange with HCO_3^- in the terminal ileum to buffer products of fermentation in the cecum and colon.[17,18]

Compared with other animals, content and output of enzymes in pancreatic secretion is small in the horse,[17] and cellular concentration and specific activities of pancreatic enzymes are low, with the exception of lipase.[19] Nonetheless, nutrient digestion and absorption appear to be as efficient as in other species,[17,20] although horses are unique because they also ferment starches in the stomach and small intestine.[21] Also, starch digestibility in horses is largely related to its botanical origin, with almost complete digestion of starch from oats and wheat in the small intestine.[22]

In the liver, cholic acid and chenodeoxycholic acid combine with glycine and taurine to form conjugated bile salts.[23] Lipolysis in the small intestine requires emulsification of fat by bile salts.[7] Micelles form when the bile salts reach the critical micellar concentration (CMC) in the small intestine to aid in fat absorption (see "Fat Absorption," later). Approximately 94% of bile salts are reabsorbed by the small intestinal mucosa, pass to the liver, and are then resecreted, a process called *enterohepatic circulation of bile*.[7,23] Active transport by a Na^+-dependent process in the ileum and by passive absorption in the jejunum combine to reclaim intraluminal bile. Secondary bile salts are produced from bacterial deconjugation and dehydroxylation of bile salts that are not absorbed in the small intestine and enter the colon. These secondary bile salts are lost in feces and are replaced continually by liver cells.[7,23]

Absorption of Ions and Water

Most water absorption takes place in the distal third of the small intestine, but the bulk of intestinal water is absorbed by the large intestine.[18] However, Na^+ and water absorption in the small intestine is important in absorption of nutrients and other ions.

The routes for transepithelial movement of ions and water (see Figure 36-1) are through the cells (transcellular) and through the paracellular space (extracellular).[13,14] Transcellular movement of Na^+ involves entry from the lumen into the cell, down an electrochemical gradient. However, exit of Na^+ from the cell is against an electrochemical gradient and is therefore an active process that requires energy from the Na^+ pump (Na^+/K^+–ATPase), located along the basal and lateral membranes.[14,18] The active transport of Na^+ across the cell creates a transmucosal electrical potential difference.

The electrochemical gradient for Na^+ across the apical membrane has sufficient potential energy to move Cl^- into the cell against its electrical gradient. The cotransport of both ions is brought about by a carrier mechanism on the luminal membrane that is specific for Na^+ and Cl^- (see Figure 36-1). The net movement of Cl^- by this mechanism is called *secondary active transport*.[24] The Na^+ gradient also energizes uptake of hexoses (glucose), amino acids, and most B vitamins against their chemical gradients into the cell.[24]

Water transport is passive, closely coupled to solute movement, and is primarily paracellular (see Figure 36-1).[14] As absorbed Na^+ is pumped across the basolateral membrane, it creates an osmotic gradient that draws water into the intercellular space.[14,24] Water accumulation in this space increases the hydrostatic pressure, and this pressure forces the water across the basolateral membrane toward the capillary bed. Although the tight junction restricts backflow of absorbed water and electrolytes into the lumen, paracellular permeability and back-leak through this route in the jejunum is high. Therefore Starling forces have a considerable influence on ion and fluid transport in the proximal bowel, just as in the proximal tubule of the kidney.[13,18]

Net water movement from lumen to plasma through the paracellular route will "drag" permeant ions and low-molecular-weight substances with it (sugars, amino acids, Ca^{2+}, and Mg^{2+}), and this mechanism is called solvent drag, or convection.[13,18] Fluid absorbed by the epithelium moves into the central lacteal of the villus, from which it moves into the deeper lymphatics.[14] There is little proof that the intestinal villus has a countercurrent multiplier that could enhance water and solute absorption, at least in the dog.[25]

Carbohydrate Absorption

Several brush border oligosaccharidases and disaccharidases hydrolyze initial products of starch digestion by amylase to produce monosaccharides, such as D-glucose, D-fructose, and D-galactose (see Figure 36-2).[8] In horses on a grass-based diet, sucrase activity in the proximal small intestine is comparable to that reported in other nonruminants.[26] Maltase distribution is constant along the equine small intestine, and its activity is higher than in other species.[26] Lactase activity is higher in equine jejunum than in other parts of the small intestine, and it does decrease with maturity.[26]

Transport of D-glucose and D-galactose across the enterocyte membrane is through a high-affinity, low-capacity, Na^+-dependent cotransporter type 1 isoform, SGLT1 (see Figure 36-1).[18,26,27] The major site of D-glucose uptake by the SGLT1 transporter in grass-fed horses is the duodenum, followed by the jejunum and then the ileum.[26] Fructose moves from the lumen into the cell by facilitated diffusion through the GLUT5 transport system, which is a high-capacity, low-affinity, Na^+-independent transporter with highest activity in the duodenum in horses.[8,28] The monosaccharides diffuse down their intracellular concentration gradient in the absorptive cell to leave

through the basolateral GLUT2 passive transport mechanism and enter the portal venous system.[7,8]

Compared with a pasture forage diet, a dietary increase in soluble carbohydrate in the form of oats, corn, or barley can change glucose transport along the equine small intestine without affecting disaccharidase activities.[29] The change induced by hydrolyzable carbohydrate compared with pasture forage diet is evident as increased glucose transport rates and SGLT1 expression from approximately two times higher in the jejunum to three to five times higher in the ileum, but with no change in the duodenum.[29] A gradual switch in hydrolyzable carbohydrate in the diet from low to high has a similar effect on SGLT1 expression.[29] In all, these findings indicate that horses can adapt during a change to a high-carbohydrate diet in a manner that could reduce their risk of diet-induced colic, provided the dietary transition is gradual.[29]

Protein Absorption

Small neutral peptides yielded by pancreatic peptidases are broken down by brush border oligopeptidases (see Figure 36-2) into dipeptides and tripeptides or amino acids.[7,15] In the enterocyte, the peptides are hydrolyzed by cytoplasmic oligopeptidases into their constituent amino acids, which move passively into portal blood down a concentration gradient. Consequently, many dipeptides and tripeptides are transported into the cell, but mostly free amino acids enter portal circulation.[15] The intestine has several distinct amino acid transport systems, with varying degrees of specificity for amino acids and differences in Na^+ dependence.[15] The transport systems for small peptides are H^+ dependent,[15] although additional work is needed to confirm this in equine jejunum.[27] Circulating and intraluminal amino acids can also be used by the intestinal epithelial cells as energy sources.[11]

Fat Absorption

Long- and medium-chain triglycerides are split into constituent fatty acids and monoglycerides by pancreatic lipase interacting with colipase.[7] The resulting long-chain fatty acids and 2-monoglycerides, as well as fat-soluble vitamins and cholesterol, must combine with bile acids to form a water-soluble mixed micelle.[30] This micellar solubilization facilitates movement of lipids through the unstirred water layer to the brush border, where the micelles release their components for absorption.[23] Because of their fat solubility, the released long-chain fatty acids and 2-monoglycerides can traverse the membrane down a concentration gradient. Fatty acids and 2-monoglycerides in the mucosal cell undergo reesterification and formation of chylomicrons, and chylomicrons are taken up by the lymphatics.[23]

Fat digestion in horses is affected by compostion of the diet and concentration of dietary fat. It is lowest for forages followed by grains, and with a true digestibility approaching 100% in diets with added triglycerides.[31] Digestibility is low when dietary fat is low, which could have a negative effect on vitamin A and E status.[31] Increasing dietary fat slowly through supplementation can increase lipolysis of fat in the equine small intestine after accomodation is complete, without any negative associative effects on fiber digestion in the large intestine.[31] Adding fat increases its digestibility through added substrate concentration and through delayed gastric emptying and increased residence time in the small intestine.[31] These observations indicate that lipolytic activity in horses is upregulated in response to increasing dietary fat and that horses might require greater supplementation with fat-soluble vitamins, especially during growth and pregnancy.[31]

Absorption of Iron, Calcium, and Magnesium

Iron transport is closely regulated to meet the body's needs. Some absorbed iron combines with an intracellular protein, called *apoferritin*, to form an iron complex called *ferritin*.[12,24] If no binding protein is available, absorbed ferrous iron is transported out of the cell through transferrin receptors and is released to the circulation.[24,32] If iron stores are low in the body, crypt cells migrating to the villus tip are low in apoferritin but high in basolateral transferrin receptor.[32] As ferrous iron enters the cell from the lumen, it is not bound and is therefore free to enter the circulation to replenish iron stores. On the other hand, with a large iron store in the body, the crypt cell reaches the villus tip as a mature cell high in apoferritin. As ferrous iron enters the cell from the lumen, it is therefore bound and stored, and this bound iron is lost as the cell exfoliates at the end of its life span.[12,32]

Lumen-to-plasma flux of calcium is highest in the duodenum.[12,20] A high concentration of dietary magnesium can decrease calcium absorption through competition for the calcium transport site.[12] Absorbed calcium enters the cell down an electrochemical gradient and is transported through the cytoplasm complexed with a specific calcium-binding protein.[12] This protein is regulated by the biologically active form of vitamin D, 1,25-dihydroxycholecalciferol. Extrusion from the cell is against an electrochemical gradient through the Ca^{2+}-ATPase.[12]

Magnesium absorption is of interest because magnesium sulfate is used as a laxative in horses, and the extent of absorption could alter its laxative effects and the risk for magnesium toxicity.[33] Mean magnesium absorption is approximately 40% to 70% in growing foals and mature ponies, and the absorption occurs primarily in the proximal small intestine.[34]

Intestinal Secretion

Crypts of Lieberkühn are located over the mucosal surface of the small intestine and they secrete an almost pure extracellular fluid that maintains chyme in a fluid state, delivers secretory IgAs, and flushes crypts of noxious and infectious agents.[14] Intestinal water and electrolyte secretion is determined primarily by Cl^- secretion by crypt cells, with movement of Na^+ and water following. In equine jejunum, as in other species, chloride secretion can be mediated through cyclic adenosine monophosphate (cAMP) and a calcium-activated pathway.[27] Intestinal secretion is under a degree of neural and eicosanoid tone.[35]

Protective mucus is secreted by mucous cells located extensively over the surface of the intestinal mucosa, and by Brunner glands, which are located in the first 9.6 m of the equine small intestine.[9] The function of mucus from Brunner glands is to protect the duodenal wall from digestion by gastric secretions.

Motility

Small intestine has an outer longitudinal smooth muscle and an inner circular muscle layer, and the latter is divided into a thick outer lamella and a thin inner lamella.[36] Rhythmic segmentation in circular smooth muscle and synchronous shortening of the longitudinal muscle causes aborad transit during

peristalsis.[37] The interstitial cells of Cajal (ICC) are thought to act as pacemakers for the generation of electrical slow waves that propagate into the longitudinal muscle layer.[36,38] The equine small intestine has two types of ICC, one associated with the muscle layers and one with the myenteric plexus,[39] and the latter is probably responsible for pacemaker activity.[40] The density of myenteric ICC was found to be higher throughout the equine small intestine than in other parts of the intestinal tract and the density of ICC in the muscle layers was highest in the ileocecal junction.[39] The ICC distribution and density in the small intestine seems to have reached full development in the neonatal foal, whereas ICC development in the distal parts of the large intestine continues after birth.[40]

Two basic components of myoelectrical activity of gastrointestinal smooth muscle are the slow wave (basic electrical rhythm, or electrical control activity) and the action potential ("spike potential"). Slow waves are phasic oscillations of the smooth muscle resting membrane potential that are considered responsible for the rhythmicity and polarity of intestinal contractions.[38,41] In the pony, as in other animals, slow-wave frequency is greater in the duodenum and decreases toward the ileum.[41] This determines the direction and propagation rates of associated motility.[42]

An action potential is generated if something lowers the threshold of the smooth muscle cells, allowing a slow-wave oscillation to reach a threshold and cause a rapid depolarization that triggers intestinal contractions.[42] Mechanical (stretch), neural, and hormonal stimuli determine if that threshold will be reached. Action potentials do not necessarily occur with every slow wave, but slow-wave frequency determines the maximal frequency of action potentials.[42]

These wave forms produce a distinct pattern of myoelectrical activity called the *migrating motor complex (MMC)*.[42] Three phases of activity are recognized. Phase I, or no spiking activity (NSA), has slow waves only and no action potentials (Figure 36-3). Phase II, or intermittent or irregular spiking activity (ISA), has intermittent action potentials on some slow waves. Phase III, or regular spiking activity (RSA), is evident as an action potential for every slow wave and thus is associated with intense, sustained contractions.[42-44] The MMC is always present during an interdigestive state (time between meals when stomach and small intestines are essentially empty) and plays an important role in reducing bacterial colonization in the small intestine.[42] Phases II and III are associated with intestinal contractions, and most of an ingested meal is moved through the small intestine by phase II activity.

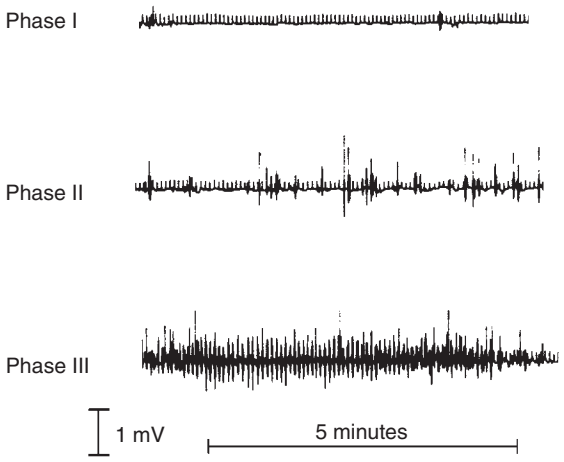


Figure 36-3. Myoelectrical activity of the duodenum of a clinically normal horse, demonstrating all components of the migrating motor complex (phases I through III). The slow wave frequency is about 14 per minute in all segments. (Modified from Merritt AM, Burrow JA, Hartless CS: Effect of xylazine, detomidine, and a combination of xylazine and butorphanol on equine duodenal motility. Am J Vet Res 59:619, 1998, with permission.)

Control of Motility

Control of motor events in the small intestine involves myogenic, neural, and hormonal mechanisms. Neural control involves the vagus nerve and components of the sympathetic nervous system, as well as an intrinsic system called the *enteric nervous system*.[38] The enteric nervous system consists of ganglia in the myenteric (Auerbach) plexus and the submucosal (Meissner) plexus, and it mediates reflex behavior independent of input from the brain or spinal cord.[38] Myenteric neurons are the main source of innervation of longitudinal muscle and the outer lamella of circular muscle.[36] Submucosal neurons innervate the inner lamella of circular muscle.[36] Hormonal control of motility involves neurotransmitters and other neural agents that act on intestinal smooth muscle. Motilin, a peptide produced by endocrine cells in the mucosa of the proximal small intestine, is released during fasting, and it regulates the interdigestive migrating motor complex.[45] A coordinated release of excitatory and inhibitory transmitters coordinates the peristaltic reflex responsible for propulsive activity.[36] Histochemical and immunohistochemical studies have identified patterns of cholinergic, nitrergic, and peptidergic innervation of the horse small intestine that correlate with functional observations.[46] The cholinergic subpopulation is larger than the nitrergic in several locations of the small intestine, and ganglia are more numerous in the small intestine than in the large.[47]

Motility of the Ileum

The migrating action potential complex (MAPC) can be recorded in the pony ileum as intense spike bursts of short duration that propagate rapidly in aboral direction, but they are absent in the pony jejunum.[43,48-51] In the ileum of mature ponies, all phases of the MMC can be recorded, but irregular spiking activity (phase II) predominates during the interdigestive period (the period between feeding).[48] Withholding food for 24 hours decreases the frequency of ileal MAPC, but it has no effect on mean slow-wave frequency, MMC phase distribution, MMC phase duration, MAPC conduction velocity, or conduction velocity of spike bursts in RSA.[48] Differences between the ileum and the jejunum in ponies might be related to the unique position of the ileum at the junction of the small and large intestines.

The MAPC is seen directly before retrograde cecal myoelectric activity 73% of the time, indicating a possible myoelectric coupling of the ileum and cecum, and it may be responsible for transit of digesta from the ileum to the cecum.[48] Also, ileal contents are discharged into the cecum at times when the cecum is inactive.[4] Ileal and cecal filling appear to be more important in regulating ileocecal motility events in ponies than stimuli associated with feeding.[48]

Local stimulation of chemoreceptors could be important in the regulation of ileal motility in the horse and humans.[5,6,52] Also, the human ileum, and possibly the equine ileum, act as reservoirs, discharging contents into the cecum in response to intraluminal stimuli.[6] At neutral pH, tonic activity of the human ileum, but not of the jejunum, is increased by intraluminal short-chain fatty acids, whereas bile acids and lipids can induce ileal relaxation.[6] In the pony, serotonin and luminal fatty acids increase ileal peristalsis.[5] The response to luminal acidification could stimulate ileal emptying of refluxed bacteria and cecal contents[6] and could thereby augment the sphincter function of the ileal papilla.[5,52] Although tone of the ileal papilla increases during contractions of the cecal base in horses, some cecal contents do reflux into the ileum.[5,52]

Sympathetic relaxation of ileal smooth muscle is mediated mainly by activation of atypical β-adrenergic receptor subtypes, and increased ileal sphincter tone is mediated through α_1-adrenergic receptors.[6,53] Whereas epinephrine decreases ileal and cecal activity, it increases ileal sphincter tone through α_1-adrenergic receptors.[5,52]

Altered Motility

Xylazine, atropine, meperidine, butorphanol, and pentazocine can increase the mean duration of the MMC in pony distal jejunum, whereas flunixin meglumine, dipyrone, metoclopramide, and panthenol have no effect.[43,51] Xylazine and butorphanol (as separate treatments) reset the duodenal MMC in the horse, but without serious disruption of proximal gastrointestinal tract motility.[54,55] However, routine doses of xylazine, detomidine, or a xylazine and butorphanol combination significantly reduce pressure peaks in the duodenum.[56] The marked, immediate decrease in amplitude of duodenal contractions following detomidine administration can last for 50 minutes.[57] In one study on conscious horses, acepromazine, butorphanol, or *N*-butylscopolammonium bromide did not affect duodenal motility.[58] Common anesthetic regimens decrease myoelectric activity in horse ileum for up to 150 minutes after induction.[44] Fasting decreases motility in the jejunum and large intestine, based on ultrasonographic examination, and renders the jejunum and cecum sensitive to the depressant effects of xylazine.[59] Drugs that can enhance small intestinal motility are discussed in Chapter 40.

After small intestinal resection and anastomosis in the dog, the aboral progression of the MMC stops at the anastomotic site, and alternate pacemakers initiate regular spiking activity distal to the anastomosis.[60] In the distal segment, the number of phases are reduced, disrupted, and delayed, and transit time is delayed also.[60,61] Therefore timing of the MMC appears to depend on bowel continuity and integrity of intrinsic innervation.[62]

Nonstrangulating obstruction of distal jejunum causes immediate continuous spiking activity proximal to the obstruction.[63] This response is not associated with fluid distention and does not cause signs of colic until accompanied by increased intraluminal pressures.[63] Occlusion of blood supply to the pony ileum decreases motility in the ischemic bowel, increases motility in the more proximal segment, and has no effect on the distal segment.[64] A low dose of endotoxin and intravenous infusion of prostaglandin E_2 alter regular spiking activity in pony jejunum, which suggests a role for endotoxin in postoperative ileus.[65,66] The effects of endotoxin can be partly blocked by pretreatment with phenylbutazone and flunixin meglumine. Increased sympathetic stimulation could decrease intestinal motility in horse jejunum and be relevant to development of ileus.[37] Nitric oxide also appears to act as an inhibitory neurotransmitter to circular smooth muscle of equine jejunum and could be released from invading macrophages in distended or inflamed small intestine.[67]

PATHOPHYSIOLOGY

Small Intestinal Distention

Intestinal obstruction causes prestenotic distention with gas, fluid, and ingesta that can compress intestinal veins.[68] Most of the resulting increase in venous pressure is transmitted to the capillary beds, and consequently capillary pressure increases.[69] The intestinal wall becomes thicker because of edema induced by increased capillary filtration.[69] The resulting filtration of fluid from capillaries into the interstitium is opposed by changes in Starling forces that attempt to maintain the tissues in a more dehydrated state.[69] As these compensatory mechanisms become overwhelmed, continued capillary filtration increases tissue edema and movement of fluid in large volumes into the intestinal lumen, producing a net secretion of fluid into the intestine.[70,71]

An experimentally induced intraluminal pressure of 25 cm H_2O (18.4 mm Hg) for 120 minutes in equine jejunum causes shortening of villi, loss of mesothelial cells, neutrophil infiltration, edema in the seromuscular layer, and a decreased number of perfused vessels in the seromuscular layer as well as in the mucosa.[72] Decompression of distended equine jejunum may not prevent progression of the morphologic lesions, but it permits partial recovery of vascular density.[72] Serosal edema and infiltration with erythrocytes and leukocytes increases significantly after distended equine jejunum is decompressed, and to a greater extent than in reperfused ischemic jejunum.[73] These serosal changes could contribute to complications in distended bowel proximal to an obstructive lesion, such as serosal adhesions.[72,73]

Small Intestinal Ischemia

Venous occlusion occurs rapidly when thin-walled veins are compressed by the edges of a hernial ring or by twisting of mesentery. The increased venous pressure causes edema and net secretion of fluid, as well as a myogenic response (the Bayliss effect) that protects the tissues from an increased vascular load.[69] This effect increases vascular resistance to arterial inflow, which combines with the decreased pressure gradient from artery to vein to reduce blood flow.[69] This response, coupled with arterial compression caused by displacement, eventually blocks arterial inflow and causes ischemia. Erythrocytes extravasate through altered capillaries and cause the characteristic intramural hemorrhage, which is known as hemorrhagic infarction.[74] Equine jejunum strangulated in a closed space develops tissue changes more slowly than in the open abdomen model and therefore might be more representative of a naturally acquired strangulation.[74-76]

Because ischemia reduces oxygen delivery to the metabolically active mucosa, morphologic changes develop that can be graded in severity from grade I (development of a subepithelial space, called Gruenhagen space, and slight epithelial lifting at the villus tip) to grade V (complete loss of the villus architecture, with severe mucosal hemorrhage and loss of the lamina propria).[77] The intermediate grades represent a progressive loss

of the epithelial layer, starting at the villus tip.[77] As a consequence of this mucosal damage, the important epithelial barrier is lost, and endotoxin can leak across to the peritoneum and be absorbed into the circulation, causing endotoxemia.[78]

In a pattern similar to that seen in other animals, the equine jejunal villus is supplied by an eccentrically placed arteriole that spirals to the villus tip and then "fountains" into a meshlike capillary network that descends along the periphery of the villus to drain into one to three venules.[79] This creates a countercurrent mechanism that allows capillaries near the villus base to shunt oxygen from the tip, rendering it more sensitive to hypoxia relative to the rest of the villus.[78] However, this does not appear to be the only or even the most important explanation for the sensitivity of villus cells to anoxia, because anoxic injury to equine jejunum *in vitro* causes the same progression of epithelial damage.[80] Most likely, cells on the villus tip are the first to succumb to anoxia because they are closer to the end of their life span.

Intestinal smooth muscle is more resistant to hypoxia than mucosa, and crypt cells are more resistant than villus cells—factors that can play a part in recovery from an ischemic insult.[76,77] In the early stages of mucosal repair, viable cells migrate from the crypts to cover the exposed villus stroma, whereas damaged cells are removed by detachment and phagocytosis.[81] This repair process is called *restitution*, and it explains how equine jejunum subjected to a grade IV injury becomes covered with stunted villi lined with cuboidal epithelium within 12 hours.[76,81] Normal architecture of the mucosal lining is restored rapidly from this point. Although the intestinal wall can recover from venous strangulating obstruction, mesentery subjected to hemorrhage and edema is prone to fibrosis, shortening, and adhesions.[74,76] Serosal edema and infiltration with erythrocytes and leukocytes increase significantly after ischemia and reperfusion in equine jejunum to an extent that could predispose to adhesions.[73]

Other factors that can contribute to the mucosal injury in ischemic intestine are those that alter capillary permeability, such as endotoxin, reactive oxygen metabolites, histamine, and activated neutrophils (for an in-depth discussion of intestinal injury, see Chapter 35).[78]

DIAGNOSIS OF SMALL INTESTINAL OBSTRUCTION

Preoperative prediction of a small intestinal versus large intestinal lesion is highly successful, but identification of the specific intestinal lesion is rarely possible, except for strangulation in an external hernia.[82] Clinical findings vary enormously, and small intestinal diseases that require surgery are difficult to distinguish from those that can zrespond to medical treatment, such as duodenitis–proximal jejunitis and ileal impaction. Although degree of pain, response to analgesic drugs, cardiovascular condition, abdominal ultrasonographic findings, and peritoneal fluid changes can be strongly predictive of the need for surgery, no single diagnostic test should be interpreted in isolation from others.

Horses with small intestinal strangulating obstruction usually exhibit signs of acute, severe abdominal pain and hypoperfusion, have an elevated heart rate and packed cell volume (PCV), and develop acidosis. The signs of distal obstructions develop more slowly, and the electrolyte imbalances and cardiovascular responses are less severe initially than with proximal lesions. Horses with proximal jejunitis may have severe abdominal pain initially; this progresses to depression and less pain than in horses with ileal impaction or strangulating obstruction.[83,84] After gastric decompression, horses with proximal enteritis usually improve in overall attitude and their heart rate decreases, whereas horses with mechanical obstructions do not improve.

The need for surgery should be considered more strongly in older horses with colic compared to younger mature horses.[85] Horses 16 years or older can have similar durations of colic and admitting heart rate, PCV, plasma creatinine, and blood lactate concentrations as younger mature horses.[86] However, they are more likely to demonstrate signs of moderate pain, are less likely to have normal borborygmi, and are less likely to be bright and alert compared with younger mature horses.[86] Also, peritoneal fluid total protein concentration can be higher in older horses.[86] Physical examination findings in geriatric horses are suggestive of a more serious disease compared with mature horses,[86] and this is consistent with the finding that they are 1.5 times more likely to need surgery and 3 times more likely to be euthanized if surgery is not an option.[85] On a percentage basis, horses at least 20 years old are 3 times more likely to have a primary small intestinal lesion compared with younger mature horses and a greater likelihood that the small intestinal lesion will be strangulating.[87] Strangulating lipomas are most likely responsible for these differences and should be high on the differential diagnosis list when examining an old horse with colic.[88]

Distended loops of small intestine on rectal palpation indicate small intestinal obstruction from any cause. In studies on horses with small intestinal strangulation, distended loops were palpated in 50% to 98% of horses on which a rectal examination was performed.[89-91] In some horses with small intestinal strangulation, a large colon abnormality can be found on palpation *per rectum* that may not be evident at surgery.[89,90] Many severely dehydrated horses with small intestinal diseases develop a "vacuum-packed" large colon, evident on rectal palpation as dehydrated colon contents contained within prominent colon haustra. These impactions should not be mistakenly interpreted as the primary cause of colic but might be considered as highly suggestive of small intestinal strangulation.[90] The degree of intestinal distention is very variable, with severe distention more likely to be associated with a strangulating lesion than with proximal enteritis. The amount of gastric reflux varies from none in the early stages of a lesion in the distal part of the small intestine, to several liters with a long-standing lesion or a lesion in the proximal part of the small intestine. Borborygmi are usually diminished or absent in all small intestinal diseases.

Abdominal distention is negligible to moderate in adult horses with small intestinal obstruction but can be pronounced in foals. Abdominal pain can be difficult to assess in foals with small intestinal strangulation because they can alternate between periods of violent colic and periods of depression. The latter can be interpreted erroneously as evidence of improvement. Inability to perform a rectal palpation in foals further complicates their examination, but ultrasonography is very informative in foals.

Peritoneal Fluid Analysis

Analysis of peritoneal fluid is important, but abdominocentesis is not necessary if other clinical findings indicate the need for surgery. Although the complications of enterocentesis are rare, peritonitis and abdominal wall cellulitis may develop after puncture of distended bowel.[92] The risk of enterocentesis is greater in foals than in adult horses, although ultrasonography

can identify fluid pockets to be sampled and also gives an impression of the volume and solid contents of the fluid.[93] Omentum can prolapse through the puncture site in a foal if a needle of 18 gauge or larger is used. For adults, an 18-gauge 3.75-cm (1½-inch) needle, a teat cannula (7.5 cm [3 inch]), or bitch catheter is inserted using aseptic technique on the midline in the most dependent part of the abdomen, behind the xiphoid cartilage.

The nucleated cell count in peritoneal fluid of normal adult horses ranges from 5000 to 10,000 cells/μL, but more than 1500 nucleated cells/μL should be considered elevated in foals.[93,94] Peritoneal fluid protein concentration is within the same range in foals as adults (0.3 to 1.8 g/dL).[93,94] Peritoneal fluid usually becomes serosanguineous with a strangulating lesion, but this is not a consistent finding, especially early in the disease. Accidental blood contamination does not appear to significantly affect nucleated cell count and total protein concentration in a peritoneal fluid sample.[95] Although strangulated bowel in an inguinal hernia or intussusception is less exposed to the peritoneal cavity than other intra-abdominal strangulating lesions, these diseases can produce serosanguineous fluid with elevated nucleated cell count and total protein.[96-99] Small intestinal rupture or perforation will produce changes in peritoneal fluid suggestive of digesta contamination, such as cloudy or green discoloration, increased protein and cell count, and intracellular bacteria; however, normal cell counts and protein can also be recorded and plant material may not be evident.[100]

Peritoneal fluid from horses with proximal enteritis is rarely serosanguineous, and nucleated cell count and total protein concentration increase to a lesser extent than with a strangulating obstruction.[84,101] Therefore peritoneal fluid is of some value in distinguishing between these diseases. Total protein can be elevated and eosinophils can be found in peritoneal fluid from horses with focal eosinophilic enteritis, a cause of small intestinal obstruction in horses.[102] Peritoneal and serum phosphate levels can be used to predict major intestinal injury, although the ranges in horses with surgical and nonsurgical colic can have considerable overlap.[103]

Factors that strongly correlate with the presence of a strangulating obstruction are changes in the gross appearance of the peritoneal fluid and values of peritoneal fluid chloride, pH, and $\log_{10}$ lactate.[104] Peritoneal fluid lactate is a better predictor of intestinal ischemia secondary to a strangulating obstruction than blood lactate and therefore could be used for early detection of small intestinal lesions.[104] Also, peritoneal fluid lactate is a more useful and sensitive prognostic indicator than plasma lactate in horses with colic.[105] A portable analyzer can be suitable for biochemical analysis of samples of blood and peritoneal fluid from horses with colic and provides lactate measurements comparable to an in-hospital analyzer.[106] Other potential peritoneal markers of severe intestinal damage are myeloperoxidase (MPO), which would be expected to indicate neutrophil activation,[107] and D-dimer concentration, a useful marker of the intense fibrinolytic activity that follows intestinal ischemia and peritonitis.[108] (For an in-depth discussion of diagnostic approaches to the horse with colic, see Chapter 33.)

Gastrointestinal Tract Imaging

Ultrasonography

Ultrasonography is invaluable for examining horses with small intestinal obstruction. Liberal application of gel or alcohol to the horse's hair coat are sufficient to improve acoustic coupling in most horses without clipping, but clipping of long hair coats can improve image quality.[109] Most abnormalities gravitate to the ventral abdomen, where they are accessible for ultrasonography.[109] A lower-frequency ultrasound transducer should be used (2 to 3.5 MHz) to penetrate deeper into the abdomen (approximately 24 cm or 9 inches), and higher-frequency transducers (5.0 or 7.5 MHz) can improve image detail in foals or superficial structures.[109]

The *jejunum* can be observed along the ventral and visceral surface of the spleen and dorsal to the left dorsal colon.[110] Time of feeding relative to the examination could explain inconsistencies in locating the jejunum, because it is seen more frequently in the ventral abdomen of fasted horses compared with fed horses.[59] Jejunal motility can be difficult to assess consistently because of depth and motion effects, but it can be decreased in the jejunum by fasting alone and combined with xylazine.[59] The jejunum should not be distended in a normal horse, although isolated hypomotile jejunal segments with hypoechoic contents can be found infrequently.[110] The clinical significance of static images of dilated loops of small intestine can be supported by an associated lack of motility.[111] An indwelling nasogastric tube causes minimal alterations in jejunal, cecal, or colonic visibility and activity.[59]

The *duodenum* can be found consistently in a relatively superficial location between the liver and the right dorsal colon, in the 14th to 15th intercostal space, on a line from the olecranon to the tuber coxae (dorsal part of middle third of the right side of the abdomen).[110-112] It can also be located ventral to the caudal pole of the right kidney at the 16th to 17th intercostal spaces.[112,113] Wall thickness in the duodenum, jejunum, cecum, and stomach is less in ponies than the published values for normal horses.[110] The mean duodenal wall thickness is greater in that segment deep to the liver than at the base of the cecum and the caudal pole of the kidney.[110] The mean duodenal contractility is greater for ponies than the published normal range for horses (0.5 to 2.8 contractions per minute).[110] Contractions and distentions of the duodenum are reduced by fasting.[113] Peritoneal fluid can be found ventrally and around the duodenum.[110] It should be anechoic and can be transient in location.[110]

The *stomach* can be found on the left side of the abdomen between the 11th and 13th intercostal spaces, at the level of the shoulder, dorsal to the splenic hilus and its large splenic vein.[109] It is normally gas-filled and amotile on ultrasound examination.[59] The greater curvature of the stomach is seen as a bright gas echo that curves toward the skin surface, deep to the spleen. When horses are fasted, the stomach can be found ventral to the costochondral junction, and placement of an indwelling nasogastric tube causes it to return to its normal location.[59] Xylazine does not alter the position or activity of the stomach in fed or fasted horses.[59] The stomach of normal ponies can be imaged further caudally and over a greater number of intercostal spaces than reported for normal horses, possibly because of a conformational difference.[110]

If ultrasonography is used after gastroscopy, the stomach wall becomes undulating and even folded on ultrasound examination as air leaves the stomach, and rugal folds may become evident.[112] Feeding increases stomach size compared with fasting and insufflation by gastroscopy, based on an increase in dorsoventral height and increased number of intercostal spaces that it spans.[112]

Small intestinal strangulating obstruction, small intestinal distention, intestinal wall thickening, peritonitis, peritoneal

effusion, gastrointestinal rupture, intussusception, inguinal hernia, diaphragmatic hernia, cholelithiasis, abdominal neoplasia, abdominal abscess, idiopathic muscular hypertrophy, intramural hematoma, intestinal lymphangiectasia with chylous peritoneal effusion, and ascarid impactions can be detected by ultrasonography.[93,96,114-119] An intramural hematoma can be recognized as a biconvex mass of anechoic loculated fluid in the intestinal wall with laminar flow in the center of the mass.[118] Transabdominal ultrasonographic findings of small intestine that is edematous (wall thickness greater than 3 mm), distended, and amotile strongly suggest strangulating obstruction (see Figures 33-1 and 33-2).[93,114] As obstructed small intestine fills with fluid, its contents become more hypoechoic. The five ultrasonographic layers of the intestinal wall are lost in strangulated segments.[120] Ultrasonography may be helpful in assessing gastric distention and in determining the efficacy of gastric decompression. Distention of the stomach can displace the spleen caudad or obscure it.[109]

Abdominal ultrasonography can provide a sensitivity of 98%, a specificity of 84%, a positive predictive value of 62%, and a negative predictive value of 99% for small intestinal strangulation obstruction from a variety of lesions.[114] Under the same conditions, palpation *per rectum* can provide a sensitivity of 50% for small intestinal strangulation obstruction, which means that it is considerably inferior to ultrasonography in finding a small intestinal abnormality when one does exist.[114] In horses with small intestine strangulated in the epiploic foramen, ultrasonography could accurately detect small intestinal distention in 94% to 100% of horses, compared with 74% to 78% for palpation *per rectum*.[90,121] With this lesion, distended or edematous small intestine is found more often in the ventral right paralumbar fossa, caudal ventral abdomen, and middle right paralumbar fossa.[114] For strangulating lipomas, similar intestinal changes are found more often in the caudal ventral abdomen, middle ventral abdomen, and ventral right paralumbar fossa than in other regions.[114] Ultrasonographic evidence of distended small intestine in the left cranial abdomen between the stomach and the spleen could be used to make a presumptive diagnosis of strangulation in the gastrosplenic ligament[122] (see Chapter 33).

Fast localized abdominal sonography of horses (FLASH) was developed to allow clinicians without extensive experience in ultrasonography to rapidly detect major intra-abdominal abnormalities that cause colic.[111] FLASH uses seven topographic locations that can be sufficient to determine the need for surgery and cover most of the windows where small intestine might be observed in colic patients.[111] These seven locations are the ventral abdomen, the gastric window, the splenorenal window, the left middle third of the abdomen, the duodenal window, the right middle third of the abdomen, and the cranial ventral thorax.[111] In agreement with previous studies,[114] the presence of dilated turgid small intestinal loops was highly sensitive and specific for small intestinal obstruction and had high positive and negative predictive values.[111] Ability to distinguish ileal impaction and proximal enteritis from surgical cases was difficult.[111]

Ultrasonographic examination of healthy ponies that had an exploratory celiotomy revealed hypocontractile segments of jejunum that had hypoechoic contents on postoperative day 1, consistent with mild functional ileus.[123] In general, there were minimal changes in bowel wall thickness, contractility, degree of distension, luminal contents, and peritoneal fluid in these ponies in the first few days after an exploratory celiotomy.[123]

Radiography

Abdominal radiography is useful in the foal with gastroduodenal obstruction, necrotizing enterocolitis, enteritis, impaction, displacement, intussusception, ruptured viscus, congenital anomalies, and inguinal hernia and in the adult horse and foal with diaphragmatic hernia.[93] In foals, radiographs can distinguish small intestinal distention from large intestinal distention with gas.[124] Contrast radiography has been used in foals to demonstrate abnormalities of gastrointestinal transit time and gastrointestinal obstruction (see Chapter 33).[125]

Laparoscopy

Although diagnostic laparoscopy has a sensitivity of 82% and a specificity of 66% for horses with acute abdominal pain, localized lesions or lesions inaccessible to the selected portals cannot be detected, and ileal lesions, such as ileocecal intussusception and ileal hypertrophy, can be missed.[126] Duodenal lesions can be seen in the standing horse.[126] Standing laparsocopic techniques have been used successfully for duodenal biopsies in horses and could be applied for evaluation of small intestinal diseases caused by inflammation, neoplasia, or grass sickness.[127,128] Care must be taken when inserting the trocar assembly into the abdomen in a horse with colic to avoid damage to distended viscera (for details on this technique, see Chapters 13 and 33).[93]

SURGICAL DISEASES OF THE SMALL INTESTINE

Small intestinal diseases can account for 25% to 64% of all colic cases treated at veterinary hospitals (Table 36-1), with a median of 34%.[90,129-136] The proportion was the same in foals as in adults.[135,137] The ileum was involved in 41% to 46% of all small intestinal obstructions.[134,136,138] The majority of small intestinal colics (58% to 85%) are caused by strangulating lesions and the remainder by nonstrangulating obstruction (simple and functional obstructions).[129,130,133,136,139] Rankings of disease prevalence can vary considerably among clinics (see Table 36-1).

Nonstrangulating Obstruction

Ileal Impaction

Impaction of the ileum is usually a primary condition in an apparently normal ileum, but it can occur secondary to other ileal diseases. The impaction forms a doughy to solid, tubular mass up to 90 cm long that extends proximally from the ileocecal junction.[83,140-143] The disease has a regional distribution in the United States, with most cases diagnosed in the Southeast, where the high prevalence may be related to feeding coastal Bermuda grass hay.[83] Ileal impaction also occurs in parts of the United States and in Europe where coastal Bermuda grass hay is not fed.[130,138] The disease appears to be more common in the United States from June through November, especially in the fall, although a seasonal effect is not consistent.[140,143,144] The risk for impaction is not consistently reduced by combining coastal Bermuda hay with other hay; it appears to be increased by recent introduction or feeding poor-quality hay,

TABLE 36-1. Prevalence of Small Intestinal Diseases that Cause Colic

Disease	% of Total Cases of Colic	% of All Cases of Small Intestinal Colic	Ileum Involved as % of All Ileal Obstructions	References
Volvulus	3.8-7.3	7-28	3	129, 130, 131, 133, 138, 178, 257, 392
Volvulus (foals)	15-19	39-50	—	124, 135
Epiploic foramen	2.1-8.4	5-23	15	88, 90, 91, 121, 129-131, 133, 138, 178, 233, 245, 392
Ileal impaction (United States)	6.5-7.4	2.2-23	—	132, 140, 143, 245
Ileal impaction (Europe)	3.7-11	5.5-17	12-42	129, 130, 133, 136, 138, 392
Proximal enteritis	1.5-7	3-22	—	130, 132, 133, 392
Lipoma	0.3-11	0.5-41	9	88, 129, 130, 131, 133, 138, 178, 245, 249, 251, 257, 392
Intussusception (all types)	0.8-5.2	1.5-29*	13	99, 130, 131, 133, 138, 178
Mesenteric rent	2.4-4.6	4.8-13.4	4	130, 131, 133, 138, 178, 245, 257
Mesenteric bands	1.1-4.6	4.4-13.4	13	129, 130, 132, 138
Inguinal/scrotal hernia	0.08-2.8	2.4-11	4-15	129, 130, 131, 136, 138, 178, 245, 257
Gastrosplenic ligament	1.2-2.5	1.1-7.3	—	130, 131, 245, 293
Thromboembolic disease	1.8-2.6	3.5-6.3	—	131, 133
Ileal muscular hypertrophy	0.14-2	0.6-5.5	12	129, 133, 138, 245
Intussusception (ileocecal)	1.9-3.2	3.3-5	12.5	99, 136, 245
Umbilical hernia	0.4-1.4	1.7-3.7	3	129-131, 138, 178

Diseases are recorded for all ages combined, but prevalence among foal diseases is also given, where available. Not all diseases were listed in all reports.
*This is an unusually high prevalence for this lesion among all small intestinal lesions, but it was the most common small intestinal lesion in this study.[137]

and it appears to be lowered by feeding a pelleted-concentrate feed in addition to hay.[144] The disease has been reported in a wide age range, including newborn and older foals.[135,140,141,145] In a large series of cases, mares and Arabian horses were significantly overrepresented.[142]

In the United Kingdom, where coastal Bermuda grass hay is not fed, ileal impaction was strongly associated with both coprologic and serologic evidence of tapeworm infection in horses.[146] This was supported by a study of 78 horses in North Carolina, which showed that feeding coastal Bermuda hay and failure to administer an anthelmintic effective against tapeworms placed horses at risk for ileal impaction.[144] Mucosal ulceration and submucosal edema caused by tapeworms at the ileocecal valve is proportional to the number of parasites present and could predispose to obstruction at that site.[146] Orbatid mites are the intermediate hosts for *Anoplocephala perfoliata* and their preference for humid regions (southeast) could also contribute to the geographic predisposition to ileal impaction in the United States.[146]

Early pain from ileal impaction is attributed to spasmodic contraction of the bowel around the impaction, but pain becomes more severe and constant as small intestinal distention progresses.[83] Small intestinal distention is generally a consistent rectal examination finding, and the impacted ileum is more easily palpated early in the disease course before small intestinal distention becomes severe.[83,140,141] Gastric reflux can be obtained in most horses but not all, depending on the duration of obstruction.[140,142,143] Distinction between ileal impactions, strangulating obstructions, and proximal enteritis cases can be difficult (see preceding). Small intestinal distention can be palpated *per rectum* in 87% of cases and and can be located on ultrasound examination in 99% of cases.[147]

Medical treatment is a viable alternative if surgery is excluded by a lack of finances and can be successful early in the disease, when the impaction can be felt *per rectum*.[83,141,147] Medical treatment consists of a balanced electrolyte solution administered intravenously at 5 L every 2 hours and flunixin meglumine at a rate of 0.5 mg/kg three or four times daily.[83] Mineral oil should be administered when gastric reflux has ceased. If surgery is required, the impaction is broken down by manual massage, aided by mixing it with fluid from the proximal bowel or from saline infusion.[143] Although dioctyl sodium sulfosuccinate can be included in the infusion, this is very irritating to mucosal surfaces. Excessive manipulation can cause serosal damage and subsequent adhesion formation. Therefore the intestinal surfaces should be generously lubricated with sterile physiologic saline solution or sodium carboxymethylcellulose while manipulations are being conducted. Enterotomy is recommended for very stubborn impactions, but jejunocecostomy is not recommended in the absence of a predisposing lesion or severe changes in the muscle wall.[83,142-144]

If treated early, the prognosis is favorable, although ileus, gastric rupture, laminitis, mucosal necrosis, and perforation of the ileum are complications when the diagnosis is delayed.[140,141,143] Compared with surgery, aggressive and successful medical treatment reduces hospital stay, cost of treatment, and expense of lost time for recuperation and hastens return to athletic activity.[147] Reimpactions are rare.[148] Because tapeworms may play a role in this disease, anthelmintics are recommended, such as pyrantel pamoate at 6.6 mg/kg to 13.2 mg/kg and dewormer combinations approved by the U.S. Food and Drug Administration, with praziquantel as the active ingredient against tapeworms.[144]

Muscular Hypertrophy of the Ileum

Muscular hypertrophy of the ileum can cause ileal impaction, but it is more likely to cause recurrent colic. Unlike the muscular hypertrophy that develops proximal to a chronic

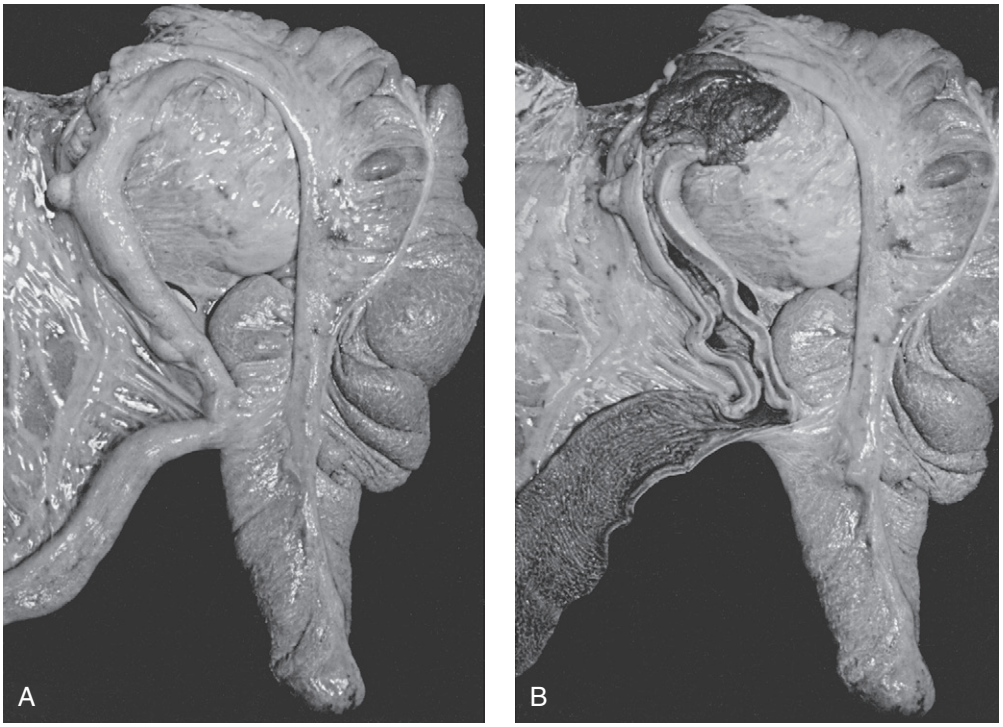


Figure 36-4. A, Ileal muscular hypertrophy treated 1 year previously by side-to-side incomplete bypass. Note the prominent diverticula and tubular appearance of the ileum. Death was from a nonrelated cause. **B,** The horse seen in **A,** with jejunum and ileum opened to demonstrate the thick wall of the ileum and the narrow lumen compared with normal bowel, as well as the diverticula.

obstruction, ileal hypertrophy causes marked luminal constriction (Figure 36-4). The condition is considered idiopathic.[149] Chronic mucosal inflammation can cause smooth muscle hypertrophy in rats, but mucosal inflammation may or may not be evident in ileal hypertrophy in horses.[150-152] Ileal hypertrophy and rupture were documented in a pony distal to an ileocecostomy that had been performed 3 years previously to bypass a short, reducible ileocecal intussusception.[153] At the time of the original surgery, that segment of ileum had been judged to be normal.

The affected segment is usually 1 m long, and both the circular and longitudinal layers are increased in thickness and diverticula are common (see Figure 35-4).[149,154] Full-thickness rupture can occur.[149,154,155] The terminal segment of the ileum and the ileocecal junction are spared in some cases.[117,134,149] The jejunum proximal to the obstruction can undergo compensatory hypertrophy in long-standing cases but with an increase in lumen diameter.[154] An unusual variant was reported in an American Miniature horse that involved multiple, separated segments of jejunum and proximal ileum, with the the ileal segment the least affected.[117]

The disease has been reported in horses over a wide age range, including foals, but it is most common in mature horses.[134,149,151,152,154,155] There does not appear to be a breed predilection.[154] Most horses are presented with a history of recurrent, low-grade colic of variable duration, up to 2.4 years in one case.[154] Partial anorexia and weight loss are common. The involved segment was palpated *per rectum* in 20% of horses in one study.[154] Abdominal ultrasonongraphy can also be helpful in diagnosing this disease based on finding severe annular thickening in the muscle wall and a collapse of the lumen in affected segments.[117] Clinical findings and history are similar to those of horses with chronic ileocecal intussusception, but most cases have been described in older horses (older than 5 years).[134,149,151,152,154]

Side-to-side ileocecostomy or jejunocecostomy with or without transection or removal of part of the ileum (incomplete bypass) is usually successful (see Figure 36-4).[134] The stoma is made proximal to the constricted segment, and if the space between the ileum and cecum is large, it is not closed.[134] *En bloc* resection of the affected bowel is required if there is extensive jejunal involvement.[117]

Ascarid Impactions

Intestinal stages of *Parascaris equorum* can cause small intestinal obstruction, intussusception, abscessation, and rupture in foals and weanlings (median age of 5 months, range of 4 to 24 months).[156] Because *P. equorum* induces absolute acquired immunity that eliminates patent infection by 1 year of age, ascarid impactions can be found in 2-year-olds.[157,158] Affected foals usually appear parasitized and unthrifty, and impaction usually (54% to 72%) follows shortly after anthelmintic treatment.[156,157] Ascarids in gastric reflux do not indicate ascarid impaction. Ultrasonographic examination can demonstrate ascarids in the small intestine.[115] The widespread resistance of *P. equorum* to ivermectin means that foals and young horses are at increased risk for this disease.[159,160]

Some ascarid impactions can be resolved at surgery by massaging the impaction into the cecum, with or without typhlotomy, and these cases seem to have the best prognosis, possibly because they have minimal small intestinal damage.[157,161] Enterotomy is required to evacuate the impaction in more severe cases (Figure 36-5), although resection is indicated if the bowel is devitalized[135,156] or there are concurrent lesions, such as volvulus or intussusception.[157] Not all the worms can be removed at surgery, regardless of method, and anthelmintic treatment is indicated after surgery.[161]

Postoperative mortality can be as high as 92%, with focal necrotizing enteritis, peritonitis, abscess formation, and

Figure 36-5. Removal of impacted ascarids through an enterotomy in the jejunum. Note that small intestine on the left is dimpled at sites where ascarids remain attached as they are drawn through the incision.

adhesions accounting for most deaths.[156,161] Reasons for such a poor prognosis after surgery include undetected ascarid-induced damage in the intestinal wall, release of toxins from the ascarids, release of antigenic proteins after organophosphate-induced rupture of the cuticle, preexisting debilitation, low-grade liver disease, low-grade pneumonia, and failure to remove all worms at surgery.[161] Although a recent study described an improved survival rate (64%) after surgery, prognosis for survival for longer than 1 year was guarded, with most deaths caused by adhesions.[157]

Prevention of ascarid impaction requires a sound deworming program based on current recommendations, not traditional practices.[158] Ascarid control should involve (1) using only those anthelmintics known to be effective against indigenous populations, (2) initiating anthelmintic treatment no sooner than 60 days of age, (3) repeating treatments at the longest intervals required to prevent serious pasture contamination with *Parascaris* eggs, and (4) recognizing that the passage of modest numbers of ascarid eggs by some foals is required to decrease selection pressure for anthelmintic resistance.[158] Between 14 and 21 days after treatment, fecal samples are collected and fecal egg counts are compared with pretreatment counts with the help of the Fecal Egg Count Reduction Test for the anthelmintic used, recognizing that correlation between egg counts and worm burdens has not been demonstrated for *P. equorum*.[158] In heavily parasitized foals at high risk for impaction, mineral oil can be given with an anthelmintic agent.[161] Ranking of anthelmintic drug activity on ascarids from highest to lowest in a 2007 study in central Kentucky was oxibendazole, fenbendazole, pyrantel pamoate (very poor), and ivermectin (no effect).[160]

Duodenitis–Proximal Jejunitis

Duodenitis–proximal jejunitis (DPJ), also called gastroduodenitis-jejunitis, hemorrhagic fibrinonecrotic duodenitis, proximal jejunitis, proximal enteritis, and anterior enteritis, involves inflammation of the proximal part of the small intestine, fluid accumulation in the stomach and small intestine, and endotoxemia. The cause is unknown. Severity of clinical signs varies over a wide range, and age, breed, and sex predispositions have not been established. Although usually diagnosed in horses older than 1½ years, it does occur in nursing foals.[84] The prevalence of the disease has been reported as 3% to 22% of all small intestinal colics.[129-132] Prevalence in California is lower than in other parts of the United States and Europe, and the disease appears to be more severe in the southeastern United States than in the Northeast.[84,101]

The clinical hallmark of the disease is nasogastric reflux of a large volume of fluid (usually greater than 48 L in the first 24 hours) in a horse that demonstrates signs of mild to severe pain initially and then depression.[162] Most horses with DPJ have tachycardia, prerenal azotemia, dehydration, hypotension, and electrolyte abnormalities. In contrast to horses with strangulating lesions, horses with DPJ are more likely to have a fever, leukocytosis, and a greater volume of gastric reflux.[84,101] Affected horses can have significantly higher serum liver enzyme activities than horses with small intestinal strangulating lesions.[163] They also have a trend toward higher prevalence of gastric ulceration compared to those with other GI lesions.[164]

After gastric decompression, a horse with DPJ usually demonstrates relief from pain and becomes quiet, and its heart rate decreases, whereas a horse with mechanical obstruction improves little if at all. On rectal examination, the degree of small intestinal distention with DPJ is subjectively less than with ileal impaction.[83] Peritoneal fluid from horses with DPJ is rarely serosanguineous, and nucleated cell count and total protein concentration increase to a lesser extent than with a strangulating obstruction.[83,84]

The most important goals in treatment of horses with DPJ are frequent gastric decompression, correction of disturbances in water and electrolyte homeostasis, and restoration of normal intestinal function. Horses must be kept off food and water until it is concluded that the condition has resolved, and attention should be paid to plasma concentrations of calcium and potassium, both of which can become depleted in horses with intestinal injury and decreased food intake. The rate of fluid administration and the types given should be largely determined by clinical and laboratory evidence of hydration status and electrolyte balance.

Flunixin meglumine can attenuate the hemodynamic responses to lipopolysaccharides (endotoxin) and reduce lipopolysaccharide-induced increases in plasma concentrations of thromboxane and prostaglandins.[165] Nonsteroidal anti-inflammatory drugs also could block the effects of lipopolysaccharides on intestinal motility that could exacerbate or sustain ileus in horses with DPJ. Antilipopolysaccharide antibodies in plasma products, such as J5 *Escherichia coli* or Re *Salmonella* mutants could be used; however, there is some controversy regarding efficacy.[165] The amphipathic cyclic polypeptide antimicrobial drug polymyxin B can avidly bind with the lipid A and thereby remove lipopolysaccharide from the circulation, although nephrotoxic side effects in dehydrated horses are valid concerns with the use of this drug.[165] Pentoxifylline, a methylxanthine derivative, can reduce lipopolysaccharide-induced production of cytokines and thromboxane in horses.[165]

Antibiotic use in DPJ is controversial, but horses in one study received metronidazole (2 g IV, followed by 1 g IV twice a day) and procaine penicillin (20,000 IU/kg IM twice a day), based on a high rate of positive cultures of gastrointestinal contents for *Clostridium perfringens*.[166] Treatment could include prokinetic drugs or lidocaine to improve gastric emptying and small intestinal motility (see paragraphs on postoperative ileus in Chapter 40). Prophylaxis against development of laminitis should be considered, including the preceding treatments, confinement in a sand stall or on deep wood shavings, application of frog support, and application of ice to the lower limbs and feet.

Surgery is performed in at least 6% of cases.[84,162] Although the prognosis is considered to be reduced by surgery in some areas in the United States, in two studies survival was similar regardless of whether horses underwent surgery.[162,167] In one report, 40 of 42 horses (95%) recovered rapidly and completely after surgical decompression, suggesting that a prompt recourse to surgery could be indicated for this disease.[166] This was not confirmed in another study, which demonstrated that survival was decreased and the risk of diarrhea was increased in surgical cases compared with medical cases.[168] Also, the longer period of inactivity necessitated by the abdominal incision and the risk for obstructive adhesions could be disadvantages of surgery. The major advantage of surgery is to use it as a diagnostic procedure when distinction between DPJ and a physical cause of obstruction is difficult.

Reported survival rates for DPJ range from 25%[101] to 94%, and recurrence is rare.[84,162,167] By univariate analysis, anion gap, abdominal fluid total protein concentration, and volume of gastric reflux in the first 24 hours were significantly associated with death.[162] Laminitis is a life-threatening complication of DPJ, and the risk is greater in horses with hemorrhagic reflux at the time of admission.[169] The considerable cost of prolonged medical treatment can necessitate euthanasia in protracted cases.

The lesions of DPJ are usually confined to the proximal half of the small intestine.[101] The serosa may appear normal or may be dark pink and edematous and have hemorrhagic patches. The mucosa can be normal, light red, dark red, hemorrhagic, and even necrotic and ulcerated.[101] Microscopic lesions range from hyperemia and edema of the mucosa and submucosa to some sloughing of villus epithelium; hemorrhage; and neutrophil infiltration from the lamina propria and submucosa to the tunica muscularis and serosa.[101,167] The liver is moderately to markedly congested in most horses, and histopathologic changes can include congestion, vacuolization, periportal fibrosis, bile duct hyperplasia, inflammatory cell infiltrates, and necrotizing hepatitis.[101,167] Liver damage could be caused by reflux of duodenal contents and ascending infection through the common bile duct, absorbed endotoxin or inflammatory mediators from the portal circulation, or hepatic hypoxia caused by systemic inflammation and endotoxemic shock.[163]

Neoplasia

Obstruction of the gastrointestinal tract by intestinal wall tumors is rare. Arabian horses are 4.5 times more likely to have a diagnosis of intestinal neoplasia than are other breeds, there is no sex predisposition, and the small intestine is the most common segment of intestine affected for all neoplasms.[170] Lymphosarcoma, lymphoma, adenocarcinoma, adenoma, leiomyosarcoma, leiomyoma, ganglioneuroma, and intestinal carcinoid have been reported to cause small intestinal obstruction in horses.[170-179] Horses with intestinal neoplasia can be presented with a single bout of acute colic or with repeated bouts of mild colic, weight loss, anorexia, and fever.[170] The most consistent clinical signs are poor body condition, tachycardia, tachypnea, fever, and diarrhea.[170] Antemortem diagnosis can be made on rectal examination, routine blood analyses, abdominocentesis, ultrasonographic examination, rectal biopsy, and exploratory celiotomy. A favorable outcome can follow surgical removal of the tumor if it is discrete, focal, and accessible.[171,173,174,176,179] Resection of an isolated lymphosarcoma lesion can be curative, but this is generally a multiorgan disease with a grave prognosis.[172]

Gastroduodenal Obstructions

Duodenal inflammation, ulcers, stenosis, and perforation can develop in foals, particularly those treated with phenylbutazone or subjected to stress, surgery, transportation, or illness.[180] Foals younger than 4 months are at greater risk, although duodenal obstruction has been reported in yearlings and in an adult horse.[125,180-182] The incidence of gastroduodenal obstructions has decreased because of increased awareness and earlier treatment with antiulcer medication.[125] Duodenal obstruction can cause reflux of duodenal contents into the bile and pancreatic ducts, inducing diffuse cholangiolitis, perilobar hepatitis, and pancreatitis, with acute erosive and fibrinous inflammation of the large ducts.[180]

Many affected foals are depressed, weak, and anorectic in the days or weeks preceding diagnosis. Clinical signs of duodenal ulcers and obstruction are teeth grinding (odontoprisis), salivation, gastric reflux, fever, diarrhea, and signs of colic (e.g., dorsal recumbency).[125] Abdominal pain may be worse after nursing. Rupture of duodenal ulcers causes signs of peritonitis.[180]

Diagnostic signs on plain radiographs include aspiration pneumonia, dilated fluid-filled esophagus, and gastric distention.[125] Gas may be evident in the hepatic duct.[125] Endoscopy is more sensitive and specific than radiography in diagnosing gastric and duodenal lesions, although ultrasonography could be used to demonstrate any abnormalities in duodenal peristalsis, wall thickness, and lumen size.[93,113,125,183]

Surgical bypass of the obstruction should be followed by medical treatment of gastric ulcers (see Chapter 32).[125,182] Contrast radiographs are recommended 24 hours after surgery to confirm successful bypass of the obstruction. Barium in dilated bile ducts after surgery is indicative of cholangitis or cholangiohepatitis.[125]

Intestinal Inflammation and Fibrosis

A number of diseases characterized by inflammation or fibrosis in the small intestinal wall have been identified as causes of colic in horses. Intestinal fibrosis has been reported as a cause of weight loss and colic in horses in a small geographic distribution in Colorado, but the disease appears to be more widespread and the signs more variable than originally reported.[184,185] Equine proliferative enteropathy from *Lawsonia intracellularis* infection in the small intestine causes weight loss, colic, hypoproteinemia, and diarrhea in weanling foals.[186,187] Although erythromycin alone or with rifampin is an effective treatment, resection or bypass of the affected segment may be required.[186,187]

Eosinophilic gastroenteritis is a well-known but poorly understood cause of colic, diarrhea, and weight loss in horses.[102] In one series of cases, weight loss was not a feature, but affected horses had colic from jejunal obstruction with a red intramural mass or circumferential plaque of fibroplasia and infiltration on the antimesenteric surface.[102] The seasonal distribution of the cases corresponded to the grazing period, suggesting a dietary association.[102] Treatment by complete or wedge resection was successful because of the focal nature of the lesions.[102]

A similar type of acute or chronic colic is characterized by small, focal to multifocal, well-demarcated areas of intestinal thickening and fibrosis that form constrictive mural bands in the jejunum and other parts of the intestinal tract.[188] These are free of mucosal ulceration and granulomatous response, but they have histologic evidence of lymphocytic, plasmacytic, and eosinophilic infiltration of all layers, and most lesions are associated with the vasculature.[188] A similar idiopathic focal eosinophilic enteritis (IFEE) has also been diagnosed in the United Kingdom and Ireland.[189,190] Affected horses can be treated by resection of the diseased segment[188,189] or by decompressing the affected small intestine into the cecum, without resection.[190] The decision to resect could be based on the severity of the constrictions,[190] because obstruction can recur at nonresected lesions after surgery.[189] Medical treatment with corticosteroids can also be effective.[188]

Pythiosis caused by *Pythium insidiosum* is a granulomatous disease, typically of skin and subcutis, that is most commonly diagnosed in the Gulf Coast States but also in the Midwest.[191] Jejunal obstruction caused by *P. insidiosum* granuloma can cause weight loss and signs of mild intermittent abdominal pain.[191] Reported cases have been amenable to resection, which can be successful.[191,192]

Miscellaneous Simple Obstructions

Duodenal obstruction in horses can cause severe pain, gastric reflux is voluminous, and some horses have spontaneous gastric reflux through the nostrils.[83,193] Impactions of the duodenum and jejunum are rare, and causes include impacted feed material, compressed cracked corn, trichophytobezoar, compacted wood fragments, persimmon fruit, molasses-based stable treats, baling twine, and cholelithes.[83,193-202] Consumption of ripe persimmon fruits by horses during fall and winter in the South cause intermittent impaction of the stomach, duodenum, and jejunum, and weight loss.[199,200] The ripe persimmons at that time are palatable to animals, and water-soluble tannins in the fruit form a solid phytobezoar when exposed to gastric acid.[199,200] Diagnosis can be confirmed by identifying persimmon seeds in the gastric impaction on gastroscopy.[199]

Small intestinal perforation by wire, a porcupine quill, ulcers, and unidentified objects may lead to diffuse or localized peritonitis, localized intestinal necrosis, and even occlusion of mesenteric vessels.[203-207] Localized lesions can be resected,[206,207] although the extent of adhesions and peritonitis can complicate access and prognosis in many cases.[205] Single discrete perforations, 1.5 cm or smaller, have been described in otherwise healthy jejunum in three young horses that were presented with fever, acute colic, depression, and hypovolemic shock.[100] No cause could be identified.[100] These perforations could be similar to idiopathic perforating ulcers that have been described on the jejunal mesenteric attachment with leakage into the jejunal mesentery.[208] Affected horses present with a history of low-grade colic, depression, and inappetance, and clincial and laboratory findings that support a diagnsosis of peritonitis.[208]

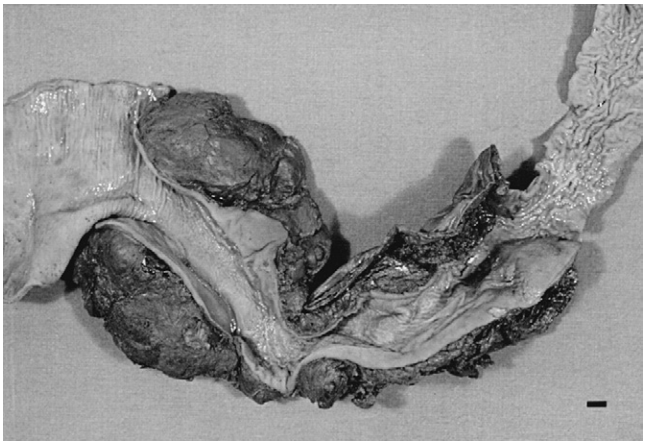


Figure 36-6. Intramural hematoma that caused an obstruction in the jejunum by collapse of the intestinal lumen. Note the healthy mucosa in the affected segment. *Bar,* 1 cm.

Congenital segmental aplasia of jejunal lymphatics may cause chyloperitoneum and colic in foals within 12 to 36 hours after birth.[119,209-212] A presumptive diagnosis can be made on the basis of ultrasonographic findings of loops of thick-walled small intestine, anechoic peritoneal fluid, and thickened mesentery.[119,211] Resection of the thickened intestine can produce a satisfactory outcome, but mild cases can respond to conservative treatment.[210,211] Ruptured lymphatics and chyloperitoneum can also develop secondary to tearing of postsurgical mesenteric adhesions and other intestinal lesions.[212] An ileal diverticulum that is not a remnant of the omphalomesenteric structures (see later), other rare forms of intestinal malformation, and congenital jejunal duplications have been described as causes of recurrent small intestinal obstruction in horses.[213-215] Extraluminal causes of simple obstruction include intra-abdominal abscesses, intramural hematoma (Figure 36-6), and adhesions.[118,133,216]

An idiopathic disease of the duodenum reported in three horses older than 12 years is characterized by mural thickening and marked distention in approximately 30 cm (12 inches) of duodenum.[217] The distention starts 20 cm (8 inches) from the pylorus and ends in a contracted segment with aganglionosis immediately proximal to the caudal duodenal flexure.[217] Primary gastric and small intestinal ileus of undetermined cause has been described as responsible for acute colic in postparturient mares.[218] The outcome following treatment by decompression is good.[218]

Strangulating Obstruction

Volvulus

Volvulus is defined as a rotation in a segment of jejunum, or in jejunum and ileum, about the mesentery so that the intestine becomes twisted into into distinct spirals (Figure 36-7). Most are 360-degree rotations and involve long segments of intestine (see Figure 36-7).[89] Volvulus may develop as a primary displacement or as secondary to preexisting lesions, such as an acquired inguinal hernia, a mesodiverticular band, a Meckel

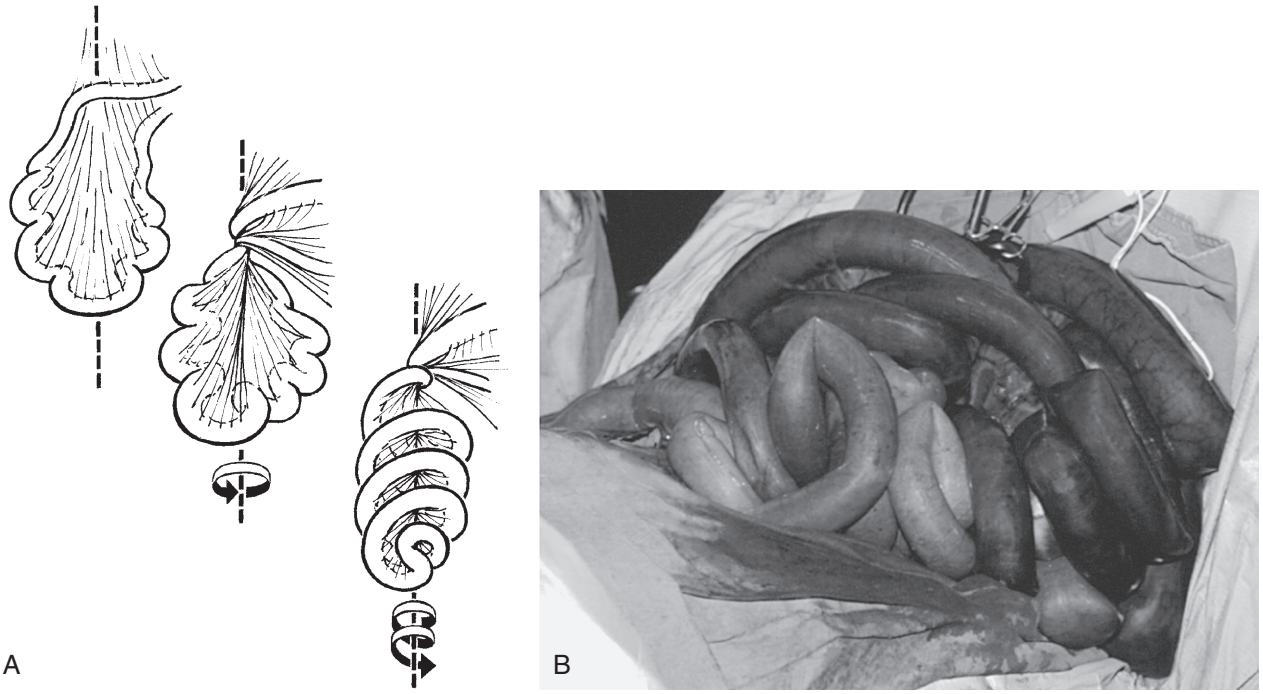


Figure 36-7. Small intestinal volvulus. **A,** Diagram. **B,** Intraoperative presentation. Note the typical appearance of numerous coils of distended and ischemic small intestine stacked one on another.

diverticulum, strangulation in mesenteric rents, distention from any cause, fixation by vitelloumbilical bands[219] and adhesions.[96,220-224] Primary volvulus can affect a horse of any age, but most affected horses are at least 3 years old.[89] It is the most common indication for intestinal surgery in foals, especially those between 2 and 4 months of age (see Table 36-1).[89,135] Volvulus nodosus is a less common form of volvulus, formed by the ileum and jejunum undergoing a 360-degree torsion that forms a mesenteric pouch in which prestenotic jejunum becomes entrapped.[225,226] Distention of the entrapped bowel draws the ileum into the mesenteric pouch to form a tight loop that knots the entrapment.[226] Volvulus nodosus is seen typically in foals 2 to 7 months of age, and it can account for 27% of small intestinal lesions that require surgery in foals.[124,226] Duodenal torsion has been reported but is rare.[217]

Although clinical signs are typical of acute small intestinal strangulating obstruction, admitting clinical signs and heart rates can vary.[89] Typically, stacks of tightly distended small intestine can be palpated *per rectum*, although distended small intestine was palpated in only 69% of horses in one study.[89] In foals, severe pain seems to alternate with periods of depression, and the abdomen becomes markedly distended. Foals with small intestinal volvulus are usually afebrile,[89] which should help distinguish them from foals with enteritis.

Treatment involves correction of the volvulus, followed by resection and anastomosis (see later), and the prognosis for survival to hospital discharge is good (80%).[89] Correction of volvulus nodosus can be difficult in foals and adults, but it can be accomplished by massaging the contents of the entrapped loop into the proximal segment, thereby allowing the bowel to be pulled through the ring formed by the ileum.[225,226] If the bowel involved in a volvulus nodosus is necrotic, time can be saved by resecting the entire lesion without reduction, followed by a jejunocecostomy.[226]

Figure 36-8. Epiploic foramen as viewed from the right side with the duodenum in the foreground, the liver dorsally, and with the cranium to the right. The *arrow* points to the ventral edge of the foramen formed by the gastropancreatic fold. *C,* Caudate lobe.

Entrapment in the Epiploic Foramen

The epiploic foramen, or foramen of Winslow, is the 4-cm–wide (1½ inch) entry into the vestibule of the omental bursa from the peritoneal cavity. Its dorsal and craniodorsal boundary is the visceral surface of the base of the caudate process of the liver (Figure 36-8).[227] The portal vein contributes to the cranioventral border, and the gastropancreatic fold becomes evident as a band where it forms the ventral border of the foramen.[227] From 8 cm (3 inches) to 18 m (60 feet) of intestine can become incarcerated in this space.[90,91,228,229] Variations include simultaneous

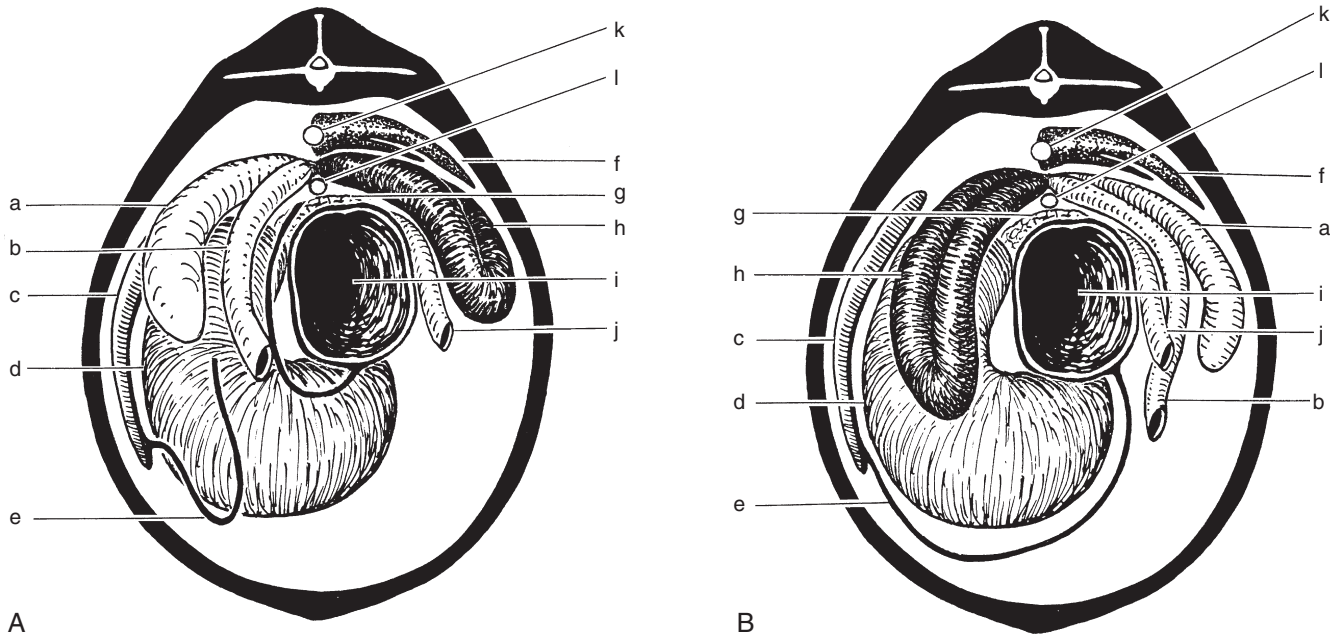


Figure 36-9. Epiploic foramen strangulation. Left to right **(A)** and right to left **(B)** herniation of the small intestine through the epiploic foramen. *a,* Jejunum proximal to strangulation; *b,* jejunum distal to strangulation; *c,* spleen; *d,* stomach; *e,* greater omentum, torn during strangulation; *f,* liver; *g,* pancreas; *h,* strangulated loop; *i,* right dorsal colon; *j,* duodenum; *k,* vena cava; *l,* portal vein.

strangulation of two loops of small intestine, strangulation of the cecum by the involved small intestine, simultaneous incarceration of the cecum and jejunum, incarceration of a Meckel diverticulum (Littre hernia), a parietal (or Richter) hernia of the ileum, a parietal hernia of the duodenum, and impaction in the incarcerated segment.[91,221,230-232] Although entrapment in the epiploic foramen (EFE) can occur from right to left in rare cases, one study reported left-to-right entrapment (Figure 36-9) in all of 53 horses, another reported it in 138 of 143 horses, and another in 97% of horses.[90,91,228,233] The ileum was involved in 66% to 81% of cases, either alone (12% to 25%) or in combination with the jejunum (41% to 60%), and involvement of the jejunum alone occurred in 28% to 34% of cases.[90,91,121,233,234] Ileal involvement for this disease (81%) was found to be significantly greater than for strangulating lipoma (31%) and miscellaneous small intestinal diseases (46%) in one study.[234] The longer the duration of colic, the more likely that ileum will become involved in the strangulation.[91]

The apparent predisposition to EFE in older horses and the predisposing enlargement of the epiploic foramen with age[235] were not confirmed in several studies.[88,227,233] Horses of all ages can be affected, including foals, and horses 11 months to 3 years.[88,90,91,124,227-229,236,237] In some of the larger studies, 47% to 71.3% of horses were younger than 11 years, and horses older than 20 years were rarely affected.[88,90,91,227,229,233] Some studies found Thoroughbreds and Thoroughbred crosses to be at a higher risk, and males constituted 68.5% and 76% of cases.[90,91,228,233,238] In one study from the United Kingdom, 77.5% of cases were seen between October and March, mainly in January, suggesting that stabling played a role in cause of this disease.[233,239] According to data from one U.S. and one U.K. university hospital, up to 68% of horses with EFE were cribbers before surgery, compared with 6% to 10.4% in the control groups, significant evidence that cribbing is associated with this disease.[240] In a prospective case-control study, a history of colic in the previous 12 months, increased time in a stall in the previous 28 days, and greater height of the horse increased the risk of EFE.[239,241] Horses that are easily frightened, sweat up when excited, or go off their food in response to a stressful situation appear to be at reduced risk of EFE.[239,241]

Clinical findings can be confusing. In one study, 38% of horses with herniation of small intestine through the epiploic foramen did not show signs of abdominal pain at the referral hospital, and in 52% of these horses no gastric reflux was obtained.[90] The signs of abdominal pain were sufficiently mild in one case to cause delay of referral for 2½ days.[242] Changes in the peritoneal fluid do not always represent the extent of intestinal damage.[90,229] Traction on the ventral band of the cecum is reported to induce a painful reaction in horses with the ileum incarcerated in the epiploic foramen[221]; however, this test was not reliable in one report.[229] With transabdominal ultrasonography, distended or edematous small intestine is found more often in the ventral right paralumbar fossa, followed by the caudal ventral abdomen and the middle right paralumbar fossa.[114]

At surgery in most cases, strangulation in the epiploic foramen can be corrected by careful traction combined with pushing of the strangulated bowel in the same direction. Careful milking of fluid into the empty and collapsed distal segment may be necessary to decompress the strangulated bowel. If possible, nonstrangulated bowel can be drawn into the foramen so that distention of the strangulated segment can be reduced by spreading its contents into the nonstrangulated bowel.[90] To avoid tearing the dorsal aspect of the portal vein and causing fatal hemorrhage during traction,[90,228,243] bowel should be drawn in a horizontal direction and not upward to the abdominal incision.[244]

When bowel cannot be drawn back through the epiploic foramen by these methods, jejunum approximately

1 m proximal to the obstruction must be emptied through an enterotomy or by transection.[244] Lumen of the empty segment of jejunum is closed and that segment can then be drawn through the foramen. After reduction, abnormal bowel is resected to include the transection or the enterotomy sites.

The short-term survival rate after completed surgery for EFE varies from 18% to 95%, and the long-term survival rate varies from 35% to 70%.[90,91,121,130,229,234,237,245] More recent trends have been toward improved survival rates to hospital discharge after surgery for this lesion, and one study reported a significantly better short-term survival after surgery for EFE (95%) than after surgery for strangulating lipoma (84%) and miscellaneous strangulating diseases of the small intestine (91%).[234] In studies on long-term survival after colic surgery, EFE was the only categorical variable associated with decreased survival, and horses with this lesion were more than 4 times as likely to have relaparotomy than horses with other colics.[246,247] In one study, horses with EFE had a shorter median survival time than those with other types of colic, and recurrence was reported in 2 of 49 horses (4%) that left the hospital.[233] Fatal hypoglycemia, a large thrombus in the portal vein, and areas of ischemic necrosis in the liver have been reported in a horse after treatment of an epiploic entrapment.[248] Rupture of the portal vein is a complication of correction, as discussed previously.[90,228,243]

Pedunculated Lipoma

A pedunculated lipoma is a benign, smooth-walled fat tumor that is suspended by a thin mesenteric pedicle of variable length. It can be a solitary tumor, or several of them can be found in the same horse, with 89% found in the small intestine and 10% in the small colon.[249] Some can develop in the omentum. Mesenteric lipomas cause strangulating obstruction when the pedicle wraps around intestine and its mesentery (Figure 36-10). A large lipoma attached close to the intestine can cause simple, recurrent obstruction, but this is rare.[250,251] In general, the length of the stalk rather than the size of the tumor seems to influence the risk for strangulation.

Intestinal strangulation by a mesenteric lipoma is a disease of older horses, with a mean age of 14 to 19.2 years at diagnosis, and the youngest reported at 8 years.[88,131,249,251,252] Incidence of lipomas is 5 times higher in horses older than 15 years compared with 10- to 15-year-old horses.[227] Ponies, Arabians, Saddlebreds, Quarter Horses, and geldings have been reported to be at a higher risk, and Thoroughbreds are at a lower risk for this disease.[227,249,251,252] Not all affected horses are overweight.[251]

Pedunculated lipomas should always be suspected in old horses with clinical evidence of strangulating obstruction, although clinical signs can be subtle and not always suggestive of this lesion.[251] In one study, 6 of 17 horses did not show signs of pain at the referral institution.[251] Consequently, this disease can be confused with duodenitis–proximal jejunitis, especially in horses with a large volume of reflux.

At surgery, the lipoma can be found wrapped in the jejunal mesentery, and the involved intestine can be difficult to exteriorize. Blind resection of the pedicle is necessary in many cases but carries the risk of creating a mesenteric rent and mesenteric bleeding. Spontaneous rupture of the lipoma pedicle and correction of the obstruction has been reported, based on surgical findings.[253] Incidental lipomas with distinct pedicles should be removed with the ligate divide stapler (LDS) (see Figure 16-14) by ligation and transection. A significant association between postoperative ileus and strangulation by lipoma has been reported, and published short-term survival rates for this strangulation range from 48% to 84%.[234,247,251,252]

Intussusception

Jejunojejunal, jejunoileal, and ileoileal intussusceptions have been reported in horses.[99] Suspected predisposing factors are segmental motility differences caused by enteritis, heavy ascarid burden, infection with the tapeworm *Anoplocephala perfoliata*, mesenteric arteritis, and abrupt dietary changes.[99,254] A transverse enterotomy, functional side-to-side anastomosis, end-to-end anastomosis, and pedunculated mucosal mass, such as a papilloma, leiomyoma, cryptococcal granuloma, granuloma of unknown cause, and carcinoid, can form the leading edge of a jejunojejunal intussusception in horses.[98,99,255-260] In one horse with weight loss and colic, jejunojejunal intussusception was associated with proliferative enteropathy with pseudodiverticula formation, jejunal muscular hypertrophy, and diverticulosis.[261]

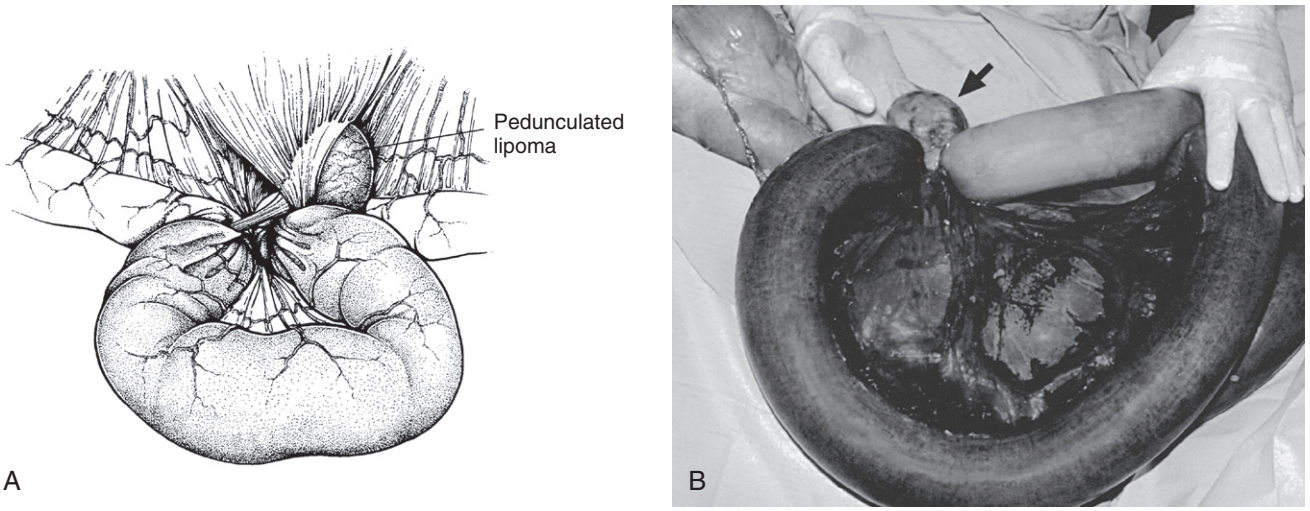


Figure 36-10. A, A pedunculated lipoma causing strangulation obstruction of the small intestine. **B,** Intraoperative presentation (the *arrow* points toward the lipoma). (A redrawn from McIlwraith CW, Robertson JT: Equine Surgery: Advanced Techniques. 2nd Ed. Blackwell, Oxford, UK, 1998.)

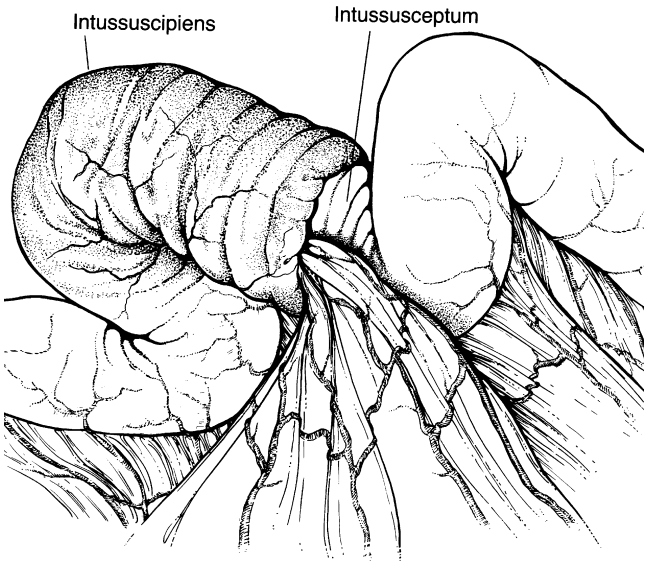


Figure 36-11. Small intestinal intussusception. (Redrawn from McIlwraith CW, Robertson JT: Equine Surgery: Advanced Techniques, 2nd Ed. Blackwell, Oxford, UK, 1998.)

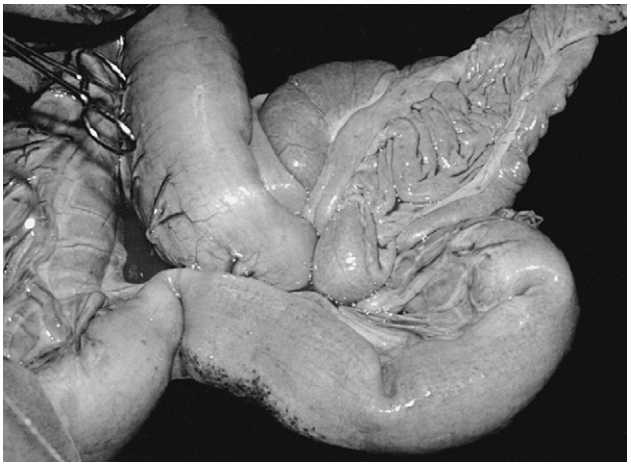


Figure 36-12. Compensatory changes in the jejunum proximal to a chronic ileocecal intussusception. The bowel is dilated but empty; its wall is thick, which is especially evident at the folds; and it has ecchymoses on the antimesenteric surface. (From Ford TS, Freeman DE, Ross MW, et al: Ileocecal intussusception in horses: 26 cases [1981-1988]. J Am Vet Med Assoc 196:121, 1990.)

Ileocecal intussusception is the most common type (74% of all small intestinal intussusceptions), with 27% of them causing chronic, intermittent colic.[97,131] Chronic ileocecal intussusceptions represented approximately 1% of all surgical colic cases in one study.[262] Horses with a chronic intussusception usually have a short intussusceptum that is not strangulated but causes an incomplete obstruction. Ileoileal intussusceptions usually form a short, doughnutlike lesion, and most cause chronic recurrent colic.[99,263-265] Jejunojejunal intussusceptions (Figure 36-11) usually involve long segments of bowel and cause complete obstruction,[98] although short jejunojejunal intussusceptions can cause recurrent colic.[98,265]

With chronic intussusceptions, the repeated bouts of obstruction cause compensatory changes in almost half the jejunum proximal to the lesion (Figure 36-12), including marked jejunal dilation, even when empty; thickening of the muscular coat; and areas of ecchymosis along the antimesenteric surface.[97,262] These changes do not appear to contribute to the obstruction.

Females accounted for six of seven (86%) intussusceptions in one study, and this was significantly different from the sex distribution in the hospital population.[238] However, the female predisposition was less pronounced in five larger studies, ranging from 45% to 68%.[97,99,254,262,264] One study reported an apparently higher risk for Thoroughbred horses, and another reported a higher risk for ponies.[99,264]

Although regarded mainly as a disease of foals, intussusceptions have been reported over a wide age range up to 24 years, with few reported in young foals.[97-99,135] Intussusceptions were the most common cause of surgical colic in horses 3 to 12 months of age in one study, particularly ileocecal intussusceptions.[137] In younger foals, intussusceptions were less common, and only jejunojejunal intussusceptions were recorded.[137] Ileocecal and ileoileal zintussusceptions, both of which are more likely to cause recurrent mild to moderate colic, are more common in horses 3 years of age and younger.[97,99,254,262-265]

Intussusceptions of long segments cause signs of severe small intestinal obstruction.[97-99] Short intussusceptions cause colic that is mild, intermittent, usually postprandial, and associated with reduced appetite and feces, elevated temperature, weight loss, and failure to thrive compared with herdmates.[97] An ileal intussusception should be strongly suspected in a horse between weaning and 3 years of age that is unthrifty and has a history of recurrent mild colic.

Rectal examination may reveal distended loops of small intestine, and the intussusception can be palpated as a firm, painful, tubular structure in some cases.[98,99] Ileocecal intussusceptions were palpated in the right dorsal quadrant of the abdomen in 31% of horses in one report and in 50% in another.[97-99] During an episode of pain in a horse with a chronic intussusception, the hypertrophied small intestine proximal to the obstruction (see Figure 36-12) can be palpated *per rectum* as large-diameter, moderately distended loops with thick walls.[97] In young horses and foals too small for rectal palpation, a cross-sectional view of a jejunojejunal or ileocecal intussusception can be seen on ultrasonography as concentric rings with a bull's-eye appearance (Figure 36-13).[93,116]

White blood cells and protein concentration may be increased in the peritoneal fluid of horses with acute ileocecal intussusceptions and jejunojejunal intussusceptions.[97,98] Peritoneal fluid protein concentration alone can increase in chronic ileocecal intussusceptions.[97] In one study, 8 of 16 horses with acute ileocecal intussusceptions had serosanguineous fluid.[97]

SURGERY: ILEOCECAL AND ILEOILEAL INTUSSUSCEPTIONS

In chronic intussusceptions of ileum, short segments (approximately 10 cm or 4 inches) are involved. Reduction is difficult, even in the absence of adhesions, because of chronic folding of the intestinal wall.[97] Reduction was impossible in 11 of 16 ileoileal intussusceptions and in 6 of 7 chronic and 10 of 19 acute ileocecal intussusceptions.[97,264] Ileocecal and ileoileal intussusceptions have been treated successfully by reduction only or by reduction and myotomy.[263,264,266] Incomplete bypass

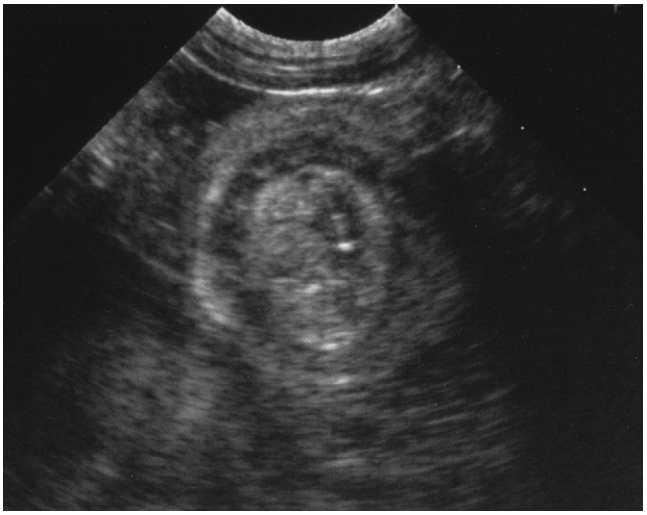


Figure 36-13. Ultrasonographic image of a small intestinal intussusception in a foal demonstrating the typical bull's-eye or target appearance. In this cross-sectional view, two concentric hyperechoic rings are evident, the outer intussuscipiens and inner intussusceptum with some hypoechoic spaces in both that represent fluid in the lumen.

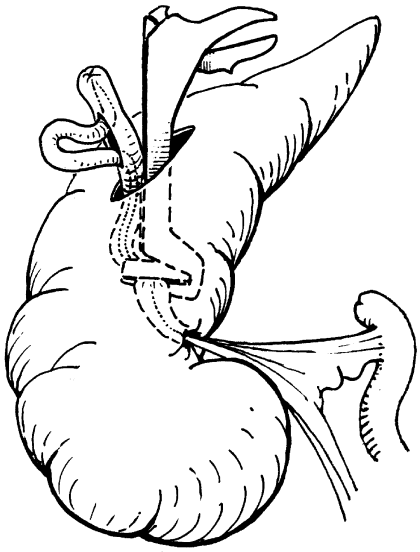


Figure 36-14. Method for resecting the intussusceptum of an acute ileocecal intussusception with the TA-90 stapler applied through a typhlotomy.

by ileocecostomy is recommended if there are concerns about permanent ileal changes and the risk of recurrence, and a hand-sewn technique is recommended over stapling instruments because the ileal wall is so thick.[97,262,267] There is no need to resect the involved ileum or the hypertrophied jejunum in chronic cases.

Ileocecostomy for chronic ileocecal intussusception has excellent short-term results, but apparently rare long-term complications include stomal impaction, ileal hypertrophy, and rupture distal to the stoma.[97,99,153] Also, the stoma can become smaller over time, which could predispose to these complications.

In acute cases, the intussuscepted ileum and jejunum can be too edematous and hemorrhagic to allow reduction. Bypass by a jejunocecostomy without reduction can cause postoperative hemorrhage.[97] Removal of the intussusceptum through a typhlotomy can cause severe contamination, although suturing an impermeable plastic drape around the typhlotomy can contain leakage.[97,218] In a technique devised to facilitate this surgery, the jejunum proximal to the intussusception is transected with the gastrointestial anastomosis (GIA) or the intestinal linear anastomosis (ILA) stapler as close to the ileocecal junction as possible.[268,269] A 10-cm (4-inch) incision is made in the cecum to allow the intussusception to be exteriorized, and another incision is made through the outer wall of the intussusceptum to expose the inner loop (Figure 36-14). This inner loop then is pulled through the incision in the outer layer until the closed transected end is distal to the selected site for transection.[269] If too much bowel is drawn through this incision, or if the jejunal transection was made far from the ileocecal junction, mesenteric vessels will tear and cause fatal hemorrhage.[268,269] Much of the necrotic bowel is removed by incision along the edge of a thoracoabdominal (TA-90) stapler applied across the inverted ileum within the cecum (see Figure 36-14).

SURGERY: JEJUNOJEJUNAL INTUSSUSCEPTION

A jejunojejunal intussusception forms a corkscrew configuration because of tension on the mesentery of the intussusceptum, and short to long segments can be involved.[259] Reduction is accomplished by slow traction on the intussusceptum and gentle massage of the distal end of the intussuscipiens.[259] If it is nonreducible, the entire intussusception can be removed by resection only,[98] although this can make ligation of mesenteric vessels difficult.[99]

Prognosis is favorable for all intussusceptions, but the bowel wall and intussuscepted mesenteric vessels can be torn during reduction.[97] Some horses can have a protracted recovery after surgery for a chronic ileocecal intussusception, with slow weight gain and mild episodes of colic.[97] Because *A. perfoliata* may be involved in the pathogenesis of ileocecal intussusceptions in horses, the treatment of affected horses and pasturemates with pyrantel pamoate and praziquantel is recommended.[97,254,270]

Mesenteric Rents

Mesenteric rents can be congenital in origin, secondary to mesodiverticular bands (Figure 36-15), or may develop as primary lesions of unknown cause. Trauma or mesenteric stretching by another lesion may play a role, and some can develop years after a small intestinal surgery at a site remote to the anastomosis.[90,271] Horses of a wide age range appear to be prone, although females are more likely to be affected, especially in the postpartum period when the tear is possibly induced by vigorous movements of the foal, as has been implicated in tears of the small colon mesentery.[271,272] Such tears in postparturient mares can cause segmental ischemic necrosis of the related segment of jejunum or predispose to later strangulation of a more distant portion of small intestine.[273] The jejunal mesentery is involved in most cases, although tears in the ileal and duodenal mesentery have been reported, as well as tears in multiple sites.[271,273]

Prognosis for surgical treatment appears to be worse than after other strangulating lesions of the small intestine.[271] Possible reasons for this are inability to reduce the hernia, the long segments of bowel involved, hemorrhage from the affected mesentery, and failure to close the entire mesenteric defect.[271]

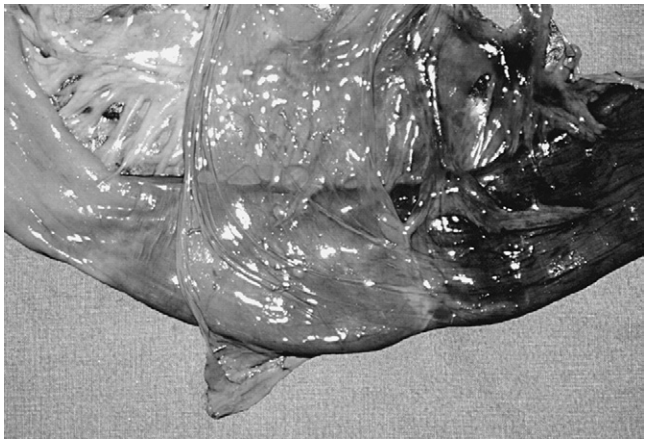


Figure 36-15. The mesodiverticular band is evident as a thin fold extending to the antimesenteric surface of the jejunum. The jejunum was strangulated in a mesenteric rent at the point of attachment of the band and mesentery.

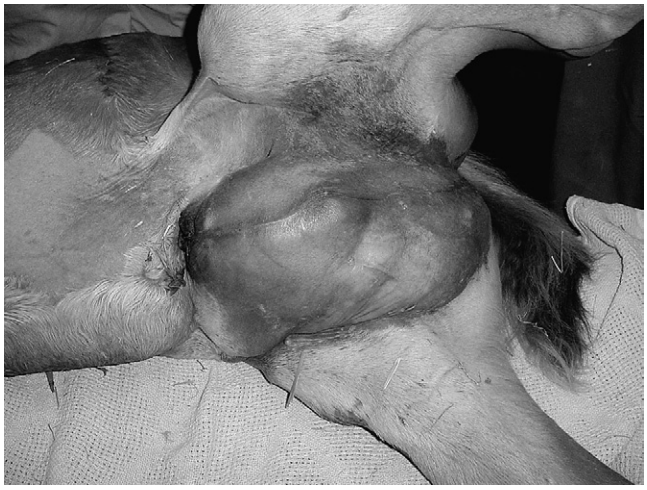


Figure 36-16. Direct inguinal hernia in a foal, with massive preputial and scrotal swelling from loops of small intestine beneath the skin. The tunic ruptured, allowing bowel to escape into a subcutaneous position. (From Bartmann C-P, Glitz F, von Oppen T, et al: Diagnosis and surgical management of colic in the foal. Clin Tech Equine Prac 1:125, 2003, with permission.)

Complete closure of the defect can be difficult or impossible through a ventral midline approach.[274] Elective laparoscopic closure of the full defect can be accomplished as a standing procedure shortly after the ventral midline celiotomy is performed to treat the original strangulation.[274] Both sides of the abdomen may need to be examined to locate the tear.[274]

Inguinal Hernia

An indirect inguinal hernia is the most common form in horses, and it involves small intestine passing through the vaginal ring into the vaginal tunic (see also Chapters 39 and 59). In the less common direct inguinal hernia, jejunum and occasionally the testicle escape through a rent in the peritoneum and transverse fascia, adjacent to the vaginal ring, to lie in the subcutaneous space of the scrotum and prepuce (Figure 36-16).[275-277] Direct hernias are more common in foals than in adults, but they can involve greater lengths of intestine in adults than are recorded with indirect hernias.[275-280]

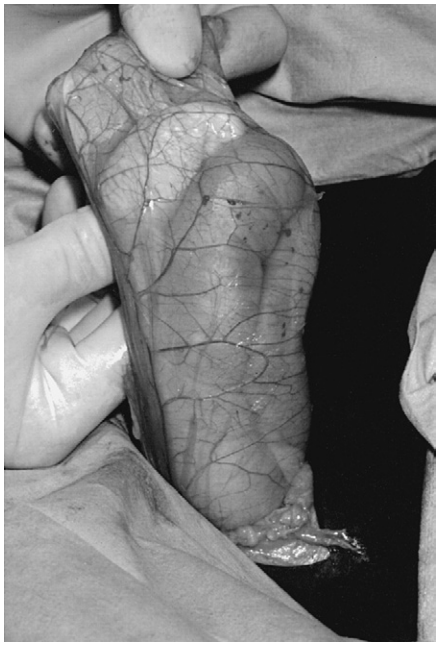


Figure 36-17. Dissected vaginal tunic in a foal with a congenital scrotal hernia, demonstrating the jejunum and the testicle above it and to the left. The cremaster muscle is evident on the left side.

Indirect inguinal hernias in adult horses involve short segments of small intestine (median of 15 cm [6 inches]) and are usually acquired and nonreducible, whereas congenital inguinal or scrotal hernias in foals involve long segments and are reducible.[280] In acquired hernias in adults, the loop of intestine rarely advances to the level of the testicle before it becomes strangulated, whereas a variable length of intestine (approximately 1 m [3 feet]) can reach the fundus of the vaginal tunic in foals and remain viable (Figure 36-17). This difference can be attributed to the shorter, wider, and more direct configuration of the foal's inguinal canal. Contrary to popular misconception, the size of the external inguinal ring is irrelevant to development of an inguinal hernia, because it is the last structure for the bowel to traverse and is always sufficiently large for intestine to negotiate easily. The vaginal ring is the first structure to be negotiated by herniated intestine, but the intestine is actually strangulated in an adult horse by a ring 2 to 3 cm further distad, formed by merging of the internal spermatic fascia into loose connective tissue in the neck of the vaginal process.[221,281]

Ileum, alone or with jejunum, was involved in 49% to 53% of adult inguinal hernias in three reports, but some hernias involve jejunum only.[96,278,280,282] Ileal involvement may be more likely with increased duration of strangulation.[221]

Many breeds can be affected, but a higher prevalence of inguinal/scrotal hernia has been reported in Standardbreds, Tennessee Walking Horses, and American Saddlebreds.[96,238,282,283] Standardbreds also appear to be at a higher risk for intestinal eventration after an open castration.[284] In acquired inguinal hernias, almost all age groups over 1 year are at risk.[227,283] Predisposing factors are not always evident but include a history of recent strenuous exercise, recent breeding, and trauma.[96,282,283] Direct and indirect inguinal hernias are rare in geldings.[279,283,285,286]

Congenital scrotal hernias are noted shortly after birth, are easily reduced when the foals are rolled onto their backs, and usually resolve spontaneously within 3 to 6 months. Intestinal strangulation is rare. Although the problem is usually unilateral, bilateral cases can occur. Direct or ruptured inguinal hernias in foals are evident within 4 to 48 hours after birth and cause intermittent colic, depression, severe scrotal and preputial swelling (see Figure 36-16), and edema, with skin excoriation and splitting caused by abrasion against the inside of the thigh.[275,276] These hernias are not reducible and are treated as surgical emergencies, although strangulation is rare.

Acquired inguinal hernias in adult horses cause signs of mild to severe colic.[282] As it becomes strangulated, the intestine compresses the testicular vessels and causes the testicle to become swollen, firm, and cold. Although the strangulated intestine may not be evident on external examination, rectal examination reveals small intestine entering the vaginal ring and distention of the small intestine. Bilateral cases are very rare.[282,287] Ultrasonography can facilitate early diagnosis and is especially useful if rectal palpation is not possible.[96]

SURGERY: CONGENITAL HERNIAS

Surgical correction is indicated if the hernia does not resolve spontaneously, the owner is concerned because of an apparent increase in size of the hernia, or the vaginal tunic ruptures. Surgical correction can be accomplished in foals through an inguinal approach with exposure of the tunic and its contents (see Figure 36-17), removal of the cremaster muscle, twisting of the testicle and tunic to force the bowel into the abdomen, and then closed castration combined with a transfixation ligature of 0 polydioxanone through the tunic (see Chapter 39). Closure of the external inguinal ring is suggested but not essential and may not prevent reherniation.[244,276,278,288] Alternative methods are laparoscopic repair with or without castration, an inguinal approach without castration, or a midline celiotomy with closure of the vaginal ring.[289,290] The testicle can atrophy after the latter two methods.

For repair of a direct or ruptured inguinal hernia in foals, the torn edges of the common vaginal tunic are identified and drawn upward to create a funnel through which the bowel is returned to the abdomen. The bowel can be directed into the abdomen by careful digital manipulation or by grasping the bowel gently in partly closed sponge forceps. The torn tunic should be repaired as much as possible to the level of the vaginal ring. Usually, the intestine is viable and does not require resection, although delayed necrosis has been reported.[276] Castration facilitates the repair and closure of the vaginal tunic.

SURGERY: ACQUIRED HERNIAS

In the early stages, a direct hernia in an adult may correct spontaneously after the anesthetized horse is placed in dorsal recumbency or can be corrected by applying gentle traction to the bowel *per rectum*, with or without external massage of the scrotum.[278,283,242] External massage alone may also be effective and can be guided by laparoscopy, which also allows assessment of intestinal viability.[281] In such cases, laparoscopic inguinal herniorrhaphy may be performed 1 week later to prevent recurrence, using either a mesh onlay graft or a cylindrical mesh plug.[291,292] Spontaneous reduction or reduction by traction does not rule out the possibility of complications from progressive intestinal necrosis.[278]

A nonreducible inguinal hernia is corrected through an inguinal incision directly over the external inguinal ring and along the spermatic cord. The abdomen should be prepared for a ventral midline celiotomy, if needed, to allow intra-abdominal traction on the bowel, to assess viability of the released bowel, to decompress or examine proximal bowel, or for a jejunocecal anastomosis. The tunic is dissected from the surrounding tissues, with care taken to not damage the pudendal vein and its branches. A cranial incision is made in the vaginal tunic, which needs to be extended far enough distad to cut the constricting ring formed by the internal spermatic fascia and release the strangulated intestine.[281] The tunic edges should be grasped with hemostats to maintain access for closure. Medial and cranial retraction on the edge of the internal abdominal oblique muscle with a Deaver retractor will improve exposure to the tunic edges, and a finger is used to direct bowel away from the suture line.

Because short segments (approximately 25 cm or 10 inches) of intestine are usually involved, resection is rarely needed if appearance of the bowel improves markedly after the strangulation has been released.[278,280] A unilateral castration is recommended to allow more complete closure of the vaginal tunic and abolish the risk of recurrence.[283] In addition, the involved testicle can become cystic or nonfunctional in time, and postoperative swelling could induce degeneration of the other testicle. Techniques to correct the hernia and prevent recurrence have been described.[289-292]

If the vaginal ring can be closed, closure of the external inguinal ring is not essential and does not ensure against evisceration.[278] However, if the vaginal ring closure is not secure, closure of the external inguinal ring is recommended. Closure of the thick subcutaneous fascia and skin incision is accomplished in two layers. After surgery, the scrotum on the castrated side usually lies against the prepuce, and swelling is minimal. Any method of correction that does not involve exploration of the abdomen would fail to detect a concurrent small intestinal volvulus, a rare complication of inguinal hernia that could cause postoperative colic.[96,220,221]

A survival rate of up to 76% has been reported for acquired inguinal hernia.[278,282,283] Adhesions at the vaginal ring are rare causes of later strangulation.[282]

Incarceration through the Gastrosplenic Ligament

Small intestinal incarceration through the gastrosplenic ligament is an rare cause of colic, with no breed or age predilection detected, although geldings appear to be at increased risk.[293] The jejunum is the segment most likely to be involved.[122,293] The gastrosplenic ligament is a broad but thin attachment between the left part of the greater curvature of the stomach and the hilus of the spleen.[1] Small intestine can pass from caudad to craniad through acute tears in this ligament so that the strangulated loop is lateral to the stomach and craniolateral to the spleen (Figure 36-18).[293,294] At surgery, the strangulation is easily corrected by traction, and enlarging the rent does not cause problems or predispose to recurrence. No attempt is made to repair the mesentreric defect, and the edges are not readily identified at surgery. If the most ventral edge of the defect in the ligament is accessible, it can be transected between two ligatures to disrupt continuity of the ring.[295] With appropriate surgical intervention and postoperative management, the short- and long-term prognosis for surgically treated horses is good.[293]

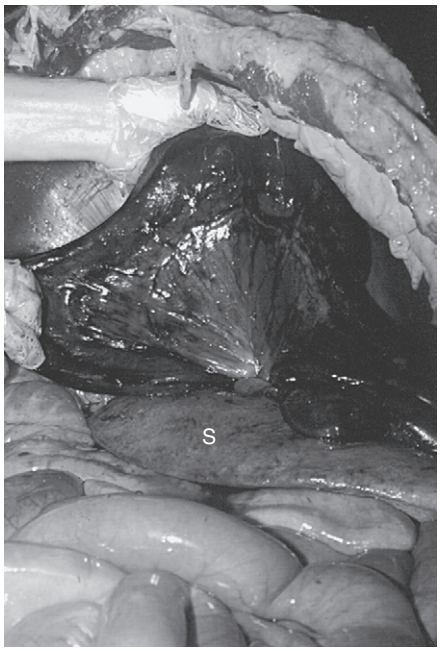


Figure 36-18. The jejunum is strangulated in a rent in the gastrosplenic ligament and lying in the left cranial part of the abdomen. *S,* Spleen.

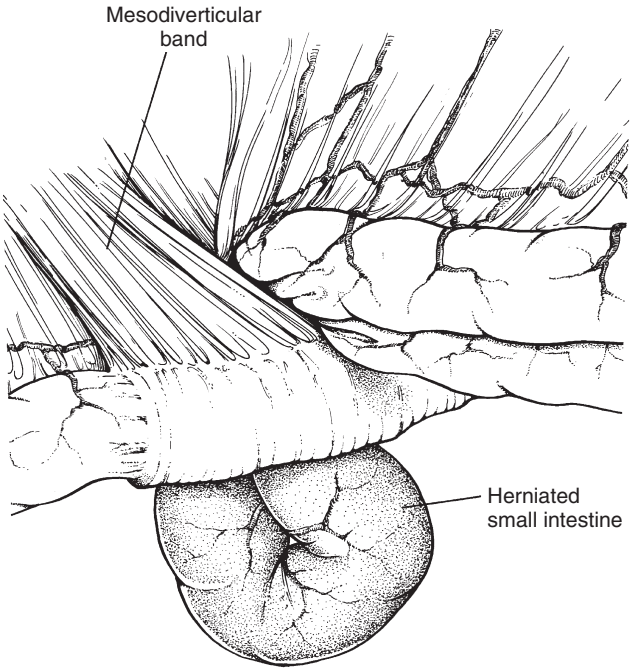


Figure 36-19. A mesodiverticular band and secondary mesenteric rent resulting in incarceration of the small intestine. (Redrawn from McIlwraith CW, Turner AS: Equine Surgery: Advanced Techniques. Blackwell, Oxford, UK, 1987.)

Vitelline Anomalies

A mesodiverticular band develops from a vitelline artery and associated mesentery that fail to atrophy during early embryonic development.[222] The band is usually found in the distal jejunum, approximately 1.5 m from the ileocecal junction. It extends from one side of the mesentery, usually the left, to the antimesenteric surface of the small intestine to form a triangular space (see Figure 36-15). Intestine may become strangulated in a mesenteric rent that forms in that space (Figure 36-19), and secondary volvulus of the small intestine follows.[222] A mesodiverticular band alone can also cause a volvulus because the band shortens the mesentery at the point of attachment.

Meckel diverticulum is a remnant of the omphalomesenteric (vitelline) duct that provides a communication between the yolk sac and early embryonic gut. It forms a conical blind extension from the antimesenteric surface of the distal jejunum or ileum, 40 to 120 cm (16 to 47 inches) from the ileocecal junction (Figure 36-20).[296,297] It can be up to 35 cm long, but it is usually 10 to 15 cm (4 to 6 inches) long and 5 to 10 cm (2 to 4 inches) in diameter.[222] Occasionally, a fibrous band persists from the apex of Meckel diverticulum to the umbilicus—the vitelloumbilical band—and creates an axis for volvulus of the jejunum and ileum.[219]

In one review of 15,000 necropsies, Meckel diverticulum was responsible for the death of all five horses (0.03%) in which it was found.[296] Meckel diverticulum can become impacted and achieve enormous proportions or become necrotic and rupture to cause fatal peritonitis.[296,298,299] Meckel diverticulum can also entangle and strangulate small intestine or form an axis for volvulus nodosus.[223,297,298,300] In one horse, a Meckel diverticulum was found adhered to and impacted in an umbilical hernia, thereby forming a Littre hernia.[301] Although Meckel diverticulum is a congenital anomaly, it can cause colic in horses of a wide age range and be an incidental finding at necropsy.[222,296-300]

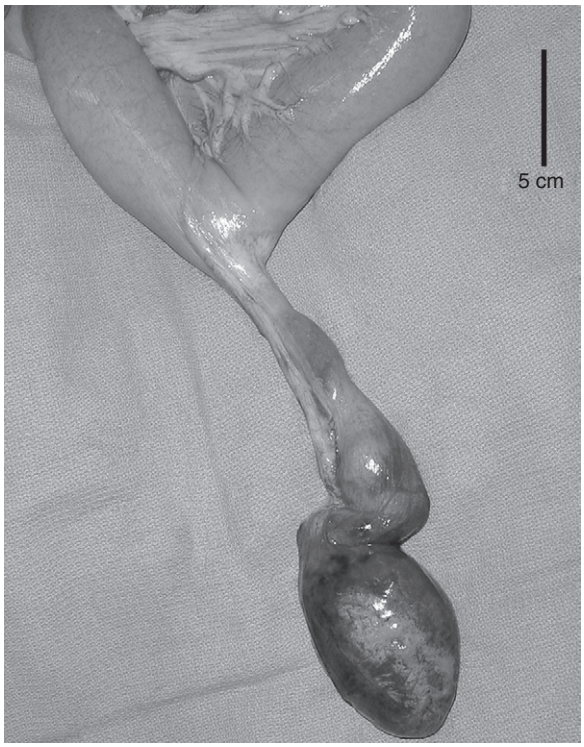


Figure 36-20. Meckel diverticulum that was knotted around the jejunum in a 5-month-old Clydesdale foal. The diverticulum was resected and the linear defect in the jejunum was oversewn. (From Bartmann C-P, Glitz F, von Oppen T, et al: Diagnosis and surgical management of colic in the foal. Clin Tech Equine Prac 1:125, 2003, with permission.)

Strangulated Umbilical Hernia

Strangulation of a loop of small intestine in an umbilical hernia is rare, and the involved intestine can rupture through the hernial sac and dissect subcutaneously in a caudal direction to cause an inguinal enlargement (see also Chapter 39).[302] More commonly, only a portion of the antimesenteric wall of the ileum is incarcerated, and this is termed a *parietal*, or *Richter*, *hernia*.[302,303] A parietal hernia should be suspected when an umbilical hernia acutely becomes nonreducible, large, firm, edematous, and painful to palpation. A parietal hernia can cause umbilical abscessation, which may lead to rupture of the hernia and formation of an enterocutaneous fistula.[302,303] Ultrasonography may be used to evaluate the hernia and its contents.[125] Colic can develop in horses with a parietal hernia, but it is not observed in all cases with an umbilical abscess or enterocutaneous fistula.[302,303] Surgical correction of these umbilical lesions is successfully accomplished by making a 10- to 15-cm (4- to 6-inch) celiotomy cranial to the ring and digitally guiding *en bloc* removal of the umbilical lesion through this incision. Resection and anastomosis of involved intestine may be required.

Diaphragmatic Hernias

A diaphragmatic defect can be congenital or acquired, although the distinction is difficult (see also Chapter 39). Small intestinal strangulation in diaphragmatic defects has been reported in foals as the cause of death at 7 hours to 8 days after birth.[304] A congenital diaphragmatic defect or a diaphragmatic tear inflicted by a rib fracture, usually at or close to the costochondral junction of ribs 3 to 8, are likely causes of diaphragmatic hernia in foals.[304,305] The most common causes of diaphragmatic hernia in adult horses are trauma, parturition (particularly dystocias), and recent strenuous activity.[306] In some horses, the presence of omental adhesions to a callus on fractured ribs on the thoracic side of the defect is suggestive of previous trauma, possibly at birth.[307,308] Although several abdominal organs can enter the diaphragmatic defect, small intestine is the most commonly reported.[309] There does not appear to be an age, breed, or sex predisposition to this disease.

Large defects are more likely to cause dyspnea from pulmonary compression by intrathoracic displacement of the colon, but they do not incarcerate bowel and cause colic.[306,310] Small defects are more likely to strangulate small intestine and manifest as acute and severe colic, clinically indistinguishable from strangulation by any other cause. Abnormalities can be detected on auscultation and percussion of the thorax in some horses.[311]

Both radiography and ultrasonography allow preoperative diagnosis.[312-315] Ultrasonography may be superior to radiography in evaluating diaphragmatic hernias in horses, especially for small tears with little visceral displacement, or when pleural effusion obscures the ventral portion of the diaphragm.[316] The diaphragm can be imaged with 5- or 7.5-MHz transducer adjacent to and parallel with the body wall ventrally, then diverging dorsally and medially to lie behind the echogenic lung.[316] The curved shape of the diaphragm may prevent complete evaluation.[316]

SURGERY

A diaphragmatic hernia can be missed at surgery.[317] If strangulated bowel is found free in the abdomen, a careful search must be conducted to ensure that it did not fall out of a diaphragmatic defect when the horse was placed in dorsal recumbency.[317]

Ideally, a respirator should be used to provide controlled positive-pressure ventilation and correct the diminished pulmonary function. An advantage of preoperative diagnosis is that the abdominal incision can be placed further craniad than the standard approach. Access to ventral defects is not difficult, but access to more dorsal defects can be improved by extending the cranial end of the incision laterally for approximately 12 to 15 cm (5 to 6 inches) at an angle of 60 to 90 degrees in a paracostal fashion (J-shaped incision). The table can be tilted to raise the cranial end of the horse's abdomen so abdominal contents can fall away from the diaphragm.

A small defect may have to be enlarged with scissors or a curved fetotome to release the bowel.[244,307,311,313] Most defects can be closed with a continuous pattern using heavy absorbable or nonabsorbable material, and the suture line should be completed at full inspiration to reduce the pneumothorax present. Mesh coverage is required for defects that cannot be sutured because of their large size or because the edges are too firm to allow apposition.[244] Successful repair of a dorsal rent that was inaccessible for suture placement was accomplished by blind fixation of a doubled polypropylene mesh (Marlex Mesh No. 1266) with 4-mm stainless steel staples (disposable skin stapler).[315] Another inaccessible defect was repaired in the standing horse by suture closure through a thoracic rib resection that was placed by guidance from thoracoscopy and manual direction through a flank incision.[318] Indwelling chest tubes covered by a Heimlich valve (see Chapter 17) can be placed to correct pneumothorax and fluid accumulation in the chest.[244]

Successful repairs of diaphragmatic hernias have been reported in adult horses and foals, and horses can race, participate successfully in various forms of competition, and deliver foals after repair.[244,306,307,310,312,313,315] One horse with a small defect that was inaccessible for repair returned to a full athletic career.[313] Unrepaired diaphragmatic defects can partly seal by adhesion of adjacent stomach, omentum, or liver to the edges, but recurrence of intestinal incarceration immediately after anesthetic recovery is also likely.[315,318]

Miscellaneous Strangulating or Ischemic Diseases

Small intestine can become strangulated in mesenteric ligamentous bands that cannot be exteriorized at surgery and must be cut blindly with scissors. Small intestine can also become strangulated by uterine torsion; through rents in the mesometrium, gastrohepatic ligament, small colon mesentery, lateral ligament of the urinary bladder, cecocolic fold, and mesentery of the large colon; by components of the spermatic cord, particularly the mesoductus deferens; and by omental adhesions.[129-131,133,221,319-323] Evisceration through a lacerated vaginal fornix, a defect in the bladder and urethra, or a castration wound may cause small intestinal strangulating obstruction.[284,324] Entrapment of small intestine within the nephrosplenic space has been reported in two horses, with the bowel passing from craniad to caudad.[325] Both horses were presented alert and in stable condition, and the affected segments of bowel did not require resection.[325] Mesenteric hematomas of unknown origin can cause colic and ischemic necrosis of affected intestine.[326] Surgical access to the source of hemorrhage may be difficult.[326]

Adhesions after small intestinal surgery or any intra-abdominal procedure can form an axis around which attached small intestine can form a volvulus, or adhesions can form fibrous bands through which small intestinal loops can become strangulated.[224,327] Nonstrangulating infarction and necrotizing

enterocolitis in the small intestine are rare and have a poor prognosis.[125,328]

SURGICAL TECHNIQUES

Success of small intestinal surgery in the horse is dependent on (1) identification and correction of the primary problem, (2) intraoperative decompression of distended small intestine, (3) resection of all intestine identified as nonviable or altered by an irreversible disease, (4) preservation of anatomic and physiologic continuity of the mesentery and intestine, (5) rapid completion of the surgery with minimal trauma, (6) early return of intestine to normal function, and (7) appropriate postoperative support, including repeat laparotomy when indicated.

If the lesion is not revealed quickly by a cursory examination of the abdomen, the cecum should be found and the ileocecal fold traced to the ileum, followed by progressive examination of the jejunum toward the duodenum until the involved segment is found. If several discontinuous loops of small intestine are exteriorized through the incision, they form tight bands of mesentery that can prevent extraction of other distended loops. Rarely, a horse has two apparently unrelated strangulating lesions of the small intestine.[221,329] However, it is more common for a horse to have a concurrent large intestinal lesion, such as volvulus, displacement, or impaction.[89,330]

Viability Assessment

Viable small intestine refers to the ability of strangulated or ischemic intestine to survive without developing adhesions and other complications (for additional information, see Chapter 33). The observation that increased duration of surgery and length of bowel resected can adversely affect prognosis provides impetus to improve viability assessment and thereby avoid resection whenever possible.[246] Clinical criteria of viability are serosal color, improvement in color after correction of the strangulation, presence or absence of mesenteric arterial pulses, and intestinal motility, spontaneous or evoked (by snapping a finger against the intestinal wall). Clinical judgment tends to be pessimistic. Fluorescein fluorescence offers little improvement over clinical judgment but is accurate when it produces a viable fluorescent pattern.[331]

Intestine can be considered viable if the predominant clinical findings are hemorrhage and edema in the bowel wall, especially if the color improves within approximately 15 minutes after release of the obstruction.[332] Spontaneous or evoked motility appears sluggish in viable strangulated bowel because of "splinting" of the muscle wall by edema and hemorrhage. Enterotomies are not recommended for viability assessment in the small intestine because of the risk for adhesion formation and because mucosal changes are usually sufficiently severe to cause an incorrect prediction in viable bowel. With long segments of questionable viability, the risk for adhesions must be balanced against the risks of additional anesthesia time and the occurrence of adhesions after resection and anastomosis.

Resection

After the lesion has been corrected, the bowel must be arranged in its correct orientation and then decompressed, if necessary. Decompression is accomplished through the strangulated bowel, but only after the bowel has been freed from its mesentery and mobilized from the surgical field. For this purpose, a ligature of 2-0 polydioxanone (PDS) is applied to the first mesenteric vessel in the strangulated bowel, level with the proposed line of mesenteric resection. The mesenteric vessel is then transected distal to the ligature with the LDS (see Figure 17-14). The short end of the ligature is secured with a hemostat, and the long end is used to gather the trimmed edge of mesentery as each mesenteric vessel is transected with the LDS instrument (Figure 36-21). Ideally, the line of mesenteric resection should remain at a constant distance from the bowel, because any deviation toward the mesenteric root will shorten the mesentery and kink the bowel.[244] The two ends of the suture are tied to close the gathered mesentery after the bowel is decompressed, and a gap will remain to be closed between the bowel and the line of mesenteric resection (see Figure 36-21). With this method, ligated vessels can be retained in view outside the abdomen for continued inspection, and formation of a large mesenteric gap, which would allow rotation or entrapment of bowel, is prevented.[244]

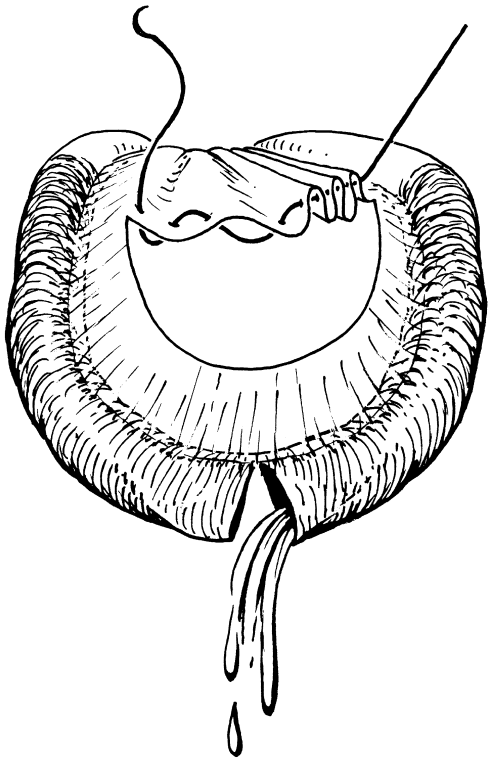


Figure 36-21. Method of resecting and gathering the mesenteric edge and then draining the bowel through the strangulated segment. Note that the line of resection should stay parallel to the bowel. (Redrawn from Freeman DE: Surgery of the small intestine. Vet Clin North Am Equine Pract 13:261, 1997.)

The nonviable intestine can now be exteriorized from the abdomen, far from the surgical field, to drain into a container, taking care not to stretch and tear remaining mesenteric attachments. As much intestine as possible proximal to the site of transection is manually stripped of its fluid and gas contents, with care taken not to tear the mesentery. This amount of intestinal handling does not appear to cause problems, and decompression seems to minimize the risk of postoperative reflux. If resection is not needed, small intestinal distention should be relieved through a typhlotomy and not a small intestinal enterotomy, to reduce the risk of adhesions to the latter.

Strangulated bowel is removed along with approximately 30 to 50 cm (12 to 20 inches) of contiguous healthy intestine at

each end. A large arcuate artery, no more than 10 cm (4 inches) from its origin from the major mesenteric vessel, is left to supply the anastomosed ends. Approximately 2 to 10 cm (1 to 4 inches) of mesentery is left beyond the last major vessels and branches to prevent inadvertent vascular occlusion or puncture during closure of the mesenteric gap. After the anastomosis is complete, the bowel is lavaged. The defect remaining in the mesentery is closed with 2-0 or 3-0 PDS in a simple continuous pattern, with care taken to avoid mesenteric vessels or creating an accordion effect that could kink the bowel.[244] If the mesenteric stump is inflamed and hemorrhagic, adjacent healthy mesentery can be wrapped around it.[130]

Hand-Sewn End-to-End Jejunojejunostomy

To prevent leakage into the surgical field, a Penrose drain may be applied 45 cm (18 inches) proximal to the anastomosis site to avoid trauma to this critical area (Figure 36-22). Penrose drains are less traumatic than Doyen clamps and facilitate manipulation and positioning of the bowel. To create a large stoma, the intestine should be transected at approximately 50 to 60 degrees from the mesenteric attachment; an S-shaped line of transection will increase the diameter further without creating sharp angulations in the incision edges. A suture is placed through the mesenteric and antimesenteric edges of both segments to draw them into alignment and to maintain them at similar diameters. With all end-to-end anastomoses, special care must be taken to appose the mesenteric border first, because rapid edema formation at this area can make it difficult to identify the seromuscular layer.

A popular method for end-to-end anastomosis of equine small intestine is a two-layer continuous pattern.[333] This involves a simple continuous pattern of 2-0 or 3-0 absorbable suture for the submucosa and mucosa, followed by a continuous Lembert or Cushing pattern in the seromuscular layer (see Figure 36-22). The Lembert pattern can be placed to minimize inversion. Mucosal and seromuscular rows are interrupted at the mesenteric and antimesenteric margins or at thirds of the circumference to prevent a purse-string effect. The Cushing pattern exposes less foreign material to the serosa and therefore poses less risk for adhesion formation than the Lembert pattern, but the Cushing is more likely to purse-string the bowel. The degree of inversion is greater with the Lembert pattern, so special care is needed to minimize this effect (see Figure 36-22). Combinations of patterns that involve two layers in the seromuscular wall, such as continuous appositional, Lembert, Cushing, or Connell oversewn with Lembert or Cushing, produce excess seromuscular inversion, stomal constriction, and a high risk for postoperative obstruction.[258,298,334,335] There is ample evidence that single-layer anastomoses work well in equine small intestine (for additional details on suture patterns, see Chapter 16).[139,244,333-336]

The Gambee and other interrupted patterns are more likely to cause anastomotic adhesions in the horse than continuous patterns,[333,337] although results of one study did not confirm this.[335] Larger sizes of suture material, such as 0 PDS or polyglactin 910 (Vicryl), could predispose to adhesions because they place a greater volume of foreign material along the anastomosis than 3-0 PDS, which is preferred.[244,333,337] An interrupted Lembert pattern is used with approximately 6- to 8-mm

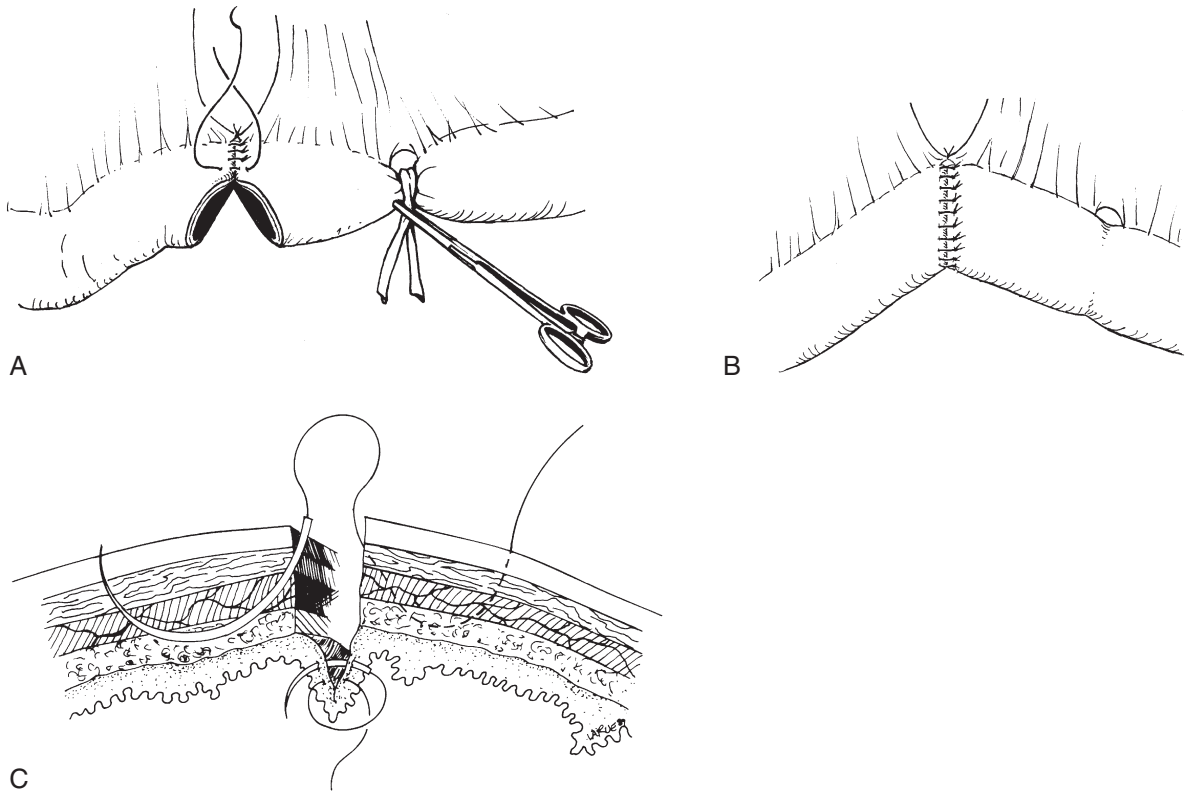


Figure 36-22. A and **B,** The lumen of the small intestine is occluded temporarily with a Penrose drain proximal to the anastomosis site. A one-layer closure consisting of interrupted Lembert sutures is carried out. **C,** Close-up of an alternative (two-layer) anastomosis technique: a simple-continuous apposition of the mucosa over three times a third of the circumference, followed by a minimal inversion pattern of the seromuscular layer.

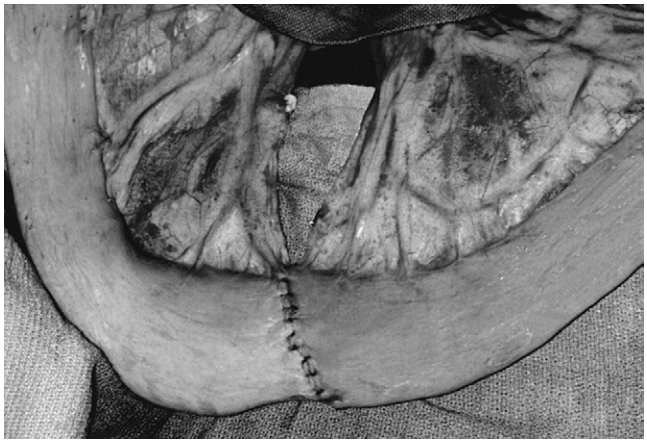


Figure 36-23. End-to-end anastomosis with interrupted Lembert sutures before the mesentery is closed. Note the darker discoloration of the bowel to the right, which is caused by prestenotic distention.

bites that penetrate the cut edge of the seromuscular layer and are 6 to 8 mm apart (Figure 36-23). This pattern may be less likely to induce adhesions than the Gambee method, and it is faster.[244]

A single-layer anastomosis with 3-0 PDS through the full thickness of the equine jejunum and wrapped with a sodium carboxymethylcellulose and hyaluronate membrane was faster than mucosal closure followed by a Cushing pattern.[336] Also, the single-layer anastomosis reduced lumen diameter from normal adjacent lumen by 35.6%, compared with a 44% reduction for mucosal closure and a Cushing pattern.[336] It also developed significantly fewer perianastomotic adhesions than a single-layer anastomosis without the membrane.[336] With this single-layer pattern, care must be taken to minimize mucosal eversion, because mucosa does tend to protrude between the seromuscular edges, which could lead to adhesion formation.[336]

Stapled Jejunojejunostomy

The major advantages of stapling instruments are speed, reduced tissue handling, improved tissue blood flow, and minimal contamination.[338] If the staple line is oversewn, the first two advantages are lost and the prevalence of adhesions becomes comparable to that with hand-sewn anastomoses in horses.[338] The most important disadvantage of staples is expense, and there is no evidence that anesthesia time is sufficiently shortened to compensate for this in colic surgery.[257,287] The security of the closure with staples can be affected by tissue thickness, and the 4.8-mm staples (green cartridge) are preferable to the 3.8-mm staples (blue cartridge) for equine gastrointestinal surgery.

With the exception of the end-to-end anastomosis instrument, which is too small for adult equine intestine, other available stapling instruments are used in a side-to-side manner. By necessity, side-to-side anastomoses disrupt the normal anatomic and physiologic polarity of the bowel. In canine jejunum, 91% of MMCs can cross an end-to-end anastomosis at 12 weeks after surgery compared with 56% at 2 years after a functional end-to-end anastomosis.[339] Stomal dilation and delayed obstruction have been reported after stapled side-to-side anastomoses in dogs and after a hand-sewn version of a side-to-side anastomosis in horses and rats.[130,339] In a study on dogs, the functional end-to-end anastomosis also altered intraluminal flora compared with a hand-sewn end-to-end anastomosis, evidence that it imposed some nonphysiologic changes on intestinal function.[339] In addition, increasing the number of raw edges (although inverted) and the number of suture lines and staple lines increases the number of potential foci of inflammation in a side-to-side anastomosis. In a retrospective study in horses, no difference was found between hand-sewn end-to-end anastomoses and stapled side-to-side and stapled functional end-to-end anastomoses with regard to short- and long-term survival, duration of surgery, or prevalence of postoperative ileus.[257] However, the stapled side-to-side anastomoses had a shorter duration of postoperative ileus than the others, a finding weakened by a lack of critical details about the types of hand-sewn anastomoses used in the study.[257] Also, a more recent study described an increased risk of postoperative ileus (POI) after side-to-side jejunojejunostomy.[340]

Side-to-Side Stapled Jejunojejunostomy

The isoperistaltic side-to-side technique involves creating blind stumps of the proximal and distal ends of the jejunum with either a Parker-Kerr technique (see Figure 16-8, *D*) or stapling instruments (see Figures 16-12 and 16-13).[244] The gastrointestinal anastomosis (GIA) instrument is used to create the stoma in the overlapped segments. This places two parallel rows of staggered staples on each side of a stoma cut simultaneously by advancement of the knife blade (Figure 36-24). If the GIA-50 is used, it is placed orad and then aborad through the same stab incisions on the antimesenteric side, effectively doubling the size of the stoma. Care must be taken to ensure that the staple lines overlap on the far side of the stab incisions or that any defect on that side is oversewn.[341] If the Multifire GIA-80 or ILA-100 is used, a single application is sufficient. The two mesenteric edges are sutured to the adjacent mesenteric surfaces at the points of overlap.

Functional End-to-End Stapled Jejunojejunostomy

With the functional end-to-end anastomosis, the bowel ends are lined up in antiperistaltic fashion, the stoma is created with the GIA instrument along apposing surfaces, and bowel ends are closed.[244,342] A closed, stapled, one-stage, end-to-end jejunojejunostomy can realign itself into an end-to-end configuration as it heals and did not cause adhesions in normal horses.[342] Intussusception of a functional end-to-end anastomosis has been reported in ponies at 2 and 26 days after surgery and in a horse at 8 months after surgery.[256,257]

Jejunocecal and Ileocecal Anastomoses

Jejunocecal and ileocecal anastomoses are indicated when a portion of the ileum is involved in a strangulating lesion. These techniques can constitute 36% to 68% of all small intestinal anastomoses.[91,245,343] Proponents of the side-to-side jejunocecostomy believe that it creates a larger stoma than is possible with the end-to-side technique and that it is associated with fewer postoperative problems from stomal edema.[130,344] In a study that compared the two techniques in clinical cases, 83% of horses with a side-to-side jejunocecostomy were discharged from the clinic, compared with 37% that had an end-to-side jejunocecostomy.[345] The latter also had more repeat celiotomies,

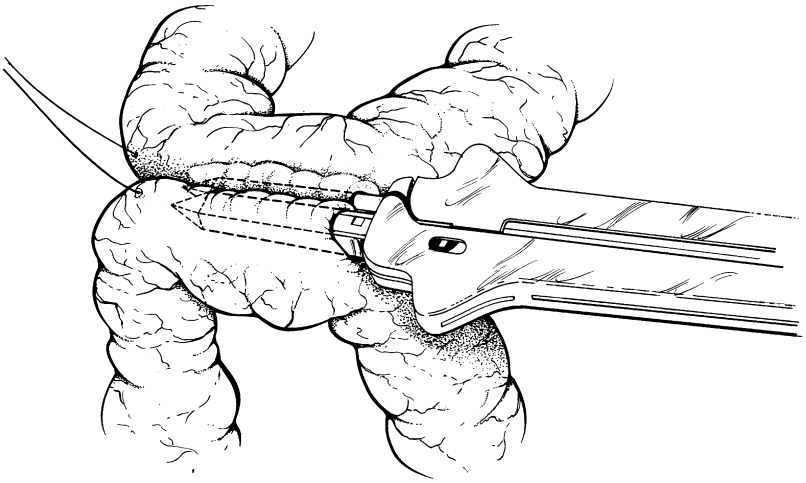


Figure 36-24. GIA-50 Premium stapler with disposable cartridge used for a side-to-side anastomosis. Stay sutures are placed at either end of the proposed anastomotic site to approximate the bowel segments side by side, and a stab is made into both bowel segments large enough to accommodate each fork of the instrument. The instrument forks are inserted fully to maximize stomal size. The stab incisions are apposed with an inverting suture pattern (not shown). (Copyright 1974, 1980, 1988, 1998, 2005 United States Surgical, a division of Tyco Healthcare Group LP. All rights reserved. Reprinted with permission of United States Surgical, a division of Tyco Healthcare Group LP.)

necessitated by such complications as bowel kinking at the anastomosis, adhesion formation, impaction, intussusception of the anastomosis, and volvulus or torsion.[345] An angled line of transection of the jejunum or ileum, with or without a longitudinal incision on the antimesenteric side, allows construction of a stoma of adequate size with the end-to-side technique.[244]

The mesentery and small intestine are prepared as for an end-to-end anastomosis except that the ileum is transected and oversewn using a Parker-Kerr technique (see Figure 16-8, *D*).[244] The ileal stump should be as short as possible, because an intussusception can form into the cecum over time,[346] and a long stump can progress into and obstruct the cecocolic orifice.[347,348] The ileal stump remains nonfunctional and therefore can tolerate more severe ischemia than would be acceptable in bowel that is expected to regain normal activity. One method proposed to reduce problems from an ischemic stump is to suture omentum over the suture line.[349]

For extensive ileal necrosis, the TA-90 can be used for a distal transection down to the ileocecal orifice.[350] The 4.8-mm staples are recommended for this purpose.[90] An incision is made in the mesentery dorsal to the ileal artery and in the ileocecal fold, and it is continued to the level of ileal transection to create a tract for passage of a loose ligature to that point.[350] This ligature is tied to compress all mesenteric tissues at the level of transection, including the ileal artery and vein. The disadvantage of this and other methods of deep ileal transection is that they create a large mesenteric defect that could entrap bowel.[350] An alternative method is to attach the ileum to a length of stomach tube that can be used to invert the necrotic stump into the cecum.[351]

End-to-Side Anastomosis

The stoma should be created as close to the base of the cecum as possible to reduce the gravitational effects of cecal contents.[244,352] The stoma is made midway between the dorsal band and the medial band, and the small intestine is aligned so that it is directed toward the base of the cecum (Figure 36-25).

A noncrushing intestinal clamp can be used to clamp off a pouch of the cecum; however, elevation of a pouch of cecal wall by upward tension on stay sutures or on Babcock forceps is preferable, because it is less traumatic and allows more proximal access on the cecal body. After the mesenteric and antimesenteric ends of jejunum are attached to the selected area on the cecum by simple-interrupted sutures of 3-0 PDS, a Lembert or Cushing pattern apposes the back side of the jejunum to the cecum. An incision is then made in the cecum 5 mm from the suture line that corresponds with the opening in the jejunum. The next row is a simple continuous, full-thickness pattern, interrupted at the mesenteric and antimesenteric borders before it is continued for 360 degrees. A Cushing pattern on the near side completes the anastomosis. The free edge of mesentery is sewn to the cecum and then to the ileocecal fold, ending at the oversewn end of the ileum and the previously gathered mesenteric stump (see Figure 36-25). Failure to close this mesenteric defect can lead to subsequent passage of small intestine through it, followed by obstruction and even strangulation.[271,353]

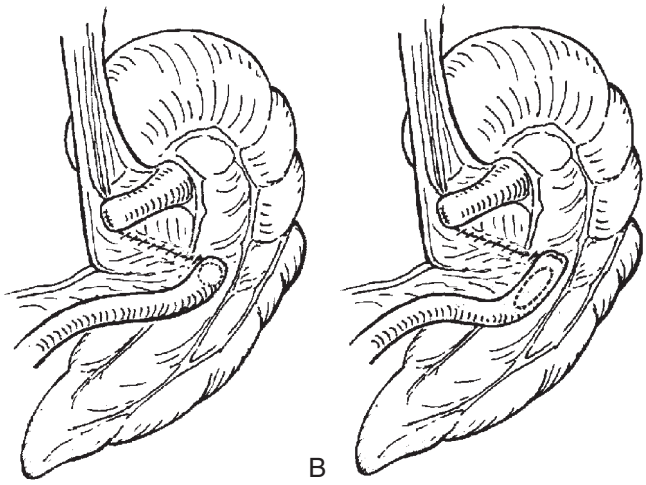


Figure 36-25. Completed end-to-side **(A)** and side-to-side jejunocecal anastomoses **(B).** In both methods, the ileum is oversewn by the Parker-Kerr technique and the mesenteric defects are closed. (From Freeman DE: Surgery of the small intestine. Vet Clin North Am Equine Pract 13:261, 1997, with permission.)

Side-to-Side Anastomosis

After the strangulated segment of small intestine is resected, the jejunum is transected and oversewn as for the ileum. The jejunum is attached to the selected area on the wall of the cecum (see "End-to-Side Anastomosis," earlier) with interrupted sutures of 3-0 PDS at the oversewn end and 10 to 12 cm (4 to 6 inches) proximal to this point (see Figure 36-25). Either a hand-sewn side-to-side method, GIA stapling instruments, or the cutting thread or saw technique can be used.[349,354] The saw

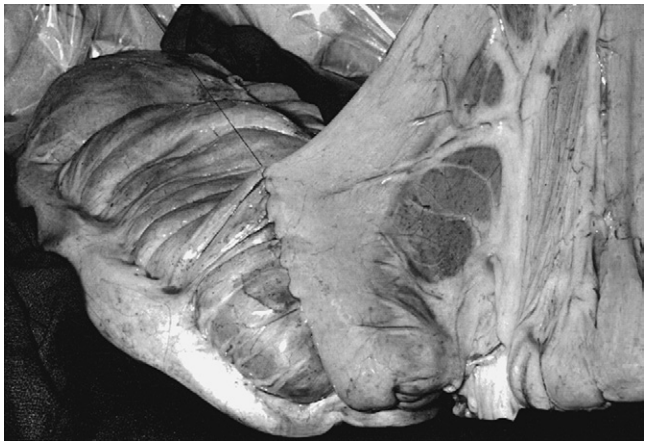


Figure 36-26. Side-to-side jejunocecostomy with the oversewn end of the jejunum in the foreground and facing the cecal base. The apparent divergence of the jejunum toward the medial band of the cecum is not real but the result of close positioning at the edge of the incision.

technique with wire or suture can effectively achieve a side-to-side anastomosis and reduces contamination more than the stapling method and without the cost.[354] A hand-sewn semi-closed single-layer jejunocecal side-to-side method has been shown to have some promise for this anastomosis.[355] Regardless of method, the oversewn end of the jejunum should not extend beyond the stoma or a stagnant loop will form, and it should be directed toward the cecal base (Figure 36-26). If the ileum is obstructed by a tumor, muscular hypertrophy, or chronic intussusception, an incomplete bypass can be used.[97]

The colon of horses with small intestinal strangulation is usually tightly contracted around dehydrated contents ("vacuum-packed" appearance), although it is not truly impacted in every case. Removal of these contents could eliminate some downstream resistance and provide a more favorable pressure gradient for emptying the small intestine into the cecum in horses with ileocecal or jejunocecal anastomoses.[356]

Jejunoileal Anastomosis

Jejunoileal anastomosis is an alternative to jejunocecal anastomosis (if sufficient ileum is available) and can be performed more quickly because fewer steps are required. The potentially greatest advantage over a jejunocecal anastomosis is preservation of normal anatomic and physiologic conditions in this critical part of the intestinal tract, particularly the ileocecal valve.[352] This anastomosis does have a tendency to obstruct,[244] although it functions very well in clinical use.[357] A two-layer anastomosis, with a simple-continuous pattern in the mucosa and a simple-continuous pattern in the seromuscular layer,[336] can prevent inversion and the associated risk of obstruction with this anastomosis.[357] In a study that compared an end-to-end jejunoileal anastomosis with an end-to-end jejunojejunal anastomosis in two groups of horses with similar preoperative parameters and types of lesions, postoperative complications and survival rates were similar between the two groups.[358]

Surgery of the Duodenum and Duodenal Bypass (See also Chapter 32)

Access to duodenum through a ventral median celiotomy is considerably easier in a foal than in an adult horse.[125,183] The abdominal incision should be extended to the xiphoid cartilage, and a J-shaped incision is required in an adult horse.[182] In addition, the table should be tilted to elevate the front end of an adult horse.[182] Access can be improved by retraction, packing of the abdomen with saline-soaked towels, and suction to remove some unavoidable contamination.[182]

Pyloric stenosis has been successfully corrected by pyloromyotomy, Heineke-Mikulicz pyloroplasty, gastrojejunostomy, or gastroduodenostomy.[125,182,183] For a side-to-side gastroduodenostomy, a hand-sutured anastomosis is preferred over GIA stapling, because stapled incision edges can heal together at the commissures of the stoma and cause stomal occlusion.[125] Additionally, stapling instruments are difficult to use in the limited space.[125] In one foal, a focal duodenal stricture was corrected by transverse closure of a full-thickness longitudinal incision that spanned the lesion.[359] Choledochojejunostomy and duodenojejunostomy have been used successfully in foals with obstruction of the common bile duct and duodenum at the level of the hepatopancreatic ampulla.[125] A duodenojejunostomy can be used to bypass a duodenal stricture beyond the hepaticopancreatic ampulla.[125] A side-to-side jejunojejunostomy can be performed distal to bypass procedures to prevent intestinal contents from stagnating in the blind loop between the obstruction and the stoma.[125] The contents of this loop can pass aborad through this stoma, without entering the stomach.

After surgical treatment of gastroduodenal ulcer disease in foals, the survival rate to hospital discharge is excellent and the long-term prognosis in restoring the patient to a reasonable level of performance is good.[360,361] Obstruction of the duodenum (rather than the pylorus), adhesions to the duodenum, and postoperative ileus were significantly associated with decreased long-term survival.[361] Factors that may improve survival compared with earlier reports include better case selection and performing the gastrojejunostomy with the jejunum oriented so that the oral end is to the left of the abdomen.[361]

A side-to-side gastrojejunostomy has been used in an adult horse with DPJ.[362] In protracted cases of DPJ that do not respond to appropriate medical therapy, bypass procedures have been developed to drain the affected segment of small intestine into more normal distal segments, so that the large volumes of fluids can be reclaimed by intestinal absorption. In one method, a temporary duodenocecostomy is performed through a 25- to 30-cm-long (10- to 12-inch-long) incision between the 17th and 18th ribs to approach the duodenum before it turns through its caudal flexure behind the base of the cecum.[363] Adjacent portions of duodenum and cecum are joined by the "cutting-thread" technique, which creates a stoma 2 cm in diameter. The goal is to allow the distended duodenum to drain into the cecum, with spontaneous closure of the fistula when the condition has resolved. A carbohydrate absorption test can be used during the recovery stage to determine if the stoma has healed spontaneously. A duodenojejunostomy through a ventral midline celiotomy is an alternative procedure for horses with DPJ and is effective in reducing gastric distention and reflux.[101] An adult horse that had a gastrojejunostomy for DPJ became lethargic for 1 to 2 weeks after a grain diet, suggesting that it had developed the "dumping syndrome," a metabolic consequence of rapid influx of gastric contents into the jejunum.[362] Although this horse did well for a follow-up period of 8 years after gastrojejunostomy, it had trouble maintaining body weight for the first year after surgery.[362]

Duodenal impactions can be treated successfully with massage of the intestinal contents into the stomach for removal

by gastrotomy or into the jejunum for manual disruption or removal by enterotomy.[193,197,199] Failure of these methods can be followed by enterotomy through an incision in the dorsal paralumbar space, 3 cm caudal to the 18th rib.[195] Location of this incision over the duodenal obstruction could be guided with some precision by ultrasonography.

Small Intestinal Enterotomy

A longitudinal enterotomy on the antimesenteric surface of the small intestine can be required for removal of impactions with food components, foreign material, or ascarids (see Figure 36-5) or to empty a segment of distended bowel to facilitate reduction of a strangulation in the epiploic foramen.[244] Methods to prevent adherence of intestinal contents to the serosa around the incision include constant lavage of the bowel with warm saline or precoating the proposed enterotomy site with sodium carboxymethylcellulose. The enterotomy can be closed with a single layer (Lembert or Cushing pattern) using 2-0 PDS on a taper needle, or in two layers, such as a simple-continuous pattern in the mucosa followed by an inverting pattern. Advantages of the Cushing pattern for the final row are that less suture material is exposed to the peritoneal cavity, and there is minimal inversion. Any closure method should minimize lumen reduction and risk for adhesions.

An enterotomy is indicated to a lesser extent in small intestinal surgery than in colon surgery, and it should be avoided if possible and if other options are available, to prevent creation of a focus for adhesion formation. As mentioned before, intestinal viability should not be assessed by mucosal inspection through an enterotomy made specifically for this purpose, and seromuscular changes can be used instead (see earlier). Extensive strangulating lesions can be corrected without removing fluid contents, although gas can be removed through a 14-gauge needle tunneled beneath the seromuscular layer and connected to a suction device. The tunnel staggers puncture sites in the different layers so suture closure is unnecessary. A focal impaction with dehydrated intestinal contents can be softened by injecting saline through an 18-gauge needle into its substance or by mixing it with more proximal liquid contents. If a resection is not indicated, distended small intestine can be decompressed by massaging contents into the cecum, from which they can be removed by typhlotomy.

AFTERCARE FOR SMALL INTESTINAL SURGERY

Routine use of an indwelling nasogastric tube does not ensure against gastric rupture in horses[96,364] and can delay gastric emptying of liquids in normal horses.[365] Therefore repeated passage of a nasogastric tube to decompress the stomach when indicated by clinical findings, such as pain and increased heart rate, might be preferable to an indwelling tube.

Most horses can be offered a small handful of good-quality hay within 18 to 24 hours after surgery and this is well tolerated.[244] This can be repeated at 3- to 4-hour intervals and the amount increased slowly to a full ration within 3 to 4 days. The refeeding process requires great care and close observation, but it can stimulate return of intestinal function.[244] A high-fiber diet stimulates intestinal propulsive motility, probably through increased intestinal bulk from the copious secretions evoked.[366] Hay in small amounts is the most potent accelerator of intestinal transit in horses, especially when divided into small meals throughout the day.[22] There is also growing evidence that healing of an anastomosis is enhanced by flow of intestinal contents through it, compared with inactivity or bypass.[367]

Broad-spectrum antibiotics and flunixin meglumine are administered before surgery and continued for 3 days afterward. Intravenous fluids are administered at 2 L/hr to a 450-kg horse for the initial 12 hours after surgery; the rate is subsequently adjusted for changes in PCV and total plasma protein (see Chapters 3 and 40).[356] Prokinetic drugs are used almost routinely by some surgeons or as needed by others.[244,356]

REPEAT CELIOTOMY

Repeat celiotomy is a lifesaving procedure and can be required for 12% to 27% of small intestinal diseases and for 4% to 12.5% of all colic surgeries.[90,91,133,139,245,353,368-371] The need for repeat celiotomy is far greater after small intestinal surgery than after large intestinal surgery, and it is greater after jejunocecostomy than after jejunojejunostomy.[139,350,370,372] Although making the distinction between postoperative ileus and mechanical obstruction from anastomotic impaction, ischemia, adhesions, or others can be difficult, horses with mechanical obstruction usually demonstrate a greater degree of pain and have a progressive increase in heart rate.[139,356,370] The median time to relaparotomy in one study was 48 hours, with 67% of horses undergoing surgery within 72 hours of the first procedure.[370] The most common findings at relaparotomy are ileus, obstruction or impaction at an anastomosis site, and secondary intestinal ischemia.[371]

Two important disadvantages of a second abdominal exploratory are the risk for incisional infection and the expense; however, the benefits of this procedure can outweigh the risks and disadvantages in most cases, including prompt termination of hopeless cases and salvage of others with treatable lesions.[133,139] Survival after a repeat celiotomy ranges from 36% to 56.4% and can be as low as 20% on a long-term basis.[90,91,139,245,368-371] However, in one study, 74% of horses that were recovered after a second celiotomy were alive at 1 year after the second surgery.[370]

COMPLICATIONS AND PITFALLS

The most commonly reported complication of small intestinal surgery in the horse is POI, although the clinical distinction between this and anastomotic obstructions (Figure 36-27) is difficult. Typically, a mechanical obstruction is associated with greater and more persistent signs of abdominal pain than with POI, but this is not a consistent finding, especially later in the postoperative course. Technical errors are not well tolerated in small intestinal surgery in horses and can account for many complications. In a study of 74 horses that recovered from general anesthesia after small intestinal surgery, technical errors and errors in judgment were responsible for 8 of 14 repeat celiotomies (57%) and for 7 of 11 deaths during hospitalization (64%).[139]

Anastomotic Obstruction

Mechanical obstruction of the anastomosis can be caused by impaction, hematoma, constriction, excessive shortening of the mesentery, and other errors in technique. Stomas of marginal size are prone to obstruction because they are further reduced by postanastomotic edema.[373] This would seem to especially

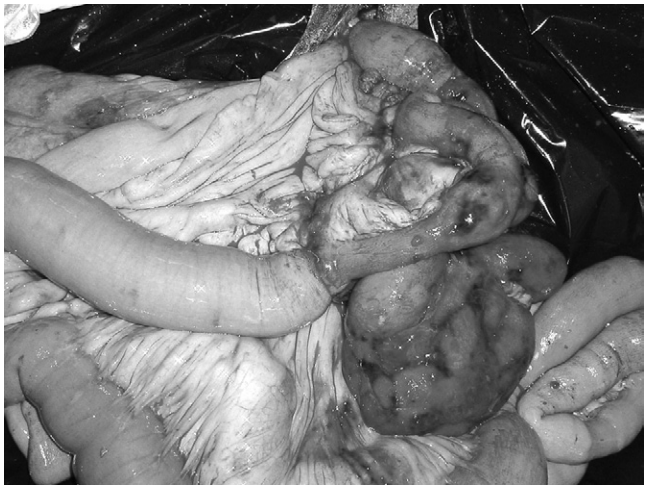


Figure 36-27. Effects of prolonged (5 days) anastomotic obstruction caused by excessive shortening of the mesentery at the anastomosis. Note the hemorrhage in the proximal bowel to the right and the fibrinous adhesions in the loops in the right foreground.

jeopardize a two-layer anastomosis with inversion, because this construction can reduce the lumen size by approximately 25%.[334] Prolonged obstruction of an anastomosis can lead to peritionitis and adhesion formation (see Figure 36-27).

Small intestinal volvulus may develop in distended jejunum and at points of small intestinal fixation, such as at a jejunocecal anastomosis. Risk of this complication is increased in a jejunocecal anastomosis by failure to decompress intestine during surgery, by decompression into the cecum, and by placement of the jejunum toward the apex of the cecum, which twists the jejunum 180 degees.[139] Intussusception of the anastomosis has been reported in horses and ponies after functional end-to-end anastomosis, hand-sewn anastomosis, and inverting closure of a transverse enterotomy[255-258,337,374]

Jejunocecal and ileocecal anastomoses are usually associated with more postoperative complications than jejunojejunostomy,[139,350,372] except when performed as a bypass procedure without resection.[178] Problems with jejunocecostomy with resection can be explained by the bypass of the ileocecal valve and by the creation of a sharp transition between intestinal segments of dissimilar functions.[244] The jejunum must overcome intracecal pressure to empty, without the coordinating mechanism of the ileum and ileocecal valve.[4,5,48] Another factor that could contribute to postoperative complications is the presence of adhesions to a devitalized ileal stump and mesentery. In addition, after a jejunocecostomy, all the remaining small intestine was subjected to distention before surgery that could delay return of function afterwards,[72,73] whereas only a portion of the remaining intestine was subjected to distention with jejunojejunostomy.

Postoperative Pain

Abdominal pain in the early postoperative period is one of the most comon early postoperative complications and has been reported in 28.2% of horses after a single surgery for treatment of colic.[340] The prevalence of postoperative pain is higher after small intestinal surgery (43%) than after large intestinal surgery, and it is higher after surgery for ischemic lesions than for simple obstructions.[340] It is usually associated with postoperative gastric reflux.[340] If postoperative pain persists especially after medication, and the pain level is regarded as moderate to severe, a repeat exploratory celiotomy should be strongly considered. This is especially true if pain is associated with postoperative endotoxemic shock and reflux. If a colonic impaction is supected, especially if identified at surgery, mineral oil (2 to 4 L as needed) should be given by stomach tube.

Postoperative Endotoxemia

Postoperative endotoxemia was diagnosed in 60% of horses after small intestinal resection in one study.[245] Injection of the oral mucous membranes, combined with depression and persistently high heart rate, would support this diagnosis. Postoperative endotoxemia would seem difficult to explain after the ischemic small intestine is removed and would suggest that a serious complication has developed in the anastomosis or proximal to it that would warrant a repeat laparotomy.

Postoperative Ileus

Postoperative ileus has a reported prevalence that varies from 10% to 55.6% and a mortality rate of 13% to 86%, accounting for 9% to 43% of all deaths after small intestinal surgery.[139,375,376] In three studies on POI in horses, high heart rate, high PCV, small intestinal involvement, increased duration of anesthesia, and increased duration of surgery emerged as significant risk factors.[376-378] Additional risk factors identified in another study were high PCV at admission, increasing age, and length of intestinal resection.[379] The prevalence of POI can be the same after surgery for ischemic lesions of the small intestine as it is after surgery for simple obstructions. In addition, prevalence can be the same in horses that have resection and those that do not.[380] In one study on small intestinal surgery, no horse developed POI after jejunojejunostomy, whereas 20% of horses that had a jejunocecostomy did develop POI.[139]

Although prokinetic drugs can be used for treatment (see Chapter 40), the role of mechanical obstruction must not be overlooked, because this delays the use of repeat celiotomy. Many horses with postoperative ileus might benefit from decompression at a second surgery.[90] Pelvic flexure enterotomy (see Chapter 37) may reduce the risk for POI,[377] although this is not a consistent finding.[378]

Adhesions

Adhesions have been documented in 6% to 26% of horses after small intestinal surgery, with a marked decline in prevalence over time.[90,133,139,327,343] Adhesions usually cause signs of intestinal obstruction in the first 2 months after surgery, but adhesion-related obstruction can arise at any time. Although foals are considered to be especially prone, adhesion formation in juvenile Thoroughbreds after surgery for lesions of all parts of the gastrointestinal tract has been reported at 8%, similar to the percentage reported for adult horses.[381,382] Foals aged 15 days to 6 months seem more likely than weanlings and yearlings to develop adhesions and require multiple surgeries.[382] Factors that could contribute to adhesions are postoperative ileus, ischemia, foreign material, serosal abrasion by towels, excessive handling of bowel, and use of large suture material. Intra-abdominal sodium carboxymethylcellulose (SCMC) at the end of surgery improved survival rate in one study, presumably by

preventing adhesions, although necropsy results were not provided to demonstrate the role of adhesions in postoperative deaths.[383] Also, the results might have been an artifact of experimental design, such as use of SCMC only on the most recent cases in the series, which could benefit from overall improvements in colic management, and disproportionately fewer resections in the SCMC group.[383]

Short Bowel Syndrome

Short bowel syndrome has been documented in horses after extensive small intestinal resection, although horses can tolerate loss of 10 m to 10.7 m of small intestine.[90,139,259,343,364] According to one study, resection of 60% or more of small intestine can lead to malabsorption.[384] A subsequent study demonstrated that resection of 70% of the small intestine of ponies is well tolerated.[385] Clinical experience supports this conclusion, although the limit for resection can vary among horses, and residual bowel can compensate over time.[139,259] Mural congestion caused by venous strangulation can increase the length of the affected segment of small intestine by as much as 36%, so some correction for an increase in length after strangulation is needed to avoid an overly pessimistic estimate of the risk for short bowel syndrome.[386] A correction factor that would apply to different severities of strangulation encountered in clinical cases is difficult to establish, and therefore assessment of the remaining length of small intestine could be a more meaningful predictor of postoperative absorptive capacity.[386] A minimal length of 4.5 m (15 feet) of remaining small intestine would be adequate in an adult horse, which approximates what would be left after resection of 75%.[90,386] Removal of longer segments does not appear to be well tolerated, although one 450-kg horse left with 3.7 m (12.3 feet) did not develop short bowel syndrome, which is evidence of a tremendous individual variation.[139]

Other potential complications of extensive small intestine resection are liver disease and anorexia.[244,384] Bypass of the ileocecal junction by jejunocecal anastomosis without resection can allow cecal bacteria to seed the small intestine and cause an abnormal xylose absorption test in ponies, which is apparently not the case in horses.[384,387] Although based on findings from laboratory animals, postoperative dietary recommendations that may apply to horses with short bowel syndrome include complex nutrients as potent stimulants of intestinal adaptation, and fiber to increase colonic production of short-chain fatty acids (see Chapter 6).[386] Carbohydrates should be fed sparingly, as they are more likely than fats and proteins to induce osmotic diarrhea.[386]

Miscellaneous

Anastomotic dehiscence and peritonitis were rare in most studies, but they were a leading cause of death when staples (oversewn or reinforced with suture) were used for all small intestinal anastomoses in horses in one study.[178] Failure to effectively ligate mesenteric vessels can lead to postoperative hemorrhage. Occlusion or kinking of vessels or postoperative thrombosis of mesenteric vessels for unknown reasons can cause anastomotic necrosis.[139] Mesenteric rents at sites remote to the anastomosis have been described as causes of recurrent small intestinal strangulation and death years after the original surgery.[90,271] Other complications include pyrexia, jugular vein thrombosis, long-bone fracture, cecal impaction, myositis, gastric ulceration, recurrent colic, incisional infections, diarrhea, and pulmonary aspergillosis.[90,121,245-247,271,388] Horses that are treated for abdominal pain and require surgery have a lower prevalence of gastric ulceration compared with those that respond to medical therapy.[164] A concurrent large intestinal displacement can be found at surgery for a small intestinal lesion, or large intestinal distention or impaction can develop after small intestinal surgery.[89,139]

PROGNOSIS

Reported survival rates to discharge range from 80% to 94.4% in horses that had surgery of the small intestine and were allowed to recover from anesthesia.* Horses with lesions involving the small intestine tend to have lower survival rates than horses with diseases of the small or large colon.[380] There is strong evidence that survival rates in recent decades have improved over previous ones,[249,343,330] which might reflect earlier referral and improvements in surgical technique and postoperative care. These same improvements probably also reduce the severity of complications such as endotoxemia, jugular thrombosis, postoperative ileus, and wound infection.[247] Colic is one of the most common complications after discharge of horses that had small intestinal surgery, especially those that had resection and postoperative ileus, most likely the result of adhesion formation.[390]

In horses with small intestinal strangulating lesions, variables associated with low survival rates are high heart rates, long capillary refill time, pale mucous membranes, severe hemoconcentration, abnormal color of peritoneal fluid, exudation of cells and protein into peritoneal fluid, and failure to achieve a mean arterial pressure of at least 100 mm Hg.[249,330,391] After small intestinal resection, postoperative factors that were associated with nonsurvival were ileus, heart rate greater than 60 beats per minute at 24 hours, postoperative colic, and repeat celiotomy.[245,383] In 143 horses with entrapment in the epiploic foramen, survival was 75.6% if surgery was performed within 8 hours from the onset of colic, compared with 45.1% survival in horses that underwent surgery 12 hours or more after onset.[91] Multiple logistic analysis demonstrated that duration of surgery was not a factor associated with short-term survival in horses that had small intestinal resection, in contrast to its negative effect on survival when all types of colic are considered.[245,392] In two studies on small intestinal surgery, surgeons with more experience had significantly better survival rates than less experienced surgeons.[139,249]

In horses with colic from small intestinal disease, survival after simple obstruction can be similar to survival after strangulation obstruction[133,139] or worse.[380] Although length of resection does not appear to influence outcome, a trend toward lower survival has been reported after resection of greater than 2 m of intestine.[245,343,330,391] The length of bowel strangulated in the epiploic foramen was longer in nonsurvivors than survivors in another study.[91] Lesions that appear to have an adverse influence on survival are strangulation in the epiploic foramen and in mesenteric rents, and ascarid impactions.[156,246,271,392] Some of the increased risk associated with strangulation in the epiploic foramen can be explained by an associated lower preoperative total protein and longer duration of surgery in these horses.[391] Survival tends to be better after a jejunojejunostomy than after a jejunocecostomy or ileocecostomy, and jejunocecostomy is associated with increased likelihood of postoperative

*References 89, 130, 139, 178, 229, 234, 257, 379, 389.

abdominal pain and repeat celiotomy.* Volvulus at the anastomosis site, infarcted jejunum at the anastomosis site, and technical errors accounted for most complications from jejunocecostomy in one study,[139] whereas problems with the ileal stump caused 47% of surgery-related complications in another.[372] However, survivals after jejunojejunostomy and jejunocecostomy become similar over the long term.[139] Survival after jejunocecostomy or ileocecostomy without resection is better than after the same procedures with resection.[178] Correction without resection, as by reduction, bypass, enterotomy, decompression, or "kneading," has a better prognosis than after resection and anastomosis.[136,139,178,249]

Both hand-sewn and stapled jejunocecal anastomoses have similar long-term survival rates, although both types of anastomosis have inferior long-term survival and greater cumulative probability of developing colic than jejunojejunostomy.[393] Another study confirmed that short-term and long-term survival were comparable between hand-sewn and stapled jejunocecostomies (the latter not oversewn), and demonstrated that the probability of survival for up to 90 months was greater than 50% for both methods.[389] However, horses in the stapled group had a significantly greater prevalence of postoperative colic and combined postoperative colic and reflux than horses in the hand-sewn group during hospitalization.[389] They also tended to have greater need for repeat celiotomy and a greater prevalence of colic after discharge, although both of these differences were not significant.[389]

The survival rate after completed surgery for small intestinal strangulating lesions, including treatment by jejunojejunostomy, is similar for horses 16 years or older compared to mature horses, although there tends to be an overall decrease in survival after surgery with increase in age.[85] However, geriatric horses presenting with colic are more likely than mature horses to be subjected to euthanasia without surgery or during surgery,[85,87] probably because of a misconception about prognosis based on age, short remaining life expectancy after surgery, financial constraints, or presence of concurrent disease (e.g., Cushing's disease). Draft horses weighing more than 680 kg that have colic surgery can have longer durations of anesthesia, more postoperative complications, and higher mortality rates than draft horses weighing less than 680 kg.[394] Heavy draft horses that undergo small intestinal surgery have a worse prognosis for short-term survival than those undergoing large intestinal surgery[394] and poorer survival rates than reported for small intestinal surgery in predominantly light breed horses.[139] Small intestinal obstruction is rare in American Miniature Horses,[395] but aftercare of these horses and ponies is complicated by their high risk for developing hyperlipemia or hyperlipidemia.

In a report on foals with surgical colic, only 10% of those younger than 14 days survived to maturity compared with 45.8% of foals 15 to 150 days old.[135] In one study, 59% of foals that had surgery for colic survived to discharge, with 25% being euthanized while under anesthesia.[124] The survival for foals that had a small intestinal lesion (46%) was worse than for those with large intestinal lesions (80%).[124] Survival in foals is also greater after nonstrangulating lesions or simple obstruction than after strangulating obstruction.[124,137]

The pattern of survival after surgery for horses with small and large intestinal colics follows a triphasic pattern.[392] Most deaths occur during the first 10 postoperative days, with 69% during the first 100 days after surgery and a slower decline in death rate after that.[392] A study on horses that had small intestinal surgery confirmed this observation and also demonstrated that the risk for death from surgery-related problems diminishes markedly after 12 months.[139]

*References 136, 139, 245, 249, 330, 343, 350, 353, 373, 379, 380, 392.

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CHAPTER
37 Large Intestine

Peter C. Rakestraw and Joanne Hardy

CECUM

Anatomy and Physiology

The cecum is positioned between the small intestine and the large colon and located primarily on the right side of the abdomen.[1] It is a large cul-de-sac with an average length of 1.25 m and an average capacity of 30 L.[1] It has a comma-shaped appearance and is divided into three parts—the base, body, and apex (Figure 37-1). The base, which is the most dorsal part, is positioned in the right iliac and sublumbar region. It has a greater curvature dorsally and lesser curvature ventrally.[2] It extends craniad to the 14th or 15th rib, forming a blind end pointing ventrad. A transverse fold arising from the floor of the cecal base craniad to the ileocecal junction divides the base into a cranial and a caudal portion.[3] The cranial part of the base is developmentally part of the ascending colon and is called the *cupula*.[1-2] The cranial and caudal parts of the cecal base appear to function separately. The body of the cecum travels cranioventrad from the cecal base. Caudally, it lies against the right flank moving to a slightly more medial position as it extends craniad along the ventral body wall, narrowing toward the cecal apex, which is situated between the right and left ventral colon approximately 20 cm (8 inches) caudal to the xiphoid cartilage.[1-2] The cecal base is attached dorsally to the ventral surface of the right kidney, to the right lobe of the pancreas, and to a part of the abdominal wall caudal to these structures.[1-2] The cecal base is attached medially to the transverse colon and the root of the mesentery, from which the vascular supply reaches the cecum.

The body of the cecum has four longitudinal bands (teniae) located on the dorsal, ventral, medial, and lateral surfaces. The dorsal and medial bands end at the cecal apex. The ventral band usually joins the medial band near the cecal apex, and the lateral band may extend to the apex or fade out before reaching the apex.[1] The ileocecal fold runs from the antimesenteric border of the ileum to the dorsal cecal band. The cecocolic fold (ligament) runs from the lateral cecal band to the lateral free band of the right ventral colon.

The cecal blood supply derives from the cecal artery, a branch of the ileocolic artery. The cecal artery divides into the medial and lateral cecal arteries, which run with the corresponding veins in the medial and lateral cecal bands, respectively. The medial cecal artery is the major vascular supply to the cecal apex. The cecum is thought to be at risk for thromboembolic disease, since both cecal arteries arise from a single vessel without collateral circulation, and since the lateral and medial cecal arteries have minimal mixing of their two circulations.[4] However, microvascular studies indicate that there is a cecal rete arising from each of the cecal arteries that supplies an extensive submucosal plexus and that may provide an alternative blood supply in the event of a cecal embolus (Figure 37-2).[4]

The ileocecal orifice is located in the lesser curvature of the cecal base approximately 5 to 7 cm to the right of the median plane and at the level of the first or second lumbar vertebra.[1] The end of the ileum is partly telescoped into the cecum, which positions the ileal orifice in an elevation of an annular fold of mucous membrane. The annular fold contains a network of veins, which can distend the annular fold when engorged.[2] In this way, the ileocecal orifice is narrowed, preventing reflux of cecal contents back into the ileum. The lack of this functional sphincter may contribute to cecal reflux into the small intestine after a jejunocecostomy. The cecocolic orifice is located distal to

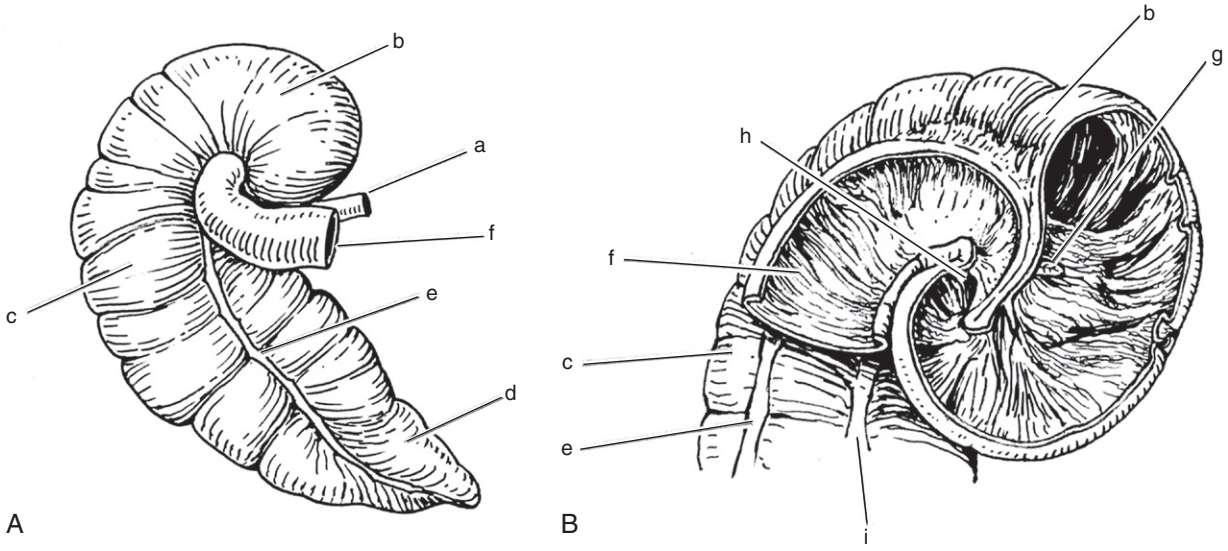

Figure 37-1. A, Cecum of the horse, right lateral aspect. **B,** Base of the cecum and the proximal part of the ascending colon (right ventral colon) of the horse, opened laterally to show the ileal and cecocolic orifices, fixed *in situ,* lateral aspect. *a,* Ileum, elevated; *b,* base of cecum; *c,* body of cecum; *d,* apex of cecum; *e,* lateral teniae; *f,* proximal part of right ventral colon; *g,* ileal orifice on papilla; *h,* cecocolic orifice; *j,* dorsal teniae with ileocecal fold.

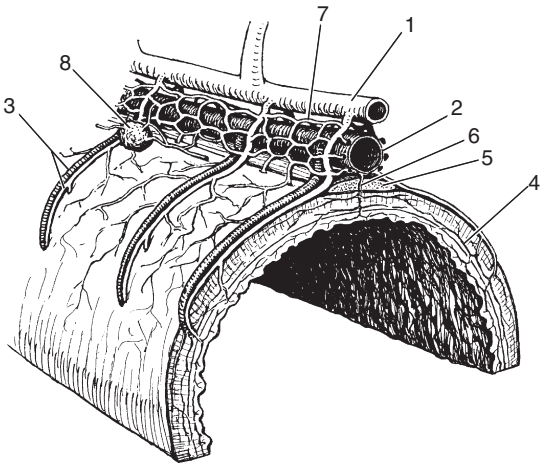

Figure 37-2. Schematic diagram of the cecal arterial rete that forms a meshlike network around the cecal veins before continuing on into the submucosal network plexus. The extensive rete and submucosal network may provide an alternative route for blood flow in the case of an arterial embolus. *1,* Marginal artery; *2,* marginal vein; *3,* secondary arcade; *4,* long artery and vein; *5,* teniae; *6,* branch supplying the teniae; *7,* vascular rete; *8,* lymph node and lymph vessels (shown only at one place).

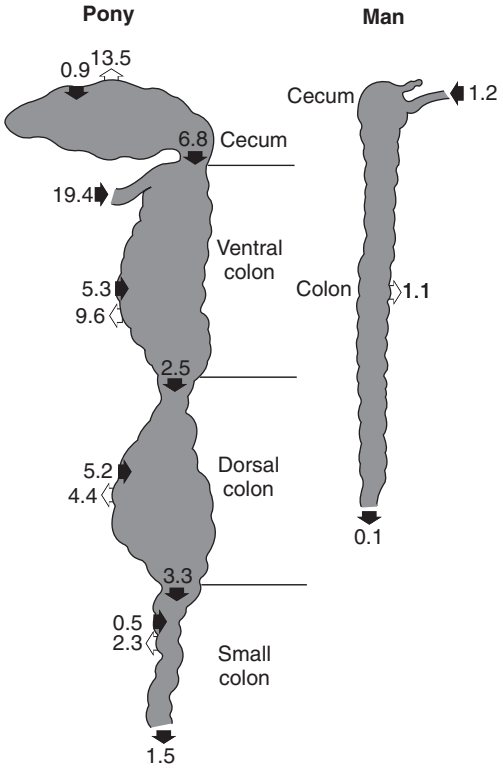

Figure 37-3. Net water movement through the large intestine of a 70-kg man and a 160-kg pony. Both species absorb approximately 90% of ileal outflow, but in a pony, this represents approximately 30 L/day. The volume is roughly equivalent to the animal's extracellular fluid volume. Note that the cecum of the pony has the greatest net water absorption. (Redrawn from Argenzio RA, Lowe JE, Prickard DW, et al: Digesta passage and water exchange in equine large intestine. Am J Physiol 226:1035, 1974.)

the ileocecal orifice. Because the cranial cecal base curves ventrally and caudally, the cecocolic orifice is positioned caudal and lateral to the ileocecal orifice (see Figure 37-1, *B*). It lies between two folds of tissue, which form the cecocolic valve. There is no sphincter at the cecocolic orifice.[2]

The two primary physiologic functions of the cecum (as well as of the large colon) are to absorb electrolytes and water and to serve as a site of microbial digestion.[5] The large intestine of a 160-kg pony reabsorbs a total of approximately 30 L of water a day, a volume equivalent to its extracellular space.[5] The cecum appears to be the site of greatest quantitative net water absorption (Figure 37-3). In an experimental model in ponies, the cecum demonstrated a net transmucosal influx of water during the first 2 hours after a meal, followed by a net absorption of approximately 600 to 800 mL/hr during the next 10 hours between meals.[6] Net increases in daily absorption of water from the ventral and small colon can compensate for decreased cecal water absorption.[6] Microbial digestion is the other important physiologic function of the cecum. A significant amount of soluble and most of the insoluble (e.g., cellulose, hemicellulose) dietary carbohydrate is digested by microbial enzymes in the cecum (and large colon) with production of organic acids (volatile fatty acids).[5] The marked decrease in bicarbonate in the cecum is because of the buffering effect of the organic acids. Although osmotic changes in organic acid (or volatile fatty acid) production has been speculated to control net water movement, sodium transport is probably more important.[5-6]

Cecal Motility

The transit time for liquid and particulate markers through the cecum is relatively rapid compared with that in the large colon.[5] There does not appear to be any retrograde movement of ingesta through either the ileocecal or the cecocolic orifice. Several studies have described normal cecal motility by examining cecal myoelectric activity patterns.[7-9] Ingesta enters the cecum from the ileocecal orifice propelled by the migrating action potential complex, a rapidly progressive electrical event extending from the ileum to the cecum. In the cecum, four different patterns of activity have been characterized. Three are associated with mixing of cecal ingesta: pattern I begins at the cecal apex and is conducted to the cranial base, and patterns II and III begin at the caudal and cranial cecal base, respectively, and are conducted to the cecal apex.

Pattern IV, a progressive pattern, begins at the cecal apex and is conducted through the cecal base and cecocolic orifice and into the right ventral colon (RVC). Pattern IV occurs once every 3 minutes in the fed horse, is associated with a loud distinctive "rush" of digesta (heard on auscultation), and is probably responsible for the transit of digesta from the cecum to the RVC.[7,9] In endoscopic studies of cecal emptying, ingesta was seen to move from the body up to the cranial aspect of the cecal base (cupula), which was separated from the caudal base by a ring of constriction. The cupula then contracted and the cecocolic orifice was elevated and opened, allowing ingesta to flow into the RVC.[10-11] In the RVC, both aborally directed propulsive spike bursts and orally directed retropulsive spike bursts were seen. The orally directed spike bursts may allow the RVC to function as a reservoir. The oral spike bursts were not propagated onto the cecum. An electrical pacemaker region is thought to be located near the cecal apex.[10] There is also indication of possible myoelectric coupling between the ileum, cecum, and RVC.[10,12] Abnormal cecal motility is thought to contribute to

several cecal problems, most notably cecal impaction caused by cecal dysfunction.

Cecal motility has been shown to be altered by pharmacologic agents. Xylazine hydrochloride (0.5 mg/kg IV) depresses the progressive motility pattern and mechanical activity for 20 to 30 minutes, and it prolongs cecal emptying.[12-16] Butorphanol tartrate (0.04 mg/kg IV) also depresses the progressive cecal motility pattern for 10 minutes.[15-16] The combination of xylazine and butorphanol prolongs the inhibition of spike bursts.[15-16] Neostigmine methylsulfate (0.025 to 0.033 mg/kg IV or SC) produces a significant increase in the frequency of progressive cecal motility and rate of cecal emptying.[14,16] Bethanechol chloride (0.025 mg/kg IV) increases the rate of cecal emptying.[14] Erythromycin lactobionate (1.0 mg/kg or 0.10 mg/kg) administered as an IV infusion over 60 minutes increases the rate of cecal emptying.[17] All of these studies have been performed in normal horses, so caution should be exercised when extrapolating the results to horses with pathology of the cecum.

Cecal Impaction

Pathogenesis

The most common pathologic condition of the cecum is cecal impaction, and it accounts for between 40% and 55% of cecal disease, 5% of all intestinal impactions, and 2% of all referral colics.[18-21] Fatalities as high as 43% have been reported, primarily because of perforation or rupture.[20-21] The etiology of cecal impaction is most likely multifactorial. Suggested predisposing factors include poor dentition, feeding of poor-quality roughage, decreased water intake, parturition, and parasite-induced thromboembolism.[22-27] In a recent study, feeding coastal hay appeared to be a risk factor for horses developing cecal impactions.[28] In this study the incidence of cecal impaction was 4.1 % of all horses examined because of abdominal pain compared to 2% in previous studies.[21] The increased incidence was thought to be related to the feeeding of coastal hay. Tapeworms *(Anoplocephala perfoliata)* located at the cecocolic orifice have been associated with cecal impaction and perforation, possibly as the result of disruption of motility.[29-31] In one study, Arabian, Appaloosa, and Morgan horses, as well as horses older than 15 years, were at an increased risk for developing cecal impactions, whereas in another study, age (older than 13.2 years) but not breed was a risk factor for cecal impaction.[19]

Another predisposing factor for cecal impaction, which may lead to cecal perforation, is hospitalization or prior surgery (within the previous 5 days). Often these horses are hospitalized or have surgery for reasons not related to the gastrointestinal tract, such as elective arthroscopy.[23,28,32-35] Some of these impactions may be related to a motility dysfunction. During and after general anesthesia, motility of the gastrointestinal tract is disrupted, with the cecum taking the longest time to return to normal function.[36] Use of nonsteroidal anti-inflammatory drugs (NSAIDs) and lack of exercise have also been associated with the development of cecal impaction or rupture in these cases.[19,34,37] The association of NSAIDs with cecal rupture has been attributed to masking of gastrointestinal pain associated with cecal impaction, exacerbation of existing ulceration associated with concurrent disease, and primary ulcer development.[3,19,24,38] There is also a group of horses that develop cecal "impactions" that do not appear to have a mechanical obstruction. In these horses, the accumulated ingesta is of normal or often fluid consistency instead of the firm, dry, compacted ingesta typically associated with feed impactions. A functional motility obstruction has been suggested in this group of horses. A disruption in the progressive motility pattern, which starts at the cecal body and propagates aborad to the cecal base and into the right ventral colon, would lead to accumulation of ingesta in the cecum.[3,8-9,19,24,38] Although altered blood flow secondary to parasite damage, dietary changes, and other clinical conditions has been hypothesized to disrupt the cecal pacemaker located at the cecal apex and to result in interruption of progressive motility, no predisposing cause can be found in many of these cases.

Chronic recurrent cecal impaction has also been associated with hypertrophy of the muscle layers in the cecal base or body.[39] The muscular hypertrophy is thought to result from chronic uncoordinated hypercontractility because of neuronal deficits in the myenteric plexus of the cecal base.[39-40] Ingesta accumulates in the cecum as a result of failure of the motility patterns to move ingesta from the cecum into the RVC. These horses have chronic weight loss with mild signs of colic. To our knowledge, this type of cecal disease has not been recognized in the United States.

Clinical Signs and Diagnosis

Often, horses with cecal impaction show only mild signs of pain, with intermittent periods of increased severity, that may continue for several days to several weeks.[19,23,28,41-42] The mild signs of colic include lying down, looking at the flank, decreased appetite, and depression. The heart rate is often normal to slightly elevated. Borborygmi are decreased, and feces may be soft with decreased production. Because gas, fluid, and some ingesta may pass through the cecum over the impaction, the small intestine proximal to the cecum is usually not distended. Although some horses may be presented with or may develop moderate to severe pain as the impaction progressively worsens, some affected horses remain relatively comfortable as the cecum becomes tightly distended. Horses with cecal impaction may be presented with cecal rupture with no history of significant abdominal pain.[3,19,38] There are reports of horses with cecal impaction deteriorating so rapidly, even when being monitored in a hospital environment, that surgical intervention before cecal rupture was not possible.[23-24,34] Laboratory evaluation is often normal in horses with cecal impactions. Although changes in peritoneal fluid may help in determining when and if surgical intervention should be performed, they are not always a sensitive predictor of cecal deterioration and may occur too late to be of any value in the decision-making process.[19,23-24,28,35]

The diagnosis is confirmed by rectal palpation in most cases. The first indication of a cecal impaction may be increased tension in the ventral cecal band. As the impaction enlarges, the cecal body begins to fill and the sacculations in the cecal body and a rounded cecal base are palpable. With increasing filling, the sacculations disappear and a large distended structure can be felt filling the right side of the abdomen. Because the cecum is attached dorsally to the body wall, the examiner is not able to pass a hand dorsally over the impaction.[38] This may help differentiate a cecal impaction from a large colon impaction. However, rectal palpation in some cases is not definitive in distinguishing between a distended cecum and a distended colon.[26] Additionally, some horses have cecal impaction involving only the cupula. Since the cupula of the cecum lies more

craniad than the body, these can be missed on rectal palpation.[39] Cecal impaction and rupture has also been reported to occur in foals, especially during the immediate postoperative period.[35] Since their size prohibits rectal palpation, the presumptive diagnosis depends on close attention to clinical signs.

Primarily on the basis of rectal palpation findings, cecal impactions have been divided into two types of obstruction.[21,43] One type has firm, dry, or doughy ingesta at the base or body of the cecum. The mass can be indented with the fingers on rectal palpation, and the cecal wall does not feel thickened. Peritoneal fluid is usually normal in these horses. These cases are thought to include the mechanical obstructions and will be referred to as type 1 cecal impactions. In the other type of cecal impaction, referred to here as type 2 cecal impactions, the cecum is tightly distended with gas and ingesta of normal or fluid consistency. These have been described as cecal dysfunction.[21,43] These horses may have an increased level of pain and an increased heart rate compared with type 1 cecal impactions. They may also show signs of endotoxemia. The cecal wall is usually tightly stretched and may feel thickened. Peritoneal fluid may have elevated protein, and as the disease progresses it becomes serosanguineous. At surgery, the cecal wall appears thickened and hyperemic, and it has decreased motility.[21,43] Although this categorization may be helpful in selecting a treatment modality (see "Treatment," next), it is not without limitations. First, it can be difficult to place a horse accurately into one of the two categories, even with physical, rectal, laboratory, and surgical findings. Second, although the progression of the disease and the response to treatment modalities may be more similar between horses within a group than between horses in different groups, clinically there are significant exceptions.

Treatment

The treatment of cecal impactions is quite controversial. Some reports suggest that cecal impactions can routinely be successfully treated with medical therapy, whereas other reports recommend surgical intervention.[3,23-24,28,38,42] To differentiate horses that will respond to medical therapy from those that will not, attempts have been made to correlate the characteristics of the impaction (the nature of the material in the cecum) with the response to medical therapy. Type 1 cecal impactions, thought to be mechanical obstructions from impacted ingesta, should be more likely to respond to medical therapy.[21,43] Type 2 cecal impactions, thought to be caused by a cecal motility dysfunction, should be less likely to respond to medical therapy and so be more likely to require surgical intervention. Although type 1 cecal impactions can initially be treated with medical therapy, some may not respond. Since these horses may not show much pain at any stage of the disease, they should be carefully monitored by rectal examination to determine which ones are not responding.

Another area of controversy is whether to perform a surgical bypass of the cecum. This question arose out of a retrospective study that identified horses that had recurrent cecal tympany or impaction after a prior typhlotomy without bypass to treat a previous cecal impaction.[44] The nature of the impaction has been used to help with this decision. Type 1 impactions should be more likely to respond to decompression only, whereas cecal dysfunction, the type 2 impactions, should be more likely to require a bypass. However, this has been questioned by some authors.[26] In our opinion, cecal impactions that occur in the postanesthetic period can be initially treated medically if they are diagnosed early while the cecum is mildly to moderately distended. However, it should be stressed that the clinical signs may be mild and their clinical significance underestimated. In a retrospective study of cecal perforation, 13 of 66 horses with cecal disease developed cecal perforation while in a hospital setting without having a prior diagnosis of cecal disease. All of these horses had some sort of painful condition, many related to a musculoskeletal condition. Seven of these 13 cases had general anesthesia several days before cecal perforation. Although these horses exhibited signs such as reduced fecal output and mild abdominal pain, the signs were not recognized as clinically significant before cecal perforation or sudden death. These horses were found to have a large and firm cecum filled with ingesta and an empty colon at necropsy.[45] In another study, 6 of 12 horses that developed cecal impaction while hospitalized survived, 2 of 4 treated medically and 4 of 6 treated surgically.[28] Increased awareness of the risk of cecal impaction and rupture in hospitalized horses leads to earlier diagnosis with improved outcome. If surgery is required for evacuation of postoperative cecal impactions, or if horses develop cecal impaction while hospitalized for other conditions, most should not need a bypass. The logic is that the dysfunction is temporary and related to anesthesia (for the horses who had surgery) or to a painful condition, to treatment with NSAIDs, or to a change in management, such as decreased exercise, and therefore the patients should respond to a more conservative surgical treatment (i.e., evacuation only).[28,36,43] These horses should be held off hay for the first 36 to 48 hours postoperatively, exercised with hand-walking and limited grazing to stimulate motility, and slowly reintroduced to a more laxative diet.

MEDICAL THERAPY

The goal of medical therapy in type 1 impactions is to soften the ingesta to allow cecal contractions to empty the cecal contents into the right ventral colon. Feed should be withheld and the horse started on intravenous fluid therapy. Oral laxatives administered by nasogastric tube may assist in softening the impaction. Mineral oil (5 to 10 mL/kg every 12 hours) is commonly used as a lubricant but may have difficulty penetrating the mass. Magnesium sulfate (1 mg/kg) is a saline laxative that exerts an osmotic effect, pulling water into the intestinal lumen. It can be administered once or twice a day for 2 to 3 days if the horse is well hydrated. Magnesium toxicity has been reported after the use of magnesium sulfate in dehydrated horses and in horses that have been treated with a combination of magnesium sulfate and dioctyl sodium sulfosuccinate (DSS), which is thought to increase the absorption of magnesium.[46] Psyllium hydrophilic mucilloid (1.0 kg every 6 to 8 hours) has also been recommended for treatment of cecal impactions.[38] Because complete fasting causes cessation of cecal motility, stimulating motility by walking and limited controlled grazing may be beneficial.

Analgesics such as flunixin meglumine (0.5 to 1.1 mg/kg IV every 12 hours) can be used if necessary for pain relief in both types of impactions. Caution should be exercised in using xylazine and butorphanol, since both drugs have been shown to decrease cecal contractile activity and their analgesic effect may inappropriately delay the decision for surgical intervention.[13,15-16] There is no consensus on the use of prokinetics. Erythromycin, bethanechol, and neostigmine have all been shown to increase contractile activity in the normal cecum as

stated previously.[16-17] Given the apparent large force of drug-induced activity in the normal cecum, some authors do not recommend the use of neostigmine in horses with cecal impaction.[14] The clinical efficacy and safety of bethanechol and erythromycin for the treatment of cecal impaction are not known.

Although separation into type 1 and type 2 cecal impactions provides some guidance in choosing which cases are more likely to respond to medical therapy, horses undergoing medical therapy for cecal impaction remain at risk for developing cecal rupture.[24,38] Consequently, close monitoring of their physical status, including heart rate and level of pain, as well as transrectal monitoring of the impaction are imperative when treating any cecal impaction medically. Surgery should be considered if there is no improvement on rectal palpation during a 24- to 36-hour period, if there is any sign of systemic deterioration, if there is a significant increase in pain, or if the cecum feels tight enough to rupture.[38] Mean systemic packed cell volume (PCV), peritoneal total protein concentration, and nucleated counts have not been found to be very sensitive indicators in determining whether a horse should be treated medically or surgically.[28]

The prognosis with medical therapy is difficult to assess from previous reports. One study reported a good prognosis for horses with cecal impactions treated with medical therapy when they lived longer than 24 hours after admission.[38] However, horses that may have been treated medically initially and that developed cecal rupture before the 24 hours were not included in the medically treated group. Consequently, it is difficult to interpret the significance of the reported survival rate (89%) of medically treated cecal impactions. Nine of 10 horses (90%) and 44 of 54 (81%) were treated successfully medically in two other studies. In the second study, surgical treatment was recommended and refused by the owners in 5 of 10 horses treated unsuccessfully medically.[28] In another study, medical therapy alone was unsuccessful in 12 out of 21 horses with cecal impaction.[23] Poor results with medical therapy have been reported by other authors.[3,24]

SURGICAL THERAPY

The ventral midline celiotomy is the most common approach for surgical access to the cecum. Because the cecal base is firmly attached to the dorsal body wall, only the apex and a portion of the body of the cecum can be exteriorized. The entire base (including the cupula), a portion of the cecal body, and the cecocolic and ileocecal orifices will remain within the abdominal cavity. A right paracostal approach made through an 18th rib resection has been described for complete typhlectomy or enlargement of the cecocolic orifice in horses with cecal impaction associated with cecal hypertrophy.[47-48] In Europe, enlargement of the cecocolic orifice has met with limited (50%) success in treating this particular type of cecal disease.[39]

Surgical options are infusion of fluid with massage of cecal contents, typhlotomy with evacuation of cecal contents, and bypass procedures including cecocolic anastomosis, ileocolostomy, and jejunocolostomy with and without ileal resection.[23,44,49-50] Poor to good results have been reported with infusion combined with massage.[23,44] This technique has been of limited value in our experience. For type 1 impactions, the mass of ingesta is usually too large and too tightly packed for infusion and massage to be of significant benefit in breaking down the impaction. Type 2 impactions already contain a significant amount of fluid and so are not likely to respond to additional fluid infused to break them down. There is still controversy concerning the criteria to use in deciding when to perform a bypass procedure. As a general rule, horses fitting into the type 2 category should be considered candidates for a cecal bypass. Some authors state that if the cecal wall appears normal and has reflex motility, and if the ventral colon contains a normal amount of ingesta, the cecum is not bypassed, whereas if the cecal wall is thickened and hyperemic and has minimal motility after evacuation, and if the ventral colon is almost empty, suggesting a cecal motility problem, the cecum should be bypassed.[21,43] These are valid guidelines, although there appear to be exceptions.[26]

Typhlotomy

The preferred surgical approach for a typhlotomy is through a ventral midline celiotomy, although the procedure can be performed through a flank laparotomy in the standing animal.[23-24] The cecum is usually quite large and tightly distended, sometimes making it difficult to exteriorize a sufficient portion of the apex to perform the typhlotomy. Extending the celiotomy incision improves the exposure. The cecum should be manipulated carefully with hands and arms placed around the body of the cecum, lifting the cecal body to allow exteriorization of the apex. Tilting the table, or tilting the horse slightly to the right side, can also facilitate exposure of the apex for the typhlotomy procedure.[43] The exteriorized apex should be isolated from the sterile field over the side of the horse. Stay sutures can be placed adjacent to the typhlotomy site to stabilize the cecum and prevent the apex from retracting back into the abdomen.[43] We prefer having an assistant stabilize the cecal apex with their hands, as stay sutures may tear, allowing the cecal apex to retract. An 8- to 12-cm (3- to 5-inch) typhlotomy incision is made between the lateral and ventral cecal bands. The size depends on the consistency of the ingesta. The surgeon manipulating the base of the cecum should continue to exteriorize an additional portion of the body of the cecum as the distention is relieved. A tube is placed through the typhlotomy site, and warm water is infused to aid in flushing out the cecal contents.[21,23,28,43] The cupula may be difficult to empty since it forms a pouch at the base of the cecum. In a rare case, we have placed a large-bore tube into the cecum through the typhlotomy along with the lavage tube to siphon cecal contents as the lavage loosened the ingesta. By using these lavage techniques, placing an arm into the cecum to remove contents from the caudal cecal base is rarely required as described in some reports.[43] Once the cecal contents have been evacuated, the contaminated region around the typhlotomy site should be thoroughly rinsed with electrolyte solution. The typhlotomy is closed with a double-inverting suture pattern or an appositional followed by an inverting pattern using 2-0 or 0 polyglactin 910. Some surgeons have recommended resecting the apex of the cecum and closure with the TA-90 stapling instrument if the apex becomes excessively contaminated during the typhlotomy.[3,43] With copious lavage, partial typhlectomy of the apex is usually not necessary.[28]

In one case series, 7 of 8 horses (88%) with cecal impaction treated only by typhlotomy and evacuation survived.[23] In another study, 36 of 37 horses (97%) treated by typhlotomy and evacuation only survived to discharge, and 25 of 28 (89%) of these survived long term.[28] Recurrent cecal impaction occurred in only 1 of the 3 that did not survive long term. The high success with evacuation only was attributed to coastal hay

causing a type 1 impaction in these horses. In another study, 9 of 10 horses (90%) that survived with evauation only had the impactions categorized as cecal dysfunction.[26] However, in two other studies, 4 of 14 horses (29%) were euthanized because of recurrence of the impaction after typhlotomy only, and cecal filling continued to occur postoperatively in horses in the other study, with only 1 of 5 surviving after typhlotomy only.[41,51]

Cecocolic anastomosis

This procedure was developed because of the poor response seen in horses with cecal impactions managed by typhlotomy only.[44] In this technique, a cecocolic anastomosis (CCA) is performed to create an alternative route for ingesta to pass from the cecum to the right ventral colon. The rationale is that cecal impactions may be caused by a cecal outflow problem, possibly because of motility dysfunction.[44] The cecum is approached by a ventral midline celiotomy. In most cases, a typhlotomy is recommended to evacuate the cecum before performing the anastomosis.[44] The CCA is performed between the lateral and dorsal bands of the cecum and between the lateral and medial free bands of the right ventral colon. Although the anastomosis can be hand-sutured with a two-layer side-to-side technique, it is recommended that the CCA be performed with intestinal stapling instruments to reduce peritoneal and serosal contamination.[44] For the stapling technique, stay sutures attaching the cecum and colon are placed at each end of the selected anastomosis site, approximately 20 cm (8 inches) apart. The seromuscular layer of the cecum and colon are apposed on one side of the intended stoma in a simple-continuous pattern using 2-0 polyglactin 910. Two small stab incisions are made on either side of the midpoint of the 20-cm (8-inch) suture line, one into the cecum and the other into the colon to allow insertion of the stapling instruments. The GIA-90 or ILA-100 stapling instrument is used twice, once orad and once aborad, taking care to overlap the staple lines, which makes a stoma between 15 and 20 cm (6 to 8 inches) in length. The stab incisions are closed and the front side of the stapled seromuscular layer is oversewn. To reduce the possibility of internal hernia formation between the cecum and the colon, the seromuscular closures are continued dorsally to the level of the cecocolic ligament.

In one study, 3 of 4 horses survived after this technique.[38] In another case series of 14 horses treated by CCA, the long-term survival (12 months) was 71%. However, 4 of the 10 long-term survivors experienced chronic, intermittent, mild abdominal pain thought to be associated with gaseous distention of the cecum.[44] This was considered to be a result of failure of normal gas transit between the two organs, or reflux of gas from the colon into the cecum.[44]

Because of the number of horses continuing to experience postoperative problems related to the cecum after the CCA, alternative techniques that bypass the cecum have been recommended in horses with cecal impactions.

Jejunocolostomy or ileocolostomy

For cecal impactions arising from abnormal cecal motility or compromised outflow at the cecocolic orifice, procedures that bypass the cecum entirely have been recommended to prevent cecal tympany or the recurrence of cecal impaction. In these bypass procedures, an anastomosis is made between the ileum or jejunum and the right ventral colon. A partial bypass has been described in which the ileum is not transected.[49] However, in an experimental study comparing a partial to a complete cecal bypass, the partial cecal bypass failed to decrease the size of the cecum or volume of ingesta in the cecum. Consequently, this was thought to be a risk for reimpaction and perforation.[52] Additionally, intermittent colic with cecal distention has been reported after partial cecal bypass.[44] The complete bypass, by taking the cecum out of the flow of ingesta, results in cecal atrophy and prevents postoperative cecal filling and rupture.[52-53] For these reasons, the complete bypass is recommended over the partial bypass.[3,24,52]

The cecum is approached through a ventral midline celiotomy and the cecal impaction is evacuated through a typhlotomy. The ileal vascular arcade is double-ligated on both sides of the area of transection. The transection can be completed by hand, with the TA-90 stapling instrument positioned on the distal end and sharply transecting proximal to the instrument, or with a GIA-90 or ILA-100 that will cut between stapled proximal and distal ends.[52,54-55] Doyen intestinal clamps are placed across the ileum oral and aboral to the line of transection for hand-sutured techniques. The proximal and distal ends of the hand-transected ileum are closed with a full-thickness continuous appositional or inverting pattern followed by a partial-thickness inverting pattern using an absorbable suture material. All stapled ends should be oversewn with a partial-thickness inverting pattern. The proximal blind end of the ileum is directed orally, toward the base of the cecum, as it is positioned on the most proximal exteriorized portion of the right ventral colon between the lateral and medial free bands (Figure 37-4). Stay sutures are placed 15 cm (6 inches) apart to attach the distal end of the ileum to the RVC. A Penrose drain is placed proximal on the ileum, and intestinal clamps are placed on the RVC to minimize contamination during the anastomosis. Although a hand-sutured side-to-side anastomosis can be performed, the use of intestinal stapling equipment shortens surgery time and decreases contamination. Stab incisions are made in adjacent areas of the attached ileum and RVC. The arm of a GIA or ILA stapling instrument is inserted into one stab incision and the anvil is inserted into the other stab incision. The stapler is discharged and the blade is pushed to form the stoma of approximately 10 cm (4 inches) in length.[55] The stab

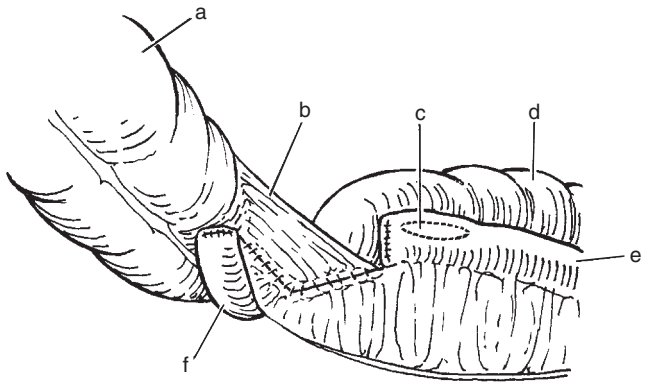

Figure 37-4. Jejunocolic anastomosis. Notice that the blind end of the small intestine is directed orally and the mesenteric defect has been closed. *a,* Cecum; *b,* cecocolic ligament; *c,* jejunocolic anastomotic stoma; *d,* right ventral colon; *e,* jejunum, *f,* blind end of the jejunum sutured to the cecum and directed toward the base of the cecum. (Redrawn from McIlwraith CW, Robertson JT [eds]: McIlwraith and Turner's Equine Surgery: Advanced Techniques. 2nd Ed. Blackwell, Oxford, UK, 1998.)

incisions are oversewn, followed by a single inverting-suture pattern circumferentially around the entire staple line at a distance of 5 to 8 mm. The ileocecal fold, portions of the cecocolic fold, and the bowel serosa are apposed in a simple-continuous pattern to close the mesenteric defect and eliminate the potential for an internal hernia.[54-55]

Several modifications of the previously described bypass procedure have been described. In one report, the authors describe the resection of a 15- to 30-cm (6- to 12-inch) segment of the ileum or ileum and distal jejunum before performing the anastomosis. By constructing the distal free end of the small intestine adjacent to a jejunal mesenteric vascular arcade, some surgeons feel the chances of an inadequate blood supply to the anastomosis are decreased.[54] In another report, a jejunocolostomy or ileocolostomy is performed without transection of the ileum and distal jejunum.[50] To make this procedure a complete bypass, once the jejunocolostomy or ileocolostomy is performed, four rows of staples are applied using the TA-90 stapling instrument immediately distal (aboral) to the anastomosis to occlude the lumen of the distal jejunum and ileum (Figure 37-5). This technique should reduce the length of the surgery and chances of contamination.

Several studies have reported on prognosis and complications after cecal bypass.[19,52,54] In one case series, 5 of 6 horses (83%) with cecal impaction treated by ileocolostomy returned to their previous activity with a minimal follow-up period of 6 months.[52] Surviving until discharge were 6 of 7 (86%) in another study.[19] In a third study, 9 horses that failed to respond to medical therapy were treated with an ileocolostomy or jejunocolostomy.[54] All horses survived until discharge, with all 7 horses available for long-term follow-up (range, 7 to 54 months; mean, 1.5 years). Several important observations were made relative to complications in this study. Positioning the small intestine aborad on the RVC makes closure of the mesenteric defect technically difficult, predisposing the horse to entrapment or strangulation of the small intestine in the mesenteric defect. To prevent this problem, the authors recommend that the small intestine be placed on the RVC facing orally and the mesentery closed. Another problem encountered was kinking at the anastomosis. Suturing a 5- to 10-cm (2- to 4-inch) segment of the distal small intestine to the colon proximal to the anastomosis is recommended to prevent this problem.[54] A further recommendation was to thoroughly evacuate the cecum before performing the anastomosis to prevent cecal impaction in the postoperative period. The authors emphasized the importance of gradual return to feed, because the most common postoperative complication was mild signs of colic within several days of reintroduction to feed. Although the cecum plays an important role in water absorption and bacterial degradation of dietary fiber, no detrimental consequences related to hydration and nutritional states have been observed with the bypass procedure.

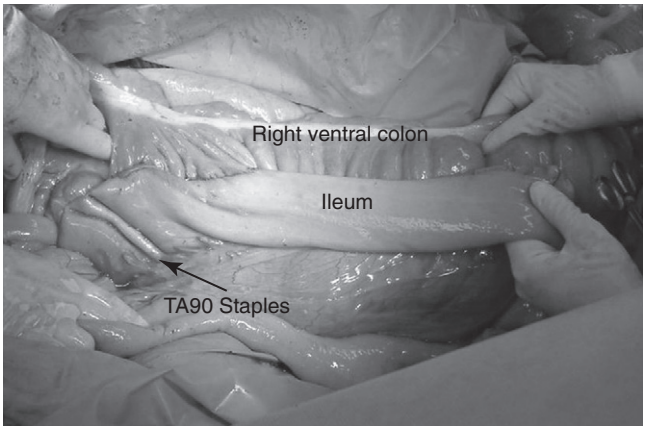

Figure 37-5. Complete cecal bypass without ileal transection. The ileum and right ventral colon are labeled. Four rows of staples are placed using a TA-90 distal to the ileocolostomy site to prevent feed from entering the cecum.

Postoperative Care

Horses are treated with broad-spectrum perioperative antibiotics and NSAIDs. The antibiotics may be discontinued 24 hours after surgery or continued for a therapeutic treatment course of 5 to 7 days, depending on the amount of contamination that occurred during the surgical procedure. The NSAIDs are continued for 3 to 5 days. During the initial postoperative period, intravenous fluids should be provided at a slow but constant rate. After 12 to 24 hours, the horse should be offered small amounts of water. Once it has demonstrated a tolerance for oral fluid, the water intake is increased and the intravenous fluids are decreased. Postoperative feeding is very important. It has been recommended that feed be withheld for a minimum of 36 to 48 hours, followed by grazing and feeding a bran mash with small quantities of good-quality hay at frequent intervals.[54]

Cecocecal or Cecocolic Intussusception

Pathogenesis

Cecocecal and cecocolic intussusception occur when the cecal apex invaginates into the cecal body (cecocecal) or continues through the cecocolic orifice and enters the large colon (cecocolic). Intussusception of the cecum was the most common cause of cecal obstruction in a report from the United Kingdom, whereas it accounted for only 3% to 3.5% of horses with primary cecal disease and for 1.3% of horses undergoing exploratory laparotomy for colic in other reports.[19,32,47,56-59] The relative frequency of cecocecal compared with cecocolic intussusception varies markedly depending on the study.[47,57,59-60] The etiology is unknown, but altered motility has been suggested to play a role. Dietary changes, cecal wall abscess, *Salmonella*, *Eimeria leuckarti*, *Strongylus vulgaris* arteritis, organophosphate exposure, and administration of parasympathomimetic drugs have all been implicated as potential risk factors.[58-59,61-62] Tapeworm infestation is often encountered in horses with cecal intussusception, leading some to speculate a causative association, possibly from mucosal inflammation initiating the intussusception.[30,58,61-63] However, other studies have not found a difference in tapeworm infestation rates between horses with and without cecal intussusception. Young horses (younger than 3 years) and Standardbreds appear to be at increased risk.[56-58,61,64]

Clinical Signs and Diagnosis

Clinical signs may vary considerably depending on the degree of vascular compromise and mechanical obstruction of the ingesta.[19,57-58,60-61,64] Approximately 55% of horses in one report presented with acute, moderate to severe pain requiring immediate surgical intervention, and 30% presented with a subacute

form (3 to 8 days) characterized by intermittent mild to moderate pain with soft feces or diarrhea. A smaller group (13%) presented with a chronic form (6 to 180 days) characterized by weight loss, scant soft feces, and mild abdominal pain.[61] Fever is common in horses suffering from this problem over a longer period of time. Cecocecal intussusceptions are more likely to be nonstrangulating, incomplete obstructions producing mild pain, and they develop into a chronic disease.[61] Cecocolic intussusceptions typically develop ischemic necrosis of the distal end of the intussusceptum, including the cecal apex and part of the body.[65] Rectal examination may be unremarkable in horses with cecocecal intussusception, as the intussuscepted cecal apex may not be palpable and the colon and small intestine are usually not obstructed. In horses with cecocolic intussusception, the cecum may feel malpositioned or may not be palpable at all. A mass or edematous bowel may be palpable in the right caudal abdomen.[64]

Ultrasonography may facilitate making the diagnosis.[56,61,64,66] Frequently, the compromised portion of the cecum is sequestered within the colon (or cecum), rendering peritoneal fluid analysis an insensitive indicator of pathology.[56] With time and progression of the disease, peritoneal fluid changes may be seen.[58,61-62] Often, the diagnosis is made at surgery after the horse fails to respond to medical therapy.[19,58,61-62,65]

Treatment

SURGICAL REDUCTION WITH PARTIAL TYPHLECTOMY

The surgical approach is through a ventral midline celiotomy. During the abdominal exploration, the cecal apex is found to be intussuscepted into the body of the cecum in the case of a cecocecal intussusception, whereas the cecum is "absent" from its normal position (with a palpable mass in the right ventral colon) in the case of a cecocolic intussusception. In rare cases, the base of the cecum may be intussuscepted into the colon while the cecal apex remains everted. In these cases, the cecum cannot be exteriorized to the normal extent while the invagination is palpable at the cecal base. Manual reduction is attempted by inserting one hand to grasp and place traction on any portion of the cecum within the intussuscipiens, while the other hand is placed on the colon over the intussuscepted cecum to gently massage the inverted cecum to an everted position. Approximately one third of cecal intussusceptions may be reduced in this manner.[61] The intussusceptum is frequently vascularly compromised, necessitating a partial typhlectomy. To prepare for the partial typhlectomy, the lateral and medial cecal vessels are double-ligated with 0 absorbable suture. Doyen intestinal clamps are placed proximal to the intended amputation site across the cecum to decrease fecal contamination, and the surgical area is draped off. The compromised cecum is often too edematous to allow the use of intestinal stapling instruments during the resection. After sharply resecting the compromised cecal apex and body, the cecum is closed using either an inverting or an appositional pattern, followed by an inverting suture pattern with No. 0 absorbable suture.

CECAL AMPUTATION THROUGH A COLOTOMY

Manual reduction of cecocolic intussusceptions is frequently not successful because of edema and adhesion formation (Figure 37-6, *A*). In these cases, reduction of the intussusception should be approached through a colotomy.[58,61,67] The large colon should be exteriorized from the abdomen and first evacuated through a pelvic flexure enterotomy if it contains an appreciable amount of ingesta. After closure of the pelvic flexure enterotomy, a second enterotomy is made on the ventral surface of the right ventral colon centered over or immediately distal to the intussusceptum. A sterile plastic bag or a plastic enterotomy drape with a 25-cm (10-inch) hole in the center can be sutured to the colon before the enterotomy is made to limit contamination during the colostomy (see Figure 37-6, *B*).[67] Attempts can be made to manually reduce the intussusception through the colotomy, but this is often unrewarding. In these cases, a partial resection of the invaginated cecum will facilitate reduction of the remaining cecum. The medial and lateral aspects of the inverted cecum are blindly ligated with two parallel transfixation sutures to occlude the cecal vessels. Overlapping mattress sutures, staples, or an encircling ligature with umbilical tape or a natural rubber ligature are placed to occlude the lumen of the cecum (see Figure 37-6, *C*).[67-68] The invaginated cecum is subsequently transected and the remaining cecal stump is reduced by both placing gentle traction on the cecal body and pushing the remaining inverted cecum out. The colotomy incision is closed with a two-layer inverting pattern using 2-0 or 0 absorbable suture material. The now everted cecal stump is subsequently examined for viability. If necessary, further resection of the cecum is performed (see Figure 37-6, *D*). The cecum is closed with a double-inverting pattern using No. 0 absorbable suture material.

If the cecum cannot be reduced after partial resection within the colon, or if the remaining cecal stump is friable and necrotic, the cecal stump is left invaginated within the colon and a cecal bypass procedure is performed.[62,69] In these cases, a partial typhlectomy is performed to remove as much of the necrotic cecum from inside the colon as possible, after which the cecal stump is sutured in a horizontal overlapping mattress pattern followed by a simple-continuous pattern. The colotomy incision is then closed and the surgical area is lavaged. To prevent leakage of ingesta, the serosal surface of the cecum at its point of invagination into the colon is oversewn.[62] However, complete oversewing at the point of intussusception may not be possible and may be unnecessary.[69] The ileum is subsequently transected, and an ileocolostomy or jejunocolostomy is performed adjacent to the closed colotomy incision (see "Jejunocolostomy or Ileocolostomy," under "Cecal Impaction"). The devitalized cecal stump is thought by some to slough inside the colon and pass in the feces, whereas others question whether this occurs.[61-62]

There are several reports where irreducible cecocolic intussusceptions were treated with bypass procedures without partial typhlectomy.[56,64] In one report, four of six horses survived long term after jejunocolostomy or ileocolostomy with ileal transection without a partial typhlectomy.[56] In a recent report describing three cases with cecocolic intussusception, irreducible by manipulation of the cecum within the colon and traction on the ileum, the horses were treated with a side-to-side ileocolic anastomosis without ileal transection and without partial typhlectomy.[64] In these cases the entire cecum, the ileocecal valve, and the distal ileum were left intussuscepted within the colon. The bypass procedure without ileal transection was performed as described earlier (see "Jejunocolostomy or Ileocolostomy," under "Cecal Impaction"), except that the ileal lumen distal to the anastomosis was not occluded with staples, because it was occluded by compression in the intussusception.[50] No attempt was made to oversew aboral to the intussusception. All three horses recovered with minimal complications and were clinically normal 12 months after the procedure.

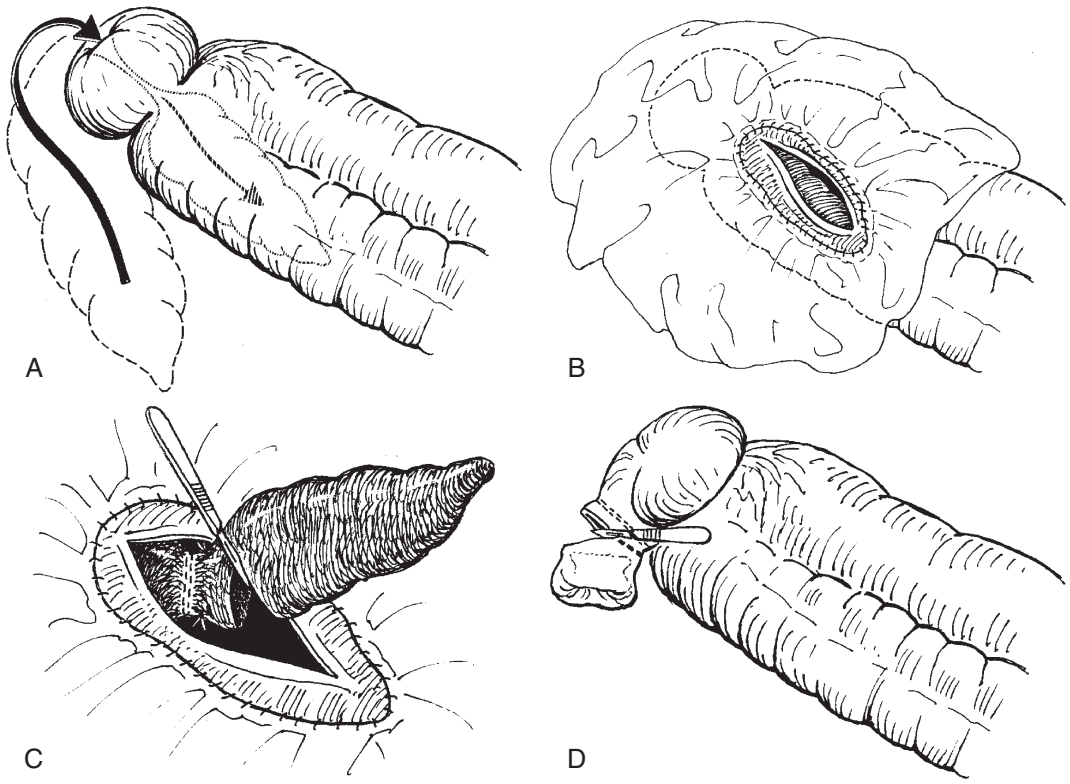

Figure 37-6. Cecal amputation within the right ventral colon (RVC) for nonreducible cecocolic intussusception. **A,** Cecum intussuscepted into the RVC through the cecocolic orifice. The *dashed lines* represent the outline of the invaginated cecum. **B,** Right ventral colostomy exposing the cecal intussusception. A sterile plastic bag or drape has been sutured to the seromuscular layer of the RVC before the colostomy to help contain contamination. **C,** Occluding mattress sutures (or TA staples) have been placed across the inverted cecum to facilitate removal of as much of the cecum as possible, thus enabling reduction of remaining invaginated tissue. The intussusception is then amputated through the RVC. **D,** After eversion of the cecal stump, the remaining compromised part of the remaining cecum is amputated, repeating the technique used for the amputation in the colon.

Postoperative Care and Prognosis

The horses are treated with intravenous fluids, broad-spectrum antibiotics, and NSAIDs. In cases of cecocecal intussusception with minimal contamination, the antibiotics may be discontinued 24 hours after surgery. Horses with cecocolic intussusceptions requiring cecal amputation within a colostomy are more likely to have been exposed to contamination and consequently require a therapeutic course of antimicrobial therapy. Postoperative abdominal lavage for 2 to 3 days also may aid in reducing the incidence and severity of postoperative peritonitis. The horse can be started back on feed 24 hours after surgery if there is no evidence of pain.

The prognosis is related to the amount of cecum intussuscepted, the ability to manually reduce the intussusception without opening the colon, the amount of contamination at surgery, and the viability of the cecum or the remaining cecal stump.[3,24,57,58,61,70] The prognosis after surgical correction of cecocecal intussusception is good.[70] Although a poor prognosis was associated with correction of cecocolic intussusceptions by partial typhlectomy through a colotomy in one report, other studies revealed a good prognosis associated with this technique.[58,61,67] Despite some contamination, which is inevitable during reduction through a colostomy, even with careful packing of the abdomen, clinical evidence of postoperative peritonitis is infrequent.[61,67] Bypass procedures with partial typhlectomy without reduction of the remaining cecal stump[62,69] or without partial typhlectomy have been reported to be effective in a limited number of cases.[56,64] More cases are needed to assess possible complications and long-term results associated with leaving part or all of the intussuscepted cecum within the colon. Until that information is available, the bypass procedures should be reserved for those cases where reduction after partial typhlectomy is not possible or for cases where ileal viability is compromised as assessed after reduction of cecal intussusception.[58,61,67,71]

Cecal Perforation or Rupture

Cecal perforation or rupture has been described in several retrospective studies.[19,32,34-35,38,72] Cecal perforation secondary to cecal impaction is well recognized.[3,19,28,35,38] Primary cecal perforation, unrelated to cecal impaction, has been described to occur in horses during treatment for an unrelated disease. In these horses, it appears to be associated with hospitalization, NSAID administration, or anesthesia. Primary cecal perforation has also been reported in periparturient mares.[19,24,27,34,73] Dystocia has been observed in approximately 50% of these horses. Horses with cecal perforation typically exhibit signs of pain soon after foaling, presumably as a result of cecal rupture from excessive pressure placed on the cecum during parturition. Primary cecal perforation has also been associated with tapeworm infestation.[74] As discussed previously, in some horses

with cecal impaction caused by cecal dysfunction, rupture can occur without significant clinical signs.[19,34] Consequently, it is not possible to know how many of these cases that have been described as primary cecal perforation have in fact been caused by a cecal dysfunction resulting in cecal distention caused by gas and fluids that went unnoticed before cecal perforation.

Cecal Volvulus or Torsion

Cecal volvulus or torsion secondary to large colon volvulus is uncommon.[3] Because of the substantial dorsal attachments of the cecal base to the body wall and colon, it is not common for the cecum to develop a primary torsion or volvulus. Incidental displacements of the cecum may be encountered at surgery and are sometimes classified as a torsion (usually 90 degrees). These typically are not a pathologic problem.[55] Primary volvulus may be related to anatomic abnormalities such as hypoplasia of the cecocolic fold and the normal cecal attachments to the body wall.[45,75] However, in one report, 9 of 96 horses (9%) with cecal disease were described as having primary cecal torsion with no apparent anatomic abnormalities.[19] Horses with cecal torsion or volvulus are presented with a sudden onset of severe abdominal pain. Rectal palpation is typically not diagnostic. Peritoneal fluid protein and nucleated cell count may be elevated. The diagnosis is made during an exploratory laparotomy. Partial typhlectomy with resection of the compromised cecum is recommended. The prognosis will depend on the viability and amount of compromised cecal tissue remaining after partial typhlectomy. In one report, three of five horses (60%) with cecal torsion treated by partial typhlectomy survived until discharge.[19] If extensive cecal damage extending to the cecal base is found at surgery, complete cecal amputation can be performed through a right flank approach with an 18th rib resection.[48] Cecal amputation leaving part of the cecal cupula to form a conduit between the ileocecal and cecocolic junctions has also been described in a case with extensive cecal ischemia from prolapse through a flank wound.[76] Recurrence has been reported after surgical correction in one horse with abnormal mesenteric attachments.[45]

Cecal Infarction

Cecal infarction has been reported to occur in 10 of 96 (11%) horses with cecal disease.[19] Verminous arteritis from *Strongylus vulgaris* infestation and multifocal infarction from larval cyathostomes are two proposed etiologies.[19,77] Mild abdominal pain increasing in severity over 24 hours is the most common clinical sign. Diarrhea, sudden severe pain, and acute cardiovascular collapse have also been described.[3,19] Peritoneal fluid evaluation reveals changes consistent with intestinal ischemia.[3,19] Rectal palpation may reveal a hard mass in the area where the cecum is located in approximately one third of the cases.[19] Surgical intervention is based on persistent abdominal pain and peritoneal fluid abnormalities, with the diagnosis of cecal infarction made at surgery. Subtotal typhlectomy is the recommended treatment.[19] In one report, seven of eight horses (88%) with cecal infarction treated by subtotal typhlectomy survived to discharge.[19] Although complete typhlectomy requiring an 18th rib resection in left lateral recumbency has been described, the favorable prognosis with partial typhlectomy indicates that the more difficult procedure performed through a right flank approach is usually not necessary.[19,48]

Cecal Tumors

Primary cecal tumors have rarely been reported in horses.[19,78-79] Three of 96 horses (3%) with cecal disease had primary tumors.[19] All three horses were 20 years or older and were presented for chronic weight loss rather than abdominal pain. Histologic diagnoses were leiomyoma, hemangiosarcoma, and papillary adenoma. Treatment should be surgical resection if possible and feasible.

Miscellaneous

Other reported conditions include cecal abscesses that may be associated with adhesions.[19,80] Horses may be presented with mild abdominal pain and peritoneal fluid changes consistent with peritonitis. Transrectal or transabdominal ultrasonography may aid in the diagnosis of cecal abscessation.[19,80] The treatment consists of surgical resection of the abscess and adhesions. Cecocutaneous fistulas have also been described, occurring after repair of body wall hernias with hernia clamps.[81] We have repaired several enterocutaneous fistulas that developed with entrapment of the cecal wall in an umbilical hernia with no history of hernial clamps. The prognosis after surgical repair is good.

LARGE COLON

Anatomy

The equine ascending colon begins at the cecocolic orifice and ends in the transverse colon. It measures approximately 3 to 3.7 m in length and has a capacity of 50 to 60 L. It is composed of the ventral and dorsal areas of the colon, which are connected by a short mesentery. Only rarely does a mesenteric defect occur between the ventral and dorsal colons (Figure 37-7). *In situ*, the large colon is further folded such that four segments are designated as the right and left ventral colon and the right and left dorsal colon. The transition from the left to the right ventral colon forms the sternal flexure, and the transition from the left to the right dorsal colon forms the diaphragmatic flexure. The maximal diameter is seen in the right dorsal colon and can reach 50 cm (20 inches). The narrowest diameter occurs in the left dorsal colon, which measures about 8 to 9 cm (3 to 3½ inches). The bands of the colon (teniae coli) support the colon. The ventral colon has four bands, of which the two ventral ones are free and the two dorsal ones are associated with the mesentery. The medial dorsal band is associated with the colonic vasculature. The pelvic flexure has one band, which can be palpated on rectal examination. The left dorsal colon has one band, which is joined by two other bands in the right dorsal colon. The roles of the teniae coli are to provide mechanical support to the colon, to maintain orientation of the colon within the abdomen, and to allow distention and contraction of the intestinal segments depending on the degree of ongoing fermentation. The teniae are composed of smooth muscle and collagen in varying proportions, depending on the role of each intestinal segment. In the ventral colon, the site of bacterial fermentation, the teniae have a greater proportion of elastin than in the dorsal colon. In the right dorsal colon, the site of transport and regulation of ingesta through the transverse colon, smooth muscle cells are present in greater proportion.[82]

The blood supply to the ascending colon is derived from the cranial mesenteric artery. The colic branch of the ileocecocolic

artery supplies the ventral colon to the pelvic flexure, where it joins the right colic artery, another branch of the cranial mesenteric artery. The middle colic artery, also a branch of the cranial mesenteric artery, provides blood supply to the transverse colon and to the initial part of the small colon. The microvascular circulation of the ascending colon has been well described.[83] The arteries branch from the colonic vessels every 2 cm and anastomose with vessels lying orally and aborally to form a colonic rete before continuing into the colonic tissue. The function of the colonic rete is unclear, but it may provide collateral blood supply, increasing the resistance of the colon to thromboembolic disease.[8] Vessels enter the submucosa through the tunica muscularis to form a complex anastomosing network of submucosal vessels. The extensive collateral supply to the colonic tissue can be expected to enhance healing, particularly where there are areas of devitalized tissue. Arterioles ascend from the submucosal plexus to the mucosa, where an extensive capillary network forms around the colonic glands (Figure 37-8). This capillary network is drained by more sparsely distributed venules (Figure 37-9). The submucosal venous plexus is characterized by regular mural helical smooth muscle constrictions that give the veins a sacculated appearance. The venous network is proposed to act as a capacitance system, storing large volumes of blood, which can be actively or passively directed into the systemic circulation as required. Alternatively, because of the large volumes of blood stored within

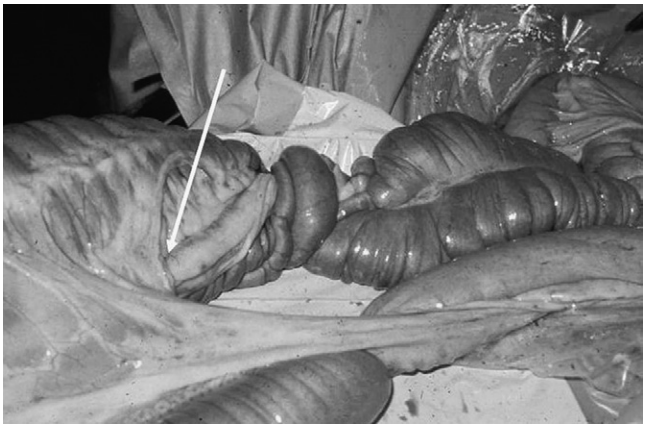

Figure 37-7. Large colon of a horse exteriorized onto a colon tray, showing a defect in the mesentery between the dorsal and ventral colon *(arrow)* that had resulted in a volvulus.

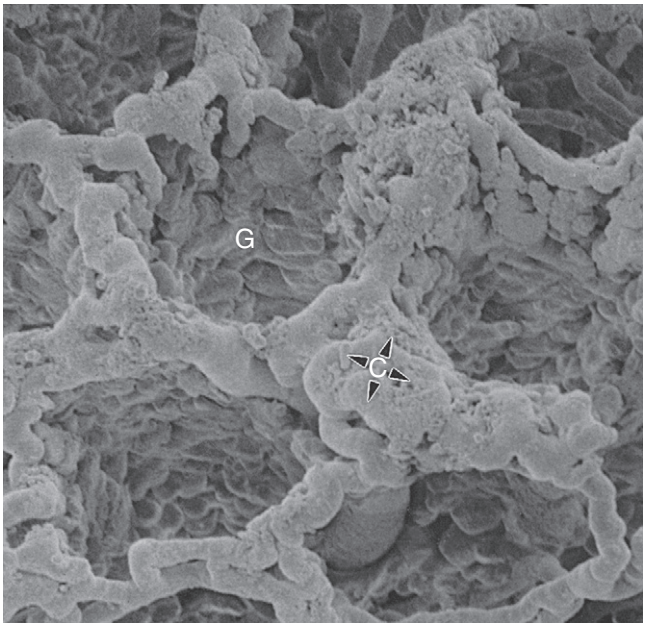

Figure 37-8. Scanning electron micrograph of a vascular replica at the luminal surface of the ascending colon. The capillary network ascends around the colonic glands *(G)* to the lumen, where the capillaries *(C)* in a network surrounding adjacent glands anastomose and look like a honeycomb.

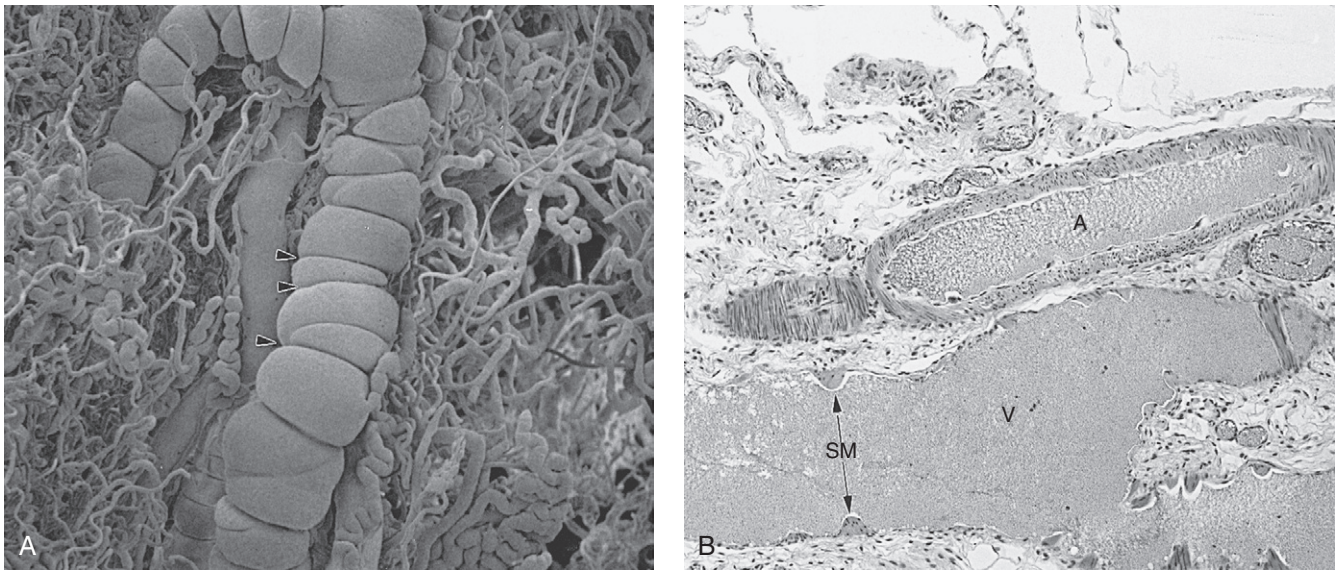

Figure 37-9. A, Scanning electron micrograph of a vascular replica of a colonic submucosal vein. Segmented sacculations are within the vessel wall *(arrowheads).* **B,** Histologic section of submucosal colonic artery *(A)* and vein *(V).* Rings of smooth muscle *(SM)* are present within the colonic vein, resulting in segmented sacculations identified on the vascular replicas.

the ascending colon, these smooth muscle constrictions may aid in maintaining blood flow through a low-pressure system.[83] The abundant colonic blood supply makes it more susceptible to severe intraluminal hemorrhage after enterotomies.[84]

Physiology

The most critical functions of the equine large colon are storage, microbial digestion, and fluid absorption. In a 24-hour period, the large intestine must recover a quantity of water approximately equivalent to the extracellular fluid volume of the horse (approximately 20% to 30% of body weight, or 90 to 135 L for a 454-kg horse). The storage function of the large colon allows time for microbial digestion and absorption of volatile fatty acids, the main source of energy in the horse.[85] The principal mechanism for delay in transit is the retropulsive activity initiated in a pacemaker region near the pelvic flexure (approximately 30 cm (12 inches) aboral to the termination of the medial and lateral free teniae of the left ventral colon).[86,87] The coordinated contractions originating at the pelvic flexure pacemaker promote physical separation of small, well-digested particles, which are propelled aborally, and of coarser particles, which are propelled orally for further digestion.[88] This may explain the roles of poor-quality feed and dentition in the development of large colon impaction.

Large Colon Tympany

Large colon tympany (gas colic, spasmodic colic) is the most commonly reported colic in horses.[89-91] It is thought to result from excessive gas fermentation in the colon, resulting in distention and pain. In one study evaluating risk factors for simple colonic obstruction and distention colic in horses (including large colon impaction), an increased risk was associated with crib-biting or windsucking, increasing number of hours spent in a stable, recent change in the regular exercise program, lack of administration of ivermectin or moxidectin anthelmintics in the previous 12 months, and a history of travel in the previous 24 hours.[92] Other factors that increased the risk of simple colonic obstruction and distention included history of previous colic, recent (less than 4 weeks) lameness, and increased time since last dental care.[92] An association between tapeworm infestation and spasmodic colic has been demonstrated.[93] Horses fed a hay and grain diet containing large amounts of soluble carbohydrates and less fiber had decreased water content in ingesta and increased gas, which predispose to gas colic.[94] Although large colon tympany can be self-limiting, it is thought to precede large colon displacements; therefore identification of risk factors in affected horses may help decrease the risk of future occurrences.

Diagnosis

Horses examined for large colon tympany have acute pain and may show signs of visible external abdominal distention. Although in pain, they are minimally compromised, and clinicopathologic parameters including abdominocentesis remain within normal reference range. The challenge for the clinician is to differentiate this type of colic from other, more serious causes of abdominal pain. The differential diagnosis for gas colic resulting in large colon distention includes large colon displacement, large colon torsion, ileus (impending colitis), and aboral obstruction (transverse or small colon obstruction). Rectal palpation reveals moderate to severe gas distention of the large colon.

Treatment

Treatment includes administering analgesics and withholding feed. The response to medication is favorable and, if the diagnosis is correct, the horse will remain comfortable. Lack of response to analgesics may indicate a more serious problem. If the response to treatment is unsatisfactory, early referral is recommended, considering that large colon volvulus is in the differential for this condition.

Large Colon Impaction

Large colon impaction is the second most commonly reported cause of colic in horses, and it is the most frequent type of simple obstruction.[90,95] It represents up to 13.4% of colics examined at referral centers.[37,96] Risk factors identified in one study included crib-biting or windsucking, increasing number of hours spent in a stable, recent change in the regular exercise program, absence of administration of ivermectin or moxidectin anthelmintic in the previous 12 months, and a history of travel in the previous 24 hours.[92] Additional factors identified in that study included history of previous colic, recent (less than 4 weeks) lameness, and increasing time since last dental care. In another study, 79 of 147 horses (53.7%) had a change in routine in the 2 weeks before development of a large colon impaction, and 17 of 147 (11.5%) developed the impaction while hospitalized for a reason other than gastrointestinal disease.[37] Decreased water intake and parasites are factors also mentioned although not well documented. High-grain diets may predispose to colonic impactions, since grain feeding was shown to be associated with an internal fluid flux and subsequent dehydration of ingesta, setting the stage for impaction.[97] Right colon dry-matter content was higher when grain was part of a free choice hay diet.[98] In another study, water consumption was 40% greater in ponies offered warm drinking water under cold weather conditions, suggesting that cold weather may predispose to colonic impaction.[99] Hospitalization and general anesthesia have been suggested as risk factors for the development of impaction colic.[37,100] In one study, 37 of 85 hospitalized horses (43.6%) developed postoperative reduced fecal output, and 10 horses (12%) developed signs of colic; 2 of 9 horses that had a rectal examination had pelvic flexure impactions. In that study, administration of phenylbutazone decreased the risk of reduced fecal output.[100] In contrast, *in vitro* studies have shown a negative effect of NSAIDs on large colon intestinal smooth muscle motility.[101,102] The benefits of NSAID administration for pain control, considering their negative effects on intestinal health, remains a matter of debate.[100,103,104]

Amitraz, an acaricide, has been used to induce experimental large colon impaction in horses.[105] Topical spraying of horses with amitraz was associated with systemic illness and impaction colic in three horses.[106] Amitraz, atropine, glycopyrrolate, and morphine significantly prolonged intestinal transit time in horses.[107] Atropine (0.044 mg/kg and 0.176 mg/kg IV) resulted in decreased intestinal motility and decreased appetite for 2 to 7½ hours after injection; signs of abdominal pain were observed in 3 of 10 ponies after administration.[108] Topical ocular administration of 1 mg of atropine hourly resulted in decreased

intestinal motility (as assessed by auscultation for 2 to 18 hours after administration), and signs of abdominal pain were observed in four of six horses.[109] Subconjunctival administration of 3 mg of atropine resulted in decreased intestinal sounds in three of six horses and signs of abdominal pain in one.[109] Lowering the luminal temperature to 20° C (room temperature) over a 100-cm (40-inch) length of colon cranial and caudal to the pelvic flexure for 2 hours significantly decreased conduction velocity as well as amplitude and duration of pressure peaks in the intestinal wall. These findings have direct relevance to surgery of the large colon, where the colon is exteriorized from the abdomen, sometimes for prolonged periods of time, and where luminal lavage is performed. Minimizing the time of exteriorization and using warm water for luminal lavage may help the colon recover from the effects of reduced temperature.

The role of intestinal parasites on pelvic flexure motility has been evaluated. In one study, arteritis of the cranial mesenteric artery induced by inoculation of *Strongylus vulgaris* larvae resulted in reduced relative colonic blood flow; but it did not cause altered motility patterns.[110] In another study, increased motility patterns were observed in the cecum and colon at intermittent intervals for several days after infection.[111] Horses with chronic (more than 24 hours) obstructions of the large colon or with previous obstruction had decreased neuron density in the pelvic flexure, which may predispose to future obstructions.[40] Megacolon with myenteric hypoganglionosis has been described in a 6-month-old foal with severe large colon impaction and colic.[112] Recurrent impactions of the large colon were diagnosed in a mare with eosinophilic enterocolitis.[113] In that mare, diarrhea, hypoproteinemia, and weight loss characteristic of the disease were absent, and the intestine appeared grossly normal at surgery and at postmortem examination. These cases support the use of intraoperative intestinal biopsies in horses with unexplained recurrent large colon impaction.

Large colon impactions usually affect horses older than 1 year, although miniature horses may be predisposed to impactions as foals. Historically, horses are presented to referral centers for chronic colic (i.e., lasting longer than 24 hours).[37]

Diagnosis

The most common location for large colon impaction is the pelvic flexure, followed by the right dorsal and transverse colons. On physical examination, horses typically show mild to moderate abdominal pain, decreased or absent intestinal sounds, decreased or absent fecal production, and occasionally mild to moderate abdominal distention.[37] Nasogastric reflux is rarely present. Rectal palpation is diagnostic in cases of pelvic flexure impaction. However, impactions of the right dorsal and transverse colons can be difficult to palpate in adult horses. When a large colon impaction is suspected on the basis of rectal palpation, it is essential to differentiate that from a large colon displacement with secondary right dorsal colon impaction.[114] Prolonged treatment of a large colon displacement with fluids and cathartics could result in preoperative or intraoperative colonic rupture.

In the case of a large colon displacement, the right dorsal colon may become severely impacted; during rectal palpation, when the hand follows the colon, it travels to the right, and lateral to the cecum. This is in contrast to a pelvic flexure impaction, where the end of the pelvic flexure can readily be identified. Another important differential diagnosis of large colon impaction is small intestinal obstruction, which can result in dry fecal contents in the large colon as identified by palpation. In contrast to large colon impaction, the teniae and haustra of the ventral colon in such cases become more prominent and distinguishable.[114] Laboratory data typically observed in horses with large colon impaction show mild to moderate dehydration, a normal leukogram, and normal electrolyte data and blood gas analysis. The abdominocentesis results should be within normal range. Deteriorating cardiovascular status or peritoneal fluid changes are an indication of bowel degeneration.[37]

Treatment

Medical treatment of large colon impaction includes fluid therapy, analgesics, cathartics, and withholding of feed until the impaction is resolved. Intravenous fluids are reserved for impactions of longstanding duration (more than 24 hours), when dehydration is documented, or when nasogastric reflux is present, precluding the use of enteral fluid therapy. Balanced electrolyte solutions are administered at twice the maintenance rate, or 120 mL/kg per day, both to restore circulating blood volume and to allow secretion of fluid into the large colon in response to cathartics. Overhydration in combination with an oral cathartic such as magnesium sulfate is thought to promote rehydration of ingesta.[115,116] Systemic rehydration should be performed before cathartics are administered.

Enteral fluid therapy can complement and even supplement intravenous fluids. Advantages of enteral fluid therapy include administration of fluid directly in the gastrointestinal tract, stimulation of colonic motility through the gastrocolic reflex, decreased expense, and decreased need for precise adjustment of fluid composition.[116] Enteral fluids can be administered by intermittent nasogastric intubation or by nasal placement of an indwelling feeding tube (18-French equine enteral feeding tube), allowing continuous fluid administration. An isotonic electrolyte solution can be made by mixing 5.27 g of NaCl, 0.37 g of KCl, and 3.78 g of $NaHCO_3$ per liter of tap water.[116] This solution results in electrolyte concentration of 135 mEq/L of Na, 95 mEq/L of Cl, 5 mEq/L of K, and 45 mEq/L of HCO_3, with a measured osmolality of approximately 255 mOsm/L, representing a slightly hypotonic electrolyte solution compared to plasma.[116] Plasma electrolyte concentrations remained within normal range with this solution compared with the marked hypernatremia and hyperchloremia observed when 0.9% saline is administered enterally.[116] Despite the fact that normal horses can tolerate up to 10 L hourly through intermittent nasogastric intubation, it is usually not possible to administer more than 5 L every 2 hours in horses with impactions, because these horses start to reflux when more fluid is administered.[117] Therefore, intermittent intubation is used, allowing administration of approximately 60 L of fluids per day. When continuous enteral fluids are given, a greater rate of administration is tolerated, and horses can be given between 4 and 10 L/hour. At the higher rate of 10 L/hour, mild signs of abdominal pain were observed in normal horses, and in horses with large colon impaction, a rate of 5 L/hour is better tolerated.[116] For enteral fluid therapy, the fluid used can be water, or water and electrolytes. In one study, right dorsal colon ingesta hydration was

significantly increased after enteral fluid therapy compared with intravenous fluid therapy combined with enteral administration of magnesium sulfate.[116] Enteral fluid therapy with an isotonic electrolyte solution with or without intravenous fluid therapy was successful in resolving 99% of colon impactions in one study.[118]

Cathartics are useful to increase the amount of water in the large colon or to promote ingesta transit. Mineral oil is a mixture of aliphatic hydrocarbons obtained from petrolatum; it is indigestible and absorbed to a limited extent. It is an intestinal lubricant that can also serve as a marker of intestinal transit. Administration of 5 to 10 mL/kg is usually recommended, and oil should be evident in the feces 12 to 24 hours after administration. Unformed feces were apparent 18 to 24 hours after its administration in normal horses.[119] Administration to normal horses decreased glucose absorption and intestinal transit time.[120,121] Chronic use can result in a foreign body reaction in the intestinal mucosa.[122] Careful administration is necessary, since inadvertent administration into the lungs results in lipid pneumonitis, which is severe and often fatal.[123,124] Mineral oil is easily confused with propylene glycol, used in the treatment of ketosis in cattle; inadvertent administration of propylene glycol was the cause of death in a horse.[125]

Dioctyl sodium sulfosuccinate is an anionic surface-active agent that by lowering surface tension may facilitate penetration of the fecal mass by water and fats. Effects on motility and secretion are also attributed to this product. The recommended dosage range is 16.5 to 66 mg/kg, and the maximal recommended dose is 200 mg/kg. Death because of circulatory shock can occur at dosages of 1 g/kg.[126] In one study in normal horses, toxic signs were observed at a dosage of 50 mg/kg.[115] It is important to correctly label the product to avoid overdosing. Its advantage is that it requires a low volume of administration. However, the drug's low margin of safety and lack of efficacy at low dosages make its use questionable. Concurrent absorption with mineral oil may result in emulsification and subsequent systemic absorption of oil. Therefore the use of the combination is frequently discouraged, although the significance of this finding is unknown.

Osmotic or saline cathartics such as magnesium sulfate or sodium sulfate are effective products to increase colonic water content.[115,127] Because of their efficacy, these products should be used after systemic rehydration. Recommended dosages are 0.5 to 1 g/kg. Absorption of magnesium resulting in signs of toxicity was reported in two horses that had received a combination of dioctyl sodium sulfosuccinate and magnesium sulfate.[128] Raw linseed oil produced from flaxseed was a commonly used laxative for the treatment of impactions. The addition of metallic salts and boil oil or boil pure oil enhances its properties as a wood preservative and is highly toxic. Only raw linseed oil should be used. Administration of 2.5 mL/kg to normal horses resulted in watery diarrhea, anorexia, mild signs of colic, and neutropenia.[119] Although raw linseed oil has greater laxative effects than mineral oil, the toxic effects may preclude its use, particularly in horses with compromised intestinal mucosa.[119]

Polyethylene glycol 3350 is an effective osmotic laxative that is used in humans for the treatment of constipation or for colonic cleansing.[129-130] This product has not been evaluated rigorously in horses, although we have used it successfully to treat large colon impactions. Its cost is much greater than that of the other cathartics mentioned. Castor oil has been used as a model for experimental colitis in ponies.[131] Its use for the treatment of impaction colic is not recommended.

Analgesics are indicated as part of the management of large colon impaction. NSAIDs are commonly used. Use of low-dose flunixin meglumine may help control pain without affecting large colon motility. In the course of treatment of severe impactions, some horses may require intermittent dosing with xylazine to relieve intestinal spasm.[132] Alternatively, a constant-rate infusion of lidocaine can be used to modulate pain.

Prognosis

Most horses respond well to medical therapy. In one study, only 24 of 147 horses required surgery.[37] Indications for surgery included uncontrollable pain, deteriorating cardiovascular status, or peritoneal fluid changes indicating bowel compromise. Of the horses that went to surgery, five were euthanized after tearing of the colon during exteriorization.[37] Surgical management of large colon impaction involves evacuation of the colon by pelvic flexure enterotomy. Complications of surgery include intraoperative rupture of the colon, postoperative diarrhea, incisional drainage, and, rarely, septic peritonitis.[37] The prognosis for large colon impaction is excellent, and the majority of horses respond to medical therapy. The prognosis is better for horses treated medically than surgically. In one study, long-term outcome for horses treated medically was 95.1%, compared with 57.8% for horses treated surgically.[37] However, the lower outcome associated with surgically treated horses reflects a worse condition rather than a surgical failure. The most common complication was jugular vein thrombophlebitis, so catheter sites should be monitored carefully. In the management of these horses, it is important to avoid risk factors that predispose to impactions. These horses are at risk for reimpaction if the same conditions remain. A small number of horses require permanent dietary modifications to prevent reimpaction.

Sand Impaction

Accumulation of sand in the equine large colon can result in variable signs, including colic, diarrhea, weight loss, and poor performance. Sand should be considered in the evaluation of chronic diarrhea in foals and adult horses.[133,134] Risk factors for sand impaction include insufficient roughage in the diet, access to sand, and mineral composition of the soil. Sand impaction has been diagnosed in horses that were exposed to sand 3 to 8 weeks before examination.[135] Higher incidences of sand colic are reported in California, Arizona, Florida, Michigan, and coastal regions. Although most horses with sand impaction are older than 1 year, sand accumulation has been documented in foals.[135-137] Miniature horses may also be predisposed to sand impaction because of environmental and management practices.

Diagnosis

Horses with sand impaction manifest signs similar to those of large colon impaction, unless a concurrent large colon displacement or torsion is present. Large colon displacements or torsion were identified in 10 of 40 (25%) and 26 of 48 (54%) of horses with sand impaction.[135,136] These clinical signs include mild to

moderate abdominal pain, reduced fecal production, and decreased intestinal sounds. The sound of sand may be heard when the ventral abdomen is auscultated behind the xyphoid. These horses are responsive to analgesics, and signs can be present for several weeks. The cardiovascular status should be normal unless dehydration or intestinal devitalization has occurred. Occasionally, horses with sand impaction show signs of endotoxemia, presumably as a result of mucosal or intestinal damage associated with the weight and abrasiveness of the sand.[137]

Diagnostic procedures that are used to detect the presence of sand include observation of sand in the feces, sand obtained or palpated during abdominocentesis, abdominal auscultation, rectal palpation of a sand-filled viscus, abdominal radiography, and abdominal ultrasonography.[136-138] In one study, 23 of 40 horses (58%) were diagnosed with intestinal sand before surgery by one or more methods.[136] Fecal sedimentation is performed by adding water to six fecal balls in a rectal sleeve, and allowing the sand to settle to the bottom. The presence of more than 1 teaspoonful of sand is considered excessive. The presence of sand on sedimentation, however, may be incidental, and horses with sand impaction may not have sand in their feces at the time of examination. The appearance of sand in the feces during treatment is considered a sign of clearance of the sand.[137] Abdominocentesis results often are normal or show an increased total protein. Abdominocentesis is not diagnostic for sand colic but rather indicates the degree of intestinal compromise. However, the weight of the colon makes it easy to perform an unintentional enterocentesis during the procedure, and sand can be palpated with the tip of the needle or cannula.[135] In one study, 13 of 23 horses with correctly diagnosed sand impaction before surgery were identified by the presence of sand obtained or palpated during abdominocentesis.[136] Abdominal auscultation for the detection of sand is performed on the ventral abdomen, with emphasis on the area caudal to the xiphoid process.[139] The sound produced has been described as similar to the sound produced by sand in a partially filled paper bag that is slowly rotated.[139] The intensity of sound is loudest with larger accumulations of coarse as opposed to fine sand. In an experimental study of sand impaction in horses, all horses had, at some time, sand sounds that could be auscultated, but in most cases only after receiving several doses of sand and after several repeated 5-minute auscultation periods.[139] Rectal palpation of horses with sand impaction most commonly reveals distention of the cecum or large colon. The impaction is rarely palpated on rectal examination; however, if coarse sand is present, it may be palpated through the intestinal mucosa.

Abdominal radiography provides the best method to evaluate the amount of sand accumulation, and it serves as a tool for monitoring disappearance of sand with treatment (Figure 37-10).[137] The cranioventral abdomen is the most useful location for imaging.[137] Abdominal ultrasonography can be used to diagnose sand impaction, but it is best used in combination with abdominal radiography to monitor clearance of the sand.[140] Ultrasonographic evidence of sand accumulation is more subjective and includes close and increased contact of the large colon with the ventral body wall, decreased or absent intestinal motility, and hyperechoic acoustic shadowing. In one study, radiography and ultrasonography outcomes were similar in only 50% of cases.[138] Because ultrasonography is more readily performed and can be easily repeated, it is thought to be a useful tool for monitoring once a diagnosis has been made, although repeated radiographs may be indicated when results of the ultrasonography are equivocal.[138]

Figure 37-10. Lateral radiographic projection of the ventral abdomen of a horse showing accumulation of sand in the ventral colon *(arrows).*

Treatment

Medical treatment of sand impaction includes removing the horse from access to sand, rehydrating by intravenous or oral methods, and using laxatives. Mineral oil is usually not effective, as it will pass around the sand. Magnesium sulfate or psyllium is used to promote evacuation of sand. In one study, psyllium failed to increase evacuation of sand in an experimentally induced model of sand impaction.[141] However, numbers were small and the model may not reflect naturally occurring disease. In a clinical study, horses that had sand impaction refractory to treatment were responsive to administration of magnesium sulfate and mineral oil.[137]

Resolution of the impaction can be monitored using abdominal radiography or ultrasonography.[137,138] Horses with sand impaction can develop abnormal motility patterns and subsequent large colon displacement. These horses have more pain and develop gas distention of the large colon. Surgical intervention is indicated when a displacement is suspected or diagnosed, when abdominal pain is uncontrolled, in the presence of deteriorating cardiovascular parameters, or when there is evidence of intestinal devitalization.[135-136] Standing flank laparotomy does not allow sufficient access to the large colon for evacuation in the presence of sand, and injection of the impaction is not successful in providing relief.

Therefore a surgical approach through a ventral midline incision is recommended. At surgery, the sand is evacuated through a pelvic flexure enterotomy (Figure 37-11). The most common location for accumulation of sand is the right dorsal colon, but any location, from the ileocecal junction to the small colon, is possible, and multiple impaction sites are commonly encountered. Care must be exercised during exteriorization of the large colon, since the weight of the sand predisposes the colon to rupture, especially in chronic cases. To facilitate exteriorization,

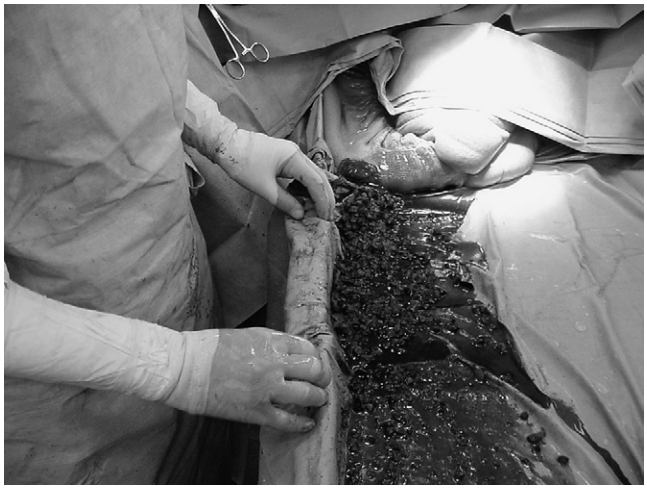

Figure 37-11. Pelvic flexure enterotomy for evacuation of a gravel impaction in the large colon of a horse.

the horse may be tilted toward the left side of the abdomen. In addition, only the minimal length of large colon necessary to safely perform a colotomy should be exteriorized; as the colon is evacuated, more of its length can be carefully exteriorized from the abdomen.

Prognosis

Reports of surgical treatment of sand impaction indicate a good long-term survival. The most common complication is postoperative diarrhea, but this complication is commonly noted after surgical evacuation of the large colon for any reason. Other complications include peritonitis associated with intestinal devitalization from pressure necrosis.

Prevention of sand impaction includes providing adequate roughage, feeding off the ground, and providing additional roughage when pastures are insufficient. The use of different formulations of psyllium (pellets or flakes) has been advocated at different dosage regimens (once a day for 3 weeks, then 1 week off, to twice a day for 2 weeks, then 1 week off), but the efficacy of these different dosage regimens in prevention of further sand accumulation has not been documented. There is concern that long-term use of psyllium alters the colonic microflora with subsequent bacterial digestion of the psyllium and decreased efficacy; this is the rationale behind interrupted administration.[141]

Enterolithiasis

Obstruction of the large or small colon by enteroliths is a well-documented cause of intestinal obstruction in the horse. Risk factors include geographic location (with California and Florida having high prevalence for this cause of colic), breed predisposition (e.g., Arabians and Arabian crosses, Morgans, American Saddlebreds, donkeys, and Miniature Horses), feeding alfalfa hay, and less than 50% of time spent outdoors.[142,143] Other factors to explain why other horses fed the same diet in the same geographic area do not develop enteroliths are as yet unidentified.

Diagnosis

Enterolithiasis can result in acute severe luminal obstruction or cause intermittent mild signs of colic, depending on the location and size of the enterolith. Those in the large colon are usually localized in the right dorsal colon and cause mild signs of intestinal discomfort. Once they migrate into the transverse or small colon, signs of acute luminal obstruction develop, with progressive abdominal distention. Although this condition causes a simple colonic obstruction, transmural pressure necrosis can occur. Critical attention to results of the abdominocentesis helps determine such occurrence. An increase in total protein above the normal range and an increase in WBC count can alert the clinician to such occurrence.

Physical examination parameters also vary, depending on the location of the enterolith and whether intestinal devitalization has occurred. Rectal palpation may be normal or may reveal large colon distention. The enterolith can rarely be palpated. Results of the abdominocentesis are often normal, although an increased total protein is an early sign of intestinal devitalization. Radiographs are a useful diagnostic method for detecting of enteroliths, although the sensitivity and specificity vary depending on the location of the enterolith and the prevalence of the disease for the hospital population.[144] In one study performed in a high-prevalence area for enteroliths, the sensitivity of radiographic diagnosis of enterolithiasis was 84.3% for those located in the large colon, compared with 50.0% for those located in the small colon.[144] The mean overall positive predictive value for radiographs was 96.4%, and the negative predictive value was 67.5%.[144] Surgical removal is indicated. If the enterolith is located in the large colon, the large colon is evacuated via a pelvic flexure enterotomy. A second enterotomy may be required in the right dorsal colon if the enterolith is too large to be evacuated through the pelvic flexure enterotomy. If the enterolith is lodged in the transverse colon, retrograde flushing by enema can facilitate its movement back into the dorsal colon. If the enterolith is in the small colon, it is removed by a small colon enterotomy. A partial-thickness enterotomy can facilitate movement of the enterolith to a more accessible portion of the small colon.[145] If there is significant pressure necrosis of the intestinal wall at the site of the obstruction, a resection may be required.

Prognosis

The prognosis is usually excellent. However, local necrosis of the intestine in an area that cannot be exteriorized, such as the transverse colon, is associated with a grave prognosis. In one study, bypass of the transverse colon by end-to-side anastomosis of the ventral colon to the small colon was successful in a Miniature Horse with intestinal necrosis after obstruction by a fecalith in the transverse colon. The recurrence rate of enteroliths is unknown, but dietary modifications such as avoidance of alfalfa hay are usually recommended. Other recommendations include removing horses from dirt or gravel, which can serve as a nidus, adding psyllium to the diet, and adding cider vinegar (1 cup twice daily) to the diet. In geographic areas where the water has a high mineral concentration, providing an alternative source of water can be useful.[143] Wheat bran should be minimized because of its high phosphorus content. Control of the dietary cation–anion balance (DCAB) has been suggested, although the benefits are unproven.[146] The target DCAB is calculated by the following equation:

$$DCAB = ([Na] + [K] + 0.15[Ca] + 0.15[Mg]) - ([Cl] + 0.25[S] + 0.5[P]),$$

is +200 to 300 mEq/kg. Grass hays and cereal grains have a DCAB within that range, whereas that of alfalfa is higher.

Large Colon Displacement

The equine large colon, because of its lack of mesenteric attachment to the body wall, is freely mobile and prone to displacement. In addition, normal longitudinal shortening of the left colon, promoted by contractions of the longitudinal layers, move the pelvic flexure toward the diaphragm, followed by backward movement toward the pelvis during relaxation; alterations in this motility pattern, initiated at the pelvic flexure pacemaker, could result in displacements and torsion.[88] The normal equine diet is composed of soluble and insoluble carbohydrates, the latter of which are digested by microbial fermentation, resulting in production of volatile fatty acids that are absorbed for energy production. When excess soluble carbohydrates are fed, alterations in the microbial population of the large colon may result in excessive fermentation, gas distention, and subsequent displacements.

Large colon displacements have been classified into left dorsal displacement of the large colon (also referred to as nephrosplenic entrapment or renosplenic entrapment), right dorsal displacement of the large colon, and nonstrangulating volvulus of the large colon. The large colon may adopt a location intermediate to these displacements, such that the exact description of the displacement can be difficult to describe.

Nephrosplenic Entrapment

Nephrosplenic entrapment (also termed renosplenic entrapment or left dorsal displacement of the large colon) is a form of nonstrangulating large colon displacement in the horse, in which the left dorsal and ventral colons migrate lateral to the spleen in a dorsal direction until entrapped in the nephrosplenic space (Figure 37-12). It has been reported in horses of any age, and in foals as young as 9 months.[147] Although one report mentions the increased prevalence in male horses, others have not substantiated that finding.[147-149] Miniature Horses and pony breeds have not been reported to acquire this problem. It is theorized that excessive gas formation within the left colon, perhaps in association with abnormal motility, causes the left colon to displace lateral to the spleen and dorsad into the nephrosplenic space; alternatively, displacement can occur during rolling episodes.[91,150-152] In most cases, there is also ventromedial rotation of the left colon, such that the left dorsal colon is rotated ventral to the left ventral colon. The weight of the colon causes the spleen to displace medially and ventrally and to become congested. Because of impaired flow of ingesta over time, with duration there is also concurrent impaction of the left dorsal colon. With continued gas formation, the sternal and diaphragmatic flexures can migrate craniad and dorsad to the stomach and become lodged between the stomach and the left lobe of the liver, a displacement classified as type II in one report.[153,154] Nephrosplenic entrapment is a nonstrangulating lesion of the large colon; however, when duration of the condition increases to longer than 24 hours, colonic congestion and edema can develop, and mural damage may follow.[152] Obstruction to gastric outflow occurs because of pressure of the colon on the duodenum or tension on the mesentery. Entrapment of the small colon or small intestine in the nephrosplenic space have been described, but these conditions are rare.[155,156]

Horses with nephrosplenic entrapment show variable degrees of pain depending on the location of the colon, the amount of gas distention, and the presence of secondary gastric distention. Location of the colon lateral to the spleen is associated with minimal to no discomfort and is found in the resolving stages of the displacement. Entrapment of the colons within the nephrosplenic space with the spleen in a relatively normal position is a very painful condition. Affected horses crouch, want to go down, and often lean to the left. When the spleen is pushed away from the body wall and displaced ventrad by the colons, effectively opening the nephrosplenic space, there is less pressure on the colon, and horses show only mild signs of abdominal pain.

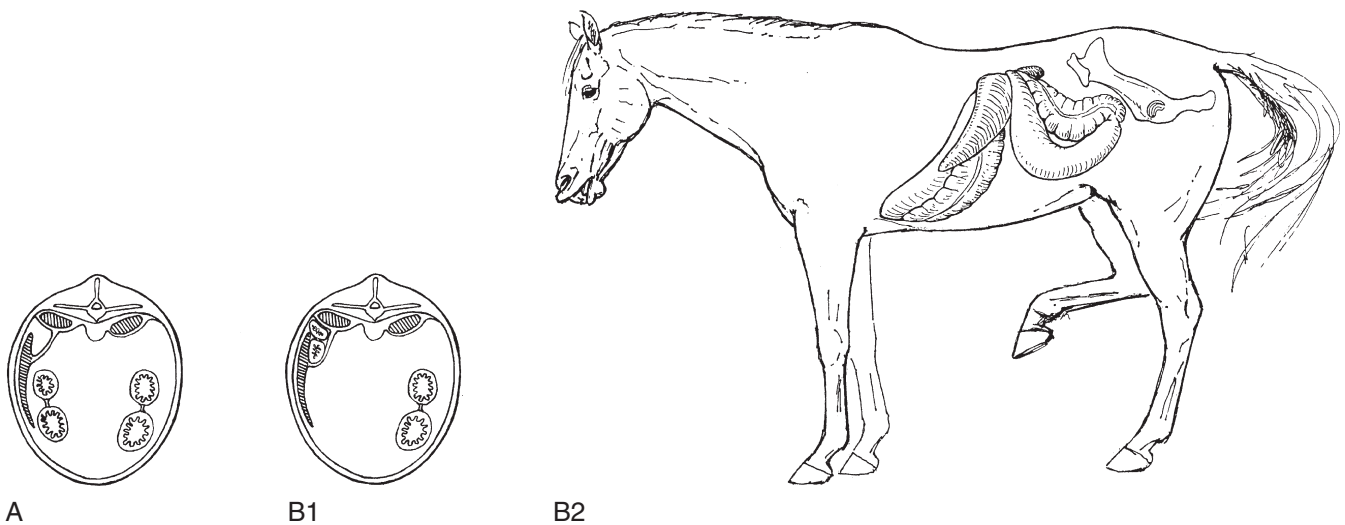

Figure 37-12. A, Left dorsal displacement of the ascending colon. The normal anatomic relationships of the colon to the spleen are shown. **B,** The dorsal and ventral colon may displace and become trapped in the nephrosplenic space. **B1,** Cross-sectional caudocranial view. **B2,** Side view.

DIAGNOSIS

The diagnosis of nephrosplenic entrapment is based on the presence of abdominal pain (which can be mild to severe, depending on the location of the colon and the degree of gas distention) and relatively normal cardiovascular parameters, consistent with a simple obstruction. The complete blood count and blood chemistry values should be normal or consistent with mild dehydration. A low PCV in the face of dehydration has been reported as an indication of red blood cell sequestration in the spleen. The abdominocentesis results should be within normal range, and collection of splenic blood (characterized by a higher PCV than in peripheral blood) supports of a diagnosis of nephrosplenic entrapment. In one study, these results were obtained in 25% of horses at admission.[148] Horses with an increased WBC in the peritoneal fluid were more likely to suffer from a longer duration of the condition and to be treated surgically.[148] Nasogastric reflux is commonly obtained in horses with nephrosplenic entrapment as a result of pressure on the duodenum or mesenteric tension. Up to 43% of horses with nephrosplenic entrapment were reported to have nasogastric reflux.[147] In one study, resistance to nasogastric intubation was encountered in horses with displacement of the sternal and diaphragmatic flexures dorsal to the stomach.

An ultrasonographic examination of the abdomen may serve as an adjunctive tool in the diagnosis of nephrosplenic entrapment, and it was diagnostic in 88% (36 of 41) horses with nephrosplenic entrapment.[157] A 2.5- or 3.5-MHz ultrasound probe is placed over the 15th to 17th intercostal space in a direction parallel to the ground, and the spleen is imaged. In the normal horse, the left kidney is imaged deep to the spleen. In horses with nephrosplenic entrapment, the presence of gas-filled colon dorsal to the spleen precludes imaging the kidney. Finding ventral displacement of the dorsal aspect of the spleen is also consistent with nephrosplenic entrapment. If the colons are displaced lateral to the spleen, then imaging of the spleen is obscured as well. It is important to direct the probe in a horizontal direction; if the probe is angled in a dorsoventral direction, the kidney can occasionally still be imaged, giving a false-negative diagnosis. The inability to image the left kidney supports only a diagnosis of a nephrosplenic entrapment and should be used in conjunction with rectal palpation. Nonspecific large colon distention or the normal presence of small colon in the nephrosplenic space can impair imaging of the left kidney.[158] In addition, if the colon is filled with fluid, imaging of the left kidney is still possible, but the entrapped bowel is also seen.[157] Rectal palpation remains the mainstay of diagnosis of nephrosplenic entrapment. On palpation, the left colon is most often gas distended, and the ventral colon is located dorsal to the left dorsal colon; often, an impaction of the left dorsal colon is present as well. The colon can be followed up into the nephrosplenic space. It is important for the correct diagnosis to follow the colon into the nephrosplenic space, because in other conditions (ranging from gas colic to large colon torsion), the colon may assume a dorsal position within the abdomen, leading to a false diagnosis of nephrosplenic entrapment.[159] This could be disastrous if nonsurgical management is attempted and the horse effectively has a large colon torsion. Rectal palpation correctly identified left dorsal displacement of the large colon in 61.2%, 68.7%, and 72% of cases in three studies, although an early study reported a much lower rate of correct identification (only 18%).[147-149,153] Rectal palpation can be impaired by the patient's size and temperament or the presence of severe distention.

A thorough physical examination, rectal palpation, and abdominal ultrasonography should be performed in horses with nephrosplenic entrapment, because a small number of them have another primary lesion involving another segment of the gastrointestinal tract. Gastric rupture, small intestinal volvulus, ileal impaction, large colon displacement, large colon volvulus, cecal torsion, and small colon obstruction have been documented in association with nephrosplenic entrapment.[148,149,157,160]

TREATMENT

Once a diagnosis of nephrosplenic entrapment has been made and the clinician is confident that there are no other abnormalities, options for treatment are evaluated. These include medical therapy with intravenous fluids and withholding of feed; exercise with or without the use of a pressor agent; rolling under general anesthesia, with or without the use of pressor agents; standing flank laparotomy; and ventral midline celiotomy.

Medical treatment

Medical therapy with intravenous fluids supplemented with calcium gluconate is indicated when the colons are located lateral to the spleen or dorsal to the spleen but not in the nephrosplenic space. This type of treatment was successful in nine affected horses.[147] Medical therapy with enteral fluids consisting of isotonic electrolyte solution, with or without intravenous fluids, was successful in 25 of 30 horses (83%) with nephrosplenic entrapments.[118] In that report, it is not clear where the left colons were located, or the number of horses with suspected as opposed to confirmed nephrosplenic entrapment.

When the colons are localized in the nephrosplenic space and the horse is in pain, nonsurgical management can be attempted. In one study, horses that were successfully treated nonsurgically had a shorter duration of clinical signs, a lower peritoneal fluid WBC, and a higher blood lymphocyte count than horses treated surgically.[148] In that study, failure at nonsurgical correction was also felt to be related to severity of gas distention, which would also increase with duration. It is important to emphasize again that the diagnosis must be certain before attempting this option.

With all nonsurgical options, *phenylephrine* is often used to cause splenic contraction and facilitate correction. Bleeding the horse was described in the early literature to reduce splenic size, but this seems unnecessary and possibly detrimental.[161] Phenylephrine (Neo-Synephrine, 10 mg/mL) is an α_1-adrenergic receptor agonist that causes vasoconstriction in most vascular beds, as well as splenic contraction. In one study, the splenic area was reduced to 28% of baseline and thickness to 48% of baseline after administration of phenylephrine at 3 μg/kg/min over 15 minutes.[162] Side effects were minimal at that dosage and included hypertension and reflex bradycardia. We use a total dose of 10 mg for horses with a body weight of 450 kg or less and 20 mg for larger horses, diluted in 50 mL of saline and given slowly intravenously over 5 minutes. There are anecdotal reports of fatal hemorrhage (hemothorax most commonly, and hemoabdomen) after phenylephrine administration to older horses. It is unknown whether these horses had preexisting systemic or pulmonary hypertension, but the drug should be used with caution in older animals.[163] It is also important to administer the drug properly, following dilution and slowly; there is one report of severe hemothorax following an intravenous bolus of the undiluted drug.[164] Vigorous exercise is an option for correction of this

problem, providing the horse is not lame. This is more successful when performed early in the course of the disease, before large colon distention develops.[147,159] If there is significant distention of the left colon, it can be trocarized before exercise. For trocarization, an area over the left flank is clipped and prepared for aseptic surgery. A 14-gauge angiocatheter with a catheter extension is used to puncture the flank in a perpendicular direction. The end of the catheter extension is placed in a water container to facilitate observation of gas exiting the colon. Once in place, the trocar portion of the catheter is withdrawn for a few millimeters to avoid lacerating the bowel. The catheter may need to be inserted farther as the colon is deflated. When doing so, care must be taken not to lacerate the tip of the catheter with the trocar. Rectal palpation can help manipulate the colon to facilitate gas evacuation. Once gas is evacuated, the trocar portion of the catheter is removed and 1 to 2 mL of gentamicin is injected as the catheter is withdrawn. Once the trocarization is completed, phenylephrine is administered and the horse is exercised either on a lunge line or in a small paddock for approximately 15 to 30 minutes. Rectal palpation is performed to ascertain the position of the colon. Exercise can be repeated if correction is not achieved.

Correction by rolling under general anesthesia is another nonsurgical option for this disorder.[160,161] This has also been reported useful in a small number of horses with other forms of large colon displacements.[165] This option is preferred if the horse is lame, or if there is marked large colon distention. Previous abdominal surgery may preclude it, however, as splenic adhesions may prevent successful correction of the displacement.[166] For this procedure, the colon is not trocarized unless the distention is severe, as gas translocation is used to achieve correction of the displacement. The horse is prepared for short-term intravenous anesthesia. Sedation is induced, followed by phenylephrine infusion as described previously. The horse is subsequently anesthetized and dropped into right lateral recumbency. Using a knee or two-handed fist, the clinician then vigorously shakes the abdomen in the region of the right flank, as the horse's hind legs are being hoisted dorsally. If a hoist is not available, the horse is slowly rolled into dorsal recumbency while abdominal compressions are performed. With the hoist, the hind legs are elevated until the horse's body reaches a 60-degree vertical position, while the clinician continues to vigorously shake the abdomen. After 1 or 2 minutes, the horse is replaced in right lateral recumbency. At this stage, some clinicians prefer to place the horse in left lateral recumbency, roll the horse into sternal position, and return it to right lateral recumbency. The procedure is repeated two more times. After the last manipulation, the horse is returned into left lateral recumbency for recovery. Between manipulations, the clinician may check the position of the colon. In our experience it is difficult to determine successful correction of the entrapment, either by ultrasonography or rectal palpation, with the horse under general anesthesia. Therefore we always recover the horse to determine if correction was achieved. Some clinicians prefer to follow immediately with surgical correction without recovery if it is suspected that correction was not achieved.[159] One author describes manipulation of the colon *per rectum*. With the horse in right lateral recumbency, the hand lifts the colon off the spleen as the horse is being turned to left lateral recumbency. The use of muscle relaxants to loosen the rectum is recommended when this procedure is used. Rectal manipulation must be performed with extreme caution, as rectal tears represent a definitive risk of the procedure.[160] Rectal palpation after manipulation often reveals that the colon is lateral to the spleen and has not completely resumed its normal position. Administration of fluids and calcium often results in complete correction within a few hours. Some horses show mild abdominal pain after manipulation, even though it is suspected that correction was achieved. This is probably a result of residual large colon distention or impaction, and it is usually responsive to analgesics.

Surgical treatment

If nonsurgical manipulations are unsuccessful, surgical intervention is indicated. Standing flank laparotomy or ventral midline celiotomy are options to consider. The advantages of the standing flank laparotomy are avoidance of general anesthesia, direct access to the problem, access to the nephrosplenic space for closure as an option for preventing recurrence, and more rapid return to use of the horse. The major disadvantage, as is the case for nonsurgical manipulation, is if an incorrect diagnosis was made, precluding correction of the condition by this approach. The advantages of the ventral midline celiotomy are that it ensures successful correction, particularly in cases where the colons are located dorsal and cranial to the stomach, and it allows correction of other undiagnosed problems. The disadvantages include the need for general anesthesia, longer postoperative recovery, and increased cost.

Standing flank laparotomy

The horse is restrained in stocks with the tail bandaged and tied to avoid contamination of the incision. Sedation with xylazine, with or without butorphanol, is sufficient for most horses. After standard preparation of the surgical site, local anesthesia is performed. This can be accomplished with a paravertebral block, an L-block, or a line block, taking care to add local anesthesia for placement of towel clamps.[167] In our experience, the paravertebral block often requires additional local anesthetic in the distal aspect of the planned incision. Draping is routine, with the addition of a drape folded to form a pocket to support the exteriorized bowel. The standard surgical approach for a flank laparotomy is midway between the last rib and the cranial aspect of the tuber coxae, starting approximately 2 cm above the palpable internal oblique muscle. In the standing horse, the modified grid approach is preferred, as the muscles will contain the intestinal contents after surgery. In the modified grid approach, the external oblique muscle is incised in a vertical direction but the internal oblique and transverse abdominal muscles are bluntly separated along the course of their muscle fibers by finger dissection. Additional topical application of anesthetic may be required before perforating the peritoneum. Once the abdomen is entered, the spleen and large colon are located. In our experience, it is easier to push the spleen down and under the large colon than to lift the large colon over the spleen. Once the colon is lateral to the spleen, it is gently pushed down into the ventral abdomen. Closure is routinely performed.

Standing laparoscopic correction

There is one report of a standing laparoscopic approach for diagnosis and correction of a nephrosplenic entrapment in an adult horse.[168] An important point in this case is that the left ventral and dorsal colons were not impacted or gas distended. This allowed the surgeon to place a 53-cm-long (21-inch-long)

stainless steel bar in a laparoscopic portal and use it to reposition the entrapped large colon off the spleen, in combination with systemic administraiton of phenylephrine. This approach would be contraindicated if significant feed impaction or gas distention of the large colon was present or if the horse showed significant abdominal pain.

Ventral midline celiotomy

This approach is recommended for correction of nephrosplenic entrapment if nonsurgical management is unsuccessful and the surgeon is not comfortable with the flank approach. It is also the suggested approach if there are clinical or clinicopathologic findings that indicate either the loss of intestinal integrity or the presence of another lesion. Ventral midline celiotomy is also used when a presurgical diagnosis is not possible because the small size of the patient precludes rectal palpation or because excessive large colon distention is present. A routine ventral midline approach is performed. To facilitate access to the nephrosplenic space the horse may be slightly rotated to the right. In the presence of excessive splenic congestion, or excessive weight of the colon, phenylephrine may be administered to reduce splenic size and facilitate correction. The base of the spleen is grasped, and the spleen is lifted and pushed medial to the colon, thus freeing it from the nephrosplenic space. The colon is subsequently cradled over the forearm and lifted out of the abdomen. Although there is often feed accumulation in the dorsal colon, evacuation is usually not necessary because the feed redistributes when the displacement is corrected. The colon is assessed for signs of devitalization. Rarely, a large colon resection has to be performed.

Reported recurrence rates of nephrosplenic entrapment are between 7.5% and 8.5%.[147,148] Considering this relatively low risk of recurrence, surgical intervention procedures for prevention of recurrence are not recommended after a first incidence. However, feeding and management practices should be carefully reviewed to minimize the risk of gas formation in the large colon and subsequent displacement.

PREVENTION OF RECURRENCE

Procedures that have been advocated to prevent recurrence of left dorsal displacement of the large colon include closure of the nephrosplenic space, large colon colopexy, and large colon resection. Refer to later sections for discussions of large colon resection and colopexy procedures.

Closure of the nephrosplenic space does not prevent migration of the large colon lateral to the spleen, or the occurrence of other forms of large colon displacement. The procedure can be performed through a flank laparotomy or though a minimally invasive laparoscopic approach.[169a-c] The flank approach is performed in the standing or laterally recumbent horse. The abdomen is entered either through a modified grid or laparotomy approach. The spleen and nephrosplenic ligament are identified, and the nephrosplenic space is verified to be free of intestine. Cruciate sutures are then placed between the ligament and the tip of the spleen, using a nonabsorbable suture material such as 2-polypropylene. Approximately 6 to 12 sutures are preplaced and tied. The abdomen is closed in a routine fashion. Alternatively, laparoscopic ablation of the renosplenic space can be performed in the standing horse.[169a-c-171] The procedure is performed using three laparoscopic portals and polyglactin 910 suture material in a simple-continuous pattern. Alternatively, laparoscopic placement of a polypropylene mesh has been used to obliterate the nephrosplenic space.[169a]

In a small series of 10 horses, where 15 episodes of left dorsal displacement of the large colon and 21 episodes of colic were recorded before laparoscopic closure of the nephrosplenic space, a significant decrease in the total incidence of colic, incidence of nephrosplenic entrapment, and incidence of ventral celiotomy was identified. However, no difference was found in the number of horses that had colic peroperatively compared with postoperatively, which was probably due to small sample size.[170]

Right Dorsal Displacement of the Large Colon

This type of displacement is thought to be initiated by retropulsive movement of the pelvic flexure, with subsequent migration of the left colon craniad and then to the right abdominal quadrant, until the right ventral and dorsal colons are located between the cecum and the body wall (Figure 37-13). The colon can also rotate on its long axis, resulting in variable degree of venous congestion.

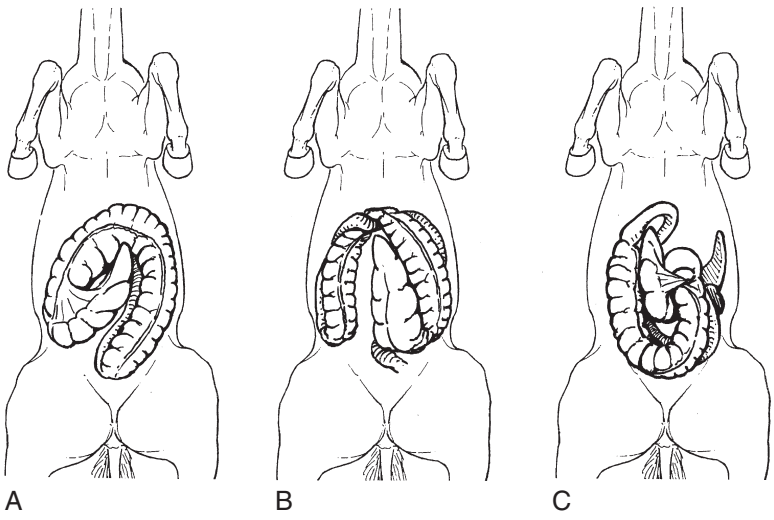

Figure 37-13. Right dorsal displacement of the ascending colon (ventral views). **A,** The normal anatomic relationship of the ascending colon. **B,** The most common direction for a right dorsal displacement is migration of the pelvic flexure in a counterclockwise direction when viewed from the caudal and ventral aspect of the horse at the time of surgery. **C,** Although less common, the ascending colon may develop a right dorsal displacement characterized by a clockwise migration of the pelvic flexure when viewed from the caudal and ventral aspects of the horse at the time of surgery.

DIAGNOSIS

The location of the colon at the time of examination is related to the clinical signs. When the colon is displaced craniad, all parameters are within normal limits, and abdominal pain is mild and intermittent. These horses may be comfortable when held off feed, and continue to pass small amounts of manure, with abdominal pain recurring when feed is reintroduced. On rectal palpation, there is no abdominal distention, but the examiner is unable to find the pelvic flexure. As the colon continues to migrate in a clockwise direction, the flow of ingesta is impaired, and a secondary impaction of the right dorsal colon may develop. Gas distention also becomes more significant, as does the associated abdominal pain. When a dorsal colon impaction develops, it is important not to mistake it for a pelvic flexure impaction, because continued medical treatment may result in rupture of the colon. This condition can be differentiated from a pelvic flexure impaction by the fact that the colon travels craniad to the right, and that the pelvic flexure cannot be identified. In right dorsal displacements, the cecum can be enlarged and filled with fluid.

Horses with right dorsal colon displacements are presented with mild to moderate abdominal pain. Depending on the degree of displacement, rectal palpation reveals absence of the pelvic flexure, presence of large colon lateral to the cecum, large colon distention, and right dorsal colon impaction. Nasogastric reflux may be present if there is large colon distention. Laboratory data are usually unremarkable, although a significant number of horses are presented with an elevated γ-glutamyltransferase, probably related to partial obstruction of the duodenum.[171]

TREATMENT

When horses are presented early, with normal parameters, mild abdominal pain, and minimal to moderate large colon distention, medical therapy may be attempted. Intravenous fluids are administered and the horse is monitored for resolution of the distention and relocation of the large colon.

When the pain is severe or there is marked large colon distention or a severe secondary impaction, surgical intervention is recommended. Although in some specific circumstances we have successfully corrected right dorsal colonic displacements via a standing flank laparotomy, a ventral midline celiotomy is preferred because of ease of exposure and correction.

At surgery, the pelvic flexure is identified, the large colon is exteriorized, and the displacement is corrected. When a severe large colon impaction coexists, the large colon is exteriorized at the pelvic flexure, but an attempt to correct the displacement is not made until the large colon has been evacuated, to avoid rupture during colonic manipulations. Although there is one report of successful treatment of a horse after intraoperative rupture during surgery, most of these cases are fatal.[172]

PROGNOSIS

The prognosis for large colon displacement is excellent. Recurrence is possible, and we have seen horses redisplace within 48 hours of the first procedure. In our opinion, it is important not to completely evacuate the large colon but to leave some ingesta and to return these horses to feed within 8 hours of surgery, again to try to maintain bulk in the large colon.

In one study of 154 horses treated surgically for right dorsal displacement of the large colon, it was found that those horses were significantly more likely to experience recurrent episodes of colic requiring veterinary intervention postoperatively compared to other types of displacements.[173]

Nonstrangulating Volvulus of the Large Colon

Nonstrangulating volvulus of the large colon is identified when the colon is rotated from 90 to 270 degrees along its long axis, a step that precedes the 360-degree large colon volvulus.

DIAGNOSIS

Horses with this type of displacement have a clinical presentation very similar to that of other simple large colon displacements; minimal cardiovascular compromise, mild to moderate abdominal pain, normal abdominocentesis, and mild to moderate large colon distention on rectal palpation. As in other displacements, medical management may be attempted. Horses that are still in pain or that have worsening of abdominal distention are candidates for surgical intervention. At surgery, the colon is replaced in its normal position.

As in all forms of large colon displacements or volvulus, prevention of recurrence should be considered. Colopexy and large colon resection are two procedures that are performed in an attempt to prevent recurrence. Usually these procedures are not recommended on a first occurrence, but they should be considered for horses that have had two or more displacements.

Other Simple Obstructions of the Large Colon

Congenital malformation of the large colon was reported as a cause of recurrent colic in a horse, and we have identified it in several horses.[174] Resection and anastamosis of the colon orad to the malformation successfully resolved the problem.

Fibrosis and stricture of the large colon caused by focal fibrosis at the pelvic flexure was identified in three horses and corrected by transverse closure of a pelvic flexure enterotomy performed at the site of the fibrosis.[175] Pelvic flexure adhesions resulting in impaction were reported in a filly with peritonitis. Laparoscopic adhesiolysis was successfully performed to allow return of transit.[176]

A massive duplication cyst of the ascending colon was reported in a 27-year-old mare with a history of recurrent colic and a pendulous abdomen. The cyst was successfully removed and the mare made an uneventful recovery.[177]

We have observed defects in the mesentery of the large colon in several horses (see Figure 37-7). The presence of the defect allows displacement or torsion of the colon upon itself, since it is no longer confined by the mesentery. The problem can be corrected by closure of the defects with the help of sutures. In one report, rupture of the mesocolon of the ascending colon was the cause of recurrent colic in a mare, and closure of the defect resolved the problem.[178]

Large Colon Torsion or Volvulus

Large colon volvulus is one of the most painful and devastating gastrointestinal problems in the horse (Figure 37-14). Successful management depends on rapid referral and prompt surgical intervention. Without intervention, death occurs in a matter of hours in horses. The prevalence of large colon volvulus is increased in geographic areas with high concentration of brood

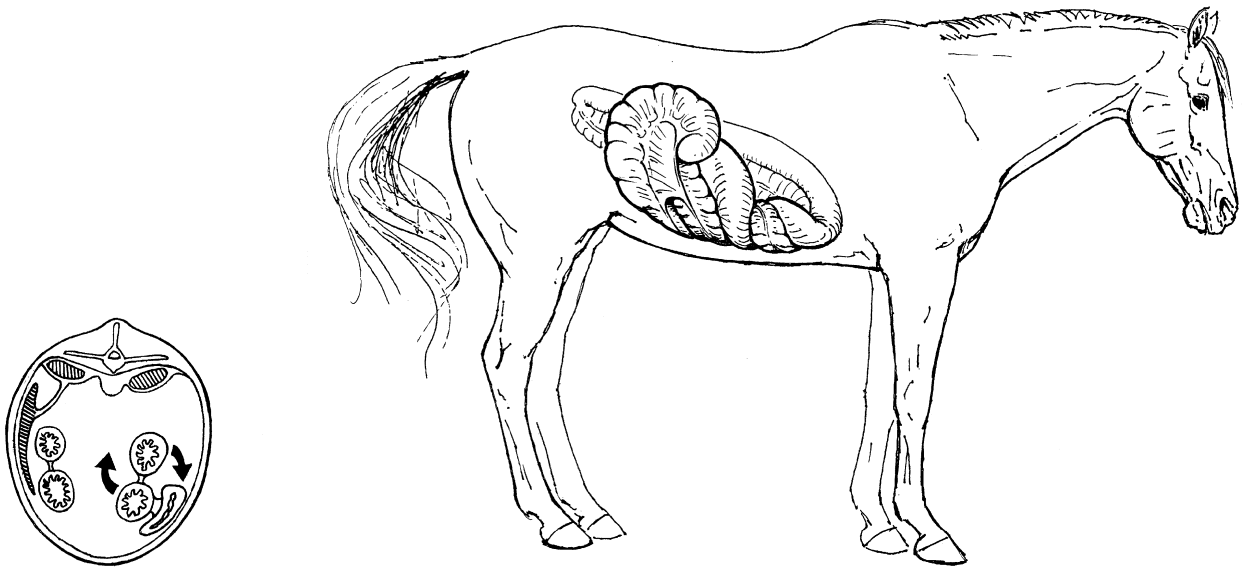

Figure 37-14. A schematic diagram representing the two directions in which the ascending colon may twist during torsion. The *arrows* indicate the more common dorsomedial torsion.

mares. Risk factors include recent parturition, recent dietary changes, and recent access to a lush pasture.

Diagnosis

The history of horses with large colon volvulus varies depending on the rapidity and totality of the development of a complete volvulus. Some horses are presented with a history of chronic (more than 24 hours) colic that suddenly worsens to intractable pain; others are presented with an acute onset of uncontrollable pain. Initially, despite the severe pain, horses maintain normal cardiovascular parameters, and rectal palpation can be unremarkable. As time elapses, these horses develop progressive large colon distention and accompanying progressive cardiovascular collapse. In mares, the color of the vaginal mucosa changes and reflects the degree of compromise of the large colon. If untreated, horses with large colon torsion die of hypovolemic shock caused by abdominal compartment syndrome associated with the severe distention, by pooling of blood in the strangulated large colon, and by poor pulmonary expansion because of severe distention.

Treatment

The treatment of large colon torsion is surgical. However, during preparation for surgery, resuscitative measures should be initiated to increase circulating blood volume and decrease abdominal pressure. Shock fluid therapy, including hypertonic saline, colloids, and crystalloids, needs to be initiated (see Chapter 3). Trocarization to relieve abdominal distention may improve lung expansion and venous return, and it can help sustain the horse during induction, until the colon can be exteriorized and decompressed.

In preparation for surgery, the horse can be tilted to one side to facilitate exteriorization of the colon and remove pressure on the caudal vena cava. Administration of intravenous lidocaine allows a decrease in the concentration of inhalant agents. A long ventral midline incision is made to help exteriorize the colon. A more cranial approach is preferred, to facilitate exposure of the colonic base.

The direction of the torsion is best described in relationship to the position of the ventral colon, which obviates the need to describe it from the observer's position. Thus a dorsomedial volvulus indicates rotation of the right ventral colon medially and dorsally.[179] Most are in a dorsomedial direction, and the location of the volvulus is at or proximal to the cecocolic ligament. Occasionally, a volvulus involving the sternal and diaphragmatic flexures is encountered. Volvuli of 270 to 720 degrees have been described.[180] Correction of the volvulus can be difficult, particularly if the colon is full or edematous, which places the colon, particularly the right dorsal colon, at risk for rupture during surgical manipulation. The ascending colon is exteriorized. If the colon is full or edematous and friable, a pelvic flexure enterotomy is performed to empty the colon before manipulation. To correct the volvulus, the surgeon, situated on the left side of the horse, places both hands in the abdomen around the base of the colon and gently manipulates it in a clockwise direction. The surgical assistant can facilitate manipulation by rotating the exteriorized portion of the colon in the same direction. Gas translocation and return of serosal color are indications that the colon is manipulated correctly. Once a 360-degree rotation has been achieved, the surgeon must ascertain that the volvulus is corrected and that another rotation of the colon is not needed. This is accomplished by examining the normal position of the cecum and the normal position of the cecocolic ligament, and by ensuring by palpation that the mesenteric attachment of the right dorsal colon to the dorsal body wall is straight. Palpation of the duodenum cranial to the right dorsal colon is possible after the volvulus is completely corrected.

Once the colon has been returned to its normal position, the surgeon must decide between euthanasia, recovery of the horse without further intervention, colopexy for prevention of recurrence, and large colon resection. It is essential to involve the owner in the decision-making process, particularly if the colon is compromised, since the cost of further intervention and

postoperative care can be significant. The surgeon must also understand that if the colon is not resected and the horse is recovered, it is unlikely that a second laparotomy will be an option. By the time clinical signs indicate that a second laparotomy with a possible large colon resection is warranted, the horse's condition will have deteriorated to the point that the chances of survival after a resection will be poor. Several factors are useful to help the surgeon reach a decision. A critical one is the horse's systemic condition both preoperatively and intraoperatively. A PCV above 50% (and increasing during surgery) associated with a decreasing total protein is a poor indicator of survival. An inability to maintain mean arterial blood pressure (despite the use of pressor agents), persistent hypoxemia, and persistent tachycardia during surgery is also associated with poor survival. Examination of the colon can provide some additional information. Return of a normal pink serosal color after volvulus correction is a positive indicator, but it does not relate to mucosal viability; severe postoperative endotoxemia may still occur if the mucosa is devitalized. Visual examination of the mucosa made through a pelvic flexure enterotomy provides additional information. The presence of dark red or black mucosa with no active bleeding is a poor prognostic indicator. Frozen sections allow calculation of the interstitial-to-crypt (I:C) ratio and percentage loss of epithelium, which correlate well with survival. An I:C ratio of 3:1 or greater, or loss of greater than 95% of the epithelium, was associated with a 95% death rate in one study. However, this requires that personnel be able to obtain and evaluate frozen sections immediately. Other research tools that have been used to evaluate colonic viability include surface oximetry, fluorescein dye, and Doppler flow; none of these tools has gained popularity in a clinical setting (see Chapter 35).

The decision to perform a colon resection should also be based on the location of the volvulus. If the line of devitalized bowel is located distal to the cecocolic ligament, the outcome of a large colon resection is much more favorable, because resection of the affected bowel is possible. However, in most instances, the line of devitalization is located at the base of the colon orad to the cecocolic ligament; resection must be performed in a compromised portion of the bowel, which places the resection site at risk for dehiscence. Surgeons who are advocates of large colon resection for the treatment of large colon volvulus make a case that removing the majority of the diseased colon decreases the endotoxic load and therefore increases the likelihood of survival; in addition, removal of the large colon prevents recurrence of the condition. Surgeons who do not routinely perform large colon resections for large colon volvulus state that the procedure in itself has a high risk of complications and that in most cases it does not result in removal of the entire diseased colon. In addition, the increased anesthesia time may be detrimental to the animal. These debates emphasize the complexity of the decision-making process in these cases, the importance of experience and familiarity with surgical techniques, and the influence of types of cases on the decision-making process. For example, horses that are referred quickly and have a short duration of illness rarely require a large colon resection.

Prognosis

Preoperative parameters that indicate poor survival include PCV greater than 50%, rectal temperature greater than 39.5° C (102° F), and a heart rate greater than 80 beats per minute.[181] Intraoperative factors associated with a poor survival include black mucosal color, poor return of perfusion after detorsion, and an increasing PCV and decreasing total protein during surgery.[155] Abdominocentesis is usually normal in horses with large colon volvulus; an increase in total protein in the abdominal fluid is associated with decreased survival.[155] Abdominocentesis therefore does not provide information that will alter the decision for surgical treatment, and it has an increased risk of enterocentesis or injury to personnel because these animals are in severe pain.

Reported mortality for large colon volvulus varies from 56% to 65%. However, in one study, the short-term survival rate was 84%; this higher survival rate was thought to be related to a shorter duration of illness and faster intervention time.[182] Although short-term mortality is high after surgical correction of large colon volvulus, a long-term probability of survival of 80% has been reported once discharge from the hospital occurred.[181] The prognosis for short-term survival (discharge from hospital) following large colon resection in horses in one study was 57.7% but was decreased to 47% when only strangulating lesions of the large colon were considered.[183]

Right Dorsal Colitis

Right dorsal colitis is a specific type of ulcerative colitis observed in the right dorsal colon of the horse, specifically the aboral segment of the dorsal colon as it joins the transverse colon. The condition is recognized in association with administration of NSAIDs, and it has been reproduced experimentally by administration of phenylbutazone. Horses with a nervous predisposition seem more prone to develop the disease despite the administration of appropriate dosages of NSAIDs. Although NSAID administration is commonly associated with this condition, it has also been documented in the absence of NSAID administration.

Diagnosis

Clinical signs of right dorsal colitis can manifest as acute colic, endotoxemia, diarrhea, and even death. The disease is more common in the chronic form, where horses are presented for weight loss, hypoproteinemia, intermittent signs of colic, and intermittent diarrhea.

The diagnosis of right dorsal colitis is based on anamnestic information, particularly related to NSAID administration, and ruling out other causes of weight loss, hypoproteinemia, and diarrhea. Diagnostic ultrasonography of the right dorsal colon may help identify the thickened colon (Figure 37-15).[184] More recently, scintigraphy with radiolabeled white blood cells has been described to diagnose the condition.[185]

Treatment

Medical management is recommended for the initial treatment of right dorsal colitis. This includes discontinuing NSAIDs, modifying diet, and giving anti-inflammatory drugs, intestinal protectants, and metronidazole. Dietary modifications are important to help control the signs of colic.

In cases of protracted colic, or when intermittent colic is so frequent as to prevent the horse from maintaining itself, surgical exploration may be recommended. At surgery, the right dorsal

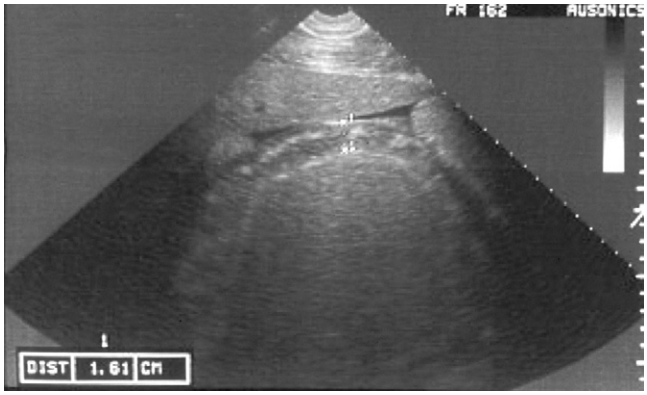

Figure 37-15. Sonographic image of the right dorsal colon showing marked thickening of the colonic wall in a horse with right dorsal ulcerative colitis associated with phenylbutazone toxicity. The image was obtained using a 7.5-MHz probe directed into the 10th right intercostal space.

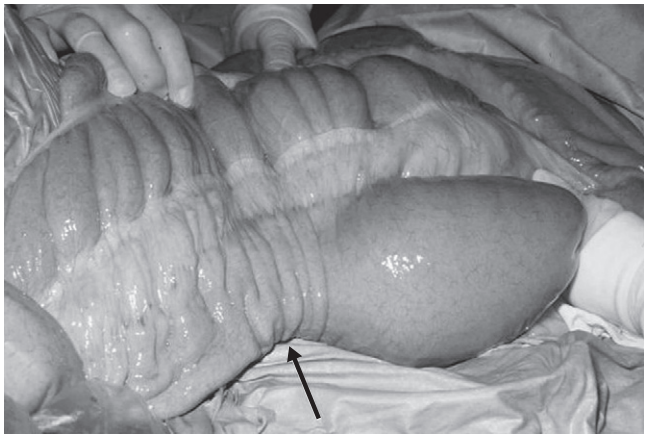

Figure 37-16. Intraoperative image of the right dorsal colon of a horse with right dorsal colitis. Note the markedly thickened colon with stricture at the diseased site *(arrow)*.

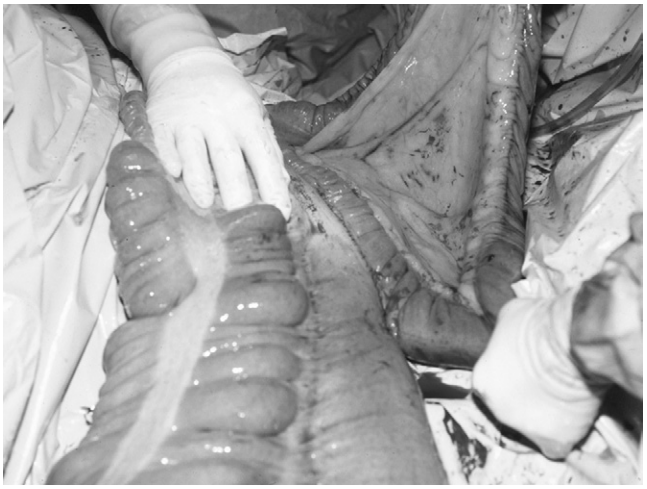

Figure 37-17. Side-to-side anastomosis of the right dorsal colon to the small colon in a horse with right dorsal ulcerative colitis.

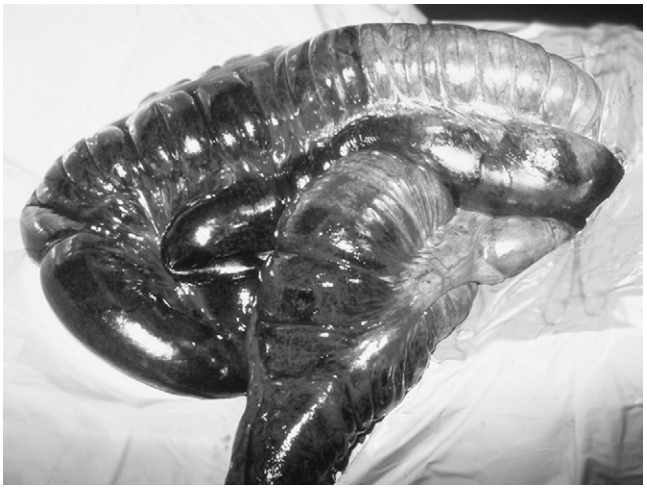

Figure 37-18. Mural infarction in a horse with colitis associated with salmonellosis.

colon feels markedly thickened, and it may be strictured with scar tissue (Figure 37-16). Once the disease is identified at surgery, treatment options include right dorsal colon bypass, resection of the affected portion of the colon, large colon resection, or large colon resection and bypass.

Bypass of the affected area of the large colon is performed by exteriorizing the large colon on a colon tray. The small colon is then exteriorized and a 20- to 30-cm (8- to 12-inch) side-to-side anastomosis between the right dorsal colon (orad to the lesion) and the small colon approximately 1 m distad to the transverse colon is performed (Figure 37-17).[186] When this procedure is performed, transient diarrhea has been observed. This procedure does not remove the affected portion of the colon, so the patient can continue to suffer from weight loss and hypoproteinemia until the colitis has resolved. Resection of the affected colon through a 16th rib resection has been reported in one horse.[187] This approach requires either prior knowledge of the disease or a second celiotomy approach once the diagnosis has been made. Resection and end-to-end anastomosis is possible only if the lesion does not extend too far aborad. In cases of severe right dorsal colitis with stricture of the right dorsal colon, large colon resection and anastomosis in a side-to-side fashion can be performed to restore intestinal transit. However, complete removal of the ulcerated area would not be possible with this approach. Another approach, which has not been described for the treatment of this condition, would be to amputate the large colon, particularly the dorsal colon, as far aborally as possible, and to perform an end-to-side anastomosis between the right ventral colon and the small colon. This approach has been reported for the management of nonfunctional dorsal colon lesions in two horses.[188,189]

Mural Infarction (Thromboembolic Colic)

Compromise to the mesenteric vasculature without evidence of strangulation has been described in association with arteritis resulting from *Strongylus vulgaris* larval migration, or in horses with severe colitis and coagulopathies (Figure 37-18).[190] Cases of larval arteritis can be acute (with signs of ischemic bowel disease and peritonitis) or chronic (with signs of recurrent colic and weight loss). Horses with acute thromboembolic colic demonstrate significant changes in their abdominocentesis,

consistent with those seen with peritonitis; horses with recurrent colic often have a normal abdominocentesis.

In acute cases, the decision for surgical intervention is based on the signs of colic and the presence of peritonitis. At surgery, mural infarction is identified, and if possible, resection of the affected segment of intestine is performed. Careful palpation of the cranial mesenteric artery should be performed; identification of severe enlargement, aneurysm, or abscessation is an indication for a guarded to poor prognosis. A regular systemic deworming program should be instituted as part of the postoperative care.

When horses with colitis show acute signs of abdominal pain and abdominal distention, thromboembolic colic and necrotizing colitis should be suspected. Many of these horses have extensive lesions that preclude surgical intervention. In addition, their systemic status, as a result of the primary colitis, makes them poor surgical candidates.

Other Strangulating Lesions of the Large Colon

Other strangulating lesions of the equine large colon that have been reported include incarceration in the epiploic foramen or the gastrosplenic ligament, large colon intussusception, and volvulus associated with abnormal mesenteric bands or defects.[45,191-195] Surgical intervention is dictated in these cases by the increasing degree of abdominal pain, abnormal rectal palpation, and abdominocentesis indicating intestinal compromise. Inguinal herniation of the large colon has been reported to occur spontaneously[196] and 4 weeks after castration.[197] Treatment was not attempted in these cases. Resection and anastomosis of the affected colon is required to correct strangulating lesions of the large colon.

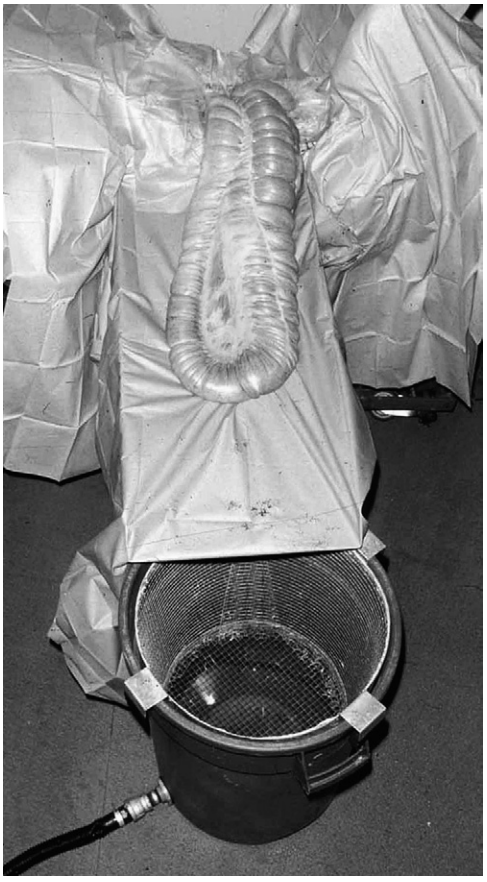

Figure 37-19. Modified colon tray used to evacuate the large colon. In this case, the contents are collected in a modified trash can with a strainer that allows fluids to be evacuated through the drainage system.

Surgical Procedures

Large Colon Enterotomy

The most commonly performed procedure in the large colon is pelvic flexure enterotomy for evacuation of the large colon. Enterotomy procedures are also performed to remove foreign bodies, usually in the right dorsal colon. Finally, right ventral colon enterotomy is used to access the cecum in cases of cecocolic intussusceptions.

PELVIC FLEXURE ENTEROTOMY AND LARGE COLON EVACUATION

For this procedure, the large colon is exteriorized and placed on a colon tray (Kimzey Enterotomy Surgery Table—see Appendix B), either on the left side of the horse or caudally between the horse's hind legs. If a colon tray is not available, the colon is best exteriorized between the hind legs. Some surgeons place the end of the laparotomy drape onto the tray to create a shelf for the colon to rest on. The enterotomy drape is then placed on top of the laparotomy drape and secured with towel clamps. The colon tray is angled at about 20 degrees. A modified trash can with an ingesta strainer and a liquid outflow hose is used in some clinics to collect and evacuate the contents of the colon (Figure 37-19).[198] Alternatively, the end of the tray can be positioned over a disposal system built into the surgery room (Figure 37-20).[199] In anticipation of colonic evacuation, a lavage system should be established. This can be done by using two garden hoses, or one hose with a Y-connection. One hose is used for

Figure 37-20. Built-in disposal system for collecting fecal material during evacuation of the intestine.

intraluminal lavage and evacuation of the colon, and the other for extraluminal lavage, using warm water (37° C (98.6° F)).[199] If these are not available, stomach tubes, buckets, and pumps are used.

A full-thickness, 8- to 12-cm (3- to 5-inch) incision is made on the antimesenteric border of the pelvic flexure.[198] One hose is inserted and gently advanced into the colon, while the other is used to continuously lavage the serosal surface to prevent fecal contamination. During the evacuation procedure, it is useful to lift the colon and ensure that the underside is lavaged as well. With sterile technique, an assistant helps to feed the hose into the colon and massage its contents. Although a modified colon tray has been described to evacuate the colon, it is not deemed necessary by most surgeons.[200] At this time, if colonic evacuation is performed as part of the correction of a large colon torsion, an intestinal biopsy can be collected. Closure of pelvic flexure enterotomies is performed using 2-0 absorbable suture material in two layers: a simple-continuous seromuscular layer followed by a Lembert or a Cushing pattern.[201] The colon is rinsed thoroughly with sterile saline and replaced in the abdomen.

Enterotomies in the right dorsal or ventral colon are performed after the colon has been draped off from the main surgical field. In the ventral colon, enterotomies are performed between teniae, since the fibrous nature of the teniae precludes successful inversion during suturing. In the dorsal colon, the location of the enterotomy is not as critical. Enterotomies performed at these sites are more likely to suffer from postoperative luminal hemorrhage; a full-thickness closure (simple-continuous or Connell) is therefore performed on the first layer to achieve better hemostasis.[84] A Lembert or Cushing pattern is used as the second layer.

A modified Heineke-Mikulicz technique for pelvic flexure enterotomy closure has been described in two horses with extensive stricture of the large colon.[175] A longitudinal incision was centered over the stricture and closed using a transverse closure, effectively increasing the diameter of the colon at that site.

Large Colon Resection

Resection of the large colon is performed to remove full-thickness mural defects in the large colon. Causes include strangulation, infarction, thromboembolic disease, neoplasia, and scar tissue formation.[177,197,196,202-208]

Removal of 50% to 95% of the large colon has been described.[208-213] Techniques for resecting the large colon include resection and end-to-end anastomosis, and resection and side-to-side anastomosis. End-to-end procedures are performed to remove the colon up to 10 to 12 cm (4 to 5 inches) from the cecocolic ligament.[179] When resection of the colon closer to or proximal to the cecocolic ligament is required, a side-to-side technique is preferred because the end-to-end technique exerts too much tension on the anastomotic site, placing it at risk for dehiscence. In our experience, it is important to ensure that apposition of the colons in an end-to-end fashion is achieved without tension, and this requires that a substantial amount of colon remain distal to the cecocolic fold.

RESECTION AND END-TO-END ANASTOMOSIS

For a *resection and end-to-end anastomosis,* the colon is exteriorized on a colon tray and draped off; the cecum is replaced in the abdomen. All ingesta are massaged into the segment to be removed. If the colon is full, a pelvic flexure enterotomy is performed to evacuate the colon; care must be taken to remove all ingesta and water from the right dorsal and ventral colons to minimize contamination during the resection. For the resection, a site is chosen that is located in the healthy part of the colon (if possible) and that is easily held by the assistant surgeon without tension on the mesentery.[179] The colon is positioned to expose the right colic artery and the colic branch of the ileocolic artery within the mesocolon; the colonic vessels are isolated by blunt finger dissection and double-ligated using 1 polyglactin 910 (Figure 37-21). Alternatively, two double rows of staggered staples (TA-90 Premium) are applied across the mesocolon, followed by a third double-staggered row 4 cm distal. The mesocolon is transected between the proximal and distal staple lines. Hemostasis is verified and any bleeding vessels are ligated. The right ventral colon is transected in a line transverse to its long axis, whereas the dorsal colon is transected at a 30-degree angle to its long axis, with the antimesenteric border shorter so that the diameters of the colons are similar (Figure 37-22, *A*). In the original description of the procedure, a V-shaped stoma was then created between the mesenteric border of the dorsal and ventral colons using an inverting linear anastomotic instrument (GIA Premium 55) reinforced with a double-layer, simple-continuous pattern. This step was omitted in later descriptions of the procedure.[214] Starting at the mesenteric border and suturing from the lumen, the colons are apposed with a double-row, simple-continuous pattern using 0 polydioxanone or Vicryl. The second layer should be full thickness to achieve hemostasis (see Figure 37-22, *B*). Once the mesenteric portion of the anastomosis is completed, suturing is approached from the serosal side, and the colons are apposed using a double layer, starting with a Connell followed by a Lembert pattern (see Figure 37-22, *C*). The colon is lavaged and replaced in the abdomen.

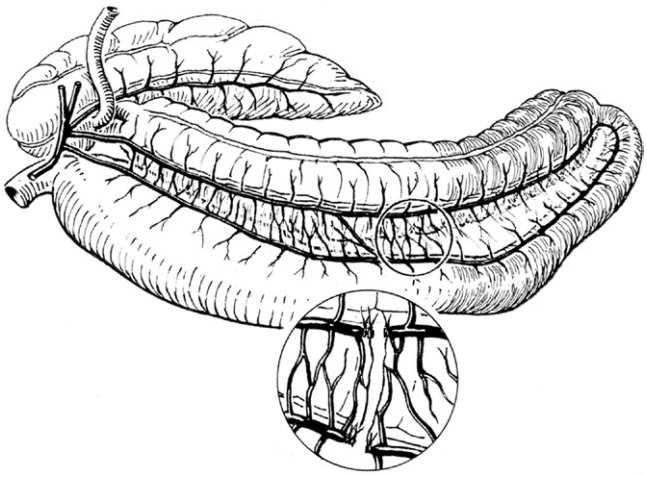

Figure 37-21. The ascending colon is placed on a colon tray with the vessels facing uppermost. The colonic vessels are isolated by blunt finger dissection and double- or triple-ligated before transection *(inset).*

SIDE-TO-SIDE ANASTOMOSIS AND RESECTION

For a *side-to-side anastomosis,* the site of resection is usually more proximal, at the cecocolic ligament or orad to it. The colon is exteriorized to the left of the horse on a colon tray and the colonic vasculature is transected as previously described. The

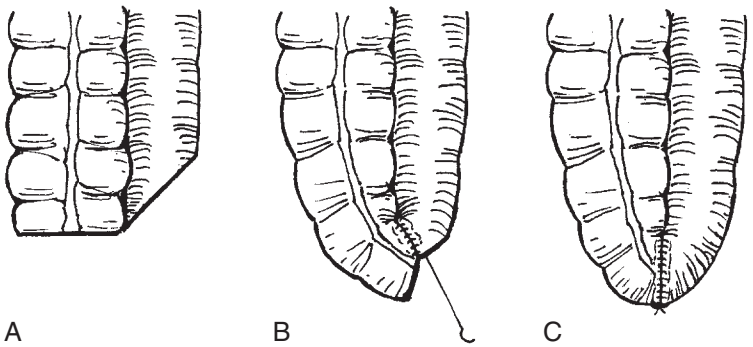

Figure 37-22. Amputation of the large colon. **A,** The right ventral colon is transected in a line transverse to its long axis, whereas the dorsal colon is transected at a 30-degree angle to its long axis. **B,** Starting at the mesenteric border, and suturing from the lumen, the colons are apposed with a double-row simple-continuous pattern using 0 polydioxanone or Vicryl. **C,** Once the mesenteric portion of the anastomosis is completed, suturing is approached from the serosal side, and the colons are apposed using a double-layer pattern, starting with a Connell and followed by a Lembert.

stoma is then created before the resection, taking advantage of the weight of the colons to facilitate exposure of the anastomotic site. The site for creating the stoma is identified, taking care to end it just proximal to colonic vessel ligation, so that a blind sac is not formed. A three-tier side-to-side stoma is created; the first layer apposes the seromuscular layers of the colons using 1 polyglactin 910 in a Lembert or Cushing layer for a length of 20 cm (8 inches) (Figure 37-23, *A*). A full-thickness incision is made in each colon and a full-thickness simple-continuous circumferential closure interrupted at 180 degrees is made to create the stoma (see Figure 37-23, *B* and *C*). The upper layer is subsequently apposed using a Cushing or Lembert pattern (see Figure 37-23, *D*). Alternatively, the stoma can be created using stapling instrumentation. This may minimize contamination, but it may not be possible if the colons are too thick and edematous as a result of the underlying disease process. For an adequate-size stoma, the stapling instrument is fired twice if the ILA-100 or the GIA-90 is used and three times if the GIA-55 is used (the staples for the GIA-55 are smaller). Staple lines should be oversewn. Once the staples have been applied, the down layer cannot be approached for oversewing. This layer should therefore be established first, before application of the stapling instrument. Once the stoma is created, the colons are resected, starting with the ventral colon, taking care to resect them at the site of colonic vessel ligation. The lumens are closed using a full-thickness simple-continuous or Connell pattern, and oversewn with a Lembert or Cushing pattern (Figure 37-24). The resected colons are lavaged and replaced in the abdomen. Although resection of the large colon has been reported using stapling instrumentation, this is usually not possible after correction of large colon torsion, since the intestinal edema and congestion do not allow proper closure of the instrument and adequate formation of the staples.

An early method of large colon resection has been described that uses a luminal approach to create the side-to-side anastomosis, followed by closing the ends of the colons.[211] The technique described earlier for resection and end-to-end anastomosis represents a modification of this technique.

Successful bypass of the right dorsal colon for the treatment of large colon volvulus has been described in one horse.[189] In that report, the large colon was judged to be nonviable after correction of a large colon volvulus. The right dorsal colon was transected as far distally as possible within the abdomen, using a TA-90 stapling instrument, and the suture line was partially oversewn. The right ventral colon was transected 10 cm (4 inches) from the cecocolic ligament, and an end-to-side anastomosis between the right ventral colon and the descending

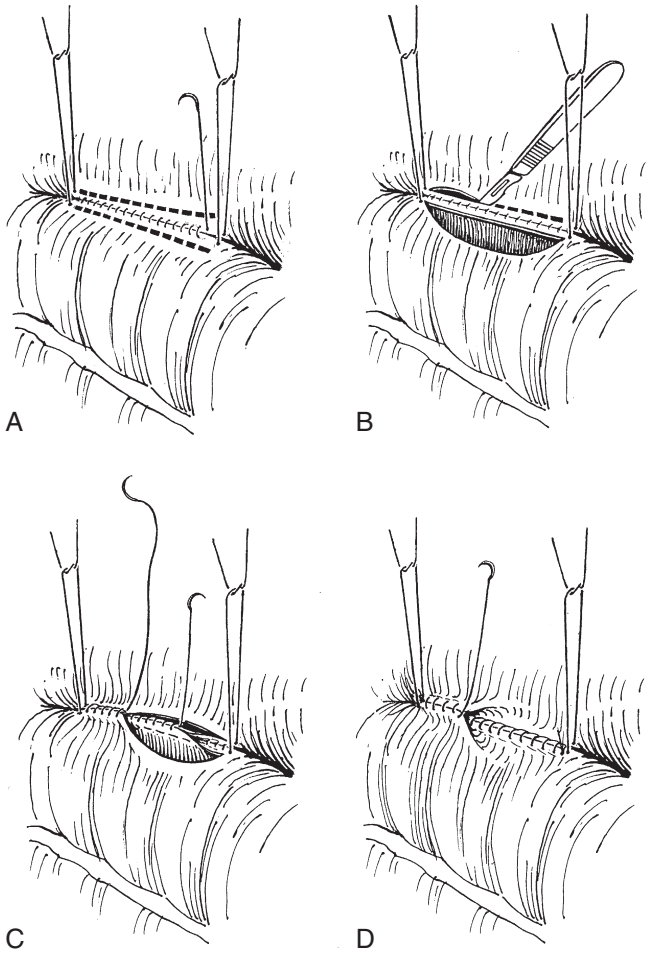

Figure 37-23. A hand-sutured side-to-side anastomosis of the ascending colon. **A,** Placement of stay sutures at either end of the intended stoma. Note that the anastomosis is performed with the diseased part of the colon still *in situ*. The seromuscular layers of the ventral and dorsal colons are apposed with a Lembert or Cushing pattern, using an absorbable suture material. **B,** A full-thickness incision is made in each colon, and a full-thickness simple-continuous circumferential closure, interrupted at 180 degrees, is made to create the stoma, shown in **(C). D,** The upper layer is then apposed using a Cushing or Lembert pattern.

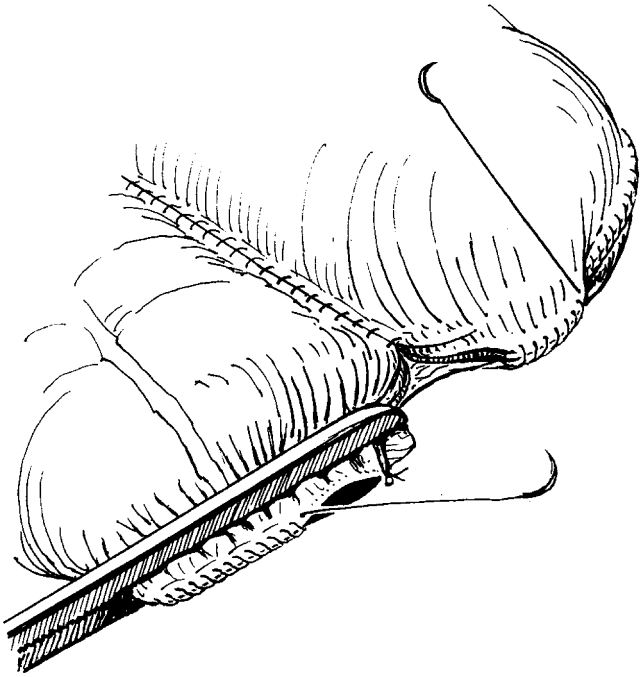

Figure 37-24. The amputated ends of the large colon are closed with a hand suture technique. Note that the amputation of the colon occurred next to the anastomosis site to prevent the formation of a blind end. The lumens are closed using a full-thickness simple-continuous or Connell pattern, oversewn with a Lembert or Cushing pattern.

colon was performed using a double-layer inverting pattern. Two mild episodes of colic and diarrhea for 1 week were the reported complications. In a similar fashion, anastomosis of the right ventral colon to the descending colon to bypass a nonfunctional descending colon anastomosis in a miniature pony was reported.[188] A two-layer hand-sewn end-to-side anastomosis was performed between the right ventral colon and the small colon. One episode of colic successfully treated with medical therapy was the only postoperative complication encountered in this case.

COMPLICATIONS

Complications from large colon resection are usually a result of the primary disease. They include persistent endotoxemia and peritonitis caused by continued bowel devitalization. This is because the site of torsion is usually at or proximal to the site of resection, so that some portion of compromised large colon cannot be removed. It is therefore essential for the surgeon to remove as much of the devitalized colon as possible. In our experience, this requires a side-to side resection at or proximal to the cecocolic fold. Even then, a segment of devitalized colon may remain in the abdomen, leading to subsequent complications. Most horses that succumb to endotoxemia and peritonitis do so within 3 to 7 days postoperatively after requiring considerable intensive care. In contrast, survivors see an improvement in clinical signs within 24 hours of the procedure.

Postoperative pain is common in horses after large colon resection. It is a painful procedure, even in normal horses. Administration of NSAIDs, lidocaine, and opiates can help alleviate the pain.

Signs of endotoxemia are common after large colon resection for large colon volvulus. Signs include fever, tachycardia, injected mucous membranes, dehydration, and hypoproteinemia. Signs of large colon ileus, such as distention, which can be quite severe, may also occur. Supportive care with fluids, plasma, and antiendotoxin modalities are important (see Chapter 2). Horses should be monitored for signs of postoperative hemorrhage, which is more prevalent with large colon procedures and occasionally requires a blood transfusion.

Postoperative diarrhea is commonly observed after large colon resection, because of mucosal damage or the reduced surface area available for fluid absorption, or both. The diarrhea is usually self-limiting and resolves within a few days if it is not of infectious origin. However, isolation procedures should be followed as dictated by hospital protocol, and infectious diseases should be ruled out.

Horses with successful resection of the large colon usually regain normal fecal consistency within 5 to 7 days. Because of the decreased surface area available for digestion and water absorption, these horses have increased water and phosphorus requirements and require a highly digestible diet.

PROGNOSIS

The prognosis after large colon resection depends on the reason for performing the resection, but the procedure does carry inherent risks, as demonstrated by a study where large colon resection performed in normal horses resulted in 3 of 10 horses dying directly as a result of the procedure.[209] In a group of horses having undergone large colon resection, there was a significant difference in short-term survival (discharged from the hospital) between horses with a nonstrangulating lesion (77.8%) compared to a strangulating lesion (47%), and horses that underwent a large colon resection for nonstrangulating lesions were 3.9 times more likely to survive.[183]

SMALL COLON

Anatomy

The transverse colon is the continuation of the right dorsal colon. It begins at the level of the 17th or 18th thoracic vertebra where the right dorsal colon narrows significantly in diameter as it turns medially.[1-2] The transverse colon is short and passes from right to left, cranial to the cranial mesenteric artery. It is connected dorsally to the pancreas, to the dorsal aspect of the abdominal cavity, and by a short transverse mesocolon to the root of the mesentery. These attachments prohibit visualization of the transverse colon during a celiotomy. To the left of the root of the mesentery, the transverse colon continues as the descending colon, also known as the small colon. The small colon occupies the left caudodorsal quadrant of the abdominal cavity. It is approximately 3.5 m long and maintains a 7- to 10-cm width throughout its entire length. It is suspended by a long descending mesocolon, which allows good surgical access to all but the most proximal and distal portions of its length. The descending mesocolon originates from the left surface of the root of the mesentery and continues caudad attached to the dorsal body wall until it turns into the mesorectum at the pelvic inlet.[1-2] The mesocolon often contains a significant amount of fat. The small colon is attached to the terminal duodenum by the duodenal colic fold, which is an important surgical landmark when exteriorizing the proximal jejunum. The small colon has two longitudinal muscular bands called teniae, one within the mesocolon and the other on the antimesenteric

border. Contractile activity of the longitudinal bands and circular muscle produce sacculations in which feces are formed into fecal balls.[215]

The vascular supply to the small colon is derived from the caudal mesenteric artery with anastomoses cranially from the cranial mesenteric artery and caudally from the middle and caudal rectal arteries. The caudal mesenteric artery divides into two major branches, the left colic artery and the cranial rectal artery, with the left colic artery supplying the proximal three fourths of the descending colon and the cranial rectal, the distal one fourth.[215] The left colic artery branches into four to eight arcuate arteries, each dividing into a cranial and caudal branch, which run parallel to the small colon to anastomose to adjacent arcuate arteries (Figure 37-25). The cranial rectal artery gives off similar arcuate vessels. These anastomosing arcades running parallel to the bowel are called *marginal arteries*. A secondary anastomosing arcade branching off the marginal arteries runs between the marginal artery and bowel wall, sending off short branches that perforate the mesenteric teniae and long branches that anastomose in the antimesenteric teniae with branches from the other side (see Figure 37-25).[215] Venous return parallels the arterial supply. The presence of fat in the mesentery makes identification of blood vessels difficult.

Pathology of the Small Colon

Horses with small colon disease make up 1.9% to 18.5% of horses admitted to referral centers with signs of gastrointestinal disease.[96,155,214-218] The incidence of small colon disease is approximately 4% in horses undergoing surgical treatment for colic.[96,214-218] Arabian horses, ponies, and American Miniature Horses are more predisposed to diseases of the small colon.[155,219] Female horses and horses older than 15 years are more likely to have small colon disease, whereas horses younger than 5 years are less likely to be affected.[155] Diseases may be broken down into congenital abnormalities, such as atresia coli and colonic aganglionosis; simple obstructions, including fecal impaction, fecaliths, phytobezoars, trichobezoars, enteroliths, and retained meconium; vascular lesions, including intramural hematomas and mesocolon tears; strangulating lesions such as lipomas, internal hernias, and volvulus; and neoplasia. (Distal iatrogenic tears, described as rectal tears, although frequently occurring in the small colon, are discussed in Chapter 38.)

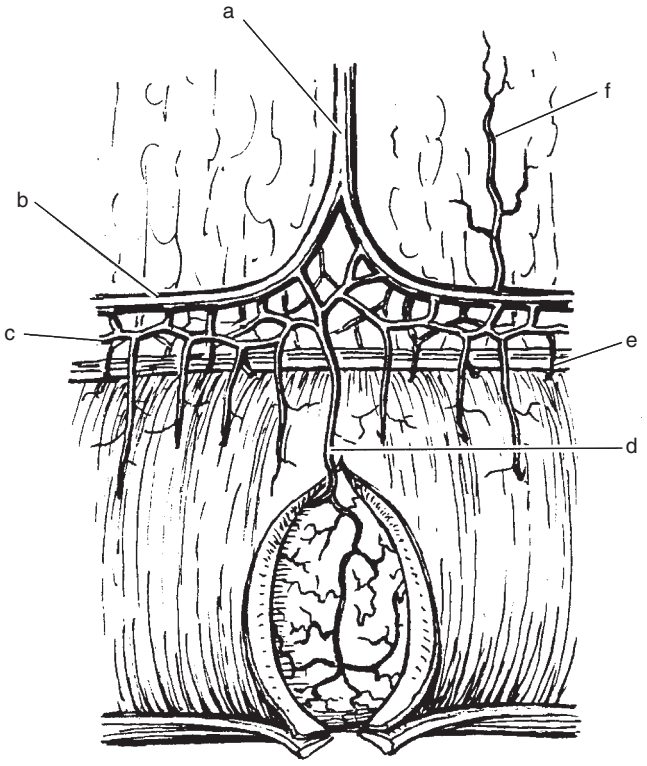

Figure 37-25. Vascular supply of the descending colon, lateral view. *a,* Arcuate artery; *b,* marginal artery; *c,* secondary arcade; *d,* long artery; *e,* small branch supplying mesenteric teniae; *f,* small branch to mesocolon.

Diagnosis

According to one report, 84% of surgical conditions of the small colon were simple obstructions, whereas 16% were vascular or strangulating lesions.[155] Because the obstruction generally affects the distal part of the gastrointestinal tract, horses with small colon disease exhibit slow deterioration of physical status and slow worsening of clinical and laboratory evaluations, making the decision for both referral and surgical intervention more prolonged than is seen with other surgical conditions of the gastrointestinal tract.[20,155,215,220,221] Early clinical signs are nonspecific and include lethargy, dullness, and inappetence. As the obstruction progresses, signs include abdominal pain, diarrhea, decreased fecal production, tenesmus, and abdominal distention. Strangulating lesions may have a more sudden and severe onset of clinical signs, although the level of pain at admission was reported to be mild or moderate in 25 of 31 cases requiring a small colon resection in a recent study.[221] Gastric reflux is uncommon with impactions but may occur as a sequela of the distal obstruction, leading to distention of the ascending colon, which places pressure on the proximal small intestine and obstructs gastric outflow. Gastric reflux has been reported in 30% of horses with strangulating lesions.[221] Changes in the nucleated cell count and the total protein concentration in peritoneal fluid, along with rectal palpation findings, are the key indicators for surgical intervention.[155,214,218] Abdominal ultrasonography may also aid in making a diagnosis.[222] Medical and surgical interventions often carry a good to excellent prognosis.

Simple Obstructions

Fecal Impaction

Horses with small colon fecal impaction represent 1.9% to 2.5% of all those admitted for signs of abdominal pain. Fecal impaction has been reported to be the most common pathologic condition of the small colon except in regions with a high incidence of enterolithiasis.[155,216,217] Suggested risk factors are poor dentition, poor-quality hay, lack of water, parasite damage, lack of exercise, submucosal edema, and motility problems.[218,220,223] Horses with small colon impactions were found to be 10.8 times as likely to have diarrhea at the time of admission than were horses with large colon impactions.[223] The fall and winter seasonal predilection observed in some studies may be the result of inadequate water consumption or change of feed.[216,217] Older horses (older than 15 years), American

Miniature Horses, and ponies were at an increased risk in several studies, but age, breed, and sex were not significant risk factors in other reports.[20,155,216-218,224]

DIAGNOSIS

The most common clinical signs, in order of decreasing frequency, are abdominal pain, decreased manure production, diarrhea, anorexia, fever, straining to defecate, and depression.[217,218] The colic is initially manifested as mild abdominal pain with a slow progression of signs. The heart rate can be mildly to moderately elevated. Leukopenia with a left shift is common in horses with small colon feed impactions, whereas serum biochemical and peritoneal fluid values are typically within normal ranges.[217,218] The diagnosis can frequently (i.e., in 75% to 87% of cases) be made by palpation of the impaction *per rectum*.[217,218,225] Other palpation findings that have been noted are distention of the large intestine and cecum, edematous or rough rectal mucosa, blood on the rectal sleeve or manure, and unformed feces.[217,218,226] Nasogastric reflux is occasionally obtained. The presence of diarrhea, fever, leukopenia, and irritated rectal mucosa often encountered in a subset of these cases suggests that colitis may predispose some horses to small colon impaction, possibly through motility disturbances.[155,223] Conversely, the gastrointestinal stasis and inflammation associated with small colon impaction may predispose horses to colitis.[217,227]

TREATMENT

Medical treatment

Most fecal impactions of the small colon are initially managed medically. The goal is to improve hydration, stimulate motility, and soften the impaction. All feed should be withheld and the impaction hydrated. This is accomplished through administering both intravenous fluids (two to three times the maintenance volume) and oral fluids (2 to 4 L every 4 to 6 hours).[217,223] Oral fluids have the additional benefit of initiating a gastrocolonic reflex, whereby gastric distention stimulates contractile activity of the colon.[115,228] Oral laxatives such as mineral oil, dioctyl sodium sulfosuccinate, or magnesium sulfate administered by nasogastric tube are commonly used to soften the impaction. Mineral oil (5 to 10 mL/kg every 12 hours) is frequently used to lubricate the impaction, and its presence in the perineum is an indication that the obstruction is incomplete. Magnesium sulfate administered to normal horses at 1 g/kg in 6 L of water has been shown to increase total weight of fecal excretion and fecal water excretion compared with administration of a lower dose (0.5 mg/kg), whereas DSS (50 mg/kg in 6 L of water) failed to increase the weight of feces excreted or fecal water excretion.[115] Magnesium sulfate can be administered once daily for 2 to 3 days if the horse is well hydrated. Magnesium toxicity appears more likely to develop in dehydrated horses or in horses treated simultaneously with DSS.[128] Flunixin meglumine (0.25 mg/kg three times a day, or 1.1 mg/kg twice a day IV) is administered to treat endotoxemia and to control pain if necessary. Antibiotics may be indicated in horses with signs of septicemia, severe leukopenia, or concurrent diarrhea. Other antiendotoxin therapy such as polymyxin B (2000 to 6000 IU/kg IV) or *Salmonella typhimurium* antiserum may also be beneficial.[217] Standing enemas may be used in selected cases to break down impactions in the distal small colon, but extreme caution should be exercised to avoid producing a rectal or small colon tear.[216,217] The horse should be adequately sedated and restrained, and epidural anesthesia should be performed. The enema should be administered, without pressure, by gravity flow. Because of the potential for iatrogenic trauma, some authors do not recommend enemas for treatment of small colon impactions.[214,216,217]

Surgical treatment

Surgical management is indicated in horses with increasing abdominal pain, increasing abdominal distention, or changes in abdominal fluid indicating deterioration of the bowel.[155,218,224] In some horses, large colon displacement or volvulus occurs secondary to gas distention proximal to the impaction. The small colon is exteriorized through a ventral midline celiotomy. The most common technique to relieve the impaction is intraluminal lavage with warm water introduced by stomach tube inserted through the anus by an assistant. The surgeon manipulates the tube to the distal extent of the impaction and then manually clamps off the small colon lumen caudal to the end of the tube to allow water distention around the distal extent of the impaction. Extraluminal massage is used to gently work the fluid into the feces to break up the distal portion of the impaction. These manipulations are repeated working orad until the impaction is relieved. To decrease mechanical damage to the serosa during the massage, sterile carboxymethylcellulose should be placed on the small colon that is being massaged.[217] In some cases, when the small colon impaction cannot be relieved by intraluminal lavage with external massage, a small colon enterotomy is performed in the antimesenteric teniae adjacent to the impacted small colon, and lavage is used to evacuate the impacted fecal material through the enterotomy (see "Small Colon Enterotomy," later). In rare cases, mural ischemia or thrombosis of the mesenteric vessels necessitates resection of a segment of small colon (Figure 37-26) (see "Small Colon Resection and Anastomosis," later). Ingesta in the large colon should be evacuated through a pelvic flexure enterotomy to prevent postoperative impaction in the edematous and inflamed small colon.

Postoperatively, horses should be started back on feed slowly. Feeding a complete pelleted diet for several weeks may help prevent reimpaction.[221] Extension of perioperative use of antibiotics (24 hours postoperatively) to therapeutic

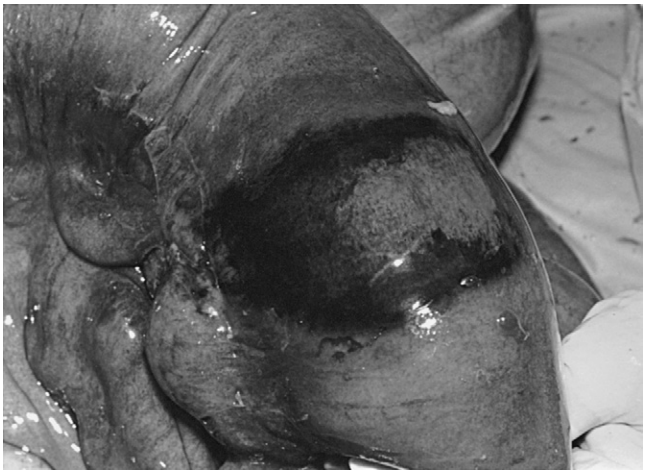

Figure 37-26. An intraoperative photograph of a horse with a descending colon impaction showing an area of compromised tissue.

use of antibiotics (5 to 7 days postoperatively) is made on a case-by-case basis, dictated by surgical findings and clinical and laboratory evaluation of the horse. In most cases, antibiotics can be discontinued within 24 hours.

PROGNOSIS

Survival after medical therapy has been reported to be good to excellent (72% to 100%), and survival after surgical therapy has been reported to be fair to excellent (47% to 95%).[155,217,218,224] The most common complications after medical or surgical therapy are diarrhea and jugular thrombophlebitis.[155,218,224] Other complications seen are fever, laminitis, peritonitis, incisional infection, incisional hernia, recurrent colic, recurrent impaction, and small colon adhesions.[155,218,224] Surgical cases in which the colon was not evacuated at surgery are more likely to develop postoperative pain.[217] Fecal cultures for *Salmonella* are positive in up to 43% of surgically treated horses.[217,223] Postoperative antibiotic therapy may increase the risk of these horses developing salmonellosis by altering gastrointestinal flora.[217,221]

Intestinal Concretions and Foreign Bodies

Enterolithiasis

The small colon is the site of the obstruction in approximately 45% to 57% of horses with enteroliths, accounting for up to 35% of all small colon diseases in certain geographic regions such as California.[142-143,155,229] The etiology was discussed in the large colon section of this chapter.

DIAGNOSIS

Horses commonly exhibit multiple episodes of mild colic, most likely from intermittent obstruction in the large colon or proximal transverse colon, before the enterolith enters the small colon. Once the enterolith enters the small colon, the clinical course is determined by the size of the enterolith. Although some are small enough to pass through the small colon without clinical problems, it is common for the enterolith to become wedged within the lumen of the transverse or small colon, usually the proximal third.[230] Horses typically show signs of increasing abdominal pain, although in some horses rupture occurs with little or no observed abdominal pain. On rectal examination, the large colon may have palpable gaseous distention. Peritoneal fluid analysis should be monitored closely. It is common for enteroliths wedged in the transverse and small colons to cause pressure necrosis at the area of obstruction.[231] A slight elevation in peritoneal fluid protein may indicate the need for surgical intervention. Radiographs successfully diagnose small colon enteroliths in approximately 40% of horses, as compared with 83% of horses with large colon enteroliths.[144] In one case series, horses with enteroliths in the descending colon had a shorter duration of clinical signs and were more likely to be tachycardic and leukopenic than horses with ascending colon enteroliths. Surgical exploration is based on signs of increasing pain, increasing abdominal distention, peritoneal fluid changes, and radiographic evaluation.[142-144,232] The most common site of gastrointestinal tract rupture in horses with enteroliths is in the descending colon.[143] Since some horses show little pain before rupture, careful monitoring of horses with suspected enterolithiasis is critical to ensure timely surgical intervention.

TREATMENT

Treatment is surgical removal through an antimesenteric small colon enterotomy or retropulsion and removal through an enterotomy in the dorsal colon or pelvic flexure.[143,230] Horses are administered broad-spectrum antibiotics, and a ventral midline celiotomy is performed. Retropulsion with lavage can be accomplished by inserting a hose up the rectum. Alternatively, a hose can be inserted through a pelvic flexure enterotomy and advanced to the transverse or small colon obstruction, and lavage can be used to distend the small colon on the oral side with the anal side being clamped off manually. The most important aspect of these techniques is letting the water gradually distend the bowel to aid in dislodging the enterolith. Pressure necrosis can make the bowel wall susceptible to rupture.[233]

When enteroliths are removed by a small colon enterotomy, it is important to ascertain if there are additional enteroliths located proximally, as nearly half of horses with descending colon enteroliths have additional stones in the ascending colon.[143,232] Particular attention should be paid to the right dorsal and transverse colon, since it may be difficult to palpate a small enterolith if there is an appreciable amount of ingesta in the large colon. Commonly, a pelvic flexure enterotomy is performed before the small colon enterotomy to evacuate ingesta from the large colon. This also will decrease the amount of fecal material passing over the small colon enterotomy site in the immediate postoperative period.

PROGNOSIS

Horses recovering from surgery for enteroliths in the small colon have a good to excellent prognosis (up to 92.5%).[142-143,232] In most cases, antibiotics are discontinued 24 hours after surgery. It is common for the area where the enterolith was wedged in the small colon to slough some of the mucosa after surgery with absorption of endotoxin. NSAIDs should be continued for 3 to 5 days, depending on the extent of intestinal insult found at surgery. Complications noted after surgery are diarrhea, incisional infection, incisional hernia formation, positive fecal cultures for *Salmonella*, laminitis, septic peritonitis, and adhesions.[142-143,230,232] In one study, there was no significant difference in short-term complications between horses that had an enterolith in the ascending compared to the descending colon, although more horses with descending colon enteroliths were euthanized on the table, and of those recovered, more had a transient fever and anorexia.[232] Dietary and management changes have been discussed in the large colon section of this chapter. Recurrence has been identified in 7.7% of cases.[143]

Fecaliths, Phytobezoars, Trichobezoars, Phytotrichobezoars, and Phytoconglobates

Fecaliths are inspissated balls of fecal material that form as a result of poor-quality diet, poor mastication, or reduced water intake.[226,234] They have been reported to occur in approximately 7% of horses with small colon disease, but they probably make up a larger proportion of small colon disorders in regions where enteroliths are not common.[155] Small colon fecaliths have a tendency to occur in young (less than 1 year old) and older (more than 15 years old) horses. Ponies, American Miniature Horses, and mixed-breed horses appear to be at an increased risk.[155,235,236] *Phytoconglobates* are concretions of matted plant residues formed into balls. *Bezoars* are combinations of concretions of magnesium ammonium phosphate and plant material

(phytobezoars) or hair (trichobezoars). Concretions composed of hair and plant material are called *phytotrichobezoars*.[220,225] Obstruction caused by ingestion of fibrous, nondigestible material occurs most commonly in horses less than 3 years old and in horses with poor dentition.[226] Fecaliths, phytobezoars, trichobezoars, and phytotrichobezoars form in the large colon and become symptomatic as they pass into the small colon and obstruct the lumen. Clinical signs are similar to those of foreign body obstructions. Treatment is surgical removal by small colon enterotomy or by retropulsion into and removal from the large colon.

Foreign Body Obstructions

Obstruction of the small colon with foreign objects has been reported in several studies.[25,236-238] The obstructing objects include rubberized fencing and nylon tires, baling twine, rope, disposable plastic sleeves, feed sacks, and cloth material.[25,236-238] Young horses (less than 3 years of age) with indiscriminate eating habits are thought to be more likely to develop these foreign body impactions. Poor pasture may also contribute to consumption of inappropriate material.[238]

Signs reported are similar to those seen in other small colon obstructions, such as getting up and down, stretching, anorexia, and scant passage of feces.[25,236-238] Walking backward and dog sitting have also been reported.[236] The heart rate can be normal to markedly elevated. Depending on the configuration of the foreign body, the obstruction may be incomplete, allowing some passage of gas and ingesta, or complete, producing tympany and impaction proximal to the obstruction. With complete obstruction and proximal distention, the abdominal pain becomes more severe. Nasogastric reflux from the distended large intestine compressing the proximal small intestine may occur. In horses with a distal obstruction, tenesmus is common.[226] On rectal palpation, horses frequently exhibit marked abdominal straining, have a large intestine distended with gas and feces, and have no fecal balls in the small colon. In some cases, the obstructing mass is palpable *per rectum*, although frequently the obstruction is in the proximal small colon out of reach of rectal palpation.[238]

Foreign bodies may produce pressure necrosis of the wall of the small colon where they are lodged. In these cases, peritoneal fluid analysis reveals an elevated protein concentration and sometimes an increase in nucleated cell count. Treatment is surgical removal via a small colon enterotomy in the majority of cases, or through retropulsion into the dorsal colon if the obstruction is at an inaccessible portion of the proximal small colon or transverse colon.

Meconium Impaction

Meconium is a mucilaginous material in the intestine of the fetus, composed of intestinal glandular secretions, bile, epithelial cells, and swallowed amniotic fluid.[214,239] Typically, peristaltic activity moves meconium into the colon or rectum before birth, and it is evacuated between 3 and 24 hours after birth.[214] Meconium is considered retained if the foal makes frequent attempts but fails to produce meconium by 12 hours of age.[239] Retained meconium is the most common cause of colic in the neonate.[96,214,239] Meconium impaction accounted for 0.8% of all cases of equine colic in one study, with a mortality rate of 15.2%.[240] Ingestion of colostrum promotes gastrointestinal activity and may have a laxative effect to aid in expulsion of meconium from higher up in the colon. Delayed ingestion of colostrum and conditions that compromise the foal such as asphyxia, dystocia, prematurity, low birth weight, and dehydration are thought to be risk factors for meconium impaction.[241] Males are more commonly affected, most likely because of their smaller pelvis.[242]

DIAGNOSIS

Clinical signs begin with restlessness, tail switching, straining to defecate, and disinterest in nursing.[239] As the condition progresses, foals develop signs of more severe colic, such as lying down and getting up, rolling, lying upside down, and abdominal distention.[239] The diagnosis is based on clinical signs, historical absence of meconium passage, and detection of a firm mass on digital rectal palpation. Plain and retrograde contrast abdominal radiography can show tympanic bowel and often dense meconium.[243,244] Ultrasonography may show a distended large colon. The differential diagnosis is ruptured bladder, atresia coli, ileocolonic aganglionosis, and enteritis.

TREATMENT

The preferred method of treatment is multiple administration of enemas along with intravenous fluids and nasogastric administration of laxatives for more persistent obstructions. Commercially available phosphate enemas (Fleet enemas) are frequently used. A 4% acetylcysteine enema solution has been recommended for treatment of retained meconium.[244,245] The compound cleaves the disulfide bonds in the mucoprotein molecules and decreases the tenacity of the meconium. The 4% solution can be made by mixing 40 mL of a commercial 20% acetylcysteine solution with 160 mL of water. Because the activity of acetylcysteine increases with increasing pH (pH 7 to 9), 20 g (1½ level tablespoons) of $NaHCO_3$ powder (baking soda) is added to the 200 mL of diluted solution. Alternatively, 8 g (1 packed tablespoon) of a powdered form (*N*-acetylcysteine) can be mixed with 200 mL of water to which 20 g of $NaHCO_3$ is added. Commercial kits are also available (E-Z Pass Foal Enema Kit). The foal is restrained, sedated with diazepam (0.2 to 0.4 mg/kg IV), and placed in lateral recumbency. A 30-French Foley catheter with a 30 mL bulb is inserted about 2.5 to 5 cm into the rectum. The balloon is slowly inflated, using care to not damage the rectum. Between 100 and 200 mL of the 4% solution is administered slowly by gravity flow and retained for 30 to 45 minutes.[239] This can be repeated up to three times over a 24-hour period. The prognosis after acetylcysteine enemas has been reported to be excellent.[239] High meconium retention can be treated with intravenous fluids and mineral oil administered by nasogastric tube.

Surgery should be performed only on foals that have failed to respond to aggressive medical therapy. Postoperative complications involving adhesion formation are common after gastrointestinal surgery of neonates.[78,246] In one study, 8 of 28 foals with meconium impactions failed to respond to medical therapy and required surgery.[247] Two of seven surgical cases available for follow-up were euthanized as a result of serosal adhesions.[247] These results suggest that if surgical treatment is required, techniques to reduce adhesion formation, such as minimal handling of the bowel, and topical 1% sodium carboxymethylcellulose during bowel manipulation, should be practiced. In one study, administration of penicillin, gentamicin, and flunixin meglumine, or DMSO (20 mg/kg diluted twice daily) were the most

effective treatments to prevent adhesions induced by ischemia and reperfusion in foals; the efficacy of sodium carboxymethylcellulose (a 3% solution was used in that study) was less effective than these treatments.[248]

Vascular Lesions

Intramural Hematoma

The incidence of small colon intramural or submucosal hematoma is very low, less than 1% of surgical colics.[47,249,250] Most often the cause of the lesion is unknown, although focal ulceration with infection and iatrogenic trauma have each been described in one horse.[249,250] Although it has been stated that older horses are at an increased risk, the low number of reported cases makes this difficult to ascertain. Four of seven cases were 10 years old or younger, and one of seven was older than 20 years in two case series.[249,250] In another report of six horses, the age range was between 9 and 18 years.[218]

The intramural hematoma causes obstruction of the small colon lumen initially with subsequent ischemic necrosis of the bowel wall leading to septic peritonitis, endotoxemia, circulatory shock, and death if not treated.[218] Horses are often presented with varying degrees of abdominal pain that may become acutely severe. Heart rate is mildly to markedly elevated. Rectal palpation findings may reveal gas distention of the large intestine, decreased fecal content, and occasionally dark or clotted blood.[218,249,250] In some cases, a solid mass is palpable *per rectum*.[249,250] During surgical exploration, the lesion is readily identified as a soft, dense, circumscribed mass occupying the bowel lumen and attached to the bowel wall. The lesion has been described as involving between 20 and 55 cm (8 and 22 inches) in a series of six cases.[218] Treatment is surgical resection and anastomosis. The prognosis can be good if the lesion is located in a region that allows its complete resection and if surgical intervention occurs before transmural necrosis or bowel rupture leads to bacterial peritonitis.[220,249] Colostomy should be considered if the lesion is too far caudal to allow exteriorization.[220]

Mesocolic Rupture

The incidence of rupture of the mesocolon has been reported to be between 0.4% and 2.5% in horses undergoing exploratory laparotomy for colic.[24,47,251] Rupture of the mesocolon leading to segmental ischemic necrosis of the small colon has been described as a complication of parturition in the mare.[47,252,253] The vigorous reflex kicking and head movement of the foal that allows it to rotate into a dorsal position for delivery is thought to mechanically tear the mesentery that is caught between the uterus and dorsal body wall.[220,251,252] Mesocolon rupture may also occur as a consequence of type III or IV rectal prolapse, often associated with tenesmus after parturition.[32,47,252,253] If more than 30 cm (12 inches) of the rectum prolapses, ischemic necrosis of the rectum and small colon should be expected from disruption of the mesocolon and vascular arcade.[32,47,220,252,253] Mares are typically middle aged and pluriparous. Foaling may have been normal or required assistance.[43,252,253]

Time from foaling to the onset of colic is variable, ranging from 0 to 24 hours, with referral 12 to 48 hours later. The severity of abdominal pain varies greatly from no demonstrable pain to severe pain. The initial mild abdominal discomfort may be incorrectly attributed to uterine involution, delaying the recognition of a surgical problem. There is a consistent lack of feces. Rectal examination findings vary from normal to feed-impacted small colon with gas-distended large intestine. Peritoneal fluid has an elevated protein and nucleated cell concentration, with intra-abdominal hemorrhage seen in some cases.[253] The decision for surgery is often based on duration of pain and peritoneal fluid changes.[252] Laparoscopy can be useful in making the diagnosis and determining the extent of damage.

Treatment consists of surgical resection and anastomosis if viable proximal and distal small colon is accessible for the anastomosis. A temporary colostomy to divert feces away from the compromised bowel has been suggested to facilitate healing if an anastomosis can be performed.[253] In most cases, tears resulting from rectal prolapse do not have sufficient viable distal small colon to allow an anastomosis. A permanent colostomy is required in these cases.[254] Often the prognosis is poor as a result of the extent of the tear or the delay in surgical intervention.[220,252,253]

Nonstrangulating Infarction

Nonstrangulating infarction of the small colon is uncommon, perhaps because the majority of the blood supply to the small colon is through the caudal mesenteric artery, which is not a common site of occlusive verminous arteritis.[214,218] In a review of 38 cases of small colon obstruction, the 3 cases with nonstrangulating infarction of the small colon showed no evidence of verminous arteritis.[218] Clinical signs are similar to those described previously for mesocolic rupture, with mild intermittent abdominal pain of 10 to 72 hours' duration. Peritoneal fluid most likely has an elevated protein concentration and nucleated cell counts. Treatment consists of surgical resection and end-to-end anastomosis. Since, in most cases, viable proximal and distal small colon is accessible for the anastomosis, the prognosis should be good as long as surgical intervention occurs before ischemic necrosis of the infarcted segment causes septic peritonitis. When the infarcted segment cannot be exteriorized through a laparotomy, a colostomy or transrectal exteriorization followed by rectocolostomy should be considered.[220]

Strangulating Lesions

Strangulating Lipomas

Strangulating lipomas of the small colon account for approximately 11% of small colon disease.[155] They are most commonly seen in horses older than 15 years and are not reported in horses younger than 8 years.[155,218,255] Geldings may be at an increased risk compared with mares and stallions.[218,255] Ponies have been reported to be at increased risk.[218] It is common for lipomas to form in the mesentery of the small colon because of the amount of fat it contains. However, unlike in the small intestine, many do not cause clinical problems, as the thickness and diameter of the small colon wall and the fecal balls within its lumen prevent the pedunculated lipoma from encircling and s trangulating the bowel. In horses with strangulating pedunculated lipomas, approximately 7% are located in the small colon and 93% are located in the small intestine.[218,255] In some cases, the pedunculated lipoma encircles the small colon, causing obstruction without significant vascular impairment.[218]

Although some horses with small colon strangulating lipomas demonstrate signs of severe colic, others do not show the amount of abdominal pain commonly seen with strangulating lesions involving other areas of the gastrointestinal tract.[221] Peritoneal fluid changes include increased nucleated cell count and protein concentration. The predominant abnormal rectal findings are gas distention of the large colon and cecum with absence of fecal balls in the small colon. In certain cases, the clinician's hand can be advanced to the area of the constriction.[221] A fibrous band encircling the colon may be palpable. Transrectal ultrasonography may indicate increased thickness of the small colon wall with intestinal distention.[222] Treatment consists of surgical removal of the lipoma with resection and anastomosis of any compromised small colon.[155,214,218,220,221]

Volvulus, Herniation, Intussusception

Volvulus of the small colon is a very rare cause of colic, most likely as a result of its fixed extremities and its relatively short length.[256] The firm fecal balls within the lumen and its fatty mesentery may also prevent the small colon from twisting around its mesentery. Adhesions and abscesses have been associated with small colon volvulus.[257] Herniation of the small colon, which may result in a strangulating obstruction, has been reported to occur with umbilical, inguinal, and body wall hernias, as well as through omental, mesenteric, uterine, vaginal, broad ligament, and gastrosplenic ligament tears.[214,257,258] Intussusception not associated with rectal prolapse has been reported in foals and one broodmare.[70,259,260] If the point of invagination is at the distal end of the small colon, the intussusceptum may protrude from the anus. In this case, a finger can be inserted between the protruding bowel and the anal sphincter and advanced cranially until reaching a blind end.[70]

Clinical signs are similar to those seen with strangulating lipomas of the small colon. There is usually a sudden onset of colic, although the clinical course may be more prolonged than is seen with strangulating lesions of the more proximal gastrointestinal tract.[220] Rectal examination reveals lack of feces and gas distention of the large colon. In some cases, the small colon is under considerable tension, limiting the rectal examination.[256,258] Blood staining of feces may be present with an intussusception.[259] Peritoneal fluid is often serosanguineous in color, with increased nucleated cell count and protein concentration.

Treatment involves surgical resection and anastomosis through a ventral midline laparotomy. Rectocolostomy has been described as a treatment for distal intussuscepted small colon protruding through the anus in an 8-week-old foal.[218] In this transrectal procedure, an encircling incision was made in the outer layer of the intussusception 4 cm caudal to the anal sphincter. Ligatures were placed around the mesenteric vessels. Continuity was restored by a colorectostomy using simple-interrupted sutures of 2-0 polyglactin.

Atresia Coli

Atresia coli is a very rare condition in the foal, involving the large, transverse, or small colon. It should not be confused with ileocolonic aganglionosis, seen in the recessive lethal white foal syndrome of the overo foal. In the lethal white syndrome, which is always fatal, foals are born with no pigment (except for the eyes) and have stenotic colons that have thin muscular walls and few myenteric plexuses, leading to megacolon and death.[226,261] Although the cause of atresia coli remains unknown, it has been proposed that a vascular injury during fetal development leads to focal ischemic necrosis of the intestinal segment.[261-263] Atresia coli does not appear to have a breed or sex predisposition.[264]

Three basic types of intestinal atresia have been recognized. Type 1, membrane atresia, is caused by a membranous diaphragm occluding the lumen of the intestine. Type 2, cord atresia, is characterized by a fibrous band or muscular cordlike remnant of gut connecting the blind ends. Type 3, blind end atresia, is caused by an absence of a segment of intestine, with disconnected ends and a gap in the mesentery. Atresia coli in foals is typically described as type 3.[263-266] Foals with atresia coli develop acute signs of colic within the first 24 hours after birth (mean, 8.2 hours; range, 2 to 26 hours).[264] The most consistent finding on physical examination is absence of meconium staining after repeated enemas. Radiographic examination may indicate gas distention of the gastrointestinal tract proximal to the atretic segment. Radiographic evaluation after a barium enema may indicate a blind end.[264,267] Surgical repair is often unsuccessful, since the remaining intestine lacks sufficient length or diameter to perform an anastomosis.[264,265,268] If surgical repair is attempted, it is recommended that the severely dilated proximal portion be resected to obtain a normal vascularized segment.[268] Also recommended is an end-to-end or end-to-oblique-end anastomosis to avoid a "blind loop" syndrome. Suggested alternative surgical approaches include colostomy and a pull-through procedure suturing the small colon to the anus.[214] Other congenital abnormalities have been found in some of these foals, such as ventricular septal defect, common truncus arteriosus, hydrocephalus, cerebellar dysplasia, dermal hemangioma, and renal agenesis.[264,268]

Neoplasia

Small colon neoplasia is very rare. Lipomas were discussed previously. A leiomyoma of the small colon found during exploratory laparotomy for a large colon displacement was successfully treated with resection and anastomosis in a 4-year-old Thoroughbred.[269] Leiomyoma has been reported to cause an intussusception in the small colon, and a lymphoma causing perforation of the small colon has been reported.[270,271]

Miscellaneous Conditions

Other conditions of the small colon that have been reported are partial obstruction by an abdominal testicular teratoma, obstruction from an ovarian pedicle in a foal, obstruction from a granulosa cell tumor in three mares, obstruction by colonic lipomatosis, and secondary chronic idiopathic intestinal pseudo-obstruction (myenteric ganglionitis) in a 4-year-old Standardbred causing recurrent impaction.[218,272-278] Colonic ganglioneuromatosis involving the small colon has been reported in a horse as an incidental finding on necropsy examination.[279]

Surgical Procedures

Small Colon Enterotomy

Small colon enterotomy is indicated to remove various types of intraluminal obstruction of the small colon, such as

fecal impactions, enteroliths, fecaliths, and foreign bodies.[155,215,216,229,280] In most cases, a ventral midline celiotomy is performed to access the small colon. However, in select cases, such as a fecalith or foreign body, if there are financial constraints, a flank incision may provide adequate access to the lesion. The preferred location for the enterotomy is a longitudinal incision on the antimesenteric teniae. Enterotomies in this location have been found to be stronger, to allow easier apposition, to develop less intraoperative hemorrhage, and to maintain an increased postoperative lumen size compared with enterotomies that do not involve the teniae.[280,281]

The segment of small colon where the enterotomy is to be performed is exteriorized and draped separately from the abdomen and the rest of the gastrointestinal tract. Intestinal clamps or Penrose drains are placed proximal and distal to the enterotomy site to prevent spillage of fecal material during the enterotomy. Stay sutures are placed approximately 1 cm from the ends of the enterotomy incision. The incision is made sharply with a No. 10 scalpel blade. A two-layer closure is performed using a full-thickness simple-continuous pattern followed by a seromuscular inverting pattern with 2-0 polyglactin 910, taking care to invert a minimal amount of tissue.

In some horses, the intraluminal obstruction, typically an enterolith, is lodged in the proximal small colon where the short mesocolon prevents adequate exteriorization, increasing the risk of peritoneal contamination during the enterotomy. If retropulsion into the right dorsal colon is not feasible, a modified teniotomy can be performed and may facilitate normograde movement of the obstruction to a more accessible location.[145] In this technique, the large colon is first evacuated to decrease fecal material proximal to the obstruction. The site of obstruction is isolated from the rest of the abdominal cavity with sterile moistened drapes. Stay sutures are placed within the antimesenteric tenia proximal to the site of obstruction and 10 to 15 cm (4 to 6 inches) distal to the site. Fluid and ingesta are stripped away from the area. Topical 2% lidocaine can first be applied to reduce muscular spasm. A 1-cm longitudinal seromuscular incision is made with a scalpel blade in the antimesenteric tenia 10 to 15 cm aboral to the obstruction. The seromuscular incision is continued orally to the widest part of the obstruction using Metzenbaum scissors. Gentle pressure applied through the wall of the small colon is used to advance the obstruction to a more accessible location, where the mucosa is incised and the obstruction is removed. The enterotomy incision is closed as described earlier. This technique is most suitable for obstructions with a smooth surface, such as enteroliths, making them more likely to move distally. In one report, enteroliths were moved distances of 4 to 15 cm (1½ to 6 inches).[145]

Small Colon Resection and Anastomosis

Several techniques have been described for small colon anastomosis. These include hand-sutured single-layer end-to-end anastomosis, hand-sutured double-layer end-to-end anastomosis, and triangulated end-to-end everting stapled anastomosis.[221,282-284] A two-layer technique consisting of a full-thickness appositional pattern oversewn with a partial-thickness continuous inverting layer has less risk for dehiscence, peritonitis, and adhesion formation than the hand-sutured single-layer and stapled closures. Also, it creates less tissue inversion than a double inverting pattern, decreasing the chances of postoperative impaction at the anastomosis site.[282-284] The vascular arcade supplying the compromised small colon is traced back to normal bowel. The marginal artery at the oral and anal side of the lesion is identified and isolated within the mesenteric fat and is triple-ligated. To prevent inadvertent puncture of a vessel to be ligated with the pointed taper needle, especially if the vessel is partly covered by fat, the needle can be advanced around the vessel with its back first, since this blunt end avoids vessels (Figure 37-27). Once the suture is placed around the vessel, tightening the loop and the knot results in secure ligation of the vessel, because firm tightening cuts through the fat tissue surrounding the vessel. The vessels are transected, and the vessels remaining *in situ* are double-ligated. Vessels supplying the intestine to be removed are triple-ligated and sectioned. The ingesta is milked out of the segment of small colon to be resected and Penrose drains or intestinal clamps are positioned approximately 10 to 20 cm (4 to 8 inches) proximal and distal to the resection area to occlude the bowel lumen and decrease contamination from luminal contents during the resection. Doyen intestinal clamps are placed across the descending colon at the oral and anal extents of the resection, angled at approximately 30 degrees from perpendicular so that the length of the small colon at the antimesenteric angle is shorter than the intestine at the mesenteric angle (Figure 37-28).[233] This should improve blood supply to the antimesenteric border and increase the luminal size of the anastomosis compared with perpendicular transection angles. Moistened sterile drapes are used to isolate this region from the abdomen and adjacent bowel. A scalpel is used to sharply transect the colon, cutting adjacent to the Doyen intestinal clamps and using them to guide a straight transection line.

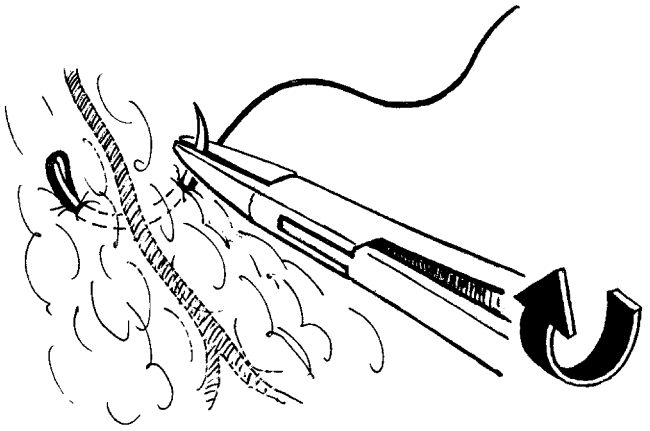

Figure 37-27. Using the blunt back end of a taper-point needle to place a ligature around a semihidden (by fat) mesenteric vessel allows safe placement of a ligature around vessels without inadvertent trauma to the vessel.

The proximal and distal segments are aligned with stay sutures placed at the mesenteric and antimesenteric borders. A slight rotation of the two segments of bowel offsets the mesocolon attachments, allowing a more secure closure at the mesenteric border.[214] The first layer placed is a full-thickness simple-continuous pattern tied at the mesenteric and antimesenteric angles, or a simple-interrupted pattern using No. 0 or 2-0 absorbable suture material. The second layer is a partial-thickness inverting layer using 2-0 absorbable suture interrupted at 180 degrees. The mesentery is closed with a simple-continuous pattern.[214,233,284]

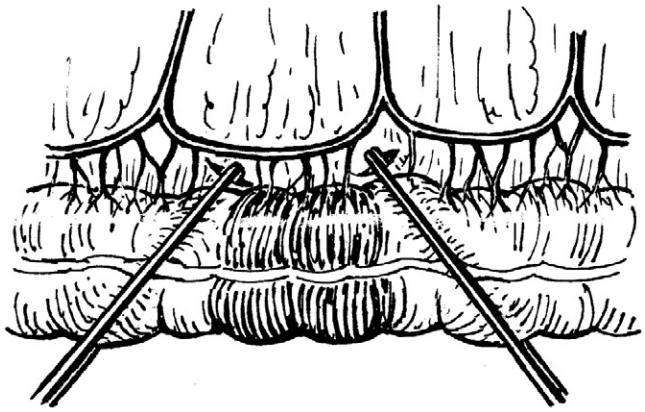

Figure 37-28. Penrose drains or intestinal clamps are positioned approximately 10 to 20 cm proximal and distal to the resection area to occlude the bowel lumen and decrease contamination from luminal contents during the resection (not shown). Doyen intestinal clamps are placed across the descending colon at the oral and anal extents of the resection, angled at approximately 30 degrees from perpendicular so that the length of the small colon at the antimesenteric angle is shorter than the intestine at the mesenteric angle. A scalpel is used to sharply transect the colon, cutting adjacent to the Doyen intestinal clamps and using them to guide a straight transection line.

Postoperative Care and Complications

Because of the high concentration of both aerobic and anaerobic bacteria, broad-spectrum antimicrobial coverage with potassium penicillin (22,000 IU/kg, every 6 hours IV), gentamicin sulfate (6.6 mg/kg, once a day IV), and metronidazole (15 mg/kg, twice a day IV) is recommended. If the bowel is healthy and the surgical procedure is performed with minimal and contained contamination, perioperative (for 24 hours) use of antibiotics can be appropriate. More extended postoperative administration (therapeutic course) of antibiotics (for 5 to 10 days) should be chosen if the horse has compromised bowel, has any signs of peritonitis, or has acquired abdominal contamination during the surgical procedure. Adjunctive antiendotoxin therapy, such as polymyxin B and hyperimmune serum, should be considered in horses with signs of endotoxemia or septicemia.

Postoperative complications with small colon surgery are dehiscence, stricture and adhesion formation, and impaction at the enterotomy or anastomosis.* Other complications, such as peritonitis, diarrhea, thrombophlebitis, and laminitis, are associated with endotoxemia and septicemia.[217,218,221] It has been proposed that several factors specifically affect small colon surgery adversely compared with surgery of other areas of the gastrointestinal tract. These are the high concentration of collagenase in the small colon, the high concentration of bacteria (including anaerobic bacteria), the mechanical stress placed on the surgical site by firm fecal balls passing over the incision, and the poor vascular supply.[226,285,286] Other authors have questioned some of these assumptions. One study has shown that the small colon vascular arcade system has adequate collateral blood supply.[215] If the lesion is in an area that allows adequate surgical access, the blood supply should not be a limiting factor.[215] The small colon is at risk for postoperative impactions because of the character of the feces and the size of the lumen. Impaction places further stress on an enterotomy or anastomosis site, increasing the risk of dehiscence. Although evacuation of the large colon decreases the risk of this occurring, postoperative feeding should also be carefully controlled to further decrease the risk of impaction at the enterotomy or anastomosis site. Reports suggest that with good surgical technique and attention to postoperative management (such as careful reintroduction to feed), horses have a good prognosis after small colon surgery, even after resection and anastomosis.[155,216,218,221,240]

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CHAPTER
38 Rectum and Anus

David E. Freeman

The rectum is approximately 30 cm (12 inches) long in an adult horse and extends from the pelvic inlet to the anus.[1] The distance from the anus to the peritoneal reflection is highly variable and is shorter in young horses and in horses with little body fat.[2,3] The peritoneal part of the rectum is attached dorsally by the mesorectum, which is a continuation of the mesocolon.[1] The retroperitoneal part of the rectum forms a dilation called the rectal ampulla, which has thick longitudinal muscle bundles.[1] The anal canal is approximately 5 cm (2 inches) long and is enclosed by the internal anal sphincter, which is a thickening of the circular smooth muscle, and the external anal sphincter, which is composed of striated muscle.[1] The levator ani muscle arises from the ischiatic spine and sacrotuberal ligament and ends under the external anal sphincter.[1] Its action overcomes the tendency of the anus to prolapse during defecation.

RECTAL TEARS

Rectal tears appear to be less common than previously observed, although numbers are not available to support this observation. The percentage of equine malpractice claims from rectal tears in the United States does appear to have declined.[4] One possible explanation for this apparent decline is the use of transrectal ultrasonography for assessment of the mare's reproductive tract and abdominal ultrasonography for examination of horses with colic, both of which provide accurate information rapidly and safely. Evidence that palpation *per rectum* for colic examination and reproductive purposes are the most common causes of rectal tears supports this hypothesis, although the role of ultrasound examination has not been examined.[4] Nonetheless, rectal tears are life-threatening injuries, and the risk of a malpractice claim against the veterinarian can be as much determined by the standard of care applied after the tear was inflicted as by the circumstances that led to its infliction.[5] Copious lubrication of the hand and forearm and adequate restraint of the horse, including sedation if necessary, should be regarded as the minimal measures required to prevent iatrogenic rectal tears. Once a rectal tear is suspected, the veterinarian must assess the tear for severity, inform the owner about the nature of the problem (without making statements that imply admission of guilt or responsibility for payment), and apply appropriate treatment, including referral. Failure to follow these guidelines will weaken a veterinarian's legal defense. The veterinarian should also contact his or her liability insurance company promptly.

Causes

Most rectal tears caused by palpation *per rectum* result from rupture of the rectal wall as it contracts around the examiner's hand or forearm and not from penetration with the finger tips. To avoid a tear, no attempt should be made to force against straining or a peristaltic wave, and intra-abdominal structures should be palpated only when the hand has been inserted beyond the contraction and then withdrawn to that level. Special precautions should be taken with Arabian horses, ponies, small breeds, horses that have had a previous rectal tear or injury, horses that are unaccustomed to palpation *per rectum*, fractious horses, and horses with colic.[2,4,6] Arabian horses, American Miniature Horses, mares, and horses older than 9 years are the breeds, gender and age groups most prone to rectal tears.[4] Most rectal tears are caused by examinations *per rectum* of the reproductive tract in mares and the gastrointestinal tract in horses with colic.[4] Older mares might be at greater risk because of the difficulty in reaching their more pendulous reproductive tracts, the need for frequent examinations of subfertile older mares, the overall greater need to perform rectal examinations on mares than male horses, and possibly age-related degenerative changes in their rectal walls.[4]

Less common causes of rectal tears are enemas, meconium extraction by forceps, sadism, dystocia, parturition without dystocia, animal bites, chronic impaction at a stricture, misdirection of a stallion's penis, spontaneous tears, rectal thrombosis, and sand impactions.[2,3,6-11] In one report of five horses with idiopathic tears, four presented with colic, and the tear developed in one horse during a lameness examination.[12] Idiopathic tears tend to be transversely oriented.[12]

Classification and Locations

Rectal tears are divided into four grades based on severity. In *grade I* tears, only the mucosa and submucosa are torn.[2] In *grade II* tears, only the muscular layer is disrupted, causing the mucosa and submucosa to prolapse through the muscle defect and create a site for fecal impaction.[2] These are rare (2 of 89 recorded in one series,[4] 3 of 85 tears in another[13]), but chronic impactions in the diverticulum may necessitate euthanasia.[13] *Grade III* tears involve all layers except the serosa (grade IIIa) or mesorectum and retroperitoneal tissues (grade IIIb).[5,14] It is not unusual for grade IIIb tears to pack with feces and produce a large plane of dissection cranially and dorsally, even approaching the left kidney. The feces packed in retroperitoneal spaces can be a source of bacterial contamination of the peritoneal cavity (Figure 38-1). A *grade IV* tear involves all layers and is the most serious, since it allows fecal contamination of the peritoneal cavity[2] and, to a lesser extent, evisceration of small intestine or small colon through the rectum and anus.[11] This grade was documented in all six postparturient mares with rectal tears in one series.[11] Most tears involve the dorsal aspect of the rectum regardless of cause, are located 4 to 60 cm from the anus, and are oriented parallel to the longitudinal axis.[2,4,13,14] Distance from the anus is not a reliable indicator of tear location relative to the retroperitoneal reflection.[1,3,5] The size of the tear can vary from 1 to 27 cm, and the only cause that appears to influence the size of the tear is dystocia, with a median size of 25 cm in one study.[4]

Clinical Signs and Diagnosis

Early diagnosis is essential for successful treatment and to avoid legal repercussions. When a tear occurs, the veterinarian may feel a sudden release of pressure or suddenly be able to directly palpate abdominal organs.[6] Alternatively, the examiner may not sense the torn rectum.[6] A rectal tear should be suspected if a large amount of blood is evident on the rectal sleeve on withdrawal of the hand or if the rectum suddenly relaxes while the horse is straining.[2] Within 2 hours after a rectal tear occurs, the horse usually shows signs of peritonitis and endotoxic shock. Signs of colic are also present initially, but these soon give way to depression.[6] Feces can be hemorrhagic initially and defecation can be accompanied by straining.[15] Idiopathic tears are usually presented as colic of variable duration, and because there is no reason to suspect a rectal tear, referral can be delayed.[12]

Before examination of the tear, straining and rectal contractions are stopped with epidural anesthesia or xylazine (0.1 to 0.2 mg/kg body weight IV), alone or in combination with butorphanol tartrate (0.1 mg/kg IV).[13-16] Butylscopolamine bromide (Buscopan), 0.3 mg/kg slowly IV, relaxes the rectum and small colon very well, allowing examination of rectal tears. A lidocaine enema can be given (12 mL of 2% lidocaine in 50 mL of tap water) or lidocaine jelly applied to the rectal mucosa before inspecting the tear digitally or through a tube speculum or endoscope. For digital palpation, the examiner can use the bare hand or wear a surgeon's glove and apply copious lubrication with a water-soluble gel. Because the abundant mucosal folds tend to obscure the rectal tear, more information can be gained from careful palpation than by visual inspection. Failure to accurately grade a tear can cause inappropriate treatment selection. A grade I tear can feel like a flap of mucosa, but a grade III tear can feel as if its edges are rigid and thick and separated, often by packed feces. Abdominocentesis should be performed to assess peritonitis.

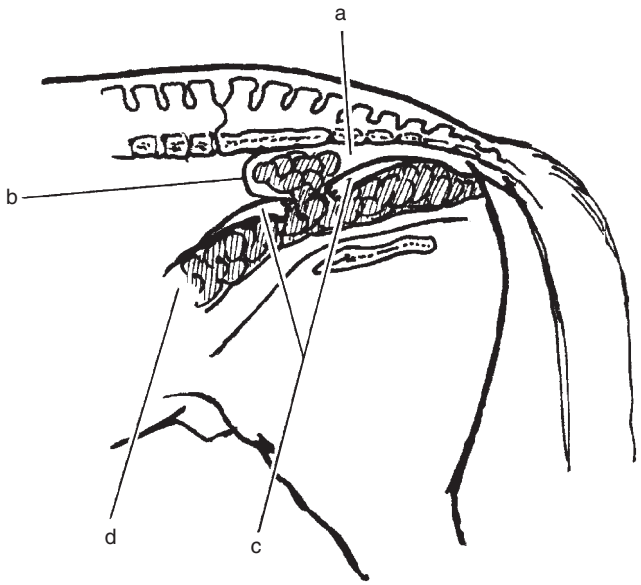

Figure 38-1. Impaction of feces into a grade IIIb rectal tear *(a)*, causing progressive enlargement of the perirectal space and bacterial leakage across the peritoneum *(b)*, swelling of the rectal wall *(c)*, and small colon impaction *(d)*. (From Freeman DE, Richardson DW, Tulleners EP, et al: Loop colostomy for management of rectal tears and small colon injuries in horses: 10 cases [1976-1989]. J Am Vet Med Assoc 200:1365, 1992, with permission.)

Initial Treatment

First aid is applied as soon as the diagnosis is made and can include (1) reduction of activity of the rectum, (2) gentle removal of feces from the tear and rectum, (3) treatment of septic shock and peritonitis, and (4) administration of epidural anesthesia and packing of the rectum.[16,17]

Flunixin meglumine (1.1 mg/kg every 24 hours IV) and antibiotics, such as sodium or potassium penicillin (22,000 IU/kg of body weight every 6 hours IV), gentamicin (6.6 mg/kg every 24 hours IV), and metronidazole (15 mg/kg every 6 hours PO) should be administered and continued as indicated. Intravenous fluids are required to treat shock. Antibiotics and laxatives may be sufficient treatment for grade I and II tears, with daily inspection and careful evacuation of the rectum as needed.[6]

Rectal packing can prevent conversion of a grade III to a grade IV tear and can protect the tear from fecal contamination in the interval before definitive treatment is applied.[5,17] The packing material of choice is a 7.5-cm (3-inch) stockinette filled with 0.25 kg of moistened rolled cotton, sprayed with povidone-iodine and lubricated with surgical gel.[17] The packing should fill the rectum without distention to a point 10 cm proximal to the tear, taking care not to pack the tear itself.[17] The anus is subsequently closed with towel clamps or a purse-string suture, and the epidural anesthetic is repeated as necessary to decrease straining.[6,17]

Bypass Procedures

Bypass procedures can be used to facilitate healing of a tear. A temporary indwelling rectal liner and colostomy divert feces

away from grade III and grade IV rectal tears and thereby prevent contamination and impaction of perirectal tissues, enlargement of the tear, and progression of peritonitis.[18-21] Each can be combined with direct suture repair and thereby used to protect the suture line during healing.

Temporary Indwelling Rectal Liner

A temporary indwelling rectal liner can be constructed by gluing a palpation sleeve with the hand portion removed, or the plastic cover for an arthroscope camera, to a 5-cm-diameter and 7.5-cm-long rectal prolapse ring with holes through which Dacron loops are laced.[6,18] With the horse anesthetized in dorsal recumbency, an unscrubbed assistant passes the well-lubricated ring and liner through the anus so the surgeon can guide it through a ventral midline celiotomy, cranial to the tear but far enough caudad that the sleeve does not retract into the rectum when the horse stands (Figure 38-2).[6,18] A circumferential suture of size 3 surgical gut is placed tightly around the small colon over the central groove of the prolapse ring, and four equidistant retention sutures (2-0 polyglycolic acid) are placed to incorporate the circumferential suture, all layers of the rectal wall, and the Dacron loops in the modified prolapse ring.[18] Then, the serosal surface is apposed over the circumferential suture with a Lembert pattern.[18] The large colon is emptied through a pelvic flexure enterotomy, and the small colon is emptied by flushing with a hose directed through the ring and liner from the anus.[6]

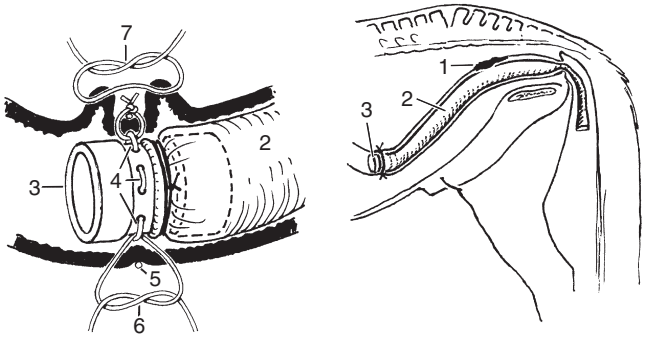

Figure 38-2. Placement of a temporary indwelling rectal liner to divert feces from a rectal tear. The *expanded view* demonstrates the construction of the liner and the method of securing it in place. *1,* Tear; *2,* rectal liner; *3,* rectal prolapse ring; *4,* Dacron anchor suture; *5,* circumferential suture; *6,* retention suture; *7,* interrupted Lembert suture.

The circumferential suture will cut through the rectal wall and allow passage of the ring and liner in feces within approximately 9 to 12 days, by which time the apposed colon walls will have healed.[19] The four retention sutures help maintain the ring in a coaxial relationship with the small colon so that it does not twist and obstruct the lumen.[6,18] Horses are usually cross-tied to prevent them from lying down, which would allow the sleeve to retract forward. Failures have been caused by tearing of the sleeve, retraction of the sleeve into the rectum and uncovering the tear, and formation of a rectoperitoneal fistula.[6,18]

Colostomy

LOOP COLOSTOMY

The loop-colostomy technique (Figure 38-3) is preferred over the end-colostomy technique because it is easier and quicker to establish and to reverse later.[19,20] Concerns about incomplete diversion of feces do not apply to a loop colostomy in horses because gravity, combined with correct construction of the stoma, prevents passage of feces into the distal small colon and rectum.[19-21]

A loop colostomy can be created in a left high flank, a left low flank, or a ventral midline incision, and all have advantages and disadvantages (Table 38-1). A single-incision colostomy

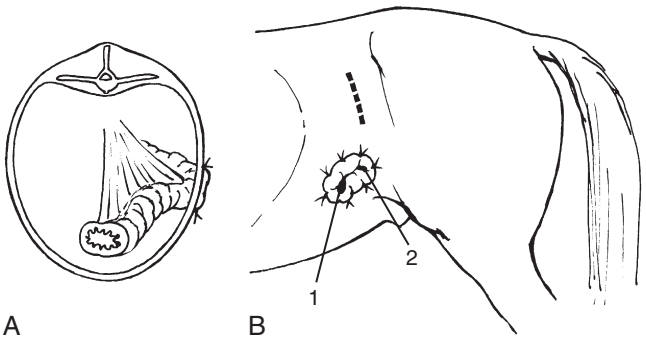

Figure 38-3. A, View through the transverse section of the abdomen shows the placement of the loop in the body wall. **B,** Loop colostomy created with a double-incision technique with the high flank approach *(broken lines)* and placement of the stoma in a small low flank incision. The left opening *(1)* is the proximal opening, and the right opening *(2)* is the distal opening through which the distal part of the small colon is flushed to prevent atrophy. (Redrawn from Freeman DE, Richardson DW, Tulleners EP, et al: Loop colostomy for management of rectal tears and small colon injuries in horses: 10 cases [1976-1989]. J Am Vet Med Assoc 200:1365, 1992.)

TABLE 38-1. Different Types of Colostomies in Horses

Type of Colostomy*	Abdominal Approach	Stoma Placement	Suitable as Standing Procedure	Suitable for Abdominal Exploration	Suitable for Emptying the Colon	Disadvantages
Single-incision	High flank	High flank	Yes	No	No	Fecal soilage of incision and skin
	Low flank	Low flank	No	No	No	Risk of prolapse and herniation
	Ventral midline	Ventral midline	No	Yes	Yes	Risk of prolapse and herniation
Double-incision	High flank	Low flank	Yes	No	No	Difficult construction
	Ventral midline	Low flank	No	Yes	Yes	Difficult construction

*Information applies to both loop and end colostomies.

involves placing the stoma in the same incision used to explore the abdomen and prepare the colon loop, whereas a double-incision colostomy involves a separate flank incision for the stoma.[19] Exploration of the abdomen through the ventral midline is necessary if the horse has a surgical colic that preceded the tear, if intra-abdominal repair of the tear is considered feasible, or if the surgeon elects to empty the large and small colons to reduce stress on the colostomy.[16,20,22] Colostomy placement in the anesthetized horse has the following disadvantages: (1) accurate placement of the stoma is difficult because muscle layers shift and landmarks become distorted in the recumbent horse compared with the standing horse, (2) it is expensive, and (3) dehiscence of the stoma is a risk of rough anesthetic recovery.[19,20] Marking the selected site on the skin with a pen or suture preoperatively while the horse is standing does not obviate the first problem, because the muscle layers disrupt the attachments when the horse stands, even when the skin incision is accurately located. With single-incision colostomies, incorporation of the colostomy in the abdominal closure weakens the body wall repair and makes the ventrally placed stomas, such as the low flank and ventral midline placements, prone to prolapse and herniation.[19] With the double-incision technique, the stoma is placed in a snug low flank incision so that it is surrounded by intact body wall and the risks of prolapse and herniation are reduced.[19]

With the double-incision technique, a high flank incision is used to prepare the colon loop and guide it into a separate low flank incision, midway between the flank fold (just in front of the stifle) and the costal arch, and at the same level as the fold (Figures 38-4 and 38-5).[19] The distal incision is approximately 8 cm long and extends through all layers, and deep dissection is guided by a hand through the high flank incision. It is angled dorsad at its caudal end by 20 to 30 degrees. Small transverse incisions are made in muscles and fascia to eliminate constricting bands that could restrict fecal passage. The stoma should not be large and loose fitting, although this would reduce the risk of stomal obstruction but predispose to the more serious complications of prolapse and herniation.[19,20]

If general anesthesia and ventral midline celiotomy are required, this stage of the surgery should be completed as needed to correct the problem. The horse is subsequently placed in lateral recumbency to complete the colostomy in a separate flank incision.[20] This somewhat reduces problems from shifting muscle layers when the horse stands. A heavy retrieval suture (size 2 nylon) is placed on the antimesenteric band of the prepared loop of small colon before closing the midline incision, so that only a small flank incision is required to form the colostomy.[6,20] The ends of the retrieval suture are placed on a long needle, and that is directed from within the abdomen through the body wall at the site marked for the colostomy.[6,20] The midline incision is closed, the horse is placed in lateral recumbency, and the low flank incision for the stoma is made as described earlier for the double-incision technique.[6,20] Unlike with the double-incision technique, the surgeon must bring the prepared loop of small colon through the small incision at the site of the stoma, without being able to direct the loop though a separate larger incision.

Regardless of the approach, the stoma is made in a segment of small colon at least 1 m from the rectum so that the small colon can be easily exteriorized for colostomy reversal. This segment of small colon is folded to form a loop, and the two arms of the loop are sutured together with an absorbable material in a continuous Lembert pattern for 8 to 10 cm, midway between the mesenteric and antimesenteric teniae.[20] This suture line is brought closer to the mesentery at the folded end of colon to turn the antimesenteric taenia along the outer edge of the fold.[19] The adhesion along this suture line creates a more complete mucosal separation between the proximal and distal segments of small colon and thereby enhances complete fecal diversion. The adhesion can also stabilize the loop within the body wall, reducing the risk of prolapse.[19] The prepared loop of small colon is subsequently inserted into the flank incision so that the proximal loop is slightly ventral to the distal loop and the antimesenteric taenia projects beyond the skin by at least 3 cm.[19]

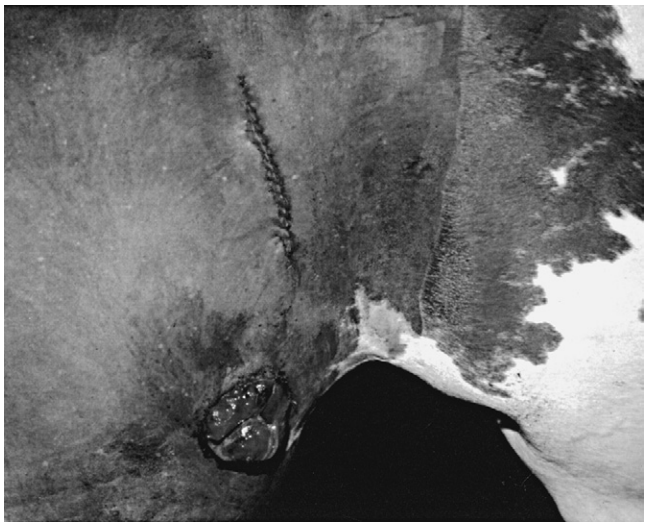

Figure 38-4. Loop colostomy placed in the low flank by a double-incision technique as a standing procedure 2 days earlier. Congestion in the mucosal shelf separating the proximal and distal parts of the small colon is typical for this stage.

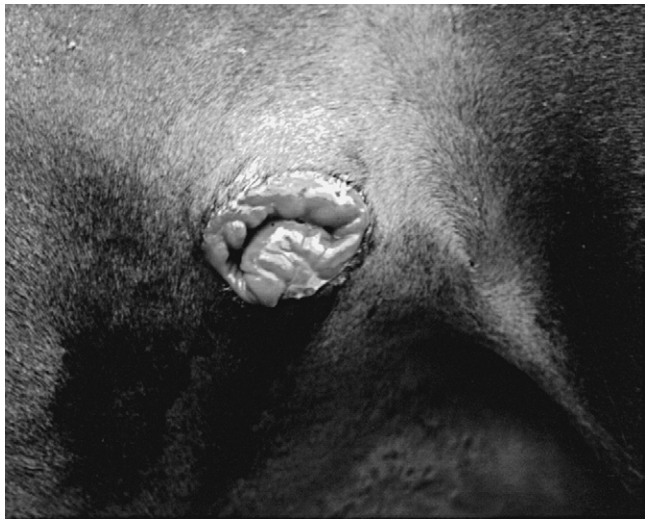

Figure 38-5. Loop colostomy placed in the low flank by a double-incision technique as a standing procedure, 60 days later (horse is not that shown in Figure 38-4). The stoma is mature and healed to the surrounding skin; the mucosal shelf is evident as a small bulge in its midpoint.

The seromuscular layer of the colon is sutured to the abdominal muscles and fascia using several interrupted sutures of 0 or 2-0 absorbable material, taking care not to puncture or occlude mesenteric vessels.[19] To form the stoma, an 8-cm incision is made along the exposed antimesenteric taenia of the colon, and the cut edges are folded back and sutured to the skin with simple-interrupted sutures of 2-0 nylon or polypropylene (see Figure 38-3).[19] The opening thus formed is approximately the same size as the small colon lumen[19] (see Figure 38-5). Because fecal balls are eliminated individually as they pass through the small colon, without being stored to form piles, as in the rectum, the risk of obstruction is low.

An alternative method for loop colostomy is to suture the colon loop to skin only and to detach and oversew the stoma at 14 days.[23] The abdominal muscle layers and skin are then closed.[23] Disadvantages of this method are that such an attachment may allow peristomal herniation and that the rectal tear is unlikely to heal in 14 days.[19-21]

END COLOSTOMY

Some surgeons prefer an end colostomy because of the concern about incomplete fecal diversion with the loop colostomy method, a concern most likely carried over from experiences with the supine human patient.[24] However, the loop colostomy method does allow complete fecal diversion, because gravity causes fecal balls to drop away from the stoma rather than turn into the distal segment. For the end colostomy, the colon is transected, the distal segment is closed by the Parker-Kerr method (see Figure 16-8, *D*), and the proximal end is incorporated into the body wall as for the loop colostomy.[24] The distal segment tends to atrophy, so its diameter and length may become reduced by 50%.[24] This makes colostomy reversal by future anastomosis difficult and may cause anastomotic impactions and dehiscence.

AFTERCARE FOR COLOSTOMY

Antibiotics and laxatives (mineral oil, 2 to 4 L/450 kg, and magnesium sulfate, 1 g/kg) are continued for 3 to 5 days. Horses are held off feed or are fed grass and alfalfa hay at half the usual amounts for the first 2 to 3 days after the colostomy is established, and petrolatum-based ointment is applied to the skin around the stoma to protect it from scalding. A cradle is applied because most horses have a tendency to mutilate the colostomy.

When the loop colostomy is created, the mucosal protrusion of the stoma becomes markedly congested over the first 5 to 7 days after surgery (see Figure 38-4) and slowly sloughs, to be replaced with healthy tissue (see Figure 38-5). Considerable ventral edema can develop, but it resolves with time.

Small colon atrophy distal to the stoma plays a large part in failure of colostomy reversal by reducing the distal lumen size to the point that fecal impaction is inevitable.[20,21] With the loop colostomy, it is possible to exercise the distal part of the small colon and prevent the atrophy that complicates anastomoses with the end colostomy.[19,20] When the tear has started to granulate, usually after 5 to 7 days, the distal loop of the small colon and the rectum are flushed daily in normograde fashion with approximately 20 L of warm water through a garden hose.[19] This is more likely to improve lumen diameter than luminal nutrients such as short-chain fatty acids,[21] which would be expected to have a positive trophic effect on the mucosa without the necessary stretching of the muscle layers achieved through lavage.

COLOSTOMY REVERSAL

For colostomy reversal, usually 6 weeks or more after the colostomy, the horse is anesthetized in right lateral recumbency, the stoma is resected *en bloc*, and a colonic anastomosis is performed through the resulting flank incision.[6,19] Even if the colon is not penetrated during dissection, incisional infection is a common complication of stomal resection.[19]

For anastomosis, the distal segment is transected along a more acute mesenteric angle than the proximal segment to correct for a slight reduction in distal diameter. A single layer of interrupted Lembert sutures with 3-0 polydioxanone (PDS) is used for anastomosis (Figure 38-6), but other methods also are suitable.[6] The stapled side-to-side technique is likely to become impacted despite the large stoma.[21] The postoperative feeding, antibiotic, and laxative regimens are similar to those used after the colostomy procedure.

COMPLICATIONS OF COLOSTOMY

Complications of colostomy are dehiscence, abscessation, peristomal herniation, prolapse, prolapse with rupture of mesenteric vessels, infarction, rupture of the colostomy, spontaneous closure, and stomal obstruction, as well as anastomotic impaction and dehiscence after reversal.[6,7,19-21] Herniation and prolapse have been attributed to ventral placement of the stoma and to a stoma that is too large.[20,21] There is a tendency to make the stoma large to prevent stomal obstruction, although stomal obstruction is rare, probably because fecal balls are voided individually.[19]

CHOICE OF DIVERTING PROCEDURE

The choice between colostomy and temporary indwelling rectal liner for definitive treatment of a rectal tear is influenced largely by cost and by the surgeon's preference, because the two methods have comparable success rates.[14,18-21] The outcome is most likely to be determined by complications of the tear that developed before application of the diverting procedure.[19] Colostomy does have inherent, life-threatening risks and is more expensive because it is a two-stage procedure. A potential advantage of colostomy is that it allows the surgeon more complete control over the duration of fecal diversion, which could be important if healing of the tear is delayed.[6,19,20] Colostomy is preferred for large tears, small horses, and a tear that is too far proximal to accommodate a rectal liner.[18]

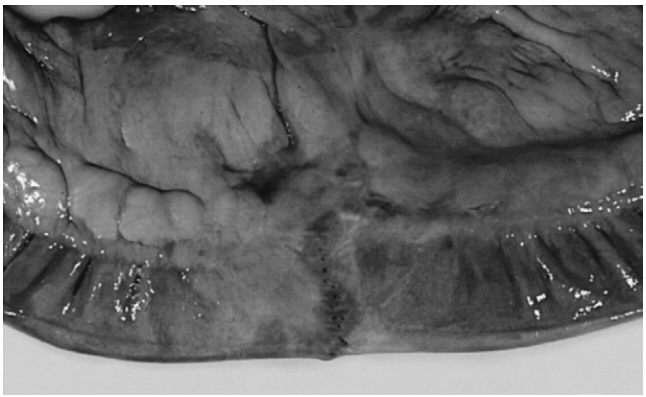

Figure 38-6. End-to-end anastomosis of the small colon with an interrupted Lembert pattern, using a 2-0 silk suture. The colostomy was reversed 21 days earlier, but the horse was euthanized because of laminitis.

The most difficult decision is whether a fecal diversion procedure is required at all, because some horses with grade III rectal tears can make a full recovery without surgery (see later).[3] Although few criteria can be used to guide treatment selection, the decision to use diverting procedures must be made quickly to derive full benefit, preferably within 12 hours after the tear occurred.[19,20] Because the cost of diverting procedures can be considerable, many owners opt for less-expensive procedures, such as direct suturing or medical treatment combined with repeated manual rectal evacuation.[25-27]

Suture Repair

Suture repair can promote healing and prevent progression of a grade III tear to a grade IV tear.[6,14,18] It should be combined with a diverting procedure if there is any concern about the integrity of the repair, but this is not always necessary and can be precluded for economic reasons. The surgery is easiest to accomplish on a thin horse with a tear that is fresh, clean, and close to the anus.[14,16] Long-handled instruments with pistol grips and a 60-cm-long expandable rectal speculum or "cage" have been developed to facilitate suture repair of rectal tears in horses.[28] The anal sphincter can be incised to improve access to a rectal tear, and this can be left open to ease defecation afterward.[29,30] A large wound of long duration can be partly closed or left open to allow drainage, and the defect can be packed with antiseptic-soaked gauze to prevent fecal impaction and dissection.[14,29]

A grade IV tear can be prolapsed through the anus to be closed with the TA-90 Premium stapling instrument, because the tear creates a pneumoperitoneum and equilibration of pressure across the rectal wall that allows it such mobility.[31] This procedure has been used successfully also for grade IV tears in mares in the immediate postpartum period, when the perirectal tissues are sufficiently relaxed to allow retraction of the rectal wall.[11] The staple line can be reinforced with a simple continuous suture pattern.[11] To improve access, four equidistant stay sutures can be used to retract the anal sphincter, which may then be transected at the dorsal commissure.[11] The latter can be sutured after surgery is completed.[11]

If the rectal tear is not accessible through the anus, it can be prolapsed through it from a caudal ventral midline laparotomy or repaired through an antimesenteric enterotomy from a ventral midline approach.[16,22] Suture repair under laparoscopic control appears to be a promising method for the future, but the instruments may be too short in some cases.[32]

A technique for nonvisual direct suturing has been reported to have favorable results and is a simple and inexpensive standing procedure.[17,25] If the tail is elevated and the anal sphincter is relaxed from the caudal epidural anesthesia, the rectum fills with air and the surgeon has more room in which to manipulate the needle.[25] This ballooning effect is poor in rectums with grade IV tears because of the equilibration between intra-abdominal and atmospheric pressure, and it diminishes in grade III tears if surgery is prolonged. Fecal material is digitally removed from the rectum and distal small colon, and the defect and lumen walls are cleaned carefully and thoroughly by wiping with moistened 4 × 4 inch gauze sponges. If the tear does not involve the full thickness of the wall, gentle gravity lavage can be used to clean the tear. In a grade IV tear, the severity of peritoneal contamination determines whether a repair should be attempted.

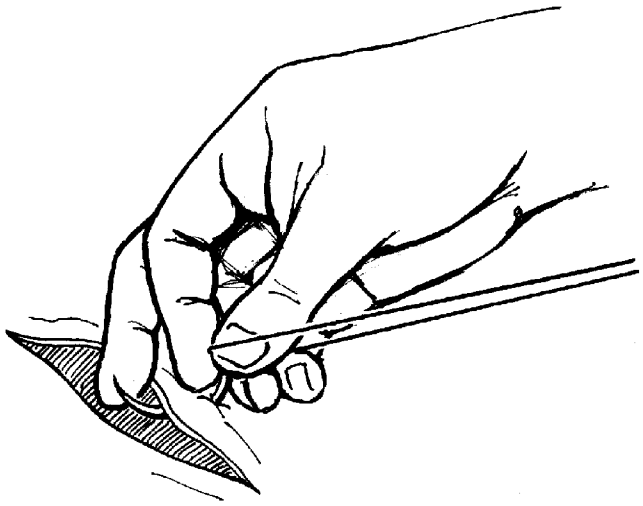

Figure 38-7. Diagram showing the first bite of the nonvisual direct suturing technique being placed in the center of the caudal edge of the tear. The needle, held with the thumb and first two fingers, is inserted approximately 1.5 cm from the edge of the wound and guided into the defect subserosally by the second or third finger. (Drawn from Eastman TG, Taylor TS, Hooper RN, Hague, BA: Treatment of grade 3 rectal tears in horses by direct suturing *per rectum*. Equine Vet Educ 12:63, 2000, with permission.)

The left hand is used for tears on the right side, and vice versa, and gloves are not worn.[25] The preferred suture is size 5 Dacron, 100 to 150 cm long, with a 6- to 8-cm half-circle cutting or trocar point needle placed in the middle of the suture.[25] A cruciate or simple-interrupted pattern is used. With both ends of the suture outside the rectum, the needle is manually advanced to the tear. The first bite is placed in the center of the caudal edge of the tear, holding the needle with the thumb and first two fingers. The needle is inserted approximately 1.5 cm from the edge of the wound and guided into the defect subserosally by the second or third finger (Figure 38-7). The needle is pulled through the tissue, again grasped with the thumb and first two fingers, and then placed in the center of the cranial edge. This bite begins subserosally within the defect and exits 1.5 cm from the edge. The third finger is used to guide the exit point and press the tissue onto the needle. The needle is subsequently brought out the rectum, leaving a single strand of suture in the tear with the other end extending 10 to 15 cm distal to the anal sphincter. The suture is then clamped with a hemostat between the needle and the point of exit from the tear and the needle remains threaded on the proximal half of the suture. An assistant holds the clamped suture to one side to close the defect into a transverse plane, and the needle is carried into the rectum. The suture is then passed through both cranial and caudal edges of the defect in one bite using digital manipulation as before, and finally the needle is brought out the rectum. The hemostat is released and the needle end of the suture is drawn through to form a cruciate suture. The knots are tied outside the rectum and pushed inside with one hand while tension is maintained on the suture with the other hand. Additional throws are placed to secure the knot.

Traction on the first suture should convert the tear to a transverse orientation, which facilitates placement of about two or three more sutures on each side of the first.[25] Care must be taken to prevent lumen reduction by the closed defect as its edges turn

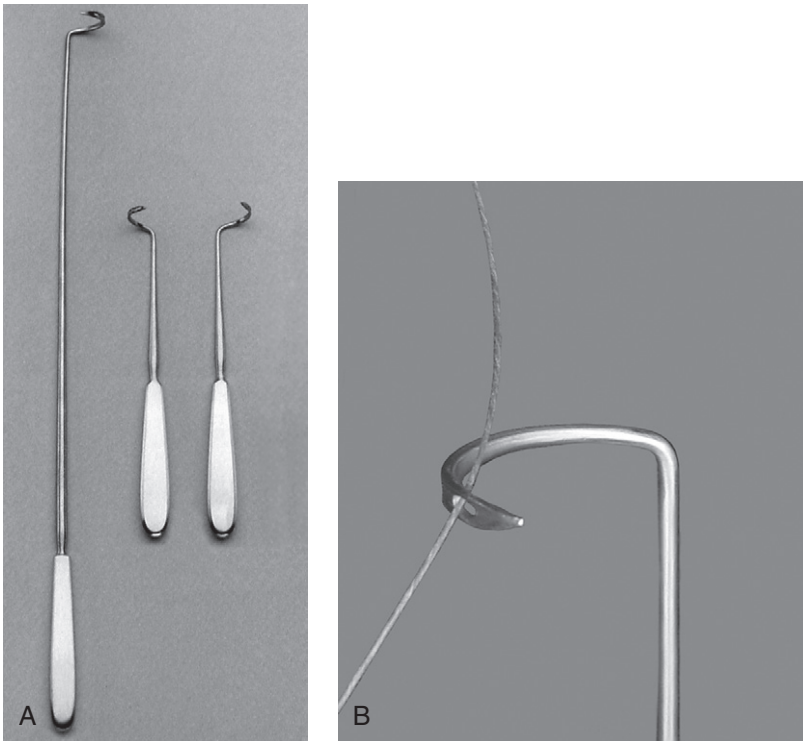

Figure 38-8. A, Deschamps needles, showing the right- and left-handed configurations with 20-cm needles *(right)* and the 45-cm needle that is suitable for rectal tears *(left)*. **B,** Close view of the end of a Deschamps needle. The threaded eye near the pointed tip allows easy retrieval of the suture without the need for complete penetration by the needle. Once the suture is grasped at the tip of the needle, the instrument is rotated backward out of the tissue and can be rethreaded for the next bite.

into the lumen. The suture ends are cut long to facilitate their removal, but if the feces cannot be kept soft, the ends should be cut short so that they do not get pulled out by fecal balls.[25] The sutures are checked at 24- to 48-hour intervals, and any suture that feels slack from loosening or reduction of edema is replaced. The sutures are removed in 12 to 14 days.[25]

Nonvisual direct suturing may not be easily mastered and can require extensive practice.[25] It is a useful technique for tears involving half or less of the rectal circumference and for tears that have very small perforations of the serosa, but it is recommended as an adjunct and not as a primary treatment for grade IV tears.[25]

The 45-cm Deschamps needle, which is made in both right- and left-handed configurations (Figure 38-8), is ideal for one-handed suturing in a deep recess (Figure 38-9) and has been used successfully for standing repair through the anus.[30] The needle tip should be sharp. Both chromic catgut and polyglycolic acid suture have been used. A right-handed surgeon guides the Deschamps needle rectally to the wound, with the left hand serving as protection and the right hand turning the handle (see Figure 38-9).[30] The caudal end of the perforation is grasped with the index finger and thumb of the left hand, and the needle is guided through the mucosa, muscularis, and serosa (if present), about 1 cm away from the wound edge.[30] The ventral wound edge is penetrated, the suture is removed from the eye of the needle, and a surgical knot placed outside the rectum is pushed in with the left hand. An assistant pulls the ends of the first knot through the anus to stabilize the repair and bring the wound edges together. Additional single sutures are placed from caudad to craniad, 1 cm apart, so that the wound closes to form a ridge (see Figure 38-9). If the suture bites exceed 1.5 cm from the wound's edge, the rectum becomes too narrow.[30]

The preferred suture for repair of rectal tears should be long, have low memory, resist stretching, and resist fecal digestion.[25]

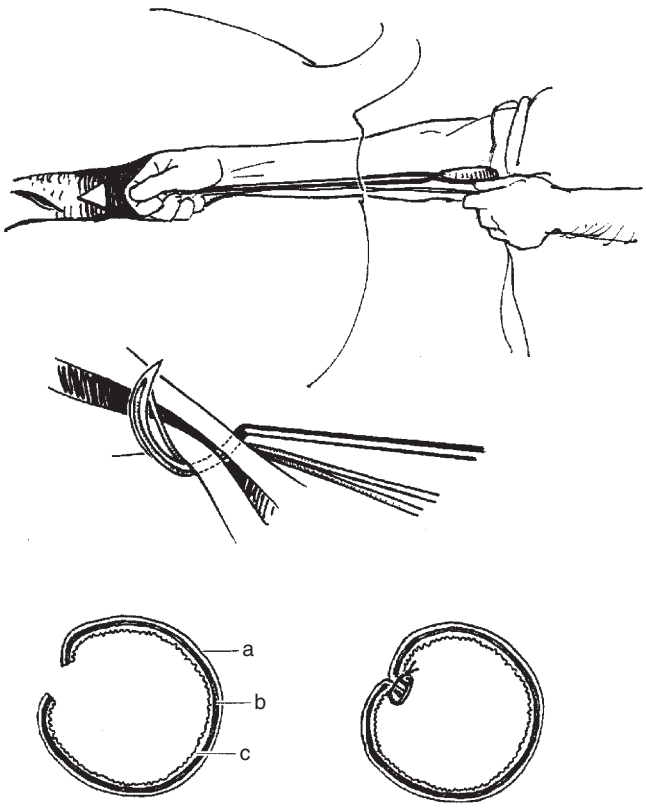

Figure 38-9. Method of inserting the Deschamps needle into the rectum and guiding it to the tear while protecting the tissues with the right hand. The close-up in the middle shows how the Deschamps needle grasps both sides of the tear and pulls the suture through it as well. The lower diagrams show the suture placement for a grade IV tear. *a,* Serosa; *b,* muscularis; *c,* mucosa.

Small suture tends to cut through the friable tissue edges, and catgut tends to stretch.[25] Continuous suture patterns are not recommended because they reduce the lumen diameter, which predisposes to impaction and dehiscence.[25] A complete pellet ration can be fed after a 2-day fasting period to produce a small fecal bulk, and mineral oil can be used to soften feces.[25] Mineral oil is not used if the repair of a grade IV tear is tenuous, because it could leak into the abdomen.[25]

Nonsurgical Treatment

Grade I and II tears rarely require surgical treatment, and grade I tears respond in most cases to antibiotics (e.g., trimethoprim-sulfadiazine, 20 mg/kg PO every 12 hours), and flunixin meglumine (1.1 mg/kg IV or PO, every 12 hours), mineral oil (4 L via nasogastric tube every 24 hours), and dietary changes such as bran mashes, moistened pellets, or grass to reduce fecal volume and soften the consistency. If all or a significant portion of the submucosa layer remains intact, grade I tears should heal with medical management without complications.

Although grade III tears usually require surgical treatment, medical management can be successful and is considerably less expensive than surgical methods.[15,26,27] In one report, six of eight horses with grade IIIb rectal tears were treated successfully with broad-spectrum antibiotics (penicillin, gentamicin, and metronidazole) and nonsteroidal anti-inflammatory agents, maintenance of soft feces with a diet of grass and bran mashes and daily administration of mineral oil by nasogastric tube, and daily manual removal of feces from the rectum after sedation and epidural anesthesia.[27] All horses developed septic peritonitis, and three of the six survivors developed a rectal diverticulum in the tear, without any apparent clinical effects.[27] The authors felt that manual evacuation of feces should be performed only if the tear became impacted, and the procedure should be conducted with extreme care.[27]

In another report on successful medical treatment of four horses with grade IIIb tears, the authors recommended frequent manual evacuation of feces on a daily basis.[26] Frequency of manual evacuation decreased from every 1 to 2 hours to every 6 to 8 hours by the 4th and 5th day, and the evacuations were discontinued between 9 and 21 days.[26] The underlying principle in this approach was to eliminate the storage function of the rectum and thereby eliminate impaction of feces into the tear.[26] Although this treatment is simpler to perform than bypass procedures, it is labor intensive and does require repeated epidurals through an epidural catheter.[26]

Full-thickness tears into the retroperitoneal space may be treated with manual evacuation of feces, antimicrobial therapy, fecal softener, and packing with gauze soaked in antiseptic solution until the defect fills with granulation tissue.[3,16] Perirectal abscesses that follow this treatment can be drained into the rectum (see later). Ventral tears in mares can be drained through the dorsal vaginal wall.[16] As with any method of treatment, peritonitis from the tear can be treated by peritoneal lavage, although this approach was not used in many cases reported in the literature.

Prognosis for Rectal Tears

Complications of grade III rectal tears are extensive and include cellulitis, abscess formation, severe toxemia, peritonitis, laminitis, and recurrent intestinal obstruction from adhesions.[14,16,18,21] The time required for healing of rectal tears varies from 2 to several weeks.[18-21] Most tears heal with little residual damage, but some can form a stricture, a diverticulum, or perirectal abscessation.[19,33] Such abscesses must be drained into the rectum by finger puncture and can delay colostomy reversal for up to 60 days.[19,20] Grade III and IV tears can heal to form a mucosal or submucosal hernia that can subsequently become impacted with feces, and a grade IV tear can form a rectoperitoneal fistula.[16,18]

In one survey of 85 horses, conservative management (medical treatment or no treatment) of 15 horses with grade I tears yielded a 93% survival rate.[13] Grade IIIa rectal tears had a 70% survival rate in this study (16 of 23 horses), and grade IIIb rectal tears had a 69% survival (9 of 13), compared with a 44% survival rate in a previous study.[13,14] Survival for grade IV tears was 2 of 31 horses.[13] In another study, all horses with grade I and II tears survived but only 38% of those with grade III tears and 2% with grade IV tears left the hospital.[4] A variety of different treatments were used, so it was difficult to draw conclusions about treatment effects on survival; however, all horses that had a colostomy (3) survived to hospital discharge.[4]

Nonvisual direct suturing of rectal tears in the horse has a higher success rate than that reported for other techniques.[25] In one study, 75% (15 of 20) of horses treated by this technique survived until discharge. Eighty-one percent of horses (13 of 16) with grade IIIa tears, and 50% of horses (2 of 4) with grade IIIb tears were discharged alive after suture repair.[25] The sutures did not hold in three horses, and the tears converted to grade IV at 1 to 7 days after surgery. One horse died of peritonitis from an unidentified grade IV tear orad to the sutured grade III tear.[25] Long-term follow-up was available for 10 of 15 survivors (66%), all of which survived at least 6 months. Horses discharged alive were hospitalized an average of 15 days (range, 9 to 24 days).

RECTAL PROLAPSE

Causes of rectal prolapse are straining from diarrhea, dystocia, intestinal parasitism, colic, proctitis, rectal tumor, and rectal foreign body.[14,33,34] In many cases, a cause cannot be identified. The condition is more common in females than in males and may affect any age group.[34]

Classification

In a *type I* rectal prolapse, only the rectal mucosa and submucosa project through the anus, sometimes more so on one side than on the other.[29,30] A *type II* lesion represents a complete prolapse of the full thickness of all or part of the rectal ampulla.[35] Type I and II prolapses are the most common.[34] In a *type III* prolapse, a variable amount of small colon intussuscepts into the rectum in addition to a type II prolapse.[35] In a *type IV* prolapse (Figure 38-10), the peritoneal rectum and a variable length of the small colon form an intussusception through the anus.[35] This type of prolapse is seen with dystocia in mares.

Clinical Signs and Diagnosis

The usual presentation of a prolapse is a mucosal mass (types I, II, and III) or tube (type IV) protruding beyond the anus, with

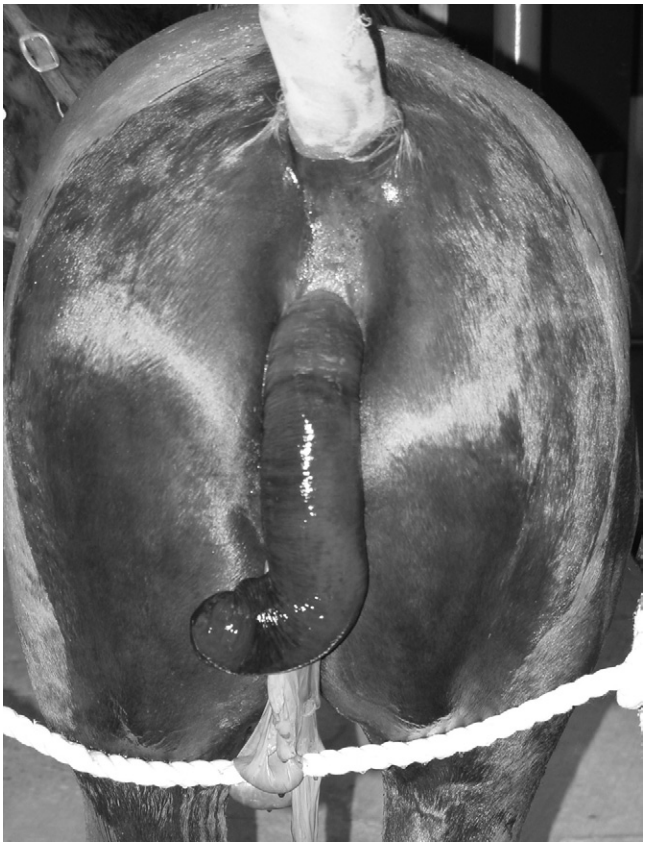

Figure 38-10. Type IV rectal prolapse in a postpartum mare.

a variable amount of inflammation, cyanosis, bruising, or necrosis. Colic and peritonitis develop with types III and IV, and abdominocentesis should be performed in such cases.[36]

Treatment

Most early type I and II prolapses respond to reduction and treatment of the primary problem. In many type I and II prolapses, mucosal edema and irritation can be reduced by topical application of glycerin, sugar, magnesium sulfate, and lidocaine jelly, or lidocaine enemas (12 mL of 2% lidocaine in 50 mL of water). Epidural anesthesia may be applied to reduce straining and to facilitate manual correction. To prevent recurrence, doubled 6-mm (¼-inch) umbilical tape can be placed with four wide bites in loose purse-string fashion, 1 to 2 cm lateral to the anus. Normal feces cannot pass through the purse-string, so it has to be opened every 2 to 4 hours to allow defecation or manual removal of feces. Mineral oil enemas and intragastric infusions of mineral oil or other laxatives should be given as needed, and the horse should not be fed for 12 to 24 hours. From then on, a laxative diet should be fed for at least 10 days. If it is well tolerated, the purse-string suture can be left in place for 48 hours. If a horse with a type IV prolapse is to be referred to a hospital for definitive treatment and if the prolapsed tissue appears nonviable, it might be better to leave it prolapsed so the surgeons can evaluate and treat it. Reduction of necrotic tissue can lead to peritonitis and complicate treatment, because access through a celiotomy is limited, especially compared with access when the rectum is prolapsed.

If type III and IV prolapses are treated by manual reduction alone, serial peritoneal fluid samples should be obtained to monitor changes in bowel wall viability.[34] Frank blood in the rectum after correction of a prolapse may be evidence of bowel necrosis.[36] Laparoscopy can be used to determine whether the mesocolon is ruptured and to assess viability of the involved small colon.[37] Access for resection of the necrotic bowel may be difficult through a celiotomy, and re-creation of the prolapse might be required to allow a submucosal resection outside the anus. A colostomy could be performed, but continued necrosis ultimately leads to failure.[19]

Submucosal Resection

A submucosal resection may be indicated if the prolapsed tissues are devitalized, the prolapse recurs after conservative treatment, or the horse continues to strain.[34] The procedure can be performed with epidural or general anesthesia. After preparation for surgery, two 18-gauge, 15.0-cm (6-inch) spinal needles, or 14-gauge, 13-cm (5¼ inch) Teflon catheters with the stylet in place are inserted at right angles to each other through the external anal sphincter and healthy mucosa to maintain the prolapse during dissection.[34] Starting at the 12 o'clock position, circumferential incisions are made in healthy tissue for one third of the prolapse circumference.[34] These incisions should be combined with deep dissection to elevate a strip of edematous and necrotic mucosa and submucosa. Remaining healthy proximal and distal edges of the mucosa and submucosa are apposed with size 1 or 2 medium chromic gut or PDS in an interrupted, horizontal mattress pattern.[34] These steps are repeated for each of the remaining thirds of the circumference until all necrotic tissue has been removed.[34] Mucosal edges are subsequently apposed with simple-interrupted sutures with buried knots, or preferably with a simple-continuous pattern interrupted at three equidistant points around the circumference.[34] A 2-0 absorbable suture material is used for this layer. The purpose of the mucosal suture is to cover all denuded areas and to prevent extensive granulation, scarring, and stricture formation.[34] Postoperative management includes laxatives, a laxative diet, and, if necessary, careful digital removal of impacted feces from the rectum.

Resection and Anastomosis

Resection and anastomosis may be indicated for type IV prolapse if the prolapsed tissues are devitalized or too much tissue is involved to allow reduction. The procedure can be performed as for submucosal resection, except that full-thickness circumferential incisions are made through the inner and outer walls of the intussusceptum in healthy tissue (Figure 38-11). The healthy proximal and distal edges are apposed with size 1 or 2 PDS in an interrupted, full-thickness, horizontal mattress pattern (Figure 38-12). These steps are repeated for each of the remaining thirds of the circumference until all necrotic tissue has been removed. Care must be taken during resection to identify and ligate any mesenteric vessels in the prolapse. Mucosal edges are then apposed in a simple-continuous pattern with 2-0 PDS, interrupted at three equidistant points around the circumference. The transfixing cross-needles and the weight of the necrotic tissue through most of the procedure maintain the line of anastomosis outside

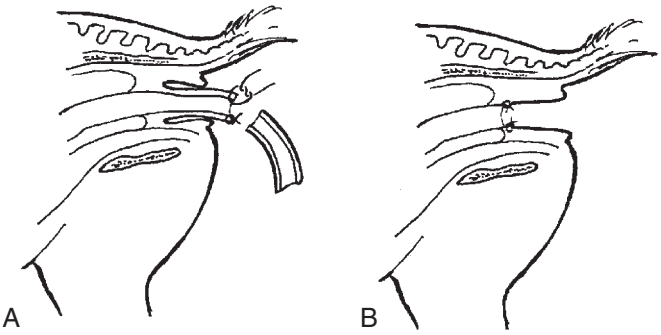

Figure 38-11. Treatment of a type IV rectal prolapse with intussusception of the distal part of the small colon and peritoneal segment of the rectum. **A,** The prolapsed tissues are resected as described and the healthy tissues anastomosed. **B,** When the stabilizing cross-catheters or needles are released, the anastomosis assumes its position in the proximal part of the rectum. (From Edwards GB: Diseases and surgery of the small colon. Vet Clin North Am Equine Pract 13:359, 1997, with permission.)

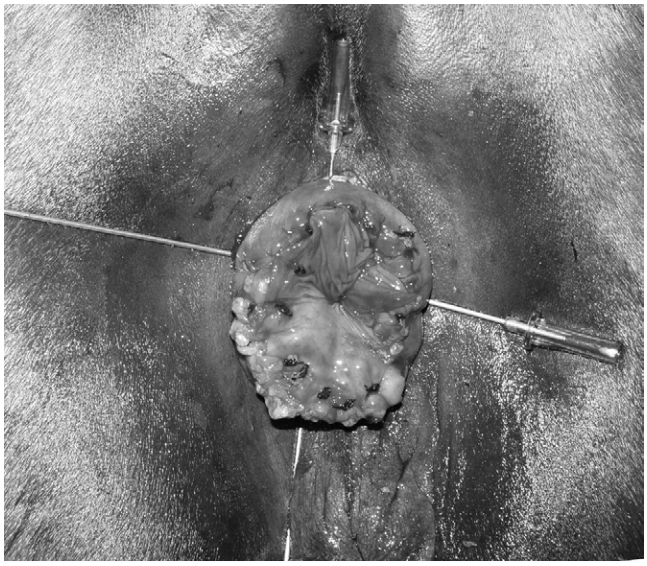

Figure 38-12. Partially completed anastomosis retained in position outside the anus with crossed 14-gauge, 5¼-inch-long Teflon catheters with the stylet in place. The mucosal closure has been completed on top and the visible sutures are full-thickness horizontal mattress sutures. This mare (the same one shown in Figure 38-10) made a complete recovery.

the rectum. Postoperative management is the same as for submucosal resection.[34]

Prognosis

The prognosis is favorable with types I and II prolapses, but the severity of vascular damage and mesenteric disruption worsens the prognosis for types III and IV prolapses.[35,38,39]

PERIRECTAL ABSCESS

In most cases, the cause of perirectal abscessation is unknown, although possible causes are rectal puncture or tear, rectal inflammation, and gravitation of a gluteal abscess after injection.[40] Enlarged anorectal lymph nodes have been reported as causes of rectal obstruction in young horses (3 to 15 months of age), and such nodes can progress to form abscesses.[41] *Streptococcus zooepidemicus* and *Escherichia coli* have been isolated.[40,41]

Clinical Signs and Diagnosis

The most common signs are low-grade abdominal pain and depression, anorexia, reduced fecal production, dyschezia, tenesmus, and fever.[40,41] Urinary tract dysfunction, manifested as dysuria, may develop from neuritis secondary to regional inflammation.[41] The abscess can form a visible swelling lateral or dorsal to the anus and can be palpated as a firm submucosal mass inside the rectum. Purulent material can be aspirated for culture and sensitivity testing through a needle inserted percutaneously (preferable) or through the rectal mucosa. Ultrasonographic examination is helpful, especially to monitor the response to treatment, but it cannot always provide a clear distinction between a reactive lymph node and an abscess, a hematoma, or a tumor.[41]

Treatment

Epidural anesthesia or sedation and local anesthesia are used for restraint. A lateral abscess can be drained lateral to the anus, a dorsal abscess can be drained into the rectum, and a ventral abscess can be drained into the vagina in mares or ventrolateral to the anus in males. Postoperatively, the abscess cavity is flushed daily for approximately 6 days with a 10% povidone-iodine solution.[40] A laxative diet is fed, mineral oil is given as needed by nasogastric tube to aid defecation, and a nonsteroidal anti-inflammatory drug such as flunixin meglumine is used to reduce discomfort.

Rarely, the abscess involves abdominal organs and causes peritonitis, which is confirmed by abdominocentesis.[40] An exploratory celiotomy may be required to drain the intra-abdominal component by marsupialization or to facilitate drainage into the rectum or vagina.[40] In horses without abdominal involvement, the prognosis for recovery is favorable.[40] Surgical drainage does not appear to be necessary in young horses with enlarged or abscessed anorectal lymph nodes, and a successful outcome is possible with treatment by antibiotics, analgesics, laxatives, and diet modification.[41]

ATRESIAS OF THE ANUS AND RECTUM

Atresia ani is rare and may be associated with atresia coli, atresia recti, persistent cloaca, absence of a kidney, renal hypoplasia and dysplasia, absence of the tail, musculoskeletal deformities, micro-ophthalmia, rectourethral fistula, and other urogenital abnormalities.[42-49] Atresia coli is also rare (0.44% to 1.3% of hospitalized foals), and affected foals have a normal anus but may have a blind and empty rectum.[50]

Clinical Signs and Diagnosis

Signs of atresia ani are evident shortly after birth and include straining to defecate, tail flagging, abdominal discomfort, and abdominal distention. The anus is absent, but an anal sphincter may be present (Figure 38-13). Signs of intestinal obstruction are not seen in foals with atresia and congenital rectovaginal

fistula because they can void feces through the vagina. Three of seven foals in one study on atresia coli had atresia of the rectum or a blind-ending rectum that could be diagnosed by digital palpation.[50] As with all foals with atresia coli, these foals had a history of failure to pass meconium and a lack of meconium staining of the perineum.[50]

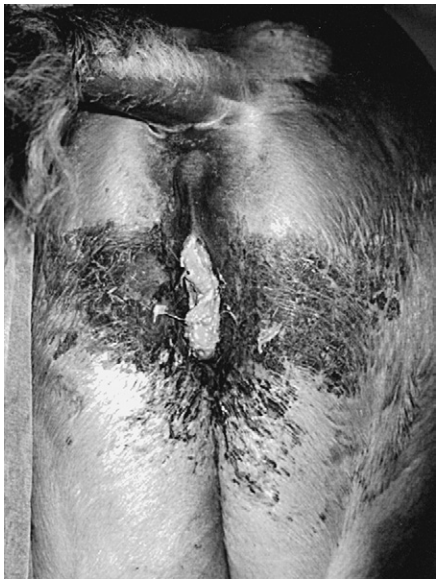

Figure 38-13. Atresia ani in a 5-day-old filly, with meconium staining of the perineum caused by defecation through a rectovaginal fistula.

Treatment

In foals with a complete rectal pouch, the persistent anal membrane is incised or a small circular piece of skin is removed, and the anal sphincter is spared, if possible. The rectal wall is subsequently sutured to the skin with simple-interrupted sutures. If the distal rectum is atretic, deeper dissection is needed. Any communication with the urogenital tract should be closed by transection between structures and closure by inverting sutures or by simultaneous closure and transection with autosuture instruments (GIA). The prognosis for life is favorable, but normal anal function may not be obtained. The prognosis for atresia coli, with or without rectal involvement, is poor.[50]

POLYPS AND NEOPLASIA

The most common neoplasms of the perineal region and anus of the horse are squamous cell carcinomas and melanomas.[51-55] Squamous cell carcinomas are necrotic, foul smelling, and locally invasive lesions that metastasize slowly.[51] Diagnosis is based on biopsy. Treatments include surgical excision, cryosurgery, electrosurgery, laser surgery, hyperthermia, radiation therapy, immunotherapy, and combinations of these methods.[51] Colostomy can be used to bypass rectal obstruction by a large tumor.[55]

Approximately 80% of Gray Horses older than 15 years have melanomas.[52] Melanomas may be solitary or multiple, and they most commonly occur in the perineum and along the ventral surface of the tail (Figure 38-14).[51] Although most equine melanocytic tumors are benign, dermal melanomatosis can occur on the perineum and have a high metastatic rate (for details, see Chapter 29).[56] Diagnosis is made by clinical features and biopsy.[56] Treatment is indicated if the lesion is large enough to

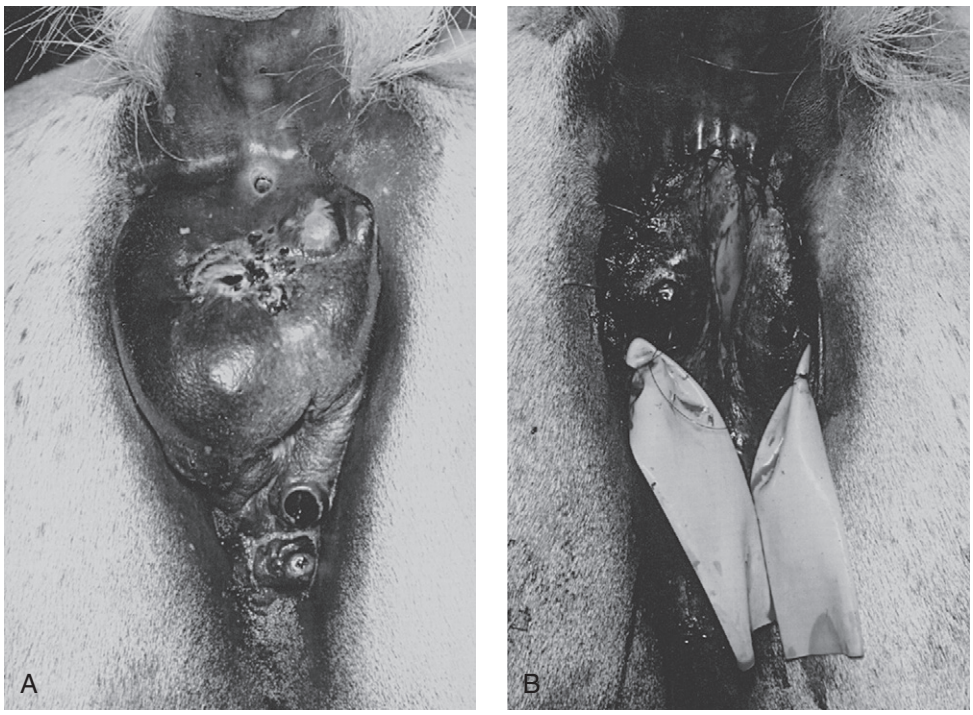

Figure 38-14. A, A large melanoma that deformed the anus and interfered with defecation. **B,** The same horse after excision. Penrose drains are placed in the dead space created by the deep perirectal dissection required to remove the large masses completely. Small tumors were not removed so that sufficient skin remained for closure.

cause clinical problems or is esthetically unacceptable. The treatment of choice is early excision or cryosurgery, although removal of all lesions in a diffuse cluster is not always feasible or necessary.[51] Successful treatment with radiation therapy, cimetidine, and bacillus Calmette-Guérin has been reported.[53]

Rectal polyps, adenocarcinoma, and leiomyosarcoma are rare in horses and can be treated successfully by transection of attachments to the rectal mucosa.[34,43,57] A hamartomatous polyp (focal disordered growth of mature tissue indigenous to the organ affected) obstructed the distal part of the small colon in a 2-day-old Standardbred foal and caused abdominal pain, tenesmus, and rectal bleeding.[58] The mass could be identified on radiographs after a contrast enema, but it was not readily accessible for removal.[58] Rectal strangulation by a mesenteric lipoma has been treated successfully by twisting the lipoma off its pedicle through a colpotomy.[59]

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CHAPTER
39 Abdominal Hernias

Martin R. Kummer and John A. Stick

A hernia represents a protrusion of an organ or tissue through an abnormal opening.[1] Hernias usually involve protrusion of abdominal contents through part of the abdominal wall, the diaphragm, or the inguinal canal. The defect or opening in the abdominal wall is an important factor in herniation, whether a protrusion through the opening is present or not.[2]

CLASSIFICATION

Various classification schemes are used to characterize hernias. One scheme names the hernia by the type of herniated tissue, such as intestine or omentum. Another classification scheme uses epidemiology of the hernia, wherein they are referred to as either congenital or acquired hernias. A *congenital* hernia is a defect that is present at birth, although the protrusion may not develop until later. In an *acquired* hernia, the defect occurs after birth and may be the result of blunt trauma, surgical trauma (wound disruption), or degeneration (prepubic tendon rupture in late-pregnancy mares), or it may occur because of an increased diameter of a normal body opening (inguinal canal).

A third classification scheme defines a hernia by the opening or defect. In *true* hernias, the protrusion occurs through a normal aperture in the abdomen and contains a complete peritoneal sac (inguinal or scrotal hernia). These hernias are sometimes referred to as *indirect* hernias. Conversely, in *false* hernias, the protrusion does not arise through a normal aperture in the abdomen. False hernias do not initially contain a complete peritoneal sac and are usually created by trauma to the abdomen or develop after breakdown of a surgical entry (incisional hernias). These are also referred to as *direct* hernias.

Hernias may be further categorized as reducible, incarcerated, or strangulated. A hernia is classified as *reducible* when the protruding hernial contents are freely movable and can readily be manipulated back into the abdominal cavity. When the hernial contents cannot be reduced, the hernia is classified as *incarcerated* (or irreducible), and this is frequently the result of adhesions that form between the contents and the surrounding tissue, effectively fixing the contents in an abnormal location. Incarceration frequently causes obstruction of the lumen of a hollow viscus, and either signs of bladder or bowel obstruction may be the presenting complaint. A hernia is classified as *strangulated* when incarceration obstructs the vascular supply to the herniated tissue, usually at the edge of the defect. Strangulation usually results in tissue necrosis and is the most serious type of hernia.

In a parietal hernia only a portion of the wall of the intestine is involved. These hernias are also called *Richter hernia* and *Littre hernia*. A parietal hernia of an intestine may manifest as draining ingesta from the abdominal wall, without contamination of the abdomen.

The anatomic site of herniation is frequently used for classification and allows the best discussion of etiology, clinical signs, and treatment. In this chapter, abdominal hernias are divided into umbilical, ventral midline (incisional), lateral abdominal, diaphragmatic (internal abdominal), and caudal abdominal (inguinal) hernias.

ANATOMY OF A HERNIA

Hernias consist of three portions: the ring, the sac, and the contents.[2] The ring forms the actual defect in the limiting wall and may be small or quite large. Wound healing leads to thickening of the leading edge of the ring through collagen maturation, which usually allows its borders to be identified through digital palpation. If this healing results in contraction of the scar, contents may become strangulated. Inflammation in the sac from trauma to the contents frequently leads to incarceration. Loops of intestine frequently move in and out of the ring of the hernia through peristalsis. Sometimes this leads to strangulation when the contents enter at a rate faster than they leave. Additionally, secretion of fluid into the bowel segment located in the hernial sac may cause distention and may eventually lead to strangulation.

The hernial sac is made up of tissues that cover the contents. This usually includes the mesothelial covering. In the initial stages, peritoneum may not line the sac of the hernia; however, chronic hernias are frequently completely lined with peritoneum. Although the contents of an abdominal hernia are usually parts of the intestine, other structures such as the omentum frequently are involved. The type of bowel in the hernial sac frequently predicts the clinical signs that are seen.

SIGNS OF HERNIATION

Most external abdominal hernias have swelling as a classic sign. However, internal abdominal hernias (diaphragmatic hernias) frequently show signs attributed to intestinal obstruction or respiratory problems. In uncomplicated external hernias, no pain may be elicited on palpation, and the consistency of the swelling depends on the contents. When inflammation or infection or luminal distention of a viscus is present, a definitive diagnosis is difficult to make on palpation alone. Also, the site of the swelling may be some distance from the actual hernial ring, for example, when contents migrate into the subcutaneous space. However, a tentative diagnosis is frequently made on the basis of clinical signs and location of the swelling. Palpation becomes an important part of the examination to determine if the hernia is reducible, incarcerated, or strangulated. Occasionally, radiography shows luminal gas distention if gastrointestinal structures are herniated. Diagnosis is frequently confirmed by reduction of the hernia and palpation of the hernial ring. The incarcerated hernia presents more of a diagnostic challenge, and ultrasonography is the diagnostic modality of choice in these instances. Use of fine-needle aspiration to rule out hernial contents is contraindicated, because it frequently results in contamination of an otherwise uninfected site.

UMBILICAL HERNIAS

The most common type of hernia in the horse is umbilical in origin and is estimated to occur in 0.5% to 2.0% of foals (Figure 39-1). Most umbilical hernias are congenital and may have a hereditary component. In a recent study of the closure of a body

Figure 39-1. Typical appearance of an umbilical hernia in a 3-day-old foal. The majority of umbilical hernias in foals at this age spontaneously regress within the first 3 weeks of life.

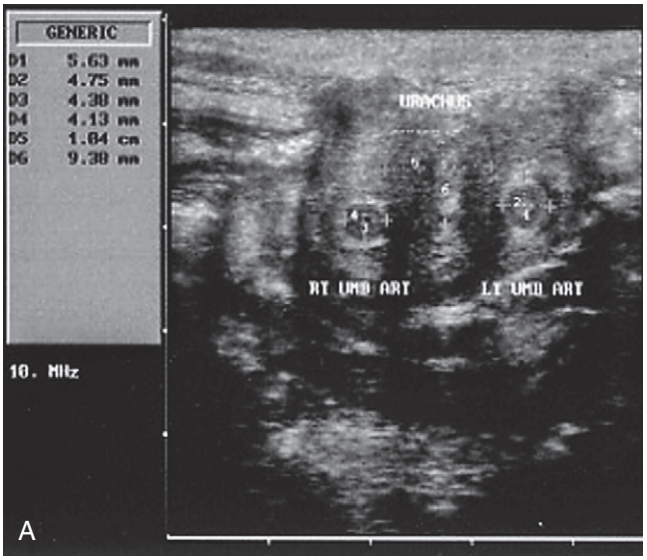
wall defect at the umbilicus, 19 of 44 foals had a defect in the body wall at the umbilicus with a palpable umbilical ring at birth.[3] This defect closed within 4 days in 18 of these foals. Additionally, in this same study, 12 of 44 foals developed an umbilical hernia between 5 and 8 weeks of age.

Etiology

The development of umbilical hernias has been attributed to trauma to the umbilical cord during birth, excessive straining, and umbilical cord infection.[4] Very rarely, evisceration occurs immediately after the birth as a result of trauma to the umbilical cord. This condition necessitates emergency surgical reconstruction in the abdominal wall. However, in most foals, umbilical hernias are chronic, small, and uncomplicated by underlying organic diseases. They usually represent a cosmetic defect but are a potential site of bowel incarceration. In fact, between 8% and 10% of umbilical hernias referred to tertiary hospitals sustain complications that are life threatening and mandate emergency surgery.[5]

Diagnosis

Digital palpation of the hernia is used to assess the size and shape of the hernia ring, the nature of the contents of the sac, and the reducibility of the hernia. Firm, thickened rings hold sutures quite well during herniorrhaphy, whereas a thin and indistinct ring is an indication that repair may be more difficult. Hernial sacs usually contain subperitoneal fat, omentum, or intestine. These hernias are almost always reducible and are rarely incarcerated. However, when incarceration does occur, strangulation of a portion of the intestine is usually suspected. In some instances, only a portion of the intestinal wall is incarcerated (Richter hernia or parietal hernia), and luminal wall destruction usually does not occur. When incarceration is present, any increase in hernia size, firmness, edema, or pain on palpation is an indication that surgery should be undertaken as soon as possible. Umbilical hernias need to be differentiated from abscessation and local infection of the umbilical cord and associated structures. Ultrasonographic examination of the



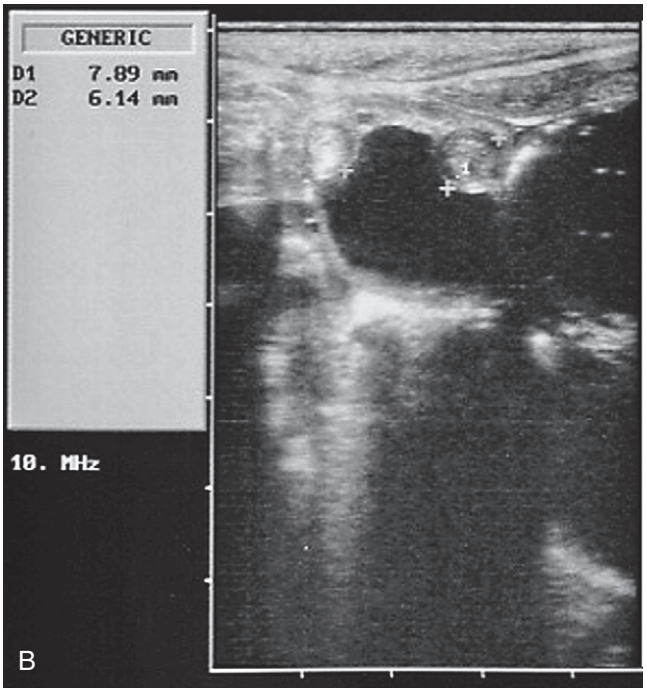


Figure 39-2. A, Sonogram of the urachus and both umbilical arteries obtained from a 1-week-old Thoroughbred foal with an umbilical hernia. The umbilical arteries are normal. **B,** Sonogram of the umbilical arteries obtained from a 12-day-old foal with omphalophlebitis. Notice the thickened left umbilical artery with the swollen hyperechoic vessel wall and hypoechoic fluid center.

hernia and ventral abdominal wall is indicated to determine the nature of the contents in complicated hernias of this type (Figure 39-2).

Treatment

Reducible umbilical hernias less than 5 cm in diameter frequently close spontaneously as the foals mature. Daily digital reduction of the hernia is indicated to monitor for changes in size or potential development of incarceration of hernia

contents. Hernias that are not spontaneously regressing by the time the foal is 4 months old should be surgically repaired.

Umbilical hernias greater than 10 cm (4 inches) in diameter are not treated with conservative methods because the risk of strangulating obstruction is so high. Additionally, a hernia of that size very rarely closes on its own. Although most umbilical hernia repairs are elective procedures and are performed on foals for cosmetic reasons, obviously strangulating obstructions of the small intestine necessitate emergency repair.

Two methods have been historically used for repairing hernias. The application of a hernia clamp is one method of management, but this is not commonly practiced and therefore is not part of this discussion. It poses a serious complication if entrapment of the intestine into the hernial clamp occurs, and this has led to intestinal obstruction, peritonitis, and enterocutaneous fistulas. Also, inadvertent dislodging of the clamp has led to evisceration and eventration. Because of these complications, this technique is strongly discouraged.

Surgical repair of umbilical hernias is accomplished with the horse in dorsal recumbency and under general anesthesia. Careful preparation of the surgical site is especially important in male foals, in which the preputial orifice should be filled with cotton and temporarily closed with sutures to reduce contamination during surgery. Surgical repair is initiated by an elliptical skin incision around the hernia, with the points of the ellipse cranial and caudal (Figure 39-3). Skin and loose subcutaneous tissue are removed to expose the external sheath of the rectus abdominal muscle. Sharp dissection generally results in less swelling than blunt dissection in this area, and this dissection is continued to about 1 cm peripheral to the hernial ring. The hernial sac can be inverted and the hernia repaired without removal (closed technique). However, the sac is usually removed (open technique). To do this, the hernial sac is incised along the edge of the hernial ring (see Figure 39-3, *E*). The contents of the sac are examined. It is unusual, but abscesses in the umbilical veins and umbilical remnants can be inadvertently entered at this point, and care should be taken, especially in the caudal aspect of the incision, to prevent such mishaps. Once the contents have been inspected and appropriately treated, the body wall should be closed. Varying suture materials and patterns can be used; however, a nicer closure without a dog-ear can be achieved if the ventral body wall defect is converted before closure to an ellipse with the point cranial and caudal, similar to the skin incision. A *simple-continuous* appositional suture pattern is recommended using appropriate-size absorbable suture material. Synthetic monofilament absorbable suture material (metric size No. 4, 5, or 6, and corresponding USP size No. 1, 2, or 3, respectively) (see Table 16-1) is selected on the basis of the body size of the patient. Thereafter, the subcutaneous tissues and skin are closed independently, once again using absorbable suture material in a simple-continuous pattern.

Aftercare

An abdominal support bandage is applied to decrease dead space and reduce edema for about 48 hours. A foal should be confined in a box stall for 30 to 45 days. Skin sutures do not need to be removed if absorbable suture material has been used. Although laparoscopic surgery is described (see Chapter 63) for disorders of the foal umbilical structures, there are few indications for laparoscopic surgery of umbilical hernias.

VENTRAL MIDLINE HERNIAS

Ventral hernias usually occur as a result of inadequate healing of a previous incision or excessive strain at the site of an abdominal wall scar (Figure 39-4). Obesity, age, and wound infection are the most common predisposing causes in people, and the incidence is between 1% and 11%.[6] Risk factors for incisional hernias in dogs include intra-abdominal pressure because of pain, entrapped fat between hernia edges, inappropriate suture material use, chronic steroid treatment, and infection.[7] Although sequelae to incisional hernias are often serious and costly, the incidence is much lower in small animals.

In horses, large abdominal wall defects commonly arise from partial incisional dehiscence after ventral midline celiotomy or after failed umbilical herniorrhaphy. Incisional herniation is strongly associated with wound separation. With the proper suture placement, suture selection (in terms of size and material), and soft tissue handling using aseptic technique, primary-intention healing of a ventral midline incision usually occurs. The experimentally determined, optimal bite size is 15 mm (½ inch) from the incision edge.[8] Ninety-three percent of sutures fail at the knot, and suture loops fail before complete fascial disruption. The strongest synthetic absorbable suture materials tested in this study were USP No. 6 polyglactin 910 and USP No. 5 polyglycolic acid. Preferably, USP No. 7 polydioxanone or USP No. 6 polyglactin 910 are recommended in adult horses for hernia closure.[9] Because the linea alba is thickest near the umbilicus and tends to become thinner cranially, it has been suggested that the cranial portion of the linea alba may be more susceptible to dehiscence. Clinically, the hernias that develop are usually located in the cranial portion of the incision rather than near the umbilicus. In one study of 210 horses undergoing midline celiotomy for abdominal disease, the rate of incisional hernias was as high as 16%, and all hernias were evident within 4 months of surgery.[10]

We suggest that the incidence of incisional infections and ventral midline hernias can be decreased by applying a belly bandage before recovery (when the horse is hoisted). It not only protects the skin suture line from contamination but may decrease the distracting forces on the sutured linea alba during recovery.

In addition to sepsis, other factors that may affect breakdown of an incision are abdominal distention immediately after recovery and incisional trauma from excessive struggling during recovery from general anesthesia or from rolling associated with uncontrolled postoperative pain. For the purposes of treatment, incisional hernias may be broken down into two types: acute total dehiscence and chronic insidious hernia formation.

Acute Total Dehiscence

Disruption of the abdominal incision that occurs within the first 4 to 7 days after surgery constitutes an emergency to prevent eventration. A belly bandage should be applied for support and the horse anesthetized for the repair. Complete acute dehiscence is detected by observing excessive peritoneal fluid leaking from the incision and palpable gaps in the sutured abdominal wall. The horse is placed in dorsal recumbency, all sutures are removed, and the site is prepared for surgery.

All devitalized tissue is sharply incised and removed. The wound is cultured and thoroughly flushed. The preferred

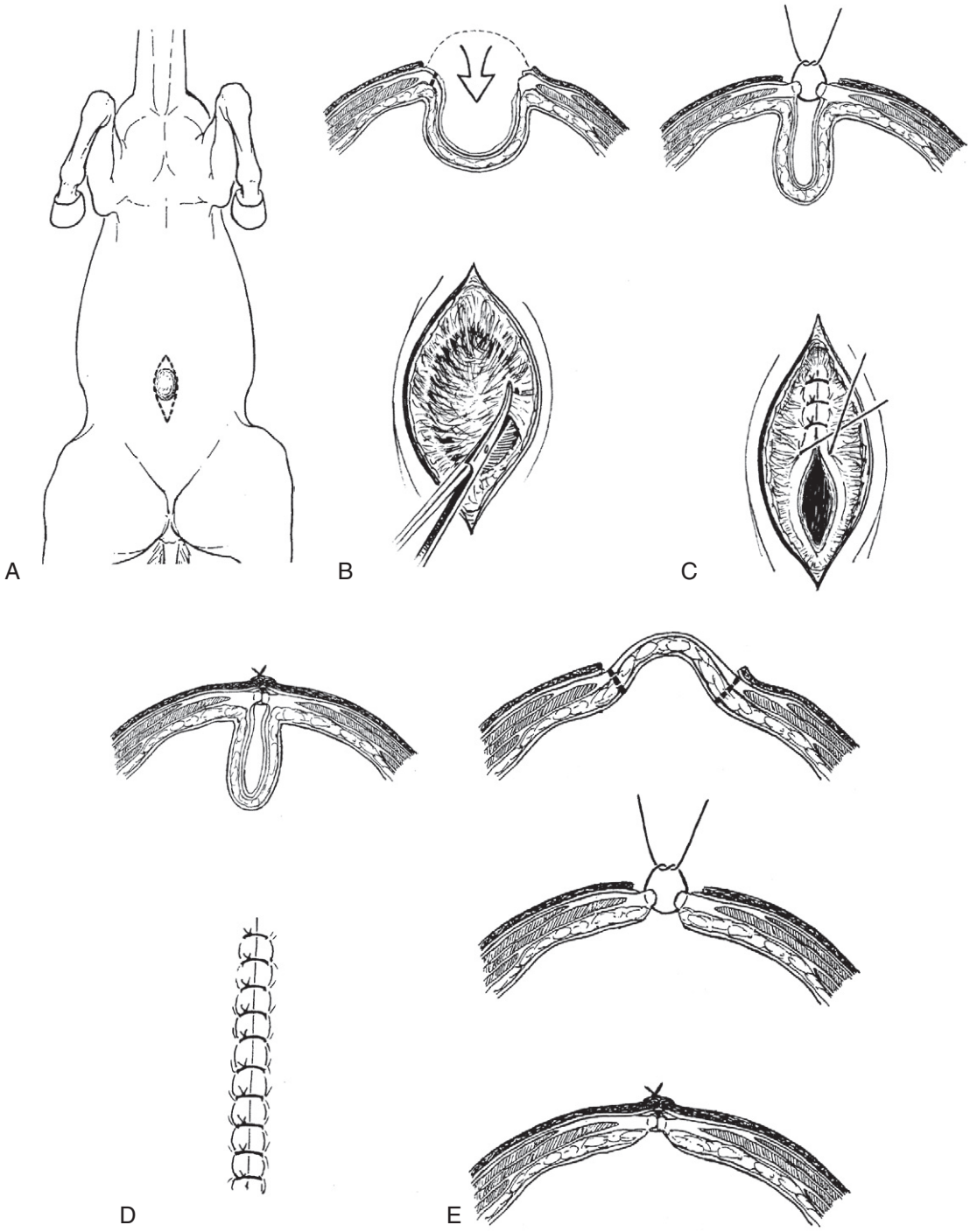


Figure 39-3. Illustrations of umbilical hernia repair. **A,** An elliptical skin incision is made around the hernial sac. The skin is dissected free from the hernial sac and discarded. **B,** The hernial ring is dissected free from the surrounding hernial sac so that the ring can be clearly identified. **C,** The sac can be inverted and the ring prepared for suturing, or the hernial sac can be removed. (The latter technique allows the round hernial ring to be converted into an ellipse for easier closure.) **D,** The ring is closed with a simple-continuous suture of appropriate-size suture material, and the subcutis and skin are closed in separate layers. **E,** In most cases, the open technique is applied: the hernia sac is excised and, after brief inspection of the abdominal cavity, the abdominal wall is closed in three layers.

surgical closure of an infected linea alba is accomplished with monofilament stainless steel wire applied as a through-and-through interrupted vertical mattress pattern, with sutures placed 2 to 3 cm apart. This suture is passed through skin, fascia, and rectus abdominal muscle about 5 cm (2 inches) from the wound edge (Figure 39-5). The wire is passed through hard rubber tubing used as stents before the bites of the vertical mattress pattern are placed. These bites are taken 2.5 cm (1 inch) from the wound edge. The rubber suture is necessary to reduce the tendency of the wire to cut through the skin and underlying tissues. The sutures are preplaced and the wound is closed by applying tension on all sutures simultaneously. The

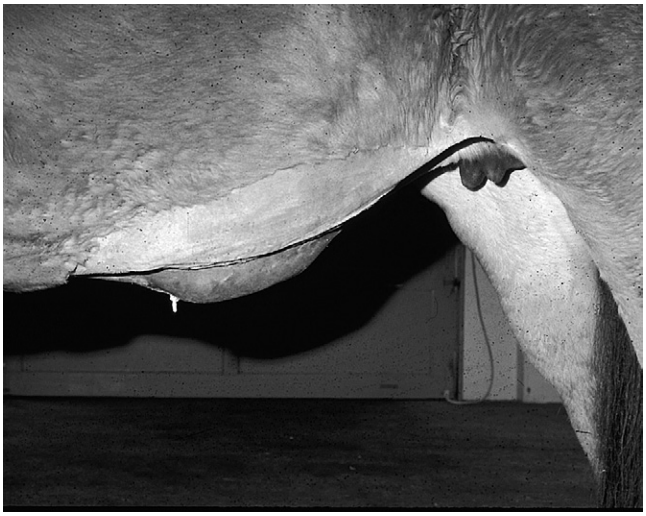


Figure 39-4. Appearance of a ventral abdominal hernia that occurred from dehiscence of a celiotomy incision. Note the purulent material at the cranial aspect of the hernia.

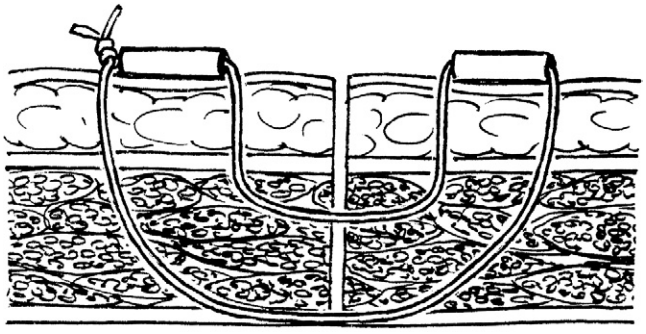


Figure 39-5. Schematic representation of the through-and-through interrupted vertical mattress suture that is used to repair acute total dehiscence of an abdominal incision.

wires are twisted rather than tied, and the cut ends of the wire are bent back into the lumen of the rubber tubing. If tissues are infected, which is usually the case, the skin edges are left unsutured to facilitate drainage.

Before recovery, a self-adhering drape is placed over the incision, and after recovery, a sterile abdominal compress is secured with Elasticon to form an abdominal bandage. The bandage compress should be changed every 24 hours until the incisions have healed (at about 30 days). Beginning on the 14th day after surgery, alternate wire sutures and rubber tubing are removed, and all remaining sutures are removed on day 21. Failure to remove these sutures this early after surgery will result in sutures and stents cutting through the skin, complicating their removal. Daily cleaning and rebandaging of the wound is necessary until the skin is healed.

Chronic Incisional Hernias

The factors responsible for acute total wound disruption usually are the same as the causes of partial dehiscence and eventual hernia formation. Incisional infection is usually part of the medical history preceding hernia formation.[11]

Many hernias can be managed conservatively. If infection is present, the wound should be cultured for bacterial growth and the animal treated with appropriate antimicrobial agents. Establishing a good drainage after removal of some skin sutures at the site of purulent discharge and periodic massaging along the suture line are very important measures in treatment of surgical site infection. Flushing with disinfectants is only recommended if there is good drainage; otherwise, there is the likelihood of disseminating the infection subcutaneously to other sutured portions of the incision. Sterile abdominal compresses may require daily changing. Continuous abdominal bandaging for 1 to 2 months during treatment of the wound infection reduces the size of the hernia. In some cases, chronic wound bandaging for up to 6 or 8 months eliminates the necessity for surgical repair of a wall defect. To decrease the amount of disposable bandaging material necessary to accomplish this, commercially available reusable elastic bandages (Surgimed) can be purchased and have been used successfully in a number of cases.

When a hernia has failed to heal or enlarges after turnout, surgical repair becomes necessary, especially if the horse is used in an athletic capacity. Large defects in the abdominal wall require prosthetic reconstruction with a synthetic mesh and are preferentially repaired in this manner. Knit polypropylene mesh (Marlex) is strong, elastic, and inert, and it resists infection. Other mesh materials with good physical properties include coated polyester (Mersilene) and polyglactin 910 (Vicryl). The main advantage of the polyglactin mesh is that it is absorbable, and in the advent of another infection, it may not have to be removed. Tissue grows through the knit mesh to be incorporated into the healing herniorrhaphy tissue. Failure to use the synthetic mesh in the repair usually results in enough tension to produce a recurrence of the hernia. All infection should be eradicated before prosthetic reconstruction of the defect is attempted. This may require surgical removal of infected suture material from sinus tracts (see Chapter 28). The earliest point in time for mesh implantation is 8 to 10 weeks after resolution of infection. If surgery is attempted sooner, the risk for postoperative infection is increased.

Prophylactic antimicrobial therapy is indicated because most incisional hernias have had some type of infection, and small pockets of bacteria may remain when surgical herniorrhaphy is undertaken. The horse is placed in dorsal recumbency under general inhalation anesthesia, and a strict aseptic technique is used.

Subperitoneal Mesh Placement with Fascial Overlay

The skin is incised for 180 degrees at the margin of the hernial ring (Figure 39-6, *A* to *C*). Likewise, the fascia and fibrous tissue overlying the hernial ring are incised along the same planes. This fascia is removed using retroperitoneal dissection, leaving the peritoneum intact. The mesh is placed retroperitoneally and subfascially. In some cases, this is not possible, because of adhesion formation between the peritoneum and the fascia, or because the fascia is so thin that it cannot be separated. In these cases only intraperitoneal and subfascial placement is possible. A single layer of mesh is cut to correspond to twice the size and contour of the defect, allowing the mesh to be folded over so the sutures pass through a double layer.

Horizontal mattress sutures are placed about 2.5 cm (1 inch) from the edge of the mesh. Each bite is approximately 2.5 cm wide. USP No. 2 sutures, made of the same material as the

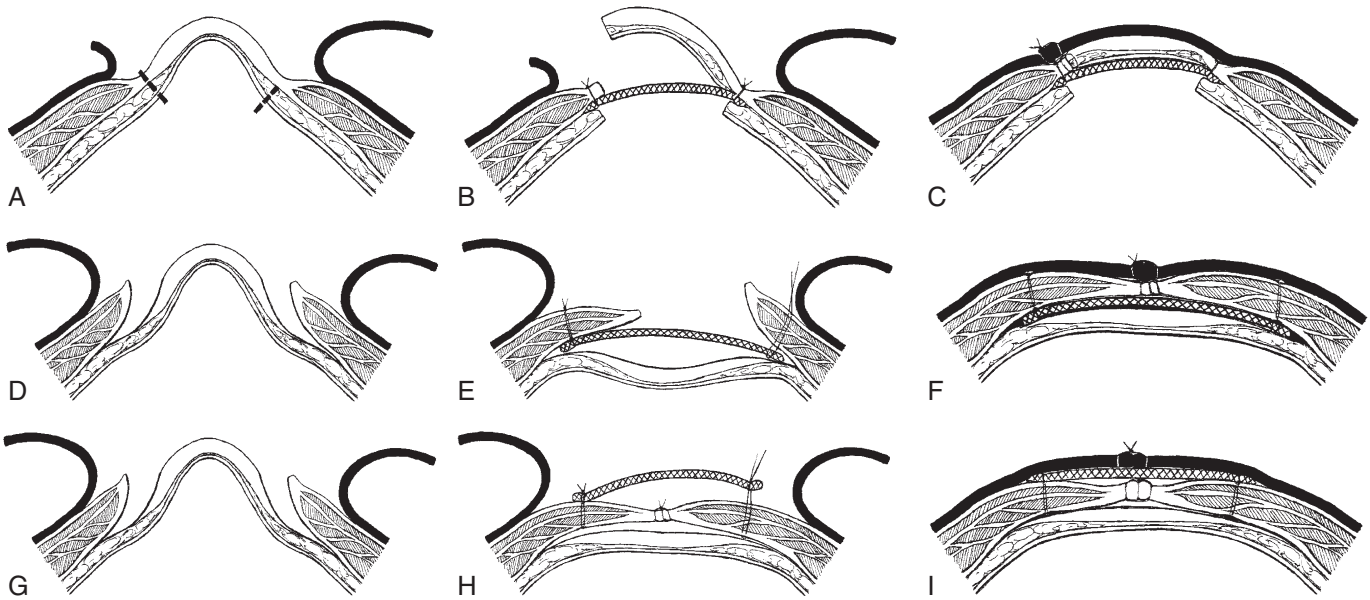


Figure 39-6. Illustrations of different techniques for herniorrhaphy using a synthetic mesh and fascial overlay. **A,** Semielliptical incision extending beyond the cranial and caudal margins of the hernia is made. The skin flap and subcutaneous tissue are reflected off the hernia and held with towel clamps. The fibrous hernial sac is then reflected toward its attached base, leaving the peritoneum in place if possible. **B,** The mesh is anchored on the inside of the abdominal wall with through-and-through horizontal mattress sutures. Some of these are preplaced. **C,** The fascial overlay is then sutured back in place using simple-interrupted or a simple-continuous suture. Routine closure of the skin follows. **D,** A straight incision extending beyond the cranial and caudal margins of the hernia is made. The skin and subcutaneous tissue is dissected from the hernial sac on both sides of the hernial ring. The hernial margin is also dissected free from the peritoneum (2 to 3 cm) on both sides. **E,** The mesh is anchored on the inside of the abdominal wall on one side. The sutures on the other side are preplaced, and then the incisional edges are apposed by tightening the sutures. **F,** The apposed linea alba is sutured and routine closure of subcutis and skin follows. **G,** After a straight skin incision, the hernial ring is prepared as described in **D. H,** After apposing and suturing the prepared linea alba, the mesh is placed in an overlay position and sutured to the abdominal tunic on both sides of the hernial closure (between fascia and subcutis) to reinforce the linea alba. **I,** The subcutis and skin are routinely closed.

mesh, are placed 0.5 cm apart (close enough together to prevent small intestine from becoming incarcerated between the sutures). The sutures are preplaced so that when they are tied, the mesh lies flat and snugly under a moderate amount of tension deep to the hernia ring. The hernial flap is subsequently trimmed and the free edge sutured to the hernial ring for reinforcement of the mesh implant. Simple-continuous synthetic absorbable sutures may be used. Subcutaneous tissues and skin are closed in a routine fashion. All redundant subcutaneous tissues and skin are trimmed so that the skin also is under a moderate amount of tension when closed.

Subperitoneal Mesh Placement with Hernial Ring Apposition

An adaptation of the technique just described starts with a straight skin incision, avoiding chronically scarred skin, which can be removed with an elliptical incision (Figure 39-6, *D* to *F*). The thickened peritoneum is sharply separated from the body wall around the whole hernial ring to expose at least 2 cm of the dorsal aspect of the body wall. The mesh is subsequently positioned between peritoneum and body wall. Using 2 to 4 layers of mesh, it is sutured to one side of the hernia with interrupted horizontal mattress sutures using USP 2 absorbable suture material. The sutures on the opposite side are placed similarly and secured after a single throw on the knots using a hemostatic forceps. After preplacing all sutures on the opposite side, the incisional edges are apposed by pulling on all of the forceps. The sutures are subsequently tightened and the apposed linea alba is closed with USP No. 6 suture material. The subcutaneous tissue and skin are closed routinely.

Subcutaneous Mesh Placement with Hernial Ring Apposition

Positioning of the mesh external to the fascia represents yet another technique for closure of chronic incisional hernias (Figure 39-6, *H* to *I*). After incising the skin incision, the hernial ring is exposed and the unopened hernial sac is inverted. Then the hernial ring is subsequently closed with inverted cruciate or near-far-far-near sutures placed at 1.5 to 2.5 cm intervals with USP 2 synthetic absorbable sutures. The mesh is then sutured to the abdominal tunic overlaying the fascia and placed subcutaneously.[12]

Laparoscopic Intraperitoneal Mesh Onlay

A laparoscopic hernioplasty of incisional hernias in five horses was described by Caron and Mehler.[13] This technique involved positioning a polypropylene mesh subperitoneally, overlaying the inner surface of the internal rectus sheath. The mesh is secured intraperitoneally using transfascial sutures tied extrafascially and subcutaneously.

With all of the described mesh techniques, some potential complications can be experienced. In one study, 62% of horses with a body weight greater than 450 kg showed some degree of

incisional drainage, and 23% experienced tearing of the internal abdominal oblique muscle.[14]

On recovery from surgery, an abdominal bandage is used for 7 to 10 days to prevent edema and seroma formation. Parenteral antibiotics are discontinued on the second or third day after surgery. Horses are confined to a stall for 60 days with hand-walking only.

LATERAL ABDOMINAL HERNIAS

Lateral abdominal hernias are usually the result of blunt traumatic injuries from farm machinery, fence posts, or kicks from other horses (Figure 39-7). Penetrating abdominal wounds may require immediate reconstruction to prevent evisceration, peritonitis, and wound infection. However, more commonly, traumatic wounds are not penetrating, and the horse can be treated with local therapy and abdominal bandaging. After 60 days, small residual defects in the body wall can be treated by direct suture apposition. When they are very large, they can be treated by prosthetic reconstruction with synthetic mesh, as described previously.

Abdominal wall hernias in pregnant mares that do not involve rupture of the prepubic tendon can be similarly repaired. However, it is suggested that the pregnancy be terminated by cesarean section if repair is necessary before parturition.

PREPUBIC TENDON RUPTURE

Rupture of the prepubic tendon occurs rarely in prepartum mares. Predisposing factors include hydrops allantois, hydrops amnions, trauma, twins, and fetal giants, but it is also seen in mares with normal pregnancy.[12,15-18] An increased prevalence of body wall defects in draft breeds and older mares is also reported.[12,16] The clinical signs include colic, development of ventral edema, stretched mammary gland, and most obviously, elevation of the tail head and ischial tuberosity (sawhorse stance).[19] Ross et al. reported that mares with prepubic tendon rupture can be successfully managed conservatively with a good prognosis for survival of the foal.[20] Intervention decreases the chances for the foals because of prematurity.[20]

DIAPHRAGMATIC HERNIAS

Internal abdominal hernias include a multitude of intestinal accidents, including mesenteric hernias, entrapment in the epiploic foramen, and diaphragmatic hernias. Although diaphragmatic hernias are rare, both congenital and acquired hernias have been described.[21-23] Diaphragmatic hernias can be congenital, resulting from failed fusion of many of the four embryonic components of the diaphragm, or acquired from trauma. Diaphragmatic hernias can be classified by anatomic location as peritoneal-pericardial or peritoneal-pleural, depending on where the tear occurs and into which thoracic compartment bowel enters. Congenital defects usually have smooth round edges, and some hernias can be quite small, only 2.5 cm (1 inch) in diameter. On the other hand, acquired diaphragmatic hernias, because they are associated with some form of violence and increased intra-abdominal pressure, are usually large and frequently originate along the dorsal body wall. There is a special form of diaphragmatic hernia called Morgagni hernia with an intact hernial sac where portions of the large colon can be incarcerated.[24]

Diaphragmatic hernias result in low-grade, chronic recurrent abdominal pain and sometimes are found incidentally at necropsy. However, this is the exception; more commonly, horses are presented in distress with either a serious abdominal crisis (found during exploratory celiotomy) or in acute respiratory distress. Diagnosis can be made on physical examination, with borborygmi auscultated during examination of the thorax. However, other signs associated with the acute abdominal crisis may be equivocal, since a rectal examination and abdominocentesis may be normal. Ultrasonography is quite accurate in revealing gas-filled loops of intestine in the thorax (Figure 39-8). Likewise, radiography may show a loss of the diaphragmatic line at the level of the hernia, or, more commonly, radiopacity in the ventral aspect of the chest. Occasionally, gas-filled loops of large colon can be visualized on thoracic radiographs (Figure 39-9).

Repair of the diaphragmatic hernia is accomplished with the horse placed in dorsal recumbency through a ventral midline

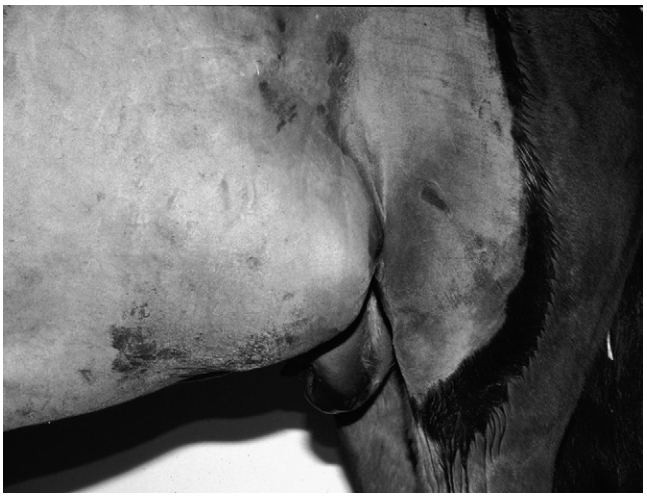


Figure 39-7. A horse that has a traumatic hernia in the lateral body wall as a result of blunt trauma.

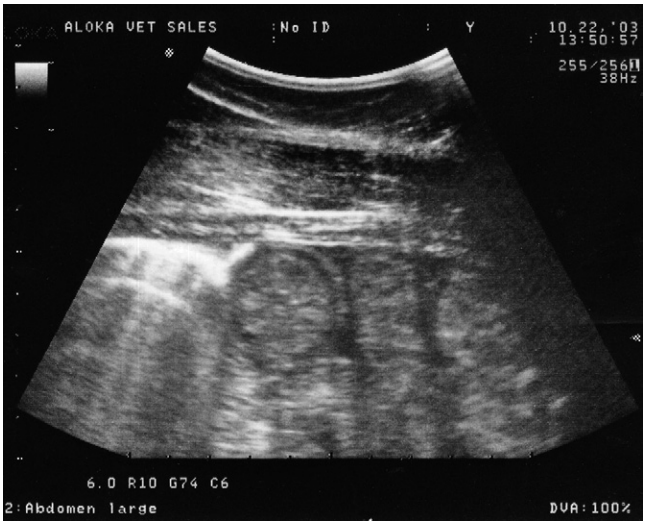


Figure 39-8. Thoracic ultrasonogram in a horse with a diaphragmatic hernia clearly demonstrates a circular viscus (bowel wall) between two ribs.

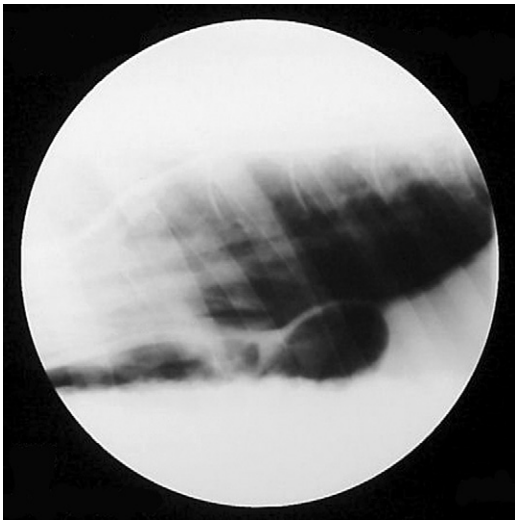


Figure 39-9. Thoracic radiographs of a horse with a diaphragmatic hernia demonstrate a gas-filled loop of bowel in the thorax and loss of diaphragmatic contour. Note fluid in the ventral aspect of the thorax.

celiotomy that extends craniad to the xiphoid. Assisted ventilation is necessary during anesthesia. Abdominal viscera should be repositioned into the abdomen, and tilting the horse with front end elevated may aid in reduction of the hernia. In some cases, especially with small chronic hernias, the incarcerated distended bowel cannot be reduced without enlarging the hernial ring either bluntly with the fingers or sharply.

Small defects (less than 5 cm [2 inches]), particularly those positioned ventrally, may be closed by primary apposition using large-gauge suture material in a simple-continuous pattern. Débridement is usually not performed, because once the hernial ring is removed, the muscular portion of the diaphragm is less likely to hold sutures as well as the hernial ring. Larger defects require prosthetic reconstruction with a synthetic mesh. The mesh is secured to the perimeter of the defect with simple-interrupted horizontal mattress sutures similar to those used for mesh herniorrhaphy for the ventral midline. Sutures are preplaced before tying to ensure that tension is maintained on the mesh. The mesh is preferentially placed on the abdominal side of the incision.

Traumatic diaphragmatic hernias are quite often associated with very large defects, dorsally positioned, and inaccessible, precluding safe closure. In these instances, thoracotomy is used to gain better access. The diaphragm holds sutures quite well if a linear tear has occurred, and mesh reinforcement may not be necessary. Before closing the abdominal incision, the lungs should be maximally inflated to expel as much air as possible from the thorax. A chest tube with a Heimlich valve (see Figure 17-7) should be used to manage a pneumothorax.

Alternatively, diaphragmatic hernia repair may be accomplished using thoracoscopic approaches and rib resection. Thoracoscopic evaluation of the chest immediately before repair helps in accurate placement of the thoracotomy incisions, facilitating mesh placement.[25,26] Repair of a diaphragmatic hernia located in the dorsal tendinous part of the diaphragm without rib resection using a thoracoscopic technique to close the hernia with interrupted extracorporeal surgical knots is described.[27]

Dehiscence of the repair occurs occasionally, and rupture of the diaphragm in another location has been reported. Prognosis for diaphragmatic hernia repair is generally guarded.

CAUDAL ABDOMINAL (INGUINAL) HERNIAS

Bilateral congenital inguinal hernias are often encountered in young colts, but they usually resolve without surgery during the first few weeks of life. The colt is confined to a box stall and observed, and the owner is instructed how to manually reduce the hernia several times daily. Larger hernias may benefit from support provided by an inguinal bandage constructed from rolled cotton and gauze. Reduction of the hernia is usually facilitated by casting the animal in lateral recumbency. For surgical descriptions of repair of inguinal hernias including via laparoscopy, please refer to Chapters 36, 39 and 59.[28]

By definition, inguinal hernias are present when there are intestinal contents in the inguinal canal. Scrotal hernias occur when the contents continue on into the scrotum. However, the term inguinal hernia is usually used to refer to both disorders. Distal jejunum and ileum are the intestinal contents most commonly encountered in inguinal hernias, but omentum and even the small colon are sometimes involved. Equine inguinal hernias usually are indirect (or true) hernias, with the contents passing through the vaginal ring and into the vaginal tunic. In a direct (or false) inguinal hernia, which occurs in humans, the contents do not pass through the ring but protrude through a weakness in the body wall. This occasionally occurs in foals, but it is highly unusual.

The clinical sign of an inguinal hernia is scrotal swelling. The testicle is often cool in the adult horse, probably because of vascular compromise. Frequently, the spermatic cord is enlarged and extremely hard and very painful to the touch. All stallions with colic should be checked for this type of inguinal hernia. Frequently, scrotal herniation in foals occurs without intestinal incarceration and may be the result of abdominal straining. Before repair is undertaken, conservative management for several weeks may be advisable. Diagnosis of inguinal hernias is made on the basis of external palpation; rectal palpation in adult horses permits further evaluation of the internal inguinal ring. Intestinal incarceration in the ring can be easily palpated. The differential diagnosis includes thrombosis of the testicle; testicular artery, seroma, or hematoma of the scrotum; neoplasia; and torsion of the spermatic cord.

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CHAPTER
40 Postoperative Care, Complications, and Reoperation

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Postoperative management of horses with abdominal disorders should be targeted toward identification and management of pain, support of the cardiovascular system, treatment of endotoxemia, identification and treatment of surgical infection, restoration of gastrointestinal function, and management of secondary complications (diarrhea, thrombophlebitis, laminitis). Disorders of the gastrointestinal (GI) tract can result in significant systemic derangements in affected horses. Strangulating lesions lead to intestinal distention proximal to the lesion, third-space pooling of fluid in the intestinal tract, and intestinal mucosal damage with subsequent endotoxin absorption. Despite correction of the primary condition, problems encountered in the postoperative period usually represent a natural progression of the events triggered by the primary lesion. This chapter reviews the management of horses after abdominal surgery and addresses complications related to specific surgical procedures.

FLUID THERAPY

Dehydration and electrolyte imbalances are commonly encountered as a result of abdominal disorders. Even though a horse is stabilized in the perioperative period and the primary problem is corrected, continued replacement of previous and ongoing fluid losses is critical to a successful outcome. Knowledge of basic electrolyte and fluid homeostasis is essential to the successful management of these cases (see Chapter 3). The daily maintenance fluid requirement in adult horses is 50 to 60 mL/kg, and in foals it is 70 to 80 mL/kg. The volume of GI secretions in adult horses is equivalent to their extracellular fluid volume, approximately 30% of the body weight. With ileus or diarrhea, these fluids are not reabsorbed by the large colon, resulting in considerable fluid losses. These losses must be added to the maintenance requirements. Fluids available for replacement therapy in horses include lactated Ringer's solution, which contains lactate as a buffer and calcium (4 mEq/L), Plasmalyte A, and Normosol-R, which do not contain calcium but have acetate or gluconate as buffer sources. Intravenous fluid administration systems are commercially available and provide a convenient method of administering large volumes of fluid to horses.

A significant complication of endotoxemia, particularly in postoperative cases of large colon torsion with significant mucosal damage, is increased capillary permeability resulting in fluid and protein loss into the interstitium. Vascular volume can be difficult to maintain in these horses because of the tremendous fluid loss in both the interstitium and the GI tract and the development of hypoproteinemia. The goal of therapy is to maintain enough vascular volume to sustain cardiac output. A reasonable indicator is the ability to maintain the heart rate at less than 80 beats per minute, packed cell volume (PCV, hematocrit) at less than 50%, and total protein at greater than

4.1 g/dL. PCV and total protein should be measured every 6 hours and therapy adjusted accordingly. If plasma protein decreases to less than 4.1 g/dL (corresponding to a plasma oncotic pressure of 12 mm Hg), it may be necessary to administer colloids to prevent severe edema and allow continued administration of fluids.[1] In surviving patients, capillary permeability is restored within 24 to 36 hours. Plasma proteins that were lost into the interstitium are quickly reabsorbed, and plasma protein concentration returns to normal range within a few days. Available colloids for administration to horses include plasma and synthetic colloids, such as dextrans and hydroxyethyl starch. Further reading on fluid therapy, colloids, and treatment of endotoxemia can be found in Chapter 2.

PAIN

Control of pain is an essential component of the management of postoperative patients. Drugs used for pain management should provide a good clinical response while minimally affecting GI motility.

Postoperative pain can originate from peritoneal inflammation, abdominal incision, or intestinal distention. *Flunixin meglumine* controls postoperative pain and improves the cardiovascular manifestations of endotoxemia. Flunixin meglumine combined with progesterone supplementation may help maintain pregnancy in mares with endotoxemia in the first trimester of gestation. Flunixin meglumine provides analgesia in a dosage-related manner up to a maximal dosage of 1.1 mg/kg. Smaller dosages (0.25 mg/kg) have been shown to improve cardiovascular parameters after endotoxemia.[1] *Butorphanol* is an agonist-antagonist narcotic that can be used to control postoperative pain and at low dosages (0.01 mg/kg) does not affect GI motility. Continuous-rate infusion of butorphanol (13 mg/kg per hour) to horses after celiotomy improved behavior scores and recovery characteristics and decreased plasma cortisol concentration.[2] However, time to first passage of feces was delayed significantly in treated horses.[2] Therefore, continuous-rate infusion of butorphanol may be contraindicated for certain GI problems. Horses with ileus occasionally require more potent analgesia. In such cases, administration of xylazine or detomidine, both α_2-agonists, may be necessary, even though these drugs can negatively affect GI motility. The duration of analgesia may be prolonged by combined intravenous and intramuscular administration.

Intravenous *lidocaine* has been advocated for the management of visceral pain as postoperative ileus.[3] *In vitro* studies have shown a potential benefit of lidocaine on normal smooth muscle contraction,[4] as well as in smooth muscle subjected to ischemia-reperfusion.[5,6] Lidocaine has also been shown to attenuate the clinical signs and inflammatory response to endotoxemia[7] and to ameliorate the inhibitory effects of flunixin meglumine on recovery of the mucosal barrier.[5] Although use of lidocaine on experimental postoperative ileus did not show benefits,[8] clinical studies support its use.[9,10] The dose of lidocaine recommended for the management of postoperative ileus in horses is a loading dose of 1.3 mg/kg IV, followed by an infusion rate of 0.05 mg/kg per minute IV.[10-12] Prolonged infusion appears to be safe, but concurrent administration of highly protein bound drugs can significantly displace the lidocaine, resulting in increased risk of toxicity.[12] The use of lidocaine is most beneficial with other analgesics and allows the dosage of analgesics to be decreased. (For further information on the mechanism of action of lidocaine on motility, see "Sodium Channel Blockers" later.)

Additional information on pain management can be found in Chapter 23.

POSTOPERATIVE ILEUS

Adynamic ileus is one of the most commonly encountered complications in horses after GI surgery and is often referred to as postoperative ileus (POI). In horses, POI occurs predominantly after correction of lesions involving the small intestine. Occasionally, POI is seen after correction of large colon problems, primarily large colon volvulus. Intestinal ischemia, distention, peritonitis, electrolyte imbalances, endotoxemia, traumatic handling of the intestine, resection and anastomosis, and anesthesia have all been proposed as potential contributing factors. Specific risk factors that have been identified for horses developing POI are age greater than 10 years, Arabian breed, PCV greater than 45%, high serum concentrations of protein and albumin, elevated serum glucose, presence of more than 8 liters of reflux at admission, anesthesia for longer than 2.5 hours, surgery longer than 2 hours, high pulse rate, resection and anastomosis, strangulating lesions of the small intestine and ascending colon, length of small intestinal resection, obstruction of the small intestine, and ischemic small intestine.[13-17] There is evidence that performing a pelvic flexure enterotomy (bowel decompression) and intraoperative administration of lidocaine decreases the risk of developing POI.[14-17]

In the current literature, the incidence of POI in horses undergoing surgical treatment of all types of colic has been reported to range from 10% to 21%.[13,16,18] In two recent studies, the incidence of POI in horses undergoing surgical treatment for small intestine lesions was reported to be 17% and 33%, respectively.[15,17] As expected, the survival rate was lower for horses that developed POI than for those that did not develop POI (87% and 84.1% survival rates in horses with POI, versus 93% and 94.1% survival rates in horses without POI). This is a marked improvement over a previous study that reported a mortality rate of 86% in horses with POI.[19] However, in one recent study of horses that had surgery to correct small intestine lesions, horses that developed POI were 28 times more likely not to survive to discharge compared to horses that did not develop POI, with only 34% of POI horses surviving to discharge.[17] Although management and prognosis of these cases has improved, POI is still associated with 38% to 40% of all postoperative deaths in horses treated for colic.[13,16]

Disruption of propulsive motility results in the sequestration of fluid, gas, and ingesta in the segment of the GI tract that is dysfunctional and in the intestine proximal to the dysfunctional area. Because small intestine strangulating lesions are the most common problems leading to POI, the distention occurs primarily in the small intestine and stomach. With colitis, endotoxemia, or ischemia following a large colon volvulus, distention may occur in the cecum and large colon. Signs of ileus are progressive and directly related to the accumulation of gas and fluid within the GI tract. Gastric distention usually occurs within 12 to 48 hours after recovery from anesthesia as fluid and gas back up from the small intestine. With increasing GI distention, the animal becomes more depressed and shows signs of increasing abdominal pain, such as pawing, flank watching, lying down, and rolling.[20-22] Borborygmi are usually decreased or absent. The heart rate is usually elevated as a result of pain associated with

distention. Hemoconcentration is reflected by increases in the PCV and total protein, as well as in elevated heart rate and increased capillary refill time. Decreases in plasma chloride and potassium are the most commonly observed electrolyte abnormalities, although sodium and calcium may also be low.[19-22] Rectal examination, ultrasonography, and abdominal radiography (in foals) can aid in determining if the small intestine or large intestine is involved. Nasogastric decompression often retrieves from 3 to 10 L of fluid. The response to nasogastric decompression provides an important clue that the problem is functional (i.e., POI). After decompression, the horse should show improvement, with decreased pain and heart rate. If no alleviation of signs is observed after repeated attempts at decompression, careful thought should be given as to the likelihood that the problem may be mechanical, such as a complication with the anastomosis, and not a functional problem. In addition, continued large volume of reflux for greater than 48 hours after surgery should alert the clinician to the possibility of a mechanical obstruction at the surgical site.

Supportive Therapy

Because of the therapeutic limitations of available prokinetic agents (discussed later), the hallmark of treatment of POI in the horse is supportive therapy. Included in this supportive therapy are fluid, acid-base, and electrolyte therapies. Antibiotics are also indicated if there is compromised intestine or the possibility of bacterial contamination resulting in peritonitis. Along with fluid therapy, nasogastric decompression remains the primary method to treat POI in the horse. Questions concerning when to place the tube, whether the tube should be left in place, and when to start feeding have not been adequately investigated. No clinically important impact of 18 hours of nasogastric intubation was found on gastric emptying in healthy horses.[23] In humans, eliminating or decreasing routine nasogastric intubation with early return to enteral feeding has been shown to be safe and may actually reduce POI.[24-26] There is some thought among equine surgeons that early removal of the nasogastric tube or passing the tube only when indicated predicated on pain, heart rate, rectal examination, and volume of previous reflux may attenuate the duration and severity of POI in the horse. This has yet to be substantiated. When providing supportive care for horses with POI, one must always question whether the horse is really experiencing a functional problem or has a mechanical problem and requires repeat laparotomy.[27,28] In a recent study evaluating horses requiring a second laparotomy, complications with the anastomosis was responsible for 16% of the cases.[29] It is likely that POI was diagnosed in many of these horses before the repeat laparotomy.

Prokinetics

It is logical to assume that during POI in the horse, an imbalance of factors controlling excitation and inhibition of GI smooth muscle occurs. Historically, pharmacologic modulation of GI motility in the horse has been directed at increasing excitatory cholinergic activity with administration of parasympathomimetic agents, such as bethanechol or neostigmine, or blocking inhibitory sympathetic hyperactivity with α-adrenergic blockers, such as yohimbine and acepromazine. However, the modulation of motility and mechanisms involved in alterations after insult are significantly more complex.[30] In addition to being affected by sympathetic and parasympathetic input, contractility of gastrointestinal muscle is directly mediated by enteric inhibitory neurotransmitters, such as vasoactive intestinal peptide (VIP), adenosine triphosphate (ATP), nitric oxide (NO), calcitonin gene-related peptide (CGRP) and enteric excitatory neurotransmitters, such as substance P (SP) and acetylcholine (ACh).[31-35] It is likely that abnormalities involving the enteric nervous system and its neurotransmitters, as well as inflammatory insult to the enteric smooth muscle cells and interstitial cells of Cajal (ICCs, astrocytes), which are responsible for generating patterns of electrical activity in the enteric smooth muscle cells that mediate contractile activity, are also involved in the pathophysiology of POI.[30,36-41] At this time, the relative importance of all the contributing mechanisms has not been sorted out. Additonally the treatments of POI in the horse are limited to pharmacologic agents that influence only a small number of the mediators of motility. For these reasons, prokinetics have produced only limited responses in the treatment of POI in the horse. The following is a summary of some of the more frequently used prokinetics in horses as well as some drugs currently being evaluated.

Parasympathomimetics

BETHANECHOL

Bethanechol chloride is a muscarinic cholinergic agonist (cholinomimetic or parasympathomimetic) that stimulates ACh receptors (primarily M_3 but also M_2 receptors) on GI smooth muscles at the level of the myenteric plexus, causing gastrointestinal contractile activity.[34,42] In an *in vitro* study, bethanechol increased contractile activity of smooth muscle preparations from the duodenum, jejunum, cecum, and pelvic flexure of horses.[42] In normal horses, bethanecol (0.025 mg/kg, IV) increased the rate of gastric and cecal emptying as measured by radiolabeled isotopes and increased myoelectrical activity in the ileum, cecum, and right ventreal colon.[43,44] In an equine POI model, bethanechol (2.5 mg SC at 2 and 5 hours postoperatively in ponies) shortened transit time as measured by the passage of beads, and it reduced the time until normal activity levels returned throughout the GI tract when administered in combination with yohimbine, an α-adrenergic receptor blocker.[45] This drug combination, however, was not as effective as metoclopramide in restoring coordinated gastroduodenal motility patterns in these ponies. Although efficacy in treating motility dysfunction has been questioned in the horse and other species, the prokinetic effects in normal horses and the clinical impression of its benefit in treating horses with ileus provides some support for its use in the treatment of certain motility dysfunctions, such as POI and gastric and cecal impactions.[34,43,46] The recommended dosage is 0.025 mg/kg SC, every 3 to 6 hours. The most common side effects of bethanechol are salivation with abdominal cramping and diarrhea.[44,47]

NEOSTIGMINE

Neostigmine methylsulfate is a cholinesterase inhibitor that prolongs the activity of acetylcholine (indirect parasympathomimetic) by retarding its breakdown at the synaptic junction. In studies on normal horses, the effects of neostigmine (0.022 mg/kg IV) varied depending on the portion of the gastrointestinal tract examined.[43,48,49] Neostigmine was shown to delay gastric emptying and decrease propulsive motility in the stomach and jejunum but to increase propulsive motility in

the pelvic flexure.[48,49] These results suggest that the drug would not be appropriate for gastric and small intestinal problems but may be beneficial for large intestinal motility dysfunction. However, neostigmine increased the amplitude of rhythmic contractions in both the resting and distended jejunum in anesthetized ponies, and it induced contractile activity in the ileum and increased the rate of cecal emptying in other studies, supporting its use for motility dysfunction in both small and large intestine.[43,50] There has been no consensus as to the recommended use of this drug for treatment of motility disorders in horses. It is rarely used as a prokinetic agent in humans.[34] There is anecdotal support that neostigmine can reduce the severity of POI in horses, particularly if the large colon is involved. However, its use for impaction colic or in cases with excess GI distention has not been recommended by some authors because of the apparent force of drug-induced contractions.[43] The dosage used clinically is 0.0044 mg/kg (2 mg per adult horse) SC or IV, repeated in 20 to 60 minutes. If there is no response and the horse is not exhibiting any side effects, the amount can be increased by 2-mg increments to a total of 10 mg per treatment. Other dosages have been recommended in a survey of prokinetic use in horses.[51] A side effect commonly seen with this drug is abdominal pain.

Adrenergic Antagonists

Acepromazine maleate is a nonselective α-adrenergic antagonist, and yohimbine, tolazamine, and atipamezole are selective α_2-adrenergic antagonists. Their use as prokinetics is based on the assumption that sympathetic hyperactivity contributes to POI. Afferent stimulation during surgery is thought to activate inhibitory sympathetic efferent neurons. Norepinephrine is released by sympathetic neurons at the enteric ganglia and inhibits the release of the excitatory neurotransmitter acetylcholine by stimulating α_2-receptors located presynaptically on cholinergic neurons. This causes a depression of smooth muscle contraction in the gastrointestinal tract. Elevated serum catecholamines that can last for the duration of POI have been temporally associated with increased synthesis of norepinephrine in the bowel wall in humans after laparotomy.[33,52]

ACEPROMAZINE

Acepromazine facilitates small intestinal transit in normal ponies.[53] On the basis of clinical impression, acepromazine administered at 0.01 mg/kg intramuscularly (IM) intraoperatively and then every 4 hours is thought to reduce the severity of POI in horses with small intestinal lesions. Because acepromazine is a nonselective α-blocker that can produce hypotension through α_1-receptor antagonism, the animal should be well hydrated before the drug is administered.

YOHIMBINE

Yohimbine hydrochloride is a competitive antagonist that is selective for α_2-adrenergic receptors. Yohimbine was shown to antogonize the inhibitory effects of detomidine on jejunal contractions *in vitro* but enhanced the inhibition evoked by xylazine.[54] When administered at 0.15 mg/kg IV at 1, 4, 7, and 10 hours postoperatively in an experimentally induced POI model, it reduced the severity of POI, especially when combined with bethanechol.[45] Yohimbine administered at 0.075 mg/kg was demonstrated to attenuate some of the negative effects that endotoxin has on propulsive motility.[55] Because yohimbine is a selective α_2-adrenergic drug, it should not produce the hypotensive response seen with acepromazine. Neither acepromzaine or yohimbine were reported to be used as prokinetics in a survery of prokinetic use in horses.[51]

TOLAZOLINE AND ATIPAMEZOLE

Tolazoline and atipamezole are selective α_2-adrenergic antagonists that have been proposed as potential prokinetic agents in horses.[54,56] Tolazoline has been shown to increase migrating myoelectrical activity in the duodenum in normal horses, and atipamezole was shown to antagonize the inhibitory effect of xylazine and detomidine on *in vitro* contractile activity of equine jejunal smooth muscle.[54] Although the research data described earlier support the use of these drugs to treat POI in horses, to date there have been no reports of their use in clinical cases in horses.

Benzamides

METOCLOPRAMIDE

Metaclopramide hydrochloride is a first-generation substituted benzamide (contains 5-hydroxytryptamine (5-HT, serotonin) whose prokinetic activity is both through dopamine 1 (DA_1) and 2 (DA_2) receptor antagonism and through 5-HT 4-receptor ($5\text{-}HT_4$) agonism and $5\text{-}HT_3$ receptor antagonism.[34,45,57] Stimulation of DA_2 receptors inhibits the release of acetylcholine, and stimulation of $5\text{-}HT_4$ receptors enhances the release of acetylcholine from the myenteric ganglia.[34] Metoclopramide has been shown to stimulate *in vitro* contractile activity of circular muscle from the stomach and small intestine in the horse.[4] In a POI model in ponies, metoclopramide was more effective in restoring GI coordination than adrenergic antagonists and cholinomimetics used individually or in combination. In horses, metoclopramide is most commonly administered at a dosage of 0.25 mg/kg, diluted in 500 mL of saline, IV over 30 to 60 minutes. In a retrospective study, metoclopramide administered as a continuous infusion (0.04 mg/kg per hour) decreased the total volume, duration, and rate of gastric reflux when used prophylactically after small intestine resection and anastomosis.[58] Metoclopramide may cause extrapyramidal side effects, such as excitement, restlessness, sweating, and abdominal cramping.

CISAPRIDE

Cisapride is a second-generation substituted benzamide, which until recently was the most popular and effective prokinetic in human medicine.[34] Although it has been taken off the human market because of its cardiotoxic effects, it is discussed here because it is still available through some compounding agencies, and drugs with similar mechanisms of action will soon be available.[59] Cisapride functions as an indirect cholinergic stimulant by selectively enhancing the release of acetylcholine from postganglionic neurons in the myenteric plexus.[60,61] Unlike that of metoclopramide, the main prokinetic activity of cisapride appears to be mediated through $5\text{-}HT_4$ receptor agonism and $5\text{-}HT_3$ receptor antagonism and not through dopamine antagonism.[62] In the horse, cisapride has been shown to act partly through a noncholinergic effect mediated by $5\text{-}HT_2$ receptors.[4] In experimental studies and in clinical trials in humans, cisapride's prokinetic effects were consistently equal to or superior to those of metoclopramide and of domperidone, a dopamine antagonist used as a prokinetic in humans.[61,63] In normal horses,

cisapride augments the amplitude of gastric contractions, stimulates jejunal activity coordinated with gastric contractions, enhances contractile activity of the large and small colon, and stimulates coordinated activity in the ileocecocolonic junction.[64] In a clinical trial in horses, cisapride (0.1 mg/kg IM every 8 hours) significantly decreased the incidence of POI.[65] There is also experimental evidence that cisapride can attenuate the motility dysfunction induced by endotoxins.[66]

Cisapride suspended in saline and administered *per rectum* is very poorly absorbed, and suspended in dimethyl sulfoxide (DMSO), it is inconsistently absorbed.[67] Mixing 400 mg of cisapride with 10 mL of tartaric acid and heating at 50° C for 20 minutes, then adding sterile water to bring the volume to 100 mL and mixing for another 20 minutes at 50° C, followed by filtration sterilization, consistently yields a 3 mg/mL solution for IV administration. This should be administered at 0.1 mg/kg every 8 hours. Because cisapride was withdrawn from the market because of a high incidence of cardiac arrhymias, it must be obtained through compounding pharmacies. As a result of this, prokinetics, such as levosulpride with dopamine (D_2) receptor antagonist activity as well as other, newer drugs, mosapride and teagaserod, that act selectively on 5-HT_4 receptors (see later) and drugs, such as levosulapride, that acts on both 5-HT and dopamine (D_2) receptors, are replacing cisapride.[68-70]

MOSAPRIDE

Mosapride citrate is a more recently developed benzamide that acts selectively on 5-HT_4 receptors in the myenteric plexus to stimulate the release of acetylcholine from cholinergic nerves, thus promoting GI motility. In a study in normal horses, mosapride (1.5 to 2 mg/kg PO) promoted motitity in the small intestine and cecum using an electrogastraph to measure percutaneous electrical potential.[71] In an experimental model, mosapride (1.5 mg/kg PO sid for 5 consecutive days) attenuated the decline of small intestinal motility following jejunocecostomy in healthy horses on postoperative days 6 through 31 compared to controls.[69] In a study comparing mosapride, cisapride, metaclopramide, and lidocaine, only mosapride promoted both jejunal and cecal motility in horses.[69] Although the efficacy of mosapride has not been demonstrated in clinical cases at this time, it appears to be a promising prokinetic without the undesirable extrapyramidal side effects of metaclopramide.

TEGASEROD

Tegaserod maleate is another selective 5-HT_4 receptor agonist that has been shown to increase frequency and amplitude of isolated muscle strips of equine ileum and pelvic flexure.[72] Tegaserod (0.02 mg/kg IV bid for 2 days) also accelerated the gastrointestinal transit time of spheres and increased the frequency of defecation and scores of gut sounds in normal horses.[73] In another study looking at the pharmacokinetics of tegaserod in normal horses, a dosage of 0.27 mg/kg PO bid was found to be appropriate to reach therapeutic concentrations.[74] Tegaserod was associated with increased cardiovascular toxicities in humans; consequently the availability of this drug is limited.[75]

Recent work has questioned the use of these selective 5-HT_4 receptor agonists as prokinetics in horses. One group has demonstrated release of serotonin (5-HT) from platelets in compromised bowel and hypothesized it to act as a proinflammatory, vascoconstrictive, and immunomodulatory agent with potential to contribute to POI by interacting with 5-HT_{1a} receptors.[76] The same group also found no functional or immunohistochemical evidence for 5-HT_4 receptors in equine jejunum whereas 5-HT_{1a} receptors were found in both longitudinal and circular muscle,[76] thus questioning the use of selective 5-HT_4 receptor agonists as prokinetics in horses. Additional research is required to assess the efficacy of drugs that interact with specific 5-HT receptors in the treatment of POI in the horse.

Motilin Agonists

ERYTHROMYCIN

Erythromycin lactobionate is a macrolide antibiotic with recognized GI side effects. It can act as a motilin agonist that influences motility partly by acting on motilin receptors on GI smooth muscles. Motilin is a hormone that is released by enterochromaffin cells and stimulates contractile activity in the stomach and small intestine. Erythromycin also acts on enteric cholinergic neurons through motilin and/or 5-HT_3 receptors to stimulate the release of acetylcholine.[34,77] Motilin and erythromycin have been shown to initiate phase III of the migrating motor complex.[78] Motilin infusion in healthy horses causes strong contractions in the proximal jejunum *in vivo*.[79] When administered at subtherapeutic antimicrobial levels, erythromycin has been shown to stimulate gastric emptying, antroduodenal coordination, and phase III activity in the duodenum in humans and laboratory animals.[78]

In vivo, cecal emptying in response to erythromycin (0.1, 1, 10 mg/kg, IV over 60 minutes) was dose dependent.[80] In normal horses, a dosage of 1 mg/kg is effective in stimulating both cecal and small intestinal contractile activity.[80] Dosages higher than 10 mg/kg can potentially disrupt propulsive activity. The dosage that has been used clinically in horses is 0.5 to 1 mg/kg in 1 L of saline infused over 60 minutes, every 6 to 12 hours.[51] The prokinetic response may diminish with repeated treatments. Erythromycin can downregulate motilin receptors, which would explain this desensitization. Additionally, downregulation and decreased production of motilin receptors has been demonstrated in ischemic and distention-traumatized equine jejunum *in vitro*.[81] Although the recommended prokinetic dosage is supposed to be below the effective antimicrobial level, there have been some anecdotal reports of severe colitis associated with its use, making some clinicians reluctant to use the drug as a prokinetic. In support of this clinical impression, an association between low-dose erythromycin (1.25 mg/kg PO every 8 hours) and *Clostridium difficile* colitis was demonstrated in one report.[82] However, in a study on prokinetic use in horses, erythromycin was one of the more frequently used drugs to treat various motility disordes in horses.[51] Erythromycin has been commonly used to treat gastroparesis in humans, but many of the prokinetic therapeutic benefits that were anticipated have failed to materialize. More recently developed motilin agonists are in preclinical trials.[75]

Sodium Channel Blockers

LIDOCAINE

Intravenous administration of the sodium channel blocker lidocaine hydrochloride shortens the duration of paralytic ileus in the colon in humans after abdominal surgery.[76,83] Stimulation of sympathetic inhibitory spinal and prevertebral reflexes as well as influx of inflammatory cells with release of inhibitory

mediators, such as nitric oxide and prostaglandins, are two of the proposed mechanisms involved in the pathogenesis of POI.[33,84-86] Lidocaine may act on several of these mechanisims by (1) reducing the level of circulating catecholamines through inhibition of the sympathoadrenal response, (2) suppressing activity in the primary afferent neurons involved in reflex inhibition of gut motility, (3) stimulating smooth muscles directly, and (4) decreasing inflammation in the bowel wall through inhibition of prostaglandin synthesis, inhibition of granulocyte migration and their release of lysosomal enzymes and cyokines, and inhibition of free radical production.[83]

In an ischemia reperfusion model in horses, intravenous lidocaine reduced plasma prostacyclin E_2 metabolite concentration and mucosal COX-2 expression.[87] In this same study, lidocaine ameliorated flunixin-induced mucosal neutrophil counts. Lidocaine increased contractility of the circular muscle from the proximal duodenum of horses *in vitro* in another study.[4] In a further study, lidocaine-stimulated contractility was more pronounced in jejunal smooth muscle after ischemia reperfusion insult compared to control tissue.[6] These observations suggest that the drug's primary methods of action in reducing POI may be through reduction of inflammation, stabilization of cell permeability, and reduction of pain.

In normal horses, lidocaine did not improve jejunal progressive motility as measured by the duration of migrating myoelectric complexes and spiking activity of the jejunum in one study and delayed transit of feces in another study.[8,88] In clinical trials in horses, lidocaine was effective in decreasing the duration of reflux in horses with POI and in horses with duodenitis–proximal jejunitis.[10] Intraoperative (prophylactic) administration of lidocaine has been shown to decrease the risk of horses developing POI (16 out of 77 [21%] treated prophylactically versus 24 out of 47 [51%] untreated),[17] as well as decreasing severity of POI and significantly enhancing short-term survival in those horses developing POI, with lidocaine treated horses having 3.33-fold higher odds to survive to discharge.[14,17] Lidocaine is reported to be the most commonly used prokinetic in horses with gastrointestinal disease.[17,51]

The recommended protocol requires an initial bolus of 1.3 mg/kg IV administered slowly over 5 minutes, followed by 0.05 mg/kg per minute in saline or lactated Ringer solution over 24 hours to reach a target blood concentration of 1 to 2 μg/mL.[11] Because intraoperative treatment has been shown to decrease the incidence of POI, if a horse is at risk for developing POI (i.e., horses with strangulating small intestinal lesions), prophylactic administration during anesthesia should be considered.[17] Lidocaine CRI should be discontinued 30 minutes prior to the end of surgery to reduce the chance of ataxia during the recovery period and restarted when the horse is stable.[89] It is recommended to administer the initial bolus followed by the continuous infusion each time the medication is stopped for more than 5 to 10 minutes because of the rapid redistribution from the plasma. Side effects include muscle fasciculations, trembling, and ataxia. Moderate to severe hypoproteinemia or reduced cardiac output may increase the risk of these complications. In these horses, lidocaine blood concentration monitoring every 4 to 6 hours should be considered.[10] Although prolonged infusion (96 hours) did not produce any observed adverse effects, accumulation of a potential toxic metabolite was mentioned in one study as a consideration when using lidocaine for extended periods.[11] In a clinical trial, horses that did not stop refluxing in 17.6 hrs and that did not defecate within 16 hours after the initiation of lidcaine CRI were reported to have a poor response rate and the authors stated that alternative therapy should be considered in these cases.[10] In another study, the authors recommended reducing the infusion concentration by 50% for 24 hours before discontinuing treatment in horses that have responded to lidocaine.[17]

Anti-Inflammatory and Analgesic Therapy

A large body of evidence has accumulated to indicate that motility disorders such as POI and ileus associated with enteritis or colitis are, in part, mediated by inflammatory cell infiltrates into the intestinal wall.[84-86] Intestinal manipulation, lipopolysaccharide administration, and ischemia-reperfusion injury have all been shown to produce significant decreases in *in vitro* contractile activity of jejunal smooth muscle. In experimental animals, blocking this influx of leukocytes by antiadhesion antibodies eliminates the disruption of motility, supporting a cause-and-effect relationship between intestinal inflammation and POI.[85] Intestinal distention, ischemia, and trauma inflicted during decompression or during resection and anastomosis induce inflammation in the bowel wall, with production of inflammatory mediators, such as prostacyclin, PGE_2, and TNF. Endotoxemia associated with necrotic intestine also stimulates production of inflammatory mediators. Infusions of endotoxin, PGE_2, and TNF have been shown to disrupt normal motility.[55,90] Both phenylbutazone and flunixin meglumine were shown to significantly attenuate the disruption of gastric, small intestine, and large colon motility elicited by endotoxin infusion.[90] Administration of these nonsteroidal drugs decreases the production of inflammatory mediators that directly inhibit smooth muscle contractility, while reducing postoperative pain and thus attenuating potential inhibitory sympathetic reflexes. On the basis of these observations, nonsteroidal anti-inflammatory drugs (NSAIDs) are recommended for prevention and treatment of motility disorders associated with GI inflammation. As mentioned previously, lidocaine has been shown to decrease the inflammatory response in ischemia models in horses and improve jejunal smooth muscle contractility, indicating lidocaine may attenuate POI through its anti-inflammatory activity.[6,87]

Adrenergic agonists are another class of drugs commonly used in horses to control pain associated with colic. Xylazine and detomidine are α_2-adrenergic agonists that may inhibit presynaptic acetylcholine release in the myenteric plexus, resulting in a decrease in motility.[91] In the horse, xylazine and detomidine have been shown to decrease motility as measured by myoelectrical activity of the distal jejunum, pelvic flexure, cecum, and right ventral colon and to decrease cecal emptying based on radiolabeled markers.[43,48,92-94] However, in another study, xylazine was not thought to seriously disrupt gastrointestinal motility.[56] Because pain stimulates the sympathetic nervous system, which has an inhibitory effect on GI smooth muscle, the potential detrimental effects of adrenergic agonists must be weighed against their benficial effects.

Opioids are occasionally used to control pain in horses. Opioid agonists should be avoided if possible, as they have been shown to depress motility in most species studied.[93,95,96] *Naloxone,* an opioid antagonist, has been suggested by some investigators to be a potentially beneficial drug for treating motility disorders.[64] There is some evidence that naloxone may enhance progressive motility in the horse; however, it has also

been shown to cause diarrhea and colic in the same species.[97] N-*methylnaltrexone*, another opioid antagonist, has been shown *in vitro* to directly stimulate equine jejunal muscle strips as well as attenuate *in vivo* the adverse effects of morphine (0.5 mg/kg, IV bid) when administered simultaneously at a dose of 0.75 mg/kg, IV bid as indicated by increasing defecation frequency fecal weight and by preventing increases in intestinal transit time.[98,99] *Butorphanol tartrate*, an opioid agonist-antagonist, has been shown to inhibit myoelectrical activity in the jejunum, whereas it has no effect on pelvic flexure activity.[100] Butorphanol prolonged xylazine-induced inhibition of myoelectrical activity of the cecum and right ventral colon in one study, but it produced no undesirable effect on antroduodenal activity in another study.[56,94]

Prognosis

In spite of a recent report, in our opinion with support from most studies, the prognosis for resolution of POI is favorable.[13,16-18] Although this discussion has been limited to pharmacologic interventions, good surgical technique and appropriate aftercare, including fluid and electrolyte replacement, antibiotic and analgesic therapy, and nasogastric decompression, are critical to the outcome of these cases. Because of the intensive postoperative medical management of these horses, successful treatment is often costly. Frequently, the limiting factor in determining the outcome is the economic constraint imposed by the owner.

ADHESIONS

Peritoneal adhesions are one of the most common complications seen after colic surgery in horses. Adhesions occur more frequently after surgery for strangulating lesions involving the small intestine compared to ischemic lesions of the colon and nonstrangulating lesions of the intestine.[29] Adhesions causing postoperative signs of pain and intestinal obstruction can occur in up to 22% of horses previously having surgery for small intestinal disease.[101-103] In a retrospective study of horses requiring repeat laparotomy after surgery for acute abdominal disease (10.1%; 113 out of 1014 cases), adhesions were the most common reason for repeat laparotomy, occuring in 28% of cases.[29] Foals seem to be more predisposed to development of adhesions than adults.[104]

Adhesion formation can be viewed as a variant of the normal physiologic healing process. An intact mesothelial cell layer is critical to the prevention of adhesion formation. Ischemia, distention, drying, or abrasion of the peritoneum during manipulation and decompression of the intestine, as well as hemorrhage, the introduction of foreign material, or infection in the peritoneal cavity, can all result in peritoneal inflammation initiating adhesion formation.[105] When the mesothelial cells are disrupted, the underlying connective tissue containing blood vessels, collagen, lymphocytes, fibroblasts, mast cells, macrophages, and plasma cells becomes exposed. The release of vasoactive substances, such as PGE_2, serotonin, bradykinin, and histamine from the exposed submesothelial tissue mediates increased vascular permeability with extravasation of a fibrinogen-rich inflammatory exudate. The release of thromboplastin (tissue factor) and exposure of subendothelial collagen activate both the intrinsic and the extrinsic clotting cascade, leading ultimately to thrombin-mediated conversion of fibrinogen to fibrin, with the fibrin adhering to sites of injury. In the normal healing process, the fibrin tags are lysed by plasmin, and the peritoneal injury is covered within 2 to 5 days by a single layer of mesothelial cells originating from metaplasia of underlying mesenchymal cells, attachment of free-floating mesothelial cells, or transformation of macrophages.[106] However, if there is inadequate fibrinolysis, fibroblasts migrate over the fibrin scaffold with neovascularization and produce collagen by day 4, initiating the formation of fibrous adhesions.[107]

Tissue plasminogen activators are proteases released by mesothelial and endothelial cells that convert inactive plasminogen to plasmin, an important part of the fibrinolysis pathway. After peritoneal injury from mechanical trauma, ischemia, inflammation, or bacterial infection, plasminogen activator activity is decreased. This was thought to occur because of damage to the cells producing plasminogen activator.[108] A more recent finding suggests that there is an increased production of plasminogen activator inhibitor, which reduces the level of plasminogen activator activity.[105,109,110] Extensive damage to either the parietal or the visceral peritoneal surface tips the balance in favor of adhesion formation, first by increasing the extent of fibrin formation and deposition and second by decreasing fibrinolytic activity. Undoubtedly, many other inflammatory mediators are involved in regulating the balance between deposition and lysis of fibrin. Recently, TGF-β was found to be increased in peritoneal fluid in horses with peritonitis and in horses with ischemic lesions of the intestine. Plasma and peritoneal TGF-β concentrations were higher in horses that had colic and did not survive than in survivors.[111] The authors speculated that increased concentration of TGF-β may influence adhesion formation by stimulating synthesis and deposition of extracellular matrix.

Several strategies are currently being used to prevent or minimize adhesion formation. The most important one involves minimizing trauma by paying attention to good surgical technique, such as keeping the bowel moist at all times, providing adequate hemostasis, leaving as little suture material as possible exposed, removing as much damaged tissue as possible, and handling the tissue as gently as possible. Consistent with the hypothesis that surgical trauma is an important stiumulus for adhesion formation, adhesions were not found to be assoicated with the site of the primary lesion or the site of resection in one study.[29] Trauma is also reduced by making the decision to perform surgery in a timely manner. Both ischemia-reperfusion and intraluminal distention have been shown to cause severe changes in the seromuscular layer of the small intestine, such as serosal edema, leukocyte infiltration, and erythrocyte leakage with fibrin accumulation, whereas a similar insult to the ascending colon does not result in comparable seromuscular lesions.[102,112] Prophylactic strategies include intervention in the inflammation, coagulation, and fibrinolysis cascades with anti-inflammatory or heparin therapy, and using therapies that provide mechanical separation of the injured areas.

Perioperative NSAIDs and Antibiotics

Perioperative NSAIDs and antibiotics are used routinely during GI surgery. Because both inflammation and infection may predispose to adhesion formation, this combination of chemotherapy should curtail the production of adhesions. Broad-spectrum antibiotics, potassium penicillin

(22,000 IU/kg IV every 6 hours) and gentamcin (6.6 mg/kg IV sid), should be administered prophylactically or therapeutically, depending on the degree of compromise to the affected segment of bowel. Flunixin meglumine (1.1 mg/kg IV bid) is continued after surgery for 3 to 5 days to reduce the inflammatory response. DMSO (20 mg/kg IV twice a day for 72 hours) may also reduce inflammation. There is experimental evidence that this combination of anti-inflammatory and antimicrobial therapy is effective in reducing adhesion formation in horses.[113,114] In an experimental study of ischemia-induced adhesions in foals, administration of flunixin meglumine and antibiotics or administration of DMSO appeared to be more effective than 3% sodium carboxymethylcellulose or heparin in preventing the formation of adhesions.[114] It is interesting to note that a 3% solution was used in this study, compared with the 1% solution in studies discussed later in this chapter.

Intravenous Lidocaine

As mentioned previously (see "Sodium Channel Blockers/Lidocaine," earlier), intravenous lidocaine reduced plasma prostacyclin E_2 metabolite concentration and mucosal COX-2 expression in an *in vivo* ischemia-reperfusion model in horses. Flunixine was shown to induce an increase in mucosal neutrophil counts. Lidocaine ameliorated the neutrophil response.[87] This would suggest that intravenous lidocaine could potentially reduce adhesions through anti-inflammatory activity. However, in an *in vivo* study, lidocaine did not inhibit neutrophil migration or adhesion formation at therapeutic concentrations, but it increased neutrophil migration and adhesion formation at higher concentrations.[115]

Heparin

Heparin is composed of a family of naturally occurring sulfated glycosaminoglycans that are synthesized by connective tissue mast cells.[116,117] By binding to antithrombin (AT), a glycoprotein synthesized in the liver and vascular endothelium, heparin markedly enhances the rate of AT-mediated inactivation of clotting factors (factors IX, X, XI, and XII and thrombin).[118,119] The AT-heparin complex, by neutralizing thrombin, suppresses thrombin-mediated amplification of the coagulation cascade and inhibits thrombin-mediated conversion of fibrinogen to fibrin.[119] Heparin sodium (40 IU/kg IV intraoperatively, followed by 40 IU/kg SC twice a day for 2 days) decreased adhesion formation in ponies after experimentally induced intestinal ischemia.[103] Based on pharmacokinetic studies using calcium heparin instead of sodium heparin, an initial dose of 150 IU/kg SC followed by 125 IU/kg SC twice a day for six doses followed by 100 IU/kg SC twice a day is recommended to maintain therapeutic anticoagulant levels.[120] Some evidence suggests that calcium ions inhibit the heparin-AT-thrombin reaction. Consequently, a higher dosage of calcium heparin than sodium heparin is required to reach therapeutic levels, although this finding is questioned.[121,122] Plasma AT levels are reported to be decreased in some horses with abdominal disease.[123,124] Because heparin acts as a cofactor to AT, heparin therapy would be of limited value if peritoneal AT were depleted significantly. However, changes in plasma AT levels do not always correlate with changes in peritoneal levels.[125] Peritoneal AT levels have actually been found to be increased in some horses with colic.[126] The most common complication reported with heparin therapy in horses is anemia induced by erythrocyte agglutination, which resolves in 3 to 4 days following cessation of heparin.[127]

Low-molecular-weight heparin (LMWH) is separated from unfractionated heparin (UFH) by solvent or gel filtration and consists of smaller molecules than UFH. It has less antithrombin activity and higher anti–factor Xa activity than UFH. In addition, LMWH has constant anticoagulant activities, greater bioavailability, and longer biological half-life.[128] In horses, LMWH causes fewer side effects and can be conveniently administered once daily.[129,130] When administered to horses with surgical colic, there were fewer jugular vein changes and fewer side effects in horses treated with LMWH compared to UFH.[131] When administered to horses following colic surgery, horses treated with LMWH had a lower prevalence and grade of laminitis compared to control horses where heparin was not administered.[132] Dalteparin can be administered subcutaneously at 50 IU/kg every 24 hours.[130]

Intraperitoneal Sodium Carboxymethylcellulose (SCMC)

Intraperitoneal SCMC is effective in preventing adhesions in experimental models using laboratory animals, and there is evidence to support its efficacy in preventing experimentally induced adhesions in horses.[133,134] The proposed mechanism of action is the "siliconizing" effect that SCMC has on the bowel. This mechanical lubricating barrier minimizes the duration of direct apposition of traumatized tissue, preventing the establishment of fibrin tags between two surfaces, although the effect is most likely transient, because the SCMC is cleared rapidly from the abdomen. A 1% solution of SCMC (7 mL/kg body weight) is infused into the peritoneal cavity through a sterile stallion catheter before tying the last suture in the linea alba,[134] or, preferably, before manipulating the bowel. In several experimentally induced adhesion studies, horses treated with 1% SCMC had reduced frequency of intra-abdominal adhesions compared with controls.[135,136] In one retrospective study, no adverse effects on abdominal incisional wound healing were observed in horses treated with SCMC, however no significant differences were found between treated and control goups for postoperative colic or long term survival.[137] In another study, intraoperative SCMC prolonged survival of horses with small intestinal disease, particularly in horses that had postoperative colic or POI, although interpretation of those results has been questioned.[138,139] The intraoperative use of SCMC can result in visible intracellular accumulation of pink or magenta colored material on cytological examination of peritoneal fluid macrophages if an abdominocentesis is performed postoperatively.[140]

Sodium Hyaluronate (HA)

Sodium hyaluronate (HA) has also been used by itself and in conjunction with SCMC to reduce adhesions. Application of a 0.4% HA solution to the serosal surface of the jejunum significantly decreased the incidence of experimentally induced intra-abdominal adhesion formation in ponies, whereas SCMC did not significantly reduce adhesions.[141] A bio-resorbable hyaluronate-carboxymethylcellulose membrane that is applied over an anastomosis site or an area of localized trauma has also been shown to be beneficial in reducing adhesion formation.[142-144] Recent data indicating that adhesion formation is often not

associated with the site of the primary lesion or with the anastomosis would suggest limitations on the use of a bio-resorbable membrane for prevention of adhesion.[29] Bio-resorbable protective membranes could be used in conjunction with intraperitoneal SCMC to provide a more diffuse protective coating.

Intraperitoneal Crystalloid Solutions

Intraperitoneal instillation of *crystalloid solutions* is often used to prevent adhesion formation, but conflicting results have been reported regarding their efficacy.[107] The proposed mechanism is hydroflotation of the abdominal organs. However, large volumes of fluid in the peritoneal cavity may compromise the host's ability to fight infection by interfering with opsonization and phagocytosis.[145]

Peritoneal Lavage

An alternative approach is the use of peritoneal lavage, which should remove fibrin and other inflammatory mediators while providing mechanical separation of the bowel at the time of lavage. Standing postoperative peritoneal lavage significantly reduced the number of adhesions in horses that received two peritoneal lavages for 2 days postoperatively with 10 L of lactated Ringer solution delivered through a 32-French chest trocar inserted in the abdomen during surgery.[146] Another technique for abdominal drainage is through the use of a closed suction intra-abdominal drain with intermittent peritoneal lavage. This technique has been associated with a high incidence of minor complications, such as partial obstruction of the drain (26%), leakage of fluid around the drain (16%), and subcutaneous fluid accumulation (12%).[147]

SURGICAL COMPLICATIONS

Significant complications, such as hemoperitoneum or peritonitis, may result from surgical procedures. Abdominal surgical procedures must adhere to basic principles of good surgical practice (after Halsted), including accurate homeostasis, gentle tissue handling, approximation of tissue, obliteration of dead space, minimal contamination, accurate ligation, and minimal foreign material. Postoperative hemorrhage can result from ligature slippage or from mucosal hemorrhage following intestinal resection and anastomosis. Fatal hemorrhage resulting from tearing of the portal vein or, less commonly, the caudal vena cava has been reported after manual reduction of epiploic foramen entrapment.[148-150] Therefore, reduction of an epiploic foramen entrapment should be performed with one hand on each side of the foramen and gentle feeding of the entrapped bowel through the foramen in a horizontal direction, because pulling the bowel in an upward direction may lead to tearing of the previously mentioned major vessels. Hemorrhage has also been reported as a complication of enterotomies, particularly those performed in the large colon. Therefore, in these locations, a full-thickness suture pattern may be indicated as a first layer.[151]

Administration of whole blood may be necessary if signs of hemorrhagic shock develop (increased heart rate, pale mucous membranes, weakness, and collapse). Fresh blood collected in plastic bags to preserve platelet activity should be given, as stored blood requires several hours to restore RBC oxygen-carrying capacity. Because signs of hemorrhagic shock develop after loss of 25% to 30% of blood volume, transfusion of 6 to 8L of whole blood may be required (see Chapter 4).

Complications have been reported that are unique to specific surgical techniques. Small intestinal volvulus may develop after side-to-side anastomosis in ponies. Intussusceptions have been reported after small intestinal surgeries. Jejunojejunal intussusception was reported after a stapled functional end-to-end anastomosis in two pony mares and in two ponies after inverting closure of small intestinal transverse enterotomies, suggesting that excessive cuff formation should be avoided during small intestinal closure.[152,153] Chronic intermittent colic with small intestinal distention can occur after functional end-to-end jejunojejunostomies. Significantly dilated anastomoses found on re-laparotomy may hinder the passage of the peristaltic wave and are related to excessive length of the anastomosis.

Internal herniation is a possible complication of small intestinal resection, and care should be taken to obliterate all possible internal spaces. Closure of the ileocecal fold after jejunocecostomy, mesenteric closure after small intestinal resection or ileocolostomy, and closure of potential spaces after intestinal bypass are recommended to avoid this problem.[154,155]

Extensive small intestinal resection (greater than 60%) has been associated with malabsorption, diarrhea, weight loss, and liver damage in ponies.[156] In a separate study, a short-interval feeding program (pelleted ration divided into 8 feedings per day) prevented the development of a clinical malabsorptive syndrome after 70% of small intestinal resections in ponies.[157]

Jejunocecostomy procedures are reported to have an increased risk for postoperative complications. An ileocecocolic intussusception was reported after jejunocecostomy in one horse.[155] Alterations in calcium absorption and fat absorption and microbial derangements in normal flora have been suggested as potential complications. Attaching the jejunum as close to the base of the cecum as possible decreases backflow pressure, discouraging fluid ingesta in the cecum from traveling orally back into the jejunum.

Ileal impactions may recur in the immediate postoperative period, particularly if a bypass procedure was not performed. Horses need to be fed carefully, using a laxative diet, to avoid this complication.

There is a 15% recurrence rate reported for large colon displacements and torsion. Factors contributing to recurrence have not been thoroughly identified. Recurrence of large colon displacements can be prevented by colopexy and large colon resection. Colopexy of the large colon has been reserved for nonperformance animals because of reported intestinal rupture after colopexy in athletes.[158] Extensive large colon resection is associated with short-term decreased absorption of water, decreased absorption of phosphorus, and weight loss.[159] Horses that have undergone large colon resection therefore have increased requirements of water, phosphorus, and better-quality protein.

Recurrence of small colon impaction is also a problem. Surgical evacuation of the large colon is recommended after surgical correction of small colon impactions. These horses should be managed with a laxative diet in the immediate postoperative period.

Management of postoperative complications may require repeat laparotomy. Clinical signs indicating the need for repeat laparotomy include persistent abdominal pain despite successful gastric decompression, marked and progressive intestinal distention, systemic deterioration associated with degenerative changes

in the abdominocentesis, and presence of bacteria. Repeat laparotomies have been reported in 8% to 10% of horses undergoing a celiotomy for colic.[29,160,161] The long-term outcome of horses subjected to a second surgery was significantly worse than that of horses undergoing a single surgery. In one study, the short-term survival rate was 50% and the long-term rate was only 22%. Nearly 40% of horses surviving repeat laparotomy developed acute episodes of colic that necessitated further surgery.[160]

SEPTIC PERITONITIS

Septic peritonitis is one of the most common fatal postoperative complications following exploratory laparotomy in horses (other common fatal complications are adhesions and ileus).[162,163] In one study, 56% of horses with septic postoperative peritonitis did not survive.[164] Postoperative peritonitis may result from necrosis of a segment of the GI tract before or after surgical intervention, contamination occurring at surgery, or leakage from the anastomosis or enterotomy site. In response to bacterial contamination, peritoneal macrophages and mast cells release histamine and prostaglandins, leading to vasodilatation and increased permeability of the peritoneal vessels. The subsequent peritoneal exudate contains fibrin, complement, immunoglobulins, and clotting factors. Chemotaxins (such as IL-1, leukotriene B_4, and TNF) produced by activated macrophages, mast cells, and neutrophils stimulate a further influx of neutrophils, exacerbating the inflammatory response.[165,166] Bacteria and other particles can be cleared from the peritoneal cavity through the diaphragmatic lymphatics. Movement of the diaphragm during respiration facilitates the uptake of peritoneal fluid into stomata, which represent intercellular gaps between mesothelial cells covering the diaphragm. The stomata communicate with lymphatic lacunae, which drain peritoneal fluid into the substernal lymph nodes and subsequently via the thoracic duct into the bloodstream. Systemic effects from peritonitis may be initiated by the entrance of bacteria into the bloodstream, by the action of inflammatory mediators produced by macrophages and neutrophils, or by absorption of endotoxin from the peritoneal cavity into the portal venous system, which then acts on the Kupffer cells of the liver, contributing to the septic response.[167]

Clinical signs commonly associated with peritonitis include mild colic, depression, anorexia, ileus, diarrhea, fever, tachycardia, and tachypnea.[164,165,168] Besides relying on clinical signs, the diagnosis of peritonitis is based on peritoneal fluid analysis.[169] Peritoneal fluid from normal horses has a nucleated cell count of less than 5000 cells/mL and a protein concentration of less than 2.5 g/dL. However, after an exploratory laparotomy, the peritoneal nucleated cell count, percentage of neutrophils, fibrinogen, and protein concentration become markedly elevated.[160,164] The mean nucleated cell count peaks at approximately 200,000 cells/mL at day 4 and remains elevated at 40,000 cells/mL through day 6; the mean protein concentration remains elevated and peaks at 6 g/dL on day 6 after an exploratory laparotomy. Therefore, absolute values and differential counts may not provide sufficient information on which to base a diagnosis of postoperative septic peritonitis. Hence, cytologic evaluation of the peritoneal fluid is crucial for a definitive diagnosis. Toxic or degenerative changes in neutrophils and the presence of intracellular or extracellular bacteria indicate septic peritonitis. Peritoneal pH and peripheral and peritoneal glucose may also be evaluated to diagnose septic peritonitis. A serum–peritoneal glucose difference greater than 50 mg/dL or a peritoneal pH less than 7.2 with a peritoneal glucose of less than 30 mg/dL is highly suggestive of septic peritonitis.[170] An increased abdominal fluid lactate compared to peripheral blood is also an indicator of septic peritonitis.[171] Peripheral blood values determined by complete blood count and fibrinogen should be compared with peritoneal fluid. A left shift, thrombocytopenia, or hypoproteinemia also indicates septic peritonitis.[167] Peritoneal fluid should be submitted for both aerobic and anaerobic cultures and sensitivity.

Treatment should be directed first toward stabilization of the animal and then toward the inciting cause. Intravenous fluids should be administered to correct dehydration and acid-base and electrolyte abnormalities. Patients are often hypoproteinemic (total protein less than 4.4 g/dL) and require plasma. If sepsis is caused by gram-negative bacteria, administration of hyperimmune serum may attenuate the degree of endotoxemia. Flunixin meglumine (0.25 mg/kg IV 3 times a day) attenuates symptoms of endotoxemia. Broad-spectrum IV antibiotics are imperative. The most common organisms isolated from horses with peritonitis include the aerobic bacteria *Escherichia coli*, *Staphylococcus* spp, *Streptococcus* spp, and *Rhodococcus equi* and the anaerobic species *Bacteroides, Clostridium,* and *Fusobacterium*.[164,167,168] If necrotic intestine or leakage of an anastomosis or enterotomy site is considered to be the cause of septic peritonitis, a second exploratory laparotomy is indicated.

Open peritoneal drainage has been described for the treatment of mild inflammatory peritonitis in an experimental model in horses, but the efficacy of this technique in clinical septic peritonitis is not known.[172] However, peritoneal lavage is an important component of the treatment protocol. Placement of an abdominal lavage system in the animal at the time of surgery is indicated if the animal is at risk for developing peritonitis (i.e., with a necrotic intestine or significant contamination at surgery). Enterotomies performed in the large and small colon to remove enteroliths do not significantly increase the risk of the horse's developing septic peritonitis, and a drain is not necessary. A closed fenestrated drain system allows infusion of fluid, and the suction drain system (Snyder Hemovac) provides a continuous active evacuation of residual fluid. Alternatively, a 32-Fr thoracic drain (Pleur-Evac Thoracic Catheter) can be placed, to which additional fenestrations are made to improve drainage. Whenever possible, the omentum should be removed during the surgery, to prevent occlusion of the drain during postoperative lavage. The drains may also be placed in a standing, sedated animal. Approximately 5 to 10 L of lactated Ringer is infused every 6 hours. After infusion of the fluid, the horse is walked for 10 minutes to help circulate the fluid before draining the infusate. Heparin is often added to the lavage solution (5000 IU) or administered systemically. Heparin decreases peritoneal fibrin formation to give the antimicrobial agents better access to the bacteria.[166] The lavage is continued until there is a decrease in the nucleated cell count and protein concentration, an increase in pH and glucose of the peritoneal fluid, and improvement in the cytologic appearance of the cells. By evacuating bacteria, cellular debris, fibrinogen, fibrin, and inflammatory mediators, this technique reduces the systemic effects of peritonitis, helps to resolve the infection, and decreases the incidence of adhesion formation, one of the complications of septic peritonitis.[147,173]

The prognosis for horses with septic peritonitis depends on the extent of the insult and how quickly the initiating cause is

TABLE 40-1. Postoperative Monitoring of Horses After Abdominal Surgery

		Monitoring				
			Hemodynamics			
Surgical Findings	Possible Problems	Exam	TPR	Lab Work	Reflux	Fluids
Small Intestine						
Simple obstruction	Ileus	q 1 hr while on fluids	q 4 hr	PCV/TP q 6 hr until no reflux; monitor electrolyes and creatinine daily until no reflux	q 4 hr	Until no reflux
Anterior enteritis	Ileus, laminitis	q 1 hr while on fluids	q 4 hr	PCV/TP q 6 hr until no reflux; monitor electrolytes and creatinine daily until no reflux	q 4 hr	Until no reflux
Strangulating obstruction	Ileus, endotoxemia, adhesions	q 1 hr	q 2-4 hr	PCV/TP q 6 hr until no reflux; monitor electrolytes and creatinine daily until no reflux	q 2-4 hr	Until cardiovascular status is stable and no reflux
Large Intestine						
Simple obstruction	Postoperative diarrhea	q 4 hr	q 4 hr	If febrile: CBC, monitor for leukopenia	No	Correct preoperative dehydration
Strangulating obstruction	Endotoxemia: dehydration, protein loss; diarrhea; laminitis	q 1 hr	q 4 hr	PCV/TP q 6 hr until no reflux; monitor electrolytes and creatinine daily until no reflux; monitor rise in PCV with protein loss	No	Until cardiovascular status stable

*Preoperative antibiotics are broad spectrum: penicillin and gentamicin. Add metronidazole if significant intraoperative contamination.
†2.2 mg/kg IV over 60 min q 6 hr.
‡Loading dose: 1.3 mg/kg IV slowly over 5 min; maintenance: 0.05 mg/kg/min infusion IV.
§0.0044 mg/kg (2 mg per adult horse) SC or IV.
¶For ileal impaction, feed a laxative diet and limit intake for 4 to 5 days.
**For cecal or large colon impaction, feed a laxative diet, give mineral oil, and monitor for reimpaction.
††For large colon resection, increase water intake and feed alfalfa hay.
CBC, Complete blood count; *IV,* intravenously; *PCV/TP,* packed cell volume/total protein; *SC,* subcutaneously; *TPR,* temperature, pulse, respiration.

corrected. Horses that have extensive contamination because of leakage of the anastomosis or necrosis of devitalized bowel that was not removed during the initial surgery are likely to have a guarded prognosis.[163] Horses with less-severe contamination often respond favorably to the preceding therapeutic protocol, although the cost of postoperative management is substantially increased.

INCISIONAL COMPLICATIONS

Incisional complications after exploratory celiotomy in horses include infection, dehiscence, suture sinus formation, and herniation.

Incisional Infections

The frequency of incisional infections in horses after colic surgery is 10% to 37%.[163,174,175] A frequency of 28.6% was reported after one colic surgery, and 87.5% after two or more celiotomies.[175] Predisposing factors for incisional complications include repeat laparotomy, increased duration of surgery, use of a near-far-far-near pattern, use of chromic catgut, leukopenia, incisional edema, postoperative pain, weight (greater than 300 kg), and older age (older than 1 year).[175-177] In one study, the use of staples for skin closure and surgical site closure by less-experienced surgeons increased the risk for incisional infection.[178] The use of an antibacterial-coated suture did not decrease the likelihood of incisional complications.[179] The use of an abdominal bandage decreased the prevalence of postoperative incisional complications in one study.[180] Incisional hernias are reported in 13% to 16% of horses after celiotomy. The single most important risk factor for herniation is incisional infection, which had a relative risk of 17.8 for causing herniation.[176]

Horses developing incisional infections are initially febrile without any localizing signs. Excessive tenderness and edema are sometimes noted. Most incisional infections develop 3 days or more after surgery, and drainage can sometimes be delayed up to 14 days. Systemic antibiotics delay the onset of drainage. After drainage is noted, it is important to provide a route for exudation by removing a few staples or sutures at the site of the drainage. Culturing of the drainage identifies potential hospital problems. Horses that are afebrile after drainage has been established and in which excessive edema or cellulitis is not evident do not require systemic antibiotics. However, fever and significant tissue reaction are indications for antibiotic therapy. Flushing of the wound should be performed with caution, because

Drugs				Feeding		
NSAIDs	Antibiotics*	Premotility Agents	Other	Time	Type	Frequency
Small Intestine						
Flunixin meglumine 0.25 mg/kg IV tid	Perioperative (24 hr)	Erythromycin† Lidocaine‡		After 2 hr no reflux¶	Wet hay or grass	q 4 hr
Flunixin meglumine 0.25 mg/kg IV tid		Erythromycin† Lidocaine‡	Prevention of laminitis; acepromazine, nitroglycerin	After 12 hr no reflux	Wet hay or grass	q 4 hr
Flunixin meglumine 0.25 mg/kg IV tid	Until 24 hr afebrile	Erythromycin† Lidocaine‡	Plasma, hyperimmune serum, polymyxin B Prevention of laminitis: acepromazine, nitroglycerin	After 12 hr no reflux	Wet hay or grass	q 4 hr
Large Intestine						
Flunixin meglumine 0.25 mg/kg IV tid	Until 24 hr afebrile	Erythromycin† Neostigmine§		At 12 hr**	Grass, hay	q 4 hr
Flunixin meglumine 1.1 mg/kg IV bid, then reduce to 0.25 mg/kg tid	Until 24 hr afebrile	Erythromycin† Neostigmine§	Plasma, hyperimmune serum, polymyxin B Prevention of laminitis: acepromazine, nitroglycerin	When no large colon distention‡	Grass, hay††	q 4 hr

it can propagate the infection along the entire incision line. An abdominal support should be provided, because dehiscence may occur.

Incisional infection rates can be decreased by shortening surgery time, using adequate draping, isolating any enterotomy incision from the clean surgical field, and minimizing trauma to the incision during exploration of the abdomen. A plastic ring drape can be used to protect the incision and is preferred by some surgeons for all exploratory surgeries. Bacterial culturing of the wound does not have predictive value for the incisional infection rate.[181] Closure of the linea alba should be performed with a suture material that is minimally reactive. Chromic gut has been associated with increased incisional drainage in horses, and braided nonabsorbable suture may cause suture sinus formation. Suture patterns do not particularly influence incisional infection rates, although in one study, a near-far-far-near pattern was associated with increased postoperative drainage.[175] Care should be taken, however, not to take overly large bites and to avoid excessive force when tightening sutures; this can lead to ischemia, predisposing to incisional infection. In one study in human abdominal closure using a single-layer running suture, there was a significant decrease in wound complications when a ratio of suture length to wound length of at least 4 was achieved by using a short stitch length rather than a long one.[182]

If incisional infection is present, or in cases of repeat laparotomy in the presence of an infected incision, the wound can be apposed with large stainless steel tension-releasing sutures, placed distant from the incision, which help support the abdominal wall (see Chapter 39). The linea alba is closed, and the subcutaneous tissues and skin can be left open for drainage.[183]

The reported rate of acute incisional dehiscence is low (2% to 2.9%), but this is the most serious acute incisional problem and can be fatal. Violent recovery, severe postoperative abdominal pain, prolonged surgery time, and use of a continuous suture pattern in the linea alba have been associated with early postoperative dehiscence.[175,184] Acute incisional dehiscence is best managed by immediate repair under general anesthesia if the intestine has not been severely traumatized.[185] For details on repair of acute total incisional dehiscence, see Chapter 39.

Incisional Herniation

Incisional herniation is the most significant problem resulting from incisional infection. Incisional hernias do not necessarily require repair if they are small and do not cause deformation

in the body wall. Large hernias that protrude from the abdomen require repair for cosmetic reasons but also because they are subject to repeated trauma. Hernias must be repaired only after the incision is healed and free of infection and the hernial ring has gained strength. This usually requires a minimum of 3 months after the original surgery. No skin abrasions, open wound, or drainage should be present at the time of hernia repair, because these may contaminate the surgical site, which could have serious implications, especially if an implant is used to repair the hernia. In preparation for surgery, the horse should be fasted to decrease abdominal weight. If the hernia is large, a complete pelleted feed can be substituted for hay 2 weeks before surgery to further decrease abdominal contents. For a description of surgical procedures used for incisional hernia repair, see Chapter 39.

A postsurgical abdominal wrap has been advocated for the management of incisional hernias (CM Heal). These abdominal bandages provide firm, even abdominal support, have a special pocket designed to apply direct pressure over the hernia site, and are available in different sizes. Anecdotal reports and our experience are favorable for healing of incisional hernias without surgical intervention. An additional benefit is the ability to start the horse on an exercise program while the abdominal support is in place. Additional indications include the need for additional support after surgery in advanced pregnancies, and correction of umbilical hernias in foals.

POSTOPERATIVE MONITORING

Principles of monitoring horses after abdominal surgery are based on restoration of circulating fluid volume, electrolyte and acid-base equilibrium, and GI function. Table 40-1 summarizes the care of horses after abdominal surgery based on surgical findings. However, these are only guidelines, and therapy should be adjusted to individual cases based on physical and laboratory examination.

As a rule, horses on IV fluid therapy should be observed hourly and their hydration status evaluated every 6 hours by PCV and total protein determination. Horses that had significant bowel damage or ileus should be examined (including body temperature, pulse, respiration rate) every 4 hours until they have stabilized. Creatinine should be monitored daily until values are within the normal range. Postoperative fever is common within the first 24 hours of surgery but should resolve thereafter. A persistent fever should be investigated (impending colitis, peritonitis, catheter sepsis). Small intestinal diseases have a high incidence of postoperative ileus, whereas diarrhea more commonly follows large intestinal disorders. Laminitis is a risk for all horses but is more likely to occur if significant bowel damage was present. For all horses that are at risk for the development of endotoxemia, hyperimmune serum or polymyxin B is administered to bind endotoxin. For all horses that are at risk for the development of laminitis, cooling of the feet with ice boots (therapeutic hypothermia) may be indicated.[186]

Perioperative antimicrobial agents should be of the broad-spectrum type and should have minimal effects on GI flora; an example would be procaine penicillin combined with gentamicin. Perioperative antibiotic therapy is applied in procedures in which the bowel lumen was not entered, and it continues until the horse is afebrile for 24 hours and all systemic parameters have improved after enterotomies or resection and anastomosis procedures. Objective criteria that can be used for the discontinuation of systemic antibiotics include a normal white blood cell count without bands, normal temperature, absence of abdominal discomfort, and return of normal appetite.

Analgesic therapy is indicated after celiotomies. Flunixin meglumine is recommended for a minimum of 24 hours and then as needed, depending on the horse's systemic condition and comfort level.

In small intestinal disorders, feeding is resumed in small amounts after 12 hours if the horse has not developed reflux. The horse should first be offered water, followed by small amounts of wet hay or fresh grass. In large colon disorders, feeding can be resumed as early as 8 hours after surgery, provided there is good intestinal health. In cases of large colon torsion, feeding is delayed until there is evidence of mucosal integrity. Ileal, cecal, and large and small colon impactions should be closely monitored when feeding is resumed, because these animals are at risk for reimpaction. The use of laxatives, such as mineral oil, is recommended in the immediate postoperative period for impactions. For details on nutritional support for the critical surgical patient, see Chapter 6.

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SECTION
VI RESPIRATORY SYSTEM

John A. Stick

CHAPTER
41 Overview of Upper Airway Function

Frederik J. Derksen

The upper airway has complex physiologic functions including olfaction, phonation, deglutition, thermoregulation, filtering and conditioning of the inspired air, and protecting the lower airway from aspiration. Most important, the upper airway also functions as a conduit for airflow to and from the lung, and, because the horse is an obligate nasal breather, a normal upper airway is critical in this species. To accommodate the many diverse demands placed on it, the upper airway was not ideally designed for any one activity. For example, the equine upper airway has a large dead space volume, it has high resistance to airflow, and it tends to collapse in response to negative pressures generated during inspiration.[1-4] Although none of these characteristics is ideal for an airflow conduit, compromises in design are necessary to accommodate the other upper airway functions. For these reasons, the upper airway may well be a limiting factor in the horse's exercise capacity, and tolerances in the upper airway are minimal. Even the smallest lesion before surgery or remaining after completion of a surgical procedure may further limit performance. This makes surgical interventions in the upper airway challenging, and if upper airway function is not fully considered, surgical outcome is often suboptimal. Consequently, surgeons must have a working understanding of upper airway physiology. The purpose of this chapter is to provide the surgeon with information that should be considered when performing upper airway surgery in the horse.

THE UPPER AIRWAY AND PERFORMANCE

The capacity of horses to increase ventilation in response to the demands of exercise is truly enormous. A normal horse at rest breathing 15 times a minute with a tidal volume of 5 L has a minute ventilation of 75 L. During exercise, respiratory rate and tidal volume increase to achieve a minute ventilation of approximately 1500 L.[3] The upper airway must accommodate this twenty-fold increase in airflow by undergoing changes in caliber, rigidity, and shape. In spite of these adaptations to exercise, evidence suggests that in exercising horses upper airway resistance becomes a large portion of total respiratory resistance, and this increase in resistance may limit performance.[5] In strenuously exercising horses, the PaO_2 decreases and the $PaCO_2$ increases. This perturbation in gas exchange is in part caused by insufficient alveolar ventilation, although diffusion limitation also plays a major role. Substituting a mixture of helium and oxygen for air reduces airway resistance, and blood oxygenation is improved.[5] Taken together, this is strong evidence to suggest that upper airway resistance limits arterial blood oxygenation in exercising horses, thereby limiting oxygen delivery to tissues and performance. Thus, if the function of the upper airway as a conduit for airflow is taxed to the maximum during exercise in normal horses, it is easy to understand that small upper airway lesions can significantly affect upper airway function during exercise and further affect performance.

MECHANICS OF AIR FLOW

The respiratory muscles, primarily the diaphragm, provide the force needed for ventilation. The pumping action of these muscles results in large pressure changes in the airways. On exhalation, pressure in the upper airway becomes positive, driving the air out against the atmospheric pressure, whereas on inhalation, pressures are negative, resulting in movement of air from the outside to inside the lung. The greater the exercise intensity, the greater the airflow rates and the larger the pressure changes. During high-intensity exercise, the pressure in the trachea is approximately 15 cm H_2O on exhalation, and it is 30 cm H_2O on inhalation (Figure 41-1).[6] Positive pressure in the upper airway during exhalation encourages upper airway dilation in this phase of the respiratory cycle. On inhalation, however, the negative

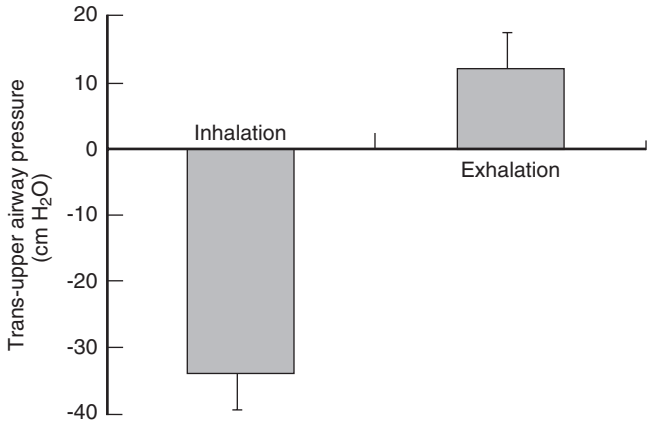

Figure 41-1. Trans-upper airway pressures of normal horses during intense exercise. Note that inspiratory pressures are greater than expiratory pressures.

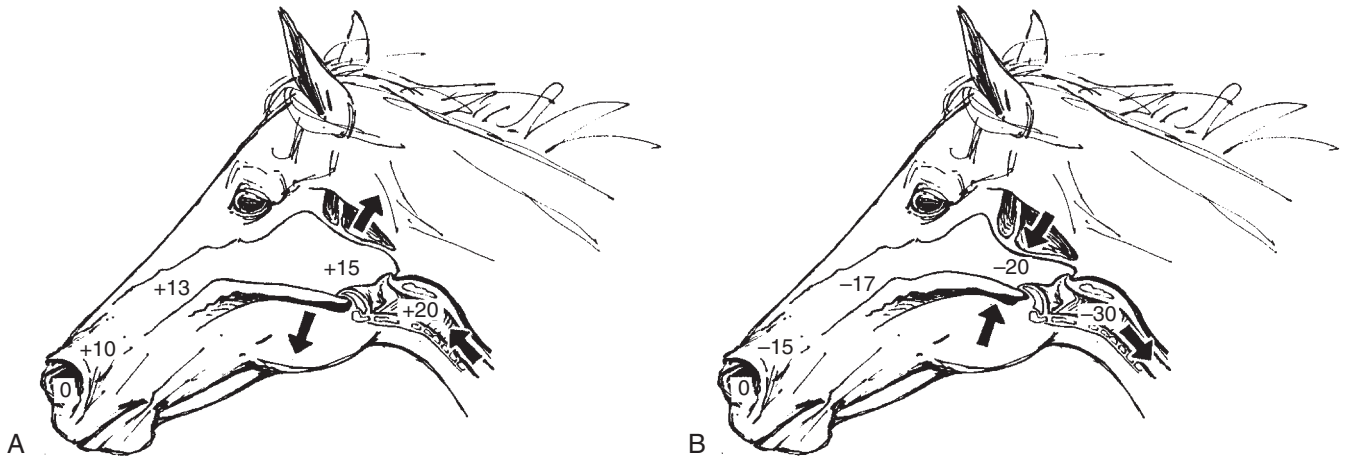

Figure 41-2. A, In exercising horses, upper airway pressures are positive relative to the atmosphere during expiration, facilitating airway dilation. **B,** During inspiration, upper airway pressures are negative, narrowing the airway lumen.

pressure in the upper airway encourages tissues to collapse, thus narrowing the upper airway (Figure 41-2).

The ratio of driving pressure and the resulting airflow is called impedance. Impedance is a very useful calculated variable, because it is a measure of how much the airflow is opposed by the respiratory system. One of the most important determinants of impedance is airway resistance. Airway resistance in turn is primarily determined by airway diameter. Many surgically correctable conditions of the upper airway (e.g., recurrent laryngeal neuropathy) narrow the airway lumen, decrease its diameter, and therefore increase impedance.[7] Measurements of upper airway impedance on inhalation and exhalation demonstrate that, even in normal horses, the upper airway is only partially able to withstand these pressure swings, especially during exercise. During inhalation, subatmospheric intraluminal pressures cause the airway to narrow, whereas positive intraluminal pressures enlarge the airway diameter during exhalation. As a consequence, impedance is higher on inhalation than during exhalation (Figure 41-3).[8]

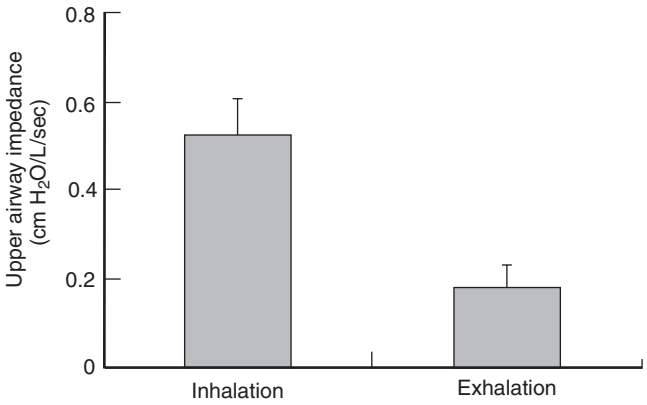

Figure 41-3. Upper airway impedance of normal horses during intense exercise. Note that inspiratory impedance is greater than expiratory impedance.

The anatomical contours of the upper airway are complex, and it is no surprise therefore that the airflow through this structure is equally complex. Recently, computer models of airflow through the upper airway have given new insight into the details of pressure and flow profiles that operate in the upper airway of exercising horses.[9,10] A detailed understanding of airflows and pressures in the upper airway is useful, as it gives clues to why certain upper airway regions are more prone to collapse than others. After the air enters the nostrils during inhalation it is forced through the relatively narrow nasal valve region, which results in a large pressure drop (Venturi effect; Bernoulli's principle) and high flow velocities. This anatomic region is where nasal strips have been used to prevent airway narrowing during inhalation.[11] As air enters the airway further during inhalation, the next narrow area it encounters is the rostral nasopharynx, at the transition between the nasal passages and the nasopharynx. As in the nasal valve area, this airway narrowing increases flow velocity and decreases wall pressure. It is possible that this pressure decrease initiates palatal instability, and predisposes the soft palate to dorsal displacement (DDSP).[10]

Next, the air enters the relatively wide nasopharynx. As airflow transitions from the nasopharynx into the relatively narrow larynx, there is another pressure drop, predisposing the laryngeal region to dynamic collapse involving such structures as the vocal folds, aryepiglottic folds, and arytenoid cartilages. Interestingly, the cross-sectional area of the larynx is approximately 7% greater than the trachea, supporting the idea that in horses with recurrent laryngeal neuropathy treated with laryngoplasty, maximal arytenoid abduction may not be necessary.[10] Computer models of the upper airway with simulated airflows generated in the author's lab provide additional interesting and sometimes unexpected information.[12] For example, the path that the airflow takes during inhalation differs from the route that it takes during exhalation.[12] Furthermore, there are flow eddies, regions of reverse flow, and areas of great turbulence, and wall stress.[9,10,12] As computer models become more sophisticated, a more detailed understanding of upper airway flow complexities is likely to improve our understanding of why upper airway regions are predisposed to collapse.

DISTRIBUTION OF AIRWAY RESISTANCE

In the horse, as in most mammals, upper airway resistance is a significant proportion of total resistance to airflow. In the resting horse, a full two-thirds of the total resistance to airflow resides

in the upper airway.[2] This proportion increases during exercise. Because of this, most animal species switch to mouth breathing during exercise, which provides a low-resistance pathway for the greater airflow required during exercise and minimizes the work of breathing. The horse cannot breathe through its mouth effectively, and the approximately twentyfold increase in airflow that occurs during exercise must be totally accommodated through nasal breathing and therefore, by the upper airway. Obligate nasal breathing is the result of a tight seal between the soft palate and the laryngeal cartilages. When the nasal passages are occluded, this seal may be broken and mouth breathing results. However, in the horse, the resistance to airflow through the mouth is apparently very high.

During exercise, the distribution of airflow resistance changes significantly. In the exercising horse, during inhalation, intrathoracic resistance is less than 25% of total resistance to flow and the remaining resistance is almost evenly divided between nasal and laryngeal resistance. On exhalation, the intrathoracic and extrathoracic resistances are about even.[2]

STABILIZING THE UPPER AIRWAY

The upper airway is designed to prevent collapse on inhalation. The simplest way to prevent a tube from collapsing in response to negative intraluminal pressure is to make the wall rigid. Indeed, for some of its length, the upper airway is rigidly supported by bone and cartilage. In other regions of the airway, such as the external nares, pharynx, and larynx, rigid support by bone or cartilage is incompatible with other functions, such as swallowing and protecting the lower airways. In these regions, the upper airway is supported by muscular activity. Neural reflexes regulate the activity of upper airway dilator muscles.[13-15] On inhalation, these muscles begin to contract just before the diaphragm contracts, and they remain active during inhalation. In this way, the upper airway stiffens before it is exposed to subatmospheric pressures. This phasic activity is finely tuned by pressure- and flow- (temperature-) sensitive receptors in the upper airway that, when stimulated, enhance reflex activation of upper airway dilator muscles. These muscles, then, provide appropriate tension to prevent dynamic collapse of tissues on inhalation.[16,17]

External Upper Airway Support

A nasal strip has been used as an external nasal dilator. It mechanically supports the soft tissue that forms the lateral wall at the narrowest portion of the nasal cavity (nasal valve), located just rostral to the naso-incisive notch. This region is bound medially by the nasal septum, ventrally by the concha, and dorsolaterally by the skin and dorsal conchal fold. The strip consists of three polyester springs sandwiched between two layers of material, and it adheres to the horse's nose. When the strip is applied, endoscopic examination shows that the strip dilates the nasal valve region.[11] Furthermore, in exercising horses, the nasal strip decreases inspiratory impedance.[11] Interestingly, the nasal strip also decreases the severity of exercise-induced pulmonary hemorrhage (EIPH).[18,19] These findings suggest that in exercising horses, the nasal strip reduces upper airway tissue collapse on inhalation. This would result in decreased inspiratory impedance during exercise, and less-negative inspiratory pressures. Less-negative inspiratory pressure may reduce the transmural pulmonary capillary pressure. Because excessive transmural pulmonary capillary pressure is responsible for EIPH, reduction in this pressure very likely accounts for the observed decrease in the severity of EIPH.[20,21]

Head Position

Horses are often required to exercise with their heads and necks in unnatural positions. For example, Standardbred horses are raced with an overcheck, which forces the head and neck into extension. Sport horses, such as dressage horses, commonly exercise with head and neck flexed. Head and neck position significantly affects upper airway impedance.[22] In resting horses that are holding their heads in a natural position, the air entering the upper airway turns approximately 90 degrees to flow from the nasal passages into the trachea. This change in direction contributes to the work of breathing. Normally, horses straighten their head and neck during exercise. When the horse's head and neck are flexed, upper airway impedance during exercise is about 50% greater than that of a horse exercising with head and neck in the extended position.[22]

Straightening of the head and neck not only provides a more direct route to the lung, it also tends to stretch upper airway tissues, making them more rigid and resistant to collapse. It stands to reason, therefore, that dynamic upper airway obstructions, especially those involving the pharynx, are often more apparent when affected horses are exercised with a flexed head and neck position.[23]

External Nares

The external nares of the horse are very mobile and can completely close the upper airway (as occurs during swimming) or fully dilate it (e.g., during intense exercise). The external nares and adjacent nasal valve are among the narrowest portions of the upper airway; therefore, lesions in this region significantly affect performance.[24] The dilator muscles of the external nares, such as the *M. dilator naris late*ralis and the *M. transversus nasi*, are innervated by the facial nerve, and horses with facial nerve dysfunction have marked inspiratory airway obstruction during exercise.

Nasal Turbinate Region

The nasal turbinate region has convoluted conchae that encourage particle deposition and conditioning of the inspired air. This portion of the upper airway is rigidly supported by cartilage and bone. However, marked changes in airway caliber are possible even in this bone- and cartilage-supported region. The upper airway is lined by mucosa that contains vascular sinuses. The purpose of this extensive vasculature is thermoregulation and humidification of inhaled air. These vessels are potentially very large and have the ability to fill with blood, thickening the upper airway mucosa to the point of occlusion. Normally, the size of capacitance vessels is kept small by tonic sympathetic innervation. During exercise, the sympathetic tone increases, capacitance vessels are constricted, and the airway diameter is increased.[25] Injury to the sympathetic trunk innervating this region—for example, in Horner's syndrome—can cause complete nasal occlusion of the affected side. Unilateral airway obstruction is often the longest-lasting condition associated with Horner's syndrome, and it causes significant exercise intolerance. Nasal vascular engorgement also has been identified as

a cause of airway obstruction in horses recovering from general anesthesia after removal of the orotracheal tube. This obstruction is sufficiently severe to cause hypoventilation and hypoxemia. Phenylephrine, an α-adrenoceptor, can be used to decrease nasal vascular engorgement after anesthesia to prevent upper airway obstruction in these horses.[26]

Nasopharynx

The nasopharynx participates in swallowing as well as breathing, and it must be able to constrict to propel a bolus of food into the esophagus and to dilate during exercise to allow unimpeded airflow to and from the lung. These functions are achieved by muscles that are uniquely positioned so that, as a group, they can both constrict and dilate the pharynx.[27-29] In the horse, the exact functions of these muscles are incompletely understood, but from studies in horses and in other species, a general picture is emerging.

The hyoid apparatus is a very important structure that supports the pharynx as well as the root of the tongue and the larynx. The muscles of the hyoid apparatus dilate the pharynx by contracting on inhalation in the following manner. The geniohyoid and genioglossus muscles pull rostrally and ventrally, whereas the sternohyoid and sternothyroid muscles pull in a caudal ventral direction. The net effect is extension of the stylohyoid-ceratohyoid articulation, a ventral movement of the hyoid bone, and nasopharyngeal stabilization or even dilation (Figure 41-4). One of the recommended surgeries for dorsal displacement of the soft palate is sectioning of the sternohyoid and sternothyroid muscles.[30,31] The objective of this surgery is to move the epiglottis craniad, thereby better engaging the soft palate. From the aforementioned discussion, it is clear that sectioning of the sternohyoid and sternothyroid muscles prevents pharyngeal dilation and may encourage pharyngeal collapse. Indeed, measurements in exercising horses have indicated that sternohyoid and sternothyroid muscle sectioning slightly increases upper airway resistance in exercising horses.[32]

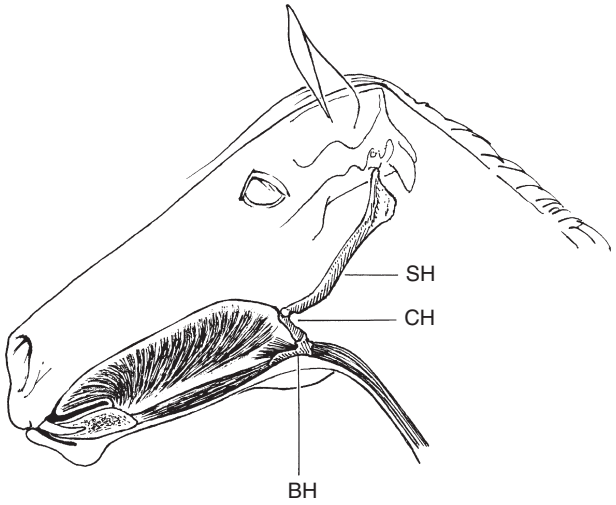

Figure 41-4. The hyoid apparatus and the pharynx. The hyoid apparatus is made up of the stylohyoid *(SH)*, the ceratohyoid *(CH)*, and the basihyoid *(BH)* bones. Contraction of the geniohyoid and sternohyoid muscles stabilizes the hyoid apparatus and the pharynx.

In racehorses, the tongue is often pulled forward and tied down to the mandible to prevent dorsal displacement of the soft palate. The tongue is attached to the hyoid bone via the genioglossus and hyoglossus muscles. Therefore, it is possible that the tongue-tie could dilate the pharynx. However, upper airway flow mechanics and computed tomographic studies have indicated that, in normal horses, the tongue-tie does not pull the hyoid apparatus forward and does not dilate the pharynx.[33,34] In horses with DDSP, the use of a tongue-tie may prevent the condition in individual horses although it is not effective in the majority.[35] Where DDSP is not prevented, application of a tongue-tie does not improve ventilation.[35]

The dorsal pharyngeal wall is supported by the stylopharyngeus muscles. These muscles insert on the dorsal nasopharyngeal wall, perpendicular to it, so that contraction of the stylopharyngeus muscles raises the wall of the dorsal nasopharynx, expanding, supporting, and preventing its collapse as pressures within the airway become more negative during inspiration (Figure 41-5). Blockade of cranial nerve (CN) IX, which innervates these muscles, results in dorsal pharyngeal collapse and inspiratory airway obstruction during exercise.[36]

The upper airway is richly endowed with receptors that, when stimulated, activate upper airway dilator muscles, thereby stiffening the upper airway. For example, when the laryngeal mucosa is anesthetized, exercising horses have nasopharyngeal collapse and upper airway obstruction.[37] During nasal occlusion, these horses can also experience DDSP. This demonstrates that, in exercising horses, sensory and motor functions must be fully coordinated to maintain upper airway patency. The message to the surgeon is that the upper airway is a finely tuned instrument that can be easily disturbed by disease or surgical intervention.

Soft Palate

The soft palate is another upper airway structure with multiple functions. It separates the nasopharynx from the oral pharynx and forms a tight seal around the larynx during breathing. However, during swallowing, the soft palate is elevated and closes the posterior nares, protecting the nasal passages from food and water. The position of the soft palate is determined by four muscles: the *M. tensor veli palatini*, *M. levator veli palatini*, *M. palatinus*, and *M. palatopharyngeus*.[38] Dysfunction of the *M tensor veli palatini* destabilizes the cranial portion of the soft palate closest to the hard palate. In affected horses, the cranial portion of the soft palate bulges into the nasopharynx on inhalation.[39] The pharyngeal branch of the vagus nerve innervates all of the muscles controlling the soft palate, except for the *M. tensor veli palatini*. Blockade of this nerve with a local anesthetic agent results in dorsal displacement of the soft palate.[40] This demonstrates that coordinated activity of these muscles is necessary to stabilize the soft palate during breathing. The pharyngeal branch of the vagus nerve courses through the guttural pouch and lies in close proximity to the retropharyngeal lymph nodes. It is thought that in clinical cases, dorsal displacement of the soft palate is the result of a functional abnormality of this nerve because of retropharyngeal lymph node or guttural pouch inflammation.

Most upper airway conditions cause inspiratory airway obstruction, and expiratory airflow is unaffected. Dorsal displacement of the soft palate is an exception to this rule, since it is an expiratory obstruction. During exhalation, the paralyzed

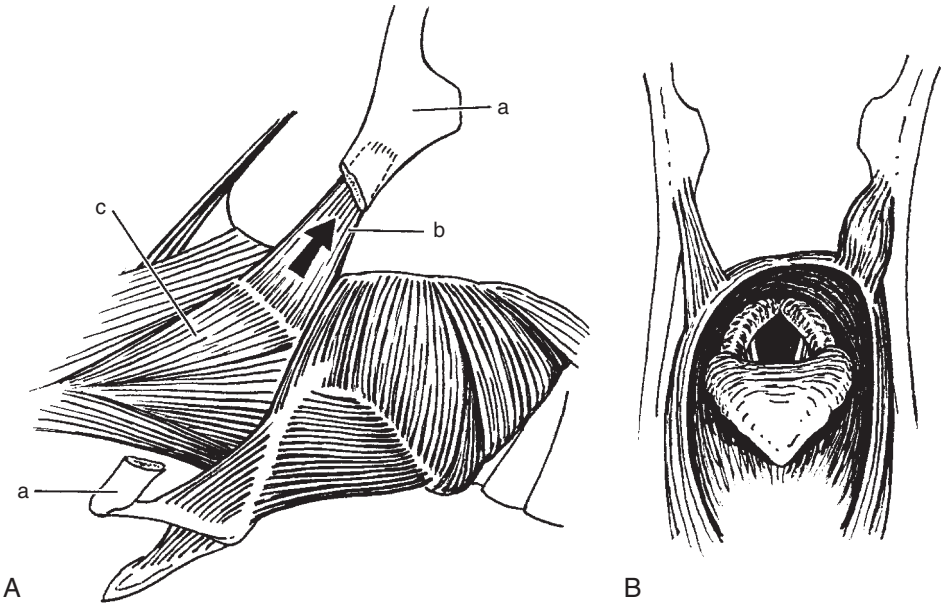

Figure 41-5. A, The stylopharyngeus muscle originates on the stylohyoid bone *(a),* and broadly inserts on the dorsal pharyngeal wall. Contraction of the stylopharyngeus muscle *(b)* stabilizes and dilates the dorsal pharynx during exercise. *c,* Palatopharyngeus muscle. **B,** Note the difference in size of the left and right dorsal pharynx when the stylopharyngeus muscle contracts unilaterally.

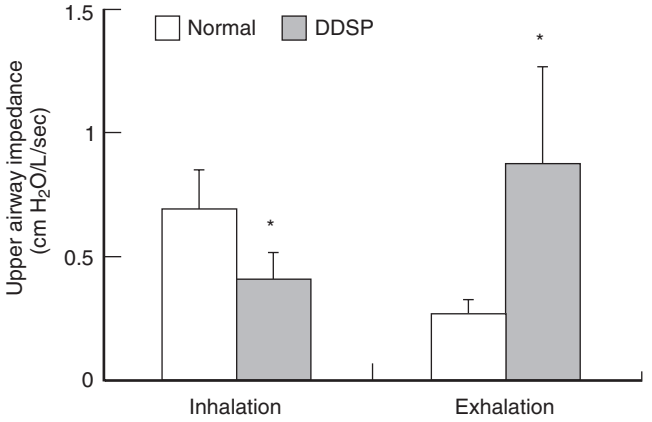

Figure 41-6. Upper airway impedance in intensely exercising horses before and after experimentally induced dorsal displacement of the soft palate. Note that expiratory impedance is increased, whereas inspiratory impedance is decreased. *DDSP,* Dorsal displacement of the soft palate; *, significantly different from normal ($p \leq 0.05$).

soft palate billows dorsally into the nasopharynx, markedly obstructing airflow and causing airway obstruction (Figure 41-6).[40]

Larynx

In the course of deglutition, the larynx functions as a very important guard, preventing food and water from entering the trachea. During swallowing, the arytenoid cartilages adduct completely, and the epiglottis pivots around its base so that the tip of the epiglottis moves dorsad and caudad. These maneuvers close the rima glottidis and prevent aspiration. Of course, during exercise the larynx must be opened maximally to allow unimpeded airflow. The larynx is one of the narrowest portions of the upper airway, and consequently lesions in this region cause significant airway obstruction. A good example of this is recurrent laryngeal neuropathy, also known as idiopathic laryngeal hemiplegia. In this condition, dysfunction of the left recurrent laryngeal nerve prevents appropriate contraction of the *M. cricoarytenoideus dorsalis*, which fails to abduct the arytenoid cartilage during inhalation.[41,42] In affected resting horses, pressure changes are insufficient to move the affected arytenoid cartilage and vocal fold into the airway lumen. On exhalation, positive pressures in the upper airway move the affected arytenoid cartilage abaxially, increasing airway caliber. However, on inhalation during exercise, luminal pressures are sufficiently negative to move the affected arytenoid cartilage and associated vocal fold into the airway, causing significant obstruction. The objective of surgical intervention in horses with recurrent laryngeal neuropathy is to prevent dynamic collapse of the affected arytenoid cartilage. In these cases, prosthetic laryngoplasty is effective because the procedure stabilizes the affected arytenoid cartilage, preventing dynamic collapse. An example of a surgical procedure that is ineffective because it fails to adhere to this principle is subtotal arytenoidectomy.[43] In this procedure, only the body of the arytenoid cartilage is removed, while the muscular process and the corniculate cartilage are left in place to help prevent postsurgical aspiration. However, during exercise the unsupported cartilage moves axially and obstructs the rima glottidis.[43]

As mentioned earlier, during swallowing, the epiglottis pivots around its base, protecting the trachea. However, during breathing and especially during exercise, the epiglottis must be stabilized. This is the function of the *M. hyoepiglotticus*, which originates on the basihyoid bone and inserts on the base of the epiglottis. When the *M. hyoepiglotticus* contracts, the epiglottis is pulled ventrad toward the base of the tongue, stabilizing the epiglottis. The *M. hyoepiglotticus* is innervated by the hypoglossal nerves, and bilateral blockade of this nerve results in epiglottic retroversion during inhalation in exercising horses.[44,45] Clinical cases of epiglottic retroversion have been described and probably are the result of disease interfering with neuromuscular control of upper airway dilator muscles, specifically the *M. hyoepiglotticus*.[46]

MEASUREMENT OF UPPER AIRWAY FUNCTION

Thus it is clear that the upper airway has often-competing functions, that small perturbations in function may cause significant airway obstruction, and that, for the surgeon, tolerance for error is small. Quantitative measurements of upper airway function help determine the effect of a particular condition on upper airway flow mechanics and the efficacy of surgical treatment. The best way to ensure that a surgical procedure is efficacious is to use quantitative methods of evaluation. The simplest method of evaluating upper airway flow mechanics is to measure the pressure needed to move air through the upper airway while the horse is undergoing a standard exercise test. Airway pressures may be measured using a multiple-hole catheter inserted into the trachea via the nostrils or percutaneously.[47] When airway pressures and airflow rates are measured simultaneously, upper airway impedance can be calculated as the ratio of pressure and flow.[7] Measurement of impedance provides a more direct evaluation of changes in airway caliber than pressure measurements alone. Pressure and flow measurements have been used to determine the efficacy of many surgical procedures used in the treatment of upper airway conditions, such as recurrent laryngeal neuropathy and DDSP.[40,48,49]

Quantitative information may also be gained from analysis of airflow rates during the respiratory cycle. Flow-volume loop analysis is a test in which airflow rate is continuously plotted against breath volume. This test is particularly useful when performed in exercising horses, and in some instances it is more sensitive than the measurement of upper airway impedance in the evaluation of upper airway function (Figure 41-7).[8,50]

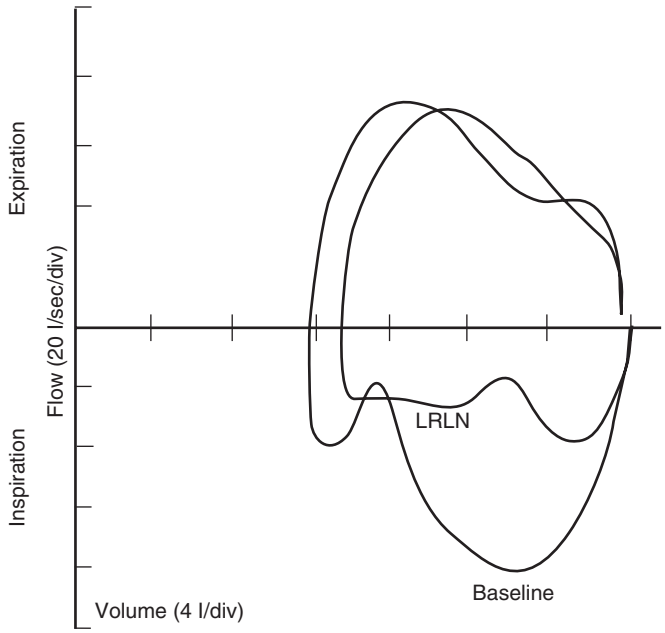

Figure 41-7. Tidal breathing flow-volume loop from a horse exercising at maximal heart rate before *(Baseline)* and after induction of laryngeal hemiplegia *(LRLN).* Note that expiration is unaffected, but there is severe inspiratory flow limitation with LRLN throughout the inspiratory portion of the tidal volume.

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CHAPTER
42 Diagnostic Techniques in Equine Upper Respiratory Tract Disease

Eric J. Parente, Samantha H. Franklin, Frederik J. Derksen, Michael A. Weishaupt, Heather J. Chalmers, and Caroline Tessier

PHYSICAL EXAMINATION

Eric J. Parente

Although a large portion of the upper respiratory tract is not easily seen or palpated, a thorough physical examination can provide critical details that assist in the overall assessment of the upper respiratory tract. A physical examination is an essential part of any evaluation, and it cannot only provide information on present pathology but also give clues on previous surgical procedures. Prior to beginning the examination, a complete history should be obtained. Specific questions should be asked about respiratory noise, dysphagia, exercise intolerance, coughing, nasal discharge, and any previous treatments or surgery. All of this information should be interpreted in conjunction with the physical examination findings and the results of the other diagnostic procedures to establish a diagnosis.

The physical examination should begin with visual assessment of any asymmetry of the head and nares and inspection for the presence of any nasal or ocular discharge. Facial deformity in front of the eye is consistent with either primary or secondary sinus disease. This may also have an associated nasal discharge via the nasomaxillary opening, or ocular discharge from occlusion of the nasolacrimal duct. More chronic sinus disease may result in nasal passage obstruction. Placing a hand in front of each nostril will provide a crude indication of airflow from each nostril. Subtle changes are very difficult to detect, but an obstructive lesion in one nasal passage may result in significant asymmetric airflow. Percussion of a normal sinus should result in a hollow sound. The presence of an abnormal structure in the sinus can change the character and intensity of the sound, but this is dependent on many factors and can often be misinterpreted. Nostril disease is rare, but palpation of each nostril and the alar folds in the dorsal aspect of the nares can often determine if a problem is present. If alar fold redundancy and obstruction during exercise is suspected, the alar folds can be tied with umbilical tape over the bridge of the nose during an exercise period. Mitigation of noise confirms the diagnosis.

Palpation of the larynx can be a difficult skill to develop, but when mastered it can be very helpful in assessing laryngeal function. An assistant should hold the horse's head slightly

elevated. The clinician can stabilize the right side of the larynx with one hand and palpate the left side of the larynx with the other hand. Then, by alternating hands, both sides of the larynx can be compared. The muscular processes of the larynx are in a fairly dorsal position and are slightly smaller than a person's knuckle. The caudal border is attached to the *cricoarytenoideus dorsalis* muscle, and when atrophy of the muscle is present, the muscular process feels more prominent than on the other side. Mild asymmetry is a common finding in horses with normal laryngeal function, but more overt asymmetry is usually consistent with the horse's inability to maintain abduction of the affected arytenoid cartilage. If the left muscular process cannot be easily palpated, it may be covered by scar tissue from a previous laryngoplasty or the arytenoid cartilage may be chondritic. A scar from previous laryngoplasty is not easily palpated at the level of the skin, and therefore it cannot be used to definitively determine if surgery was performed previously. Clipping the hair ventral to the linguofacial vein will make it easier to detect a scar if surgery was performed. If the right muscular process cannot be palpated easily, the arytenoid cartilage may be chondritic or a fourth branchial arch abnormality may be present.[1] Infrequently, stertorous breathing can be elicited with firm palpation of the larynx, and this may indicate some inability of the horse to maintain an adequate lumen from such conditions as severe arytenoid chondrosis, bilateral arytenoid paresis, or a combination of both.

Other common sites for respiratory surgery should be evaluated during the examination. Previous surgery involving a laryngotomy will often leave a palpable scar. The skin ventral to the larynx should be palpated for a scar by pinching the skin below the larynx. Often a fibrous band forms between the skin and larynx that can be palpated when drawing your fingers like a rake across the skin in this area. The accessory respiratory muscles (sternothyrohyoideus) should be examined for any sign of a defect secondary to a myectomy, and the tracheal rings should be palpated for any abnormalities associated with trauma or previous surgery.

ENDOSCOPIC EVALUATION

Videoendoscopic Examination at Rest

The endoscopic examination of the upper airway is still the primary diagnostic tool. Most diagnoses can be obtained from a complete resting endoscopic examination. Videoendoscopic equipment has been commercially available for over twenty years and provides the best view of the upper airway. Several large companies produce videoendoscopes with similar specifications, and user preference and cost often determines which system is purchased. The main components of a videoendoscopy system consist of the videoendoscope itself, the video processor, the light source, and the video monitor. A videoendoscope with an outer diameter of less than 9.5 mm can be used easily on all adult horses and most foals. Smaller videoendoscopes with an outer diameter of less than 8.5 mm are also available for younger foals, but the tradeoff is a smaller biopsy channel (2.2 mm versus 2.8 mm). Most videoendoscopes have a field of view of 120 degrees and tip deflection angles of 210 degrees to 90 degrees. The working length for most videoendoscopes is approximately 103 cm (40½ inches), which is ample for upper respiratory work. Light sources are fairly standard xenon lamps with illumination of 300 watts. Fiberoptic endoscopes typically have only half the illumination of the videoendoscopes and a much smaller field of view, which significantly limits the clinician's ability to clearly view all abnormalities.

Image storage is crucial for documentation and reevaluations. It is therefore beneficial to have a recording unit and/or printer with any videoendoscopic system. Multiple digital formats are now available and the most important consideration is compatibility with viewing and editing systems.

Like any examination, the clinician should have a plan of how and what to evaluate during the examination. The patient should not be sedated and should only be restrained with a twitch to ensure no interference with any functional assessments of the airway. It is easiest to have an assistant pass the endoscope down the ventral meatus of one nostril until the tip of the endoscope enters the pharynx. The pharynx and larynx should be evaluated first, followed by the evaluation of the nasal passage while backing the endoscope out of the horse's nose. If a suspected abnormality is present in the nasal passage, the normal side should be evaluated first to minimize any trauma or resentment from the horse before evaluating the abnormal side.

There is a variable amount of lymphoid hyperplasia within the pharynx of the horse, depending upon the age. It is common for the younger horse to have more extensive lymphoid hyperplasia than older horses, but it has not been correlated with any specific dysfunctions.[2,3] The epiglottis should always be positioned dorsal to the palate and have a distinct "serrated" edge with a clear vascular pattern on the dorsal surface. The corniculate processes of the arytenoid cartilages should be evaluated for their overall appearance, relative position, and degree of movement. Thickening, ulcerations, or granulation tissue likely indicate chondrosis (Figure 42-1). Inability to fully abduct one arytenoid cartilage relative to the other side indicates recurrent laryngeal neuropathy (RLN). Although multiple grading systems are used, a I to IV grading system for assessing laryngeal movements at rest established by Rakestraw et al[4] is the basis for most systems (see Chapter 45). Grade I refers to symmetric abduction/adduction and synchronous movement. Grade II refers to asynchronous movement but still symmetric abduction/adduction, and abduction can be maintained. Grade III refers to both asynchronous and asymmetric movement, and abduction

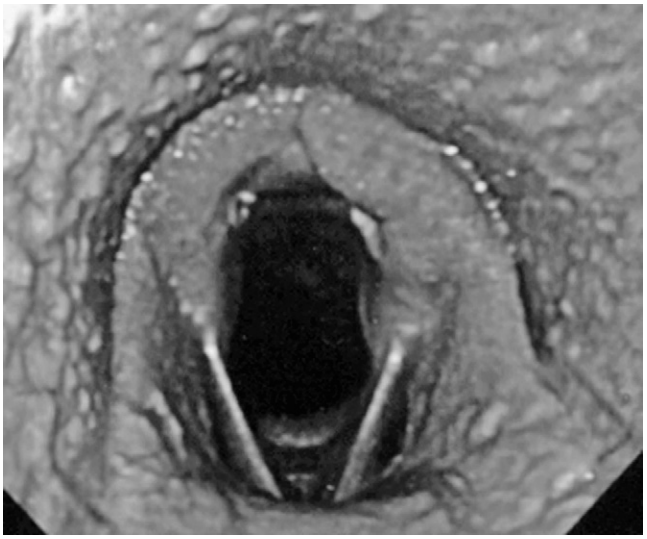


Figure 42-1. Left arytenoid chondritis with a thickened, misshapen corniculate process and incomplete abduction.

cannot be maintained. Grade IV refers to an inability to move one arytenoid hardly at all. Full abduction of the arytenoids should be induced by both swallowing and nasal occlusion. Swallowing will yield only a brief period to evaluate full abduction. Nasal occlusion may result in a longer duration of abduction, but it requires that a horse tolerate nasal occlusion for an extended period of time. Although nasal occlusion can result in negative pharyngeal pressures similar to those experienced by horses during exercise,[5] the degree of abduction after swallowing appears to have a better correlation with the degree of abduction during exercise.[6] Racehorses that are unable to fully abduct one arytenoid (grade III or IV) are much more likely to experience dynamic collapse of that arytenoid during high-speed exercise, but there are exceptions.[7]

Inability to fully adduct one arytenoid may be a result of previous laryngoplasty, and palpation of the larynx and assessing the skin for a surgical scar should help determine the diagnosis. The slap test can be used to induce adduction of the arytenoid cartilages but is not a valuable test to fully evaluate laryngeal function.[8] The vocal cords and saccules should also be examined for any evidence of previous surgery. Incomplete ablation with the laser may yield scarring of the cords, deformation of the cords, or can show an absent cord (Figure 42-2).

The endoscope should be passed into the trachea to determine the presence of any discharge or feed material, indicating aspiration. As the endoscope exits the trachea, it is common for the horse to displace its palate, but a normal horse should replace it quickly. If the horse does not replace its palate quickly, it can be stimulated to swallow by either infusing water into the pharynx or quickly bumping the pharyngeal wall with the endoscope. If the horse still does not replace the palate below the epiglottis, it may indicate some structural abnormality under the epiglottis or some pharyngeal dysfunction that may be best observed during high-speed exercise.[9] If a subepiglottic abnormality is suspected, other imaging techniques may be indicated, but the clinician can also spray the epiglottic region with topical anesthetic through the biopsy channel of the videoendoscope, and then manipulate the epiglottis with a long bronchoesophageal forceps through the contralateral nares to look underneath.

When the examination of the larynx/pharynx is complete, the remainder of the upper respiratory tract should be examined. The guttural pouch openings should be clear of any discharge. The presence of discharge at the opening of the guttural pouch may be an incidental finding caused by discharge originating from another location and becoming lodged at the opening. The suspicion of any guttural pouch disease warrants further endoscopic evaluation of the guttural pouches. This is most often done at the end of the examination, and may require a small dose of intravenous sedation. Temporohyoid disease can often be seen as an enlargement of the stylohyoid bone at the most dorsal aspect of the guttural pouch (Figure 42-3). The ethmoid recess, turbinates, septum, and region of the nasomaxillary openings (caudal middle meatus) should all be evaluated as the endoscope is slowly withdrawn from the nostril. Discharge running down the ventral turbinate, with material exiting the nasomaxillary opening, is often a sign of sinus disease. Both nasal passages should be evaluated if there is any suspicion of a sinus or nasal passage problem. It is often difficult to discern small deviations in the nasal passage because the endoscopic view provides a very magnified but small field of view. Radiographs are a helpful complementary procedure if septal deviation, or some type of sinus disease, is suspected.

Videoendoscopic Examination during Treadmill Exercise

Although a great deal of information can be gained from endoscopy in the resting horse, there are many occasions when a functional abnormality is suspected (e.g., because of exercise intolerance or abnormal respiratory noise during exercise), but no abnormalities are detected during the resting endoscopic examination. Conditions that can lead to this scenario include forms of pharyngeal collapse, axial deviation of the aryepiglottic

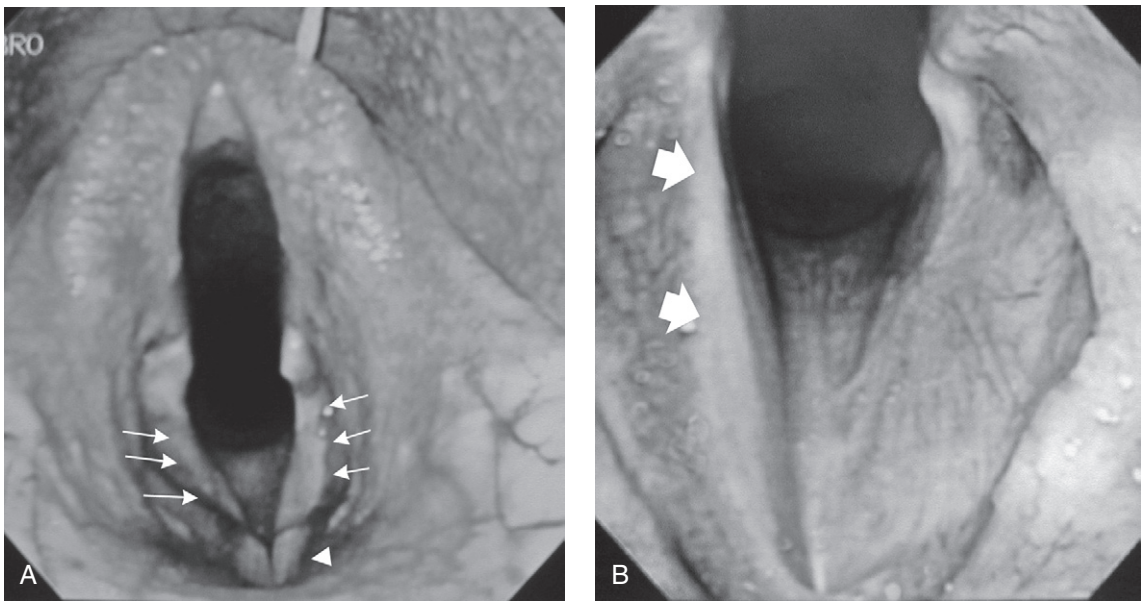


Figure 42-2. A, Scarred vocal cords *(small arrows),* and remaining normal vocal cords ventrally *(arrowhead);* **B,** A close-up videoendoscopic view of the vocal cords. Absent left vocal cord from laser resection. Normal remaining right vocal cord *(arrows).*

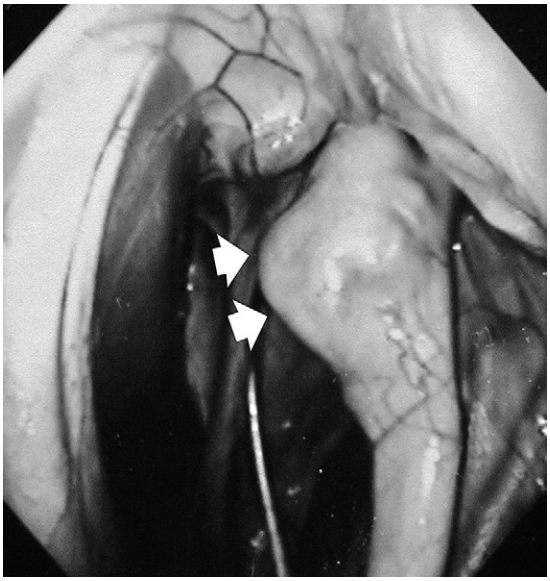


Figure 42-3. Swelling of the stylohyoid bone at the temporohyoid joint within the guttural pouch *(arrows)*.

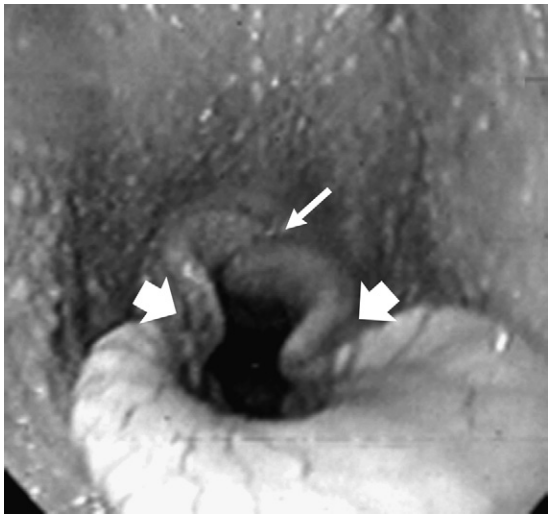


Figure 42-4. Subluxation of the apices of the arytenoid cartilages *(small arrow)* and axial deviation of the aryepiglottic folds *(large arrows)* induced during high-speed treadmill exercise only after increasing head/neck flexion. The larynx was normal without head/neck flexion.

folds, epiglottic retroversion, intermittent epiglottic entrapment, and dorsal displacement of the soft palate (DDSP). Endoscopic examination immediately after exercise may be valuable in some cases, but it can be misleading. Even normal horses have a much more flaccid-looking upper airway and displace more easily when first pulling up after exercise. Therefore, any conclusion based on a post-exercise endoscopic examination, particularly with respect to the functional stability of the pharynx, may be inaccurate.

Accurate conclusions from a "high-speed endoscopic examination" on a treadmill cannot be made from simply performing endoscopy while a horse is running without specifying exertional effort. It is critical to have the exercising conditions simulate the horse's normal working conditions as closely as possible. In the lower respiratory tract, the effects of running a longer distance at a slower speed are not equivalent to the effects of running a shorter distance at a faster speed.[10] This may also be the case in the upper respiratory tract. Furthermore, some of the upper respiratory problems are not only dynamic but also occur intermittently, and if all the same conditions of speed, head/neck flexion, fatigue, and so on are not reproduced, a false negative outcome is likely to result (Figure 42-4). A Holter monitor can be employed to record the horse's heart rate during exercise, and rates of approximately 220 beats/minute or greater, provide evidence that the horse is exercising at or near its maximum heart rate. Of the known upper respiratory abnormalities, DDSP is the most likely disease that will not be reproduced on a high-speed treadmill examination despite circumstantial evidence of the condition under field conditions.

Horses that will be examined by treadmill endoscopy must be as fit as they are for competition. A horse out of work may be too unfit to withstand a maximal exertional effort on the treadmill, and the problem to be diagnosed may not manifest itself. All horses must first be acclimatized to the treadmill before attempting an actual test. This can usually be accomplished with 1 to 2 hours of schooling, taking the horses through the different gaits, and making sure they can obtain adequate speed. During the schooling and testing periods, protective boots and leg wraps are recommended. Hobbles and harness are always recommended for pacing Standardbreds and are applied during the schooling period. The horse must be in condition and prepared for the examination as if it were the date of a competition or race.

The treadmill must be capable of obtaining a speed of at least 14 m/sec with relatively rapid acceleration and deceleration potential for any examinations of racehorses. Padded rails with polycarbonate sides, or an equivalent material, should be present to maintain the animal's position on the belt and protect individuals on either side of the treadmill. Large overhead fans in a temperature-controlled environment are also required. Sweat evaporation is the horse's primary form of body temperature regulation. Under racing conditions, the animal creates its own breeze to enhance evaporation; however, on the treadmill, the horse is stationary, and fans are required to prevent overheating. Two people, holding ropes that are attached to both sides of horse's halter, keep the horse centered on the treadmill. An individual behind the horse encourages the horse forward, and another person closely monitors the horse's gait, and controls the treadmill speed, ensuring the horse's position on the treadmill. One final person is needed to guide the endoscope during the examination (Figure 42-5). For safety reasons, and to ensure a valid test, it is extremely important that the team of individuals carrying out the examination is experienced. The specific protocol used depends on the type of competition the horse is normally engaged in and should mimic these circumstances as much as possible. Generally, after an initial warm-up period, the treadmill is stopped, the endoscope is passed via the right nostril into the pharynx, and its position is maintained by Velcro straps connecting the shaft of the endoscope and the noseband of the horse's halter (Figure 42-6). A typical exercise protocol for the Thoroughbred racehorse is as follows:

- Warm up through a combination of walk/trot/canter for 2000 meters.
- Walk again until heart rate is 80 to 90, stop, and pass the endoscope.

Figure 42-5. Horse during high-speed treadmill endoscopy.

Figure 42-6. The videoendoscope *(black arrow)* is being held in position up the horse's right nostril by a Velcro strip taped to the scope and then attached to the noseband of the halter *(white arrow).*

- Accelerate to 9 m/sec (incline treadmill to 3 degrees for Thoroughbreds).
- Accelerate to 11 m/sec for 400 meters.
- Accelerate to 12 m/sec for 400 meters.
- Accelerate to 14 m/sec for 1600 meters.
- Decelerate to 12 m/sec for 400 meters.

Most horses suffering from a poor performance problem are unable to meet the demands of this protocol. Such horses should be exercised at their own maximal exertional effort. Determination of maximum effort is a subjective assessment by the clinician in conjunction with heart rate evaluation.

Figure 42-7. Overground endoscopy performed on a racehorse exercising on the gallops. (Courtesy John Periam.)

Recording the entire endoscopic examination and reviewing it on slow-motion playback is sometimes necessary because most abnormalities occur rapidly within the respiratory cycle. Occasionally, stimulating a swallow with water through the endoscope or altering the speed of the treadmill during the test will induce the dynamic respiratory problem.

Videoendoscopic Examination during Overground Exercise

Samantha H. Franklin

Videoendoscopy during treadmill exercise enables the diagnosis of dynamic upper airway collapse in horses where resting findings are frequently unreliable or absent. However, there remains much reluctance among trainers and owners to present horses for treadmill evaluations and hence many horses still have a diagnosis made on the basis of clinical history and resting endoscopic findings alone. The reasons for this may include the need to travel long distances to a specialist center, the cost of the procedure and concerns regarding the risk of injury. Very little data are available regarding the risk of injury during treadmill exercise. However, a recent study of 2305 horses from nine different centers in the United Kingdom and Europe suggests that injury rates are no greater than those incurred during strenuous exercise, including racing and eventing, with only 13 (0.6%) horses incurring significant injuries.[11] However, horses that are presented for investigation of poor performance may have underlying or sublinical musculoskeletal disease that is contributing to their suboptimal performance.[12,13] This may be exacerbated during a strenuous exercise test, irrespective of whether this is performed on the treadmill or in the field. Therefore, examination of the musculoskeletal system is recommended prior to exercise testing to rule out any pre-existing lesions.

The need for a system that enables videoendoscopy to be performed in the horse's natural environment has led to the recent development of overground endoscopy (Figure 42-7).[14] This technique is well tolerated by horses and enables a definitive diagnosis of upper respiratory tract (URT) collapse to be made during ridden exercise.[14-18] Advantages of performing field exercise tests include the ability to perform the examination in

the environment in which the horse trains and is likely to be used in competition. This enables external factors to be standardized including jockey, equipment, and ground conditions that cannot necessarily be replicated during treadmill exercise.[19] In addition, there is no need for training sessions and horses do not need to travel such long distances to be examined, thereby reducing both the time and costs involved.

Several commercial systems are now available or are in the final stages of development. These systems incorporate a CCD video chip into an insertion tube of varying length. Some systems are mounted on the horse's head, and others have a longer tube with the processor, telemetry unit, battery pack, and recording equipment mounted either in a saddle pack or in a backpack worn by the jockey. The video image is transmitted wirelessly for real-time viewing by the veterinarian. The transmission distance varies among systems but currently appears to be in the range of 150 to 1000 m. However, the signal relies on line of sight and hence may be lost where obstacles come between the horse and the viewer. Interference may also occur as a result of other electronic equipment or because of reflection of some stray signals off solid surfaces, for example when used in an indoor arena. In addition, legal restrictions for wireless video transmission are likely to limit the transmission distance in some countries. Therefore, most systems also incorporate a recording system on the horse, whereby images are recorded onto a digital recorder or secure digital (SD) card and reviewed after completion of the test.

Factors to Consider when Choosing Equipment

EASE OF USE AND SAFETY

The system should be easy to apply, should stay in place during the examination, and should not provide a safety hazard for horse, rider, and equipment.

QUALITY OF THE IMAGE

This is determined by a combination of the CCD camera chip and lighting. Most systems use LED lighting at the endoscope, which enables miniaturization of the system by negating the need for an external light source. However, the illumination is considerably lower than that of light sources that are used with standard hospital-based videoendoscopes.

ABILITY TO FLUSH THE ENDOSCOPE DURING EXERCISE

The majority of horses have some mucus within the upper airways during exercise. This emanates from the trachea and may be increased in horses with lower airway disease. In many cases, mucus is cleared from the endoscope tip by the passage of air through the nasal passages at high flow rates during expiration. However, where mucus becomes lodged at the tip, this may significantly impair the image. In these instances it may be necessary to flush the endoscope tip with air and/or water. Ideally a system should enable flushing "on demand" by telemetry and care should be taken in interpreting clinical findings that coincide with flushing, such as swallowing or episodes of DDSP that may be induced by the presence of water within the nasopharynx.

ABILITY TO VIEW THE IMAGE IN REAL TIME: TELEMETRY DISTANCE

The ability to view the endoscopic image in real time is important (1) to ensure a diagnostic image is obtained and (2) to identify the timing of events that may occur during the exercise test. In particular, the muscles associated with the nasopharynx relax as the horse slows, which may result in pharyngeal wall collapse and/or palatal instability and displacement of the soft palate at this time, despite functioning normally during strenuous exercise. Also, dynamic airway collapse may be induced or exacerbated by other external factors including poll flexion, whether the horse is pulling hard, or whether the mouth is open.[20] It is important to be able to correlate this information to make appropriate judgments regarding treatment.

It is now possible to measure additional parameters (including speed, distance, and heart rate) during the exercise test using global positioning (GPS) and heart rate monitoring systems.[16,21-23] It would be beneficial to superimpose such data with the endoscopic image to identify the timing of events within the upper respiratory tract.

ABILITY TO VIEW THE IMAGE IN SLOW MOTION AFTER THE EXERCISE TEST

This is important because dynamic changes occur rapidly during strenuous exercise when the breathing frequency is in the region of 120 breaths per minute. It is therefore possible to miss subtle forms of dynamic collapse unless the recording is viewed in slow motion or on a frame-by-frame basis.

Setting up the Equipment and Positioning the Endoscope

Application of the equipment is best performed in a quiet environment (e.g., in a stable) rather than in the arena or start of the gallops, where horses are likely to be more excitable. As for resting endoscopy, passage of the endoscope is via the ventral meatus and is usually straightforward in the majority of horses with or without the use of a nose-twitch. It is useful to have an assistant hold the viewing screen while the scope is being positioned to visualise the nasal passages. The ability to move the endoscope tip is important for accurate positioning. However, in the majority of cases, when the position is set, there is little need to alter it during exercise.

Positioning of the tip of the endoscope (whether for treadmill or field-based endoscopy) is critical. If the endoscope is positioned too far rostrally, it may be difficult to observe all of the structures associated with the larynx (e.g., the vocal cords) and beyond (e.g., the cricotracheal membrane). This is particularly evident if lighting is suboptimal or if there is pharyngeal collapse that obscures the view of the larynx. However, if the endoscope is positioned too close to the larynx, it may not be possible to identify the presence of pharyngeal wall collapse or palatal instability. Ideally, the endoscope should be positioned so that the tip of the epiglottis is clearly visible (Figure 42-8). However, in some horses it may be necessary to readjust the endoscope and repeat the exercise test to visualise different parts of the upper airway.

Exercise Testing

The type of exercise test performed is crucial in enabling an accurate diagnosis of dynamic airway obstruction to be made. In cases where horses make obvious respiratory noise during training conditions, a diagnosis should be straightforward. However, for investigation of those cases, which make

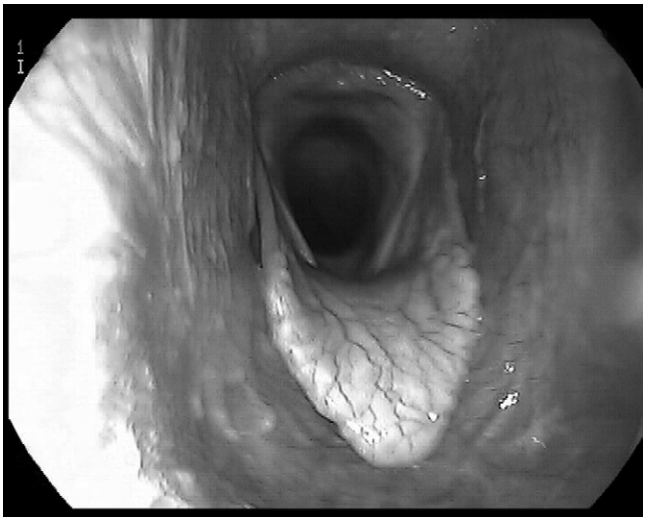


Figure 42-8. Optimal positioning of the endoscope for visualization of the pharynx and larynx during exercise. The tip of the epiglottis and caudal aspect of the soft palate should be clearly visible.

Figure 42-9. Cavison-mounted microphone used to record respiratory sounds in exercising horses.

abnormal respiratory noise only during competition or racing, it is essential to replicate the conditions encountered during competition. This also applies to those horses that present with poor performance and a history of "stopping" in races that are not reported to make abnormal respiratory noise. If the clinical signs reported during competition are not replicated, false-negative findings may occur.

Several studies have compared treadmill and overground endoscopy.[18,23] In pleasure horses, it was found that pharyngeal collapse was more readily diagnosed during overground endoscopy compared with treadmill endoscopy.[18] This is likely because of factors, including enforced poll flexion, that frequently predispose horses to develop dynamic airway collapse.[20,24,25] Changes in poll flexion are easier to recreate during ridden exercise, although it is possible to induce changes in poll flexion during treadmill exercise.[26,27] In racehorses, however, it was found that in some cases, palatal dysfunction and DDSP were easier to recreate on the treadmill than at the track.[8,28] Reasons for this may include a reluctance or inability to exercise horses to fatigue during field testing. DDSP predominantly occurs during the latter stages of a race as the horse starts to fatigue. It has been shown that DDSP is more likely to be observed in horses that undertake longer exercise tests and that tests on circular gallops are preferable to those performed in intervals on short gallops.[28] During treadmill exercise testing, horses are routinely run to the point of fatigue, and therefore there may be circumstances where treadmill assessment of poor performance is preferable. Treadmill exercise tests also have the advantages of being easier to standardize than field exercise tests[23,28] and enable the concurrent measurements of respiratory parameters that cannot currently be performed in the field.[19]

Nevertheless, it is likely that the technique of overground endoscopy will revolutionize the way dynamic airway collapse is diagnosed, thereby enabling a definitive diagnosis to be established in more horses. It also has the potential to facilitate further research into the pathogenesis of upper respiratory tract obstructions.

SOUND ANALYSIS

Frederik J. Derksen

Horses with upper airway conditions often have exercise intolerance and make a respiratory noise. Although reduced performance may be caused by dysfunction of many body systems, respiratory noise during exercise is a specific indication of upper airway disease. Furthermore, in sport horses, the respiratory noise associated with an upper airway condition is often more worrisome to the owner than reduced performance.

One cannot hear respiratory noises in exercising horses very well. Even the jockey, driver, or rider, who is nearest to the source of the noise most of the time, does not hear respiratory sounds well because the noise is obscured by other sounds associated with exercise, such as wind noise and footfall. At best, a listener standing near the track gets "a snapshot" of the noise. Also, respiratory sounds do not always occur throughout the exercise period. In some instances, the sounds are more obvious at maximum exercise, but in other cases the sounds are intermittent, or occur as the horse is finishing the exercise. For these reasons, recording of respiratory sounds in exercising horses is advantageous, and it also permits analysis after the recording. Recording of respiratory sounds in exercising horses is simple and inexpensive and requires minimal equipment. The sounds can be recorded throughout the exercise period, in the field, and under conditions in which the horse normally works. This gives the veterinarian complete and verifiable information about the respiratory noise.

Evaluation of respiratory sound during exercise is also useful in the assessment of effectiveness of surgical procedures used in the treatment of upper airway conditions.[29,30] In many horses with upper airway conditions, the primary objective of surgery is to reduce the respiratory noise. In these cases, evaluation of sound production before and after surgery is a good way to determine surgical success.

Methodology

Although several methods are described in the literature,[31-35] I record respiratory sounds in exercising horses in the following manner.[36] A unidirectional microphone with a cardioid pickup pattern is attached via a flexible wand to a cavison (Figure 42-9).

This type of microphone centers the sound pickup toward the front of the microphone and helps reduce extraneous sounds, such as footfall and track noise, that originate behind and to the side of the microphone. The microphone, covered with a windscreen, is placed equidistant between the horse's nostrils, approximately 4 cm (1½ inches) from the horse's nose. In this way, the microphone is as close to the source of sound to be recorded as possible without being placed in the respiratory air stream. The microphone is attached to an audio recorder with a compression circuit. This kind of recorder automatically adjusts the gain, decreasing the recording of extraneous noises. The advantages of this technique are that it is easily used in the field, and it records respiratory sounds that are also heard by human observers.

Just listening to the audiotape of respiratory sounds made by exercising horses is revealing. The listener can appreciate such factors as respiratory rate and consistency, the number of swallows, the frequency of stride lead changes, and whether abnormal respiratory sounds are present.

Sound can be characterized in terms of time, frequency, and intensity. A sound analysis technique that evaluates these characteristics concurrently is *spectrogram analysis*. A spectrogram is a three-dimensional plot of frequency on the vertical axis, time along the horizontal axis, and sound intensity in the third dimension. Sound intensity is often plotted in terms of color, or relative darkness. This powerful technique has been used commonly to study human voice or biological sounds, such as bird songs, and to evaluate upper respiratory sounds made by exercising horses.[37-40] Spectrogram analysis requires a personal computer and commercially available software. Listening to the audiotape and viewing the spectrogram at the same time is an effective method of evaluating sound.

Upper Airway Sounds in Exercising Horses

In normal exercising horses, most sounds are heard during exhalation and are present throughout this portion of the respiratory cycle (Figure 42-10).[29,30] Inspiratory sounds are less than half as loud as sounds made during exhalation.[29] Sound level increases as speed increases. Almost all of the sound intensity occurs at frequencies below 4 kHz, with most of the sound intensity concentrated at frequencies below 800 Hz.[29]

RLN and experimentally induced laryngeal hemiplegia (LH) are characterized by a loud inspiratory noise. This noise contains higher frequencies than normal expiratory sounds and has three bands in the frequency domain called *formants*. These formants are centered at approximately 300, 1700, and 3700 Hz (Figure 42-11).[29,30] Human hearing is most acute between 2000 and 4000 Hz. The sound formant centered at about 1700 Hz has significant sound intensity and is in the range of acute human hearing, so this is the one that contributes most to the perception of the high-frequency whistle or roar associated with RLN. The highest-frequency formant has the least sound intensity, and it is not present in all RLN-affected horses. The sound intensity of the formant centered at about 300 Hz is in a region where human hearing is less acute, so this lower-frequency noise does not appear prominent to the human ear. Whether the spectral pattern just described is unique for RLN remains to be determined. If this is the case, spectrum analysis of upper airway sounds in exercising horses could be used as a definitive diagnostic tool for RLN.

DDSP occurs intermittently during exercise; therefore, the abnormal noise associated with this condition is also intermittent.[29,35] Often, the soft palate displaces near the end of the exercise or as the horse pulls up. In some horses, the abnormal sound can be heard throughout the exercise period. In affected horses, expiratory sounds are abnormal. The noise is characterized by rattling, which is seen on the spectrogram in the time domain as bars of sound with a periodicity of about 32 msec (Figure 42-12). Most of the sound energy is below 4000 Hz, but the range is up to approximately 10,000 Hz.[29,41] In some horses with DDSP, respiratory sounds are normal, but in others, an inspiratory noise similar to the noise described for exhalation is also heard. In these cases, the inspiratory noise is often less loud than the expiratory noise. During exercise, affected horses often swallow frequently, presumably in an attempt to keep the soft palate in its normal position.

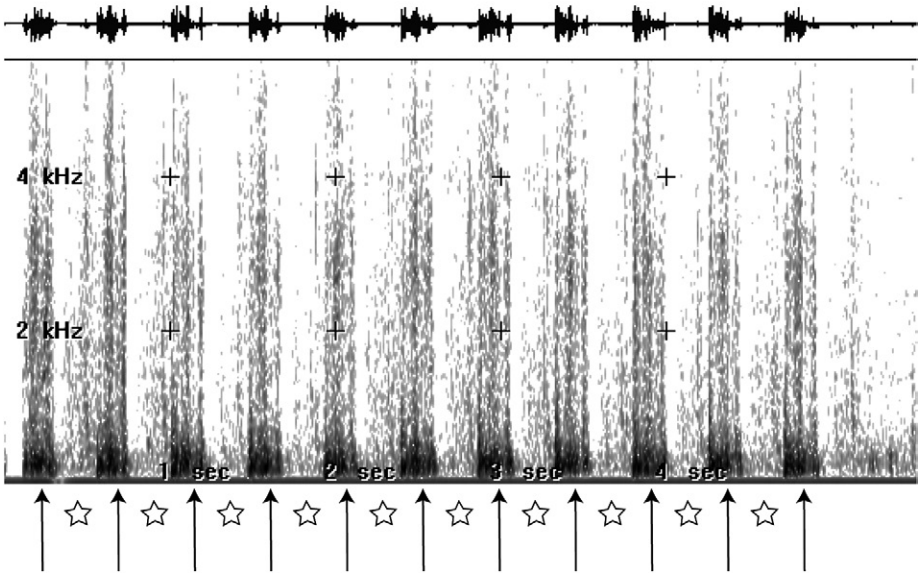


Figure 42-10. Spectrogram of respiratory sounds in a normal galloping horse. Timing is on the abscissa, frequency on the ordinate. The gray scale indicates sound intensity. Inspiration is indicated by a *star*, expiration by an *arrow*.

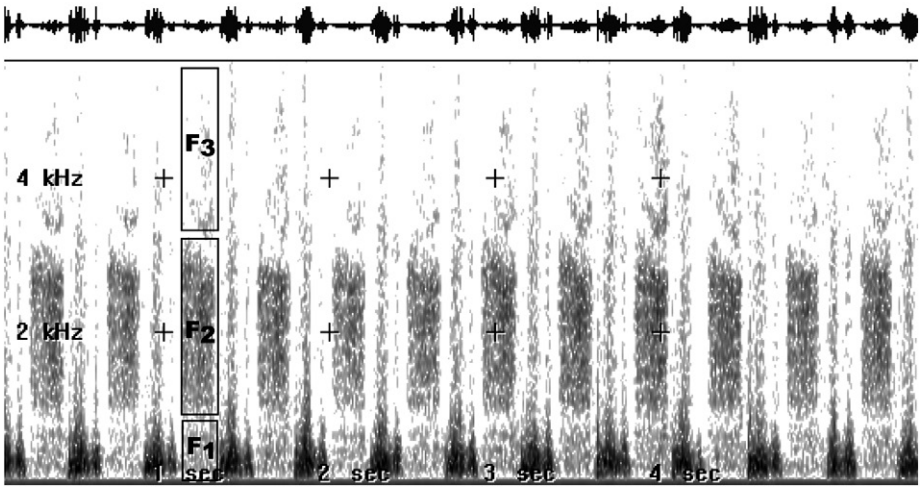


Figure 42-11. Spectrogram of respiratory sounds in a galloping horse with experimentally induced laryngeal hemiplegia. Notice the inspiratory bands of sound (formants) centered at approximately 0.3, 1.7, and 3.7 kHz (*F1*, *F2*, and *F3*, respectively).

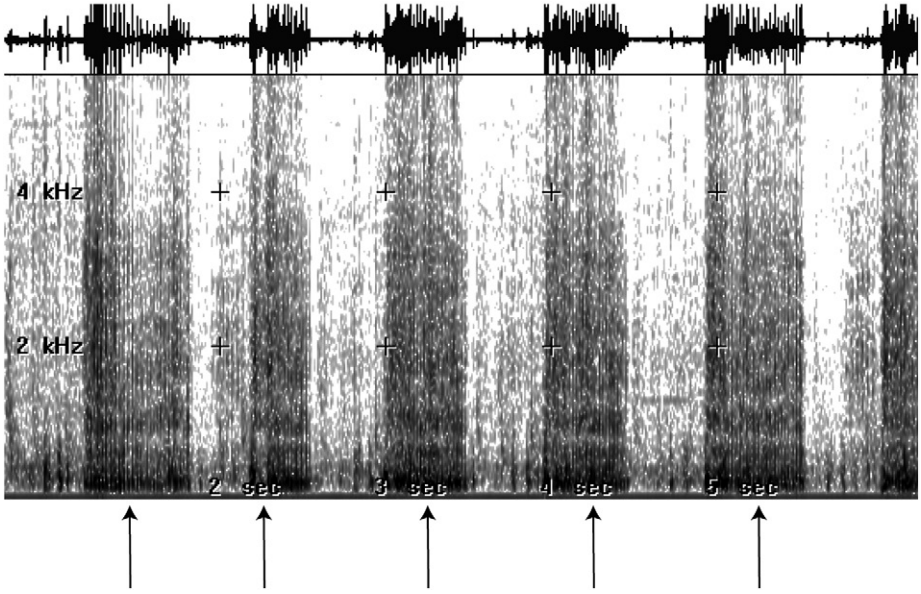


Figure 42-12. Spectrogram of respiratory sounds in a galloping horse with experimentally induced dorsal displacement of the soft palate. Note the "rattling" of the expiratory sound *(arrows)*.

It is likely that vibrations of the DDSP are the source of the upper airway noise.[41] The rate and amplitude of soft palate vibration are a function of the airflow velocity, the compliance of the DDSP, and the geometry of the individual horse's upper airway. Combined, these factors explain why some horses with DDSP make no noise, whereas in others the noise is also apparent during inhalation. Listening to the recorded respiratory sounds made by a horse with DDSP while concurrently viewing the spectrogram is an effective way to diagnose the condition.

RLN and DDSP can be experimentally reproduced and are the most common upper airway conditions of horses.[29,30] Consequently, noises associated with these conditions have been studied best. However, such upper airway conditions as pharyngeal collapse, epiglottic entrapment, aryepiglottic fold collapse, and rostral soft palate collapse are also associated with respiratory noise during exercise. It is possible that in the future each upper airway condition associated with noise production will have a well-described "voiceprint," allowing a presumptive spectrogram-based diagnosis of the condition in the field.

Effect of Surgical Treatment on Upper Airway Noise in Horses with Recurrent Laryngeal Neuropathy

Surgical procedures used to treat RLN include prosthetic laryngoplasty, various combinations of laryngeal ventricle and vocal cord excision, often combined with laryngoplasty, and nerve-muscle pedicle grafting. The effect of these surgical procedures on upper airway function has been well studied.[42-48] Other studies have investigated the possibility of these procedures to reduce respiratory noise.[29,30,49-51] In one study, bilateral ventriculocordectomy effectively reduced respiratory noise in horses with experimentally induced laryngeal hemiplegia.[30] Respiratory noises associated with the condition were almost completely eliminated, although spectrum analysis revealed some residual abnormal respiratory sounds (Figure 42-13). It takes

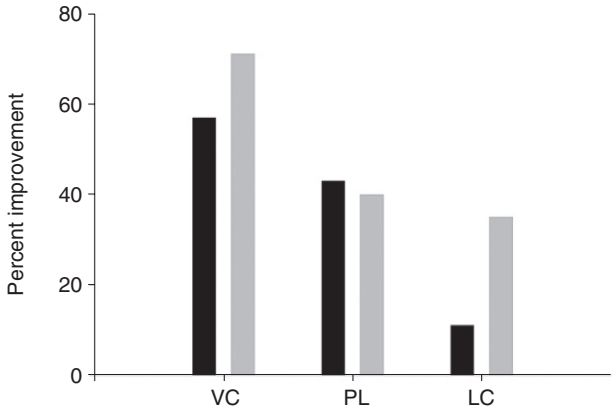


Figure 42-13. Percentage in improvement of inspiratory sound level *(black bar)* and sound intensity of formant 2 *(gray bar)* in galloping horses with laryngeal hemiplegia 90 days after treatment with bilateral ventriculocordectomy *(VC)*, prosthetic laryngoplasty *(PL)*, and unilateral laser cordectomy *(LC)*. Note that respiratory noise reduction is most effective after *VC*, and least effective after *LC*.

Figure 42-14. Horse with mask and ultrasonographic spirometer used to generate flow volume and flow time curves during high-speed treadmill exercise.

up to 90 days after the surgery until the abnormal respiratory sounds decline.

The most commonly used surgical technique to treat RLN is prosthetic laryngoplasty.[43] The procedure significantly improves upper airway flow mechanics in RLN-affected horses,[42] and many horses have raced successfully after surgery.[44,45] Prosthetic laryngoplasty reduces upper airway noise in horses with laryngeal hemiplegia, but it is not as effective as bilateral ventriculocordectomy in this regard (see Figure 42-13). Additionally, respiratory noise is reduced more rapidly than with bilateral ventriculocordectomy.[50] Residual respiratory noise after surgery is often used to determine the surgical success of prosthetic laryngoplasty.[43] This method of evaluation assumes a tight correlation between respiratory noise and airway obstruction. In fact, in horses with experimentally induced LH, the correlation between residual airway obstruction after prosthetic laryngoplasty and residual noise is weak.[50] Therefore, the residual noise during exercise cannot be used as a predictor of improvement in upper airway function in individual horses after laryngoplasty.

Endoscopically guided laser surgery has been used to perform vocal cordectomy in RLN-affected horses.[48] However, unilateral laser cordectomy does not effectively reduce upper airway noise in horses with experimentally induced laryngeal hemiplegia (see Figure 42-13).[51] Unilateral laser cordectomy results in removal of the left vocal cord but not of the entire laryngeal ventricle. Unilateral laser ventriculocordectomy is more effective.[52] These studies suggest that the laryngeal ventricle rather than the vocal cord may be the primary source of LH-associated noise. Recently, I compared the effects of bilateral ventriculectomy and ventriculocordectomy on upper airway noise in draught horses with RLN.[36] It was found that bilateral ventriculectomy and ventriculocordectomy both significantly reduce upper airway noise but that ventriculocordectomy was more effective than ventriculectomy.

In summary, respiratory sound recording in exercising horses is easy and inexpensive and can be accomplished under field conditions. The sound can be analyzed by simply listening to the recording and by spectrogram analysis. Just listening to the recording gives the veterinarian insight into breathing patterns and abnormal noises during exercise. Spectrogram analysis provides a voiceprint that in some cases suggests the presence of a specific upper airway condition. Respiratory sound analysis can also be helpful in evaluating surgical procedures aimed at reducing respiratory noise in horses with upper airway disease.

EXERCISE SPIROMETRY

Michael A. Weishaupt

Exercise spirometry is conducted with ultrasonic flow meters (Figure 42-14), which calculate the mean velocity of airflow passing through the two openings by measuring the absolute time of flight of short ultrasonic beams transmitted upstream and downstream during respiration (Exhalyzer V). Patients are trained to the high-speed treadmill wearing a face mask with an air-tight seal around the nose. When they are comfortable with the mask and with cantering on the treadmill, the spirometer is attached. In horses with obstructive diseases of the upper airways, inspiratory airflow values typically are altered to a much greater extent than expiratory values. Additionally, horses must be exercising at performance speeds for this test to be diagnosic. For example, in horses with unilateral grade 4 RLN, the first signs of inspiratory airflow limitation can be observed at the trot. However, conclusive inspiratory flow limitations usually occur only at canter speeds of 6 to 7.5 m/sec with a 6% incline (heart rates 160 to 210 min^{-1}).

Measurements of airflow mechanics have been performed to objectively assess the functional outcome of different surgical treatments for a variety of upper airway obstructive conditions.[40,42,49,53-57] Tidal breathing flow volume loops have proved to be a sensitive method to detect airway obstruction in exercising horses.[58] Exercise spirometry permits flow-volume loop and flow-time diagram data to be measured so that the significance of the upper airway obstruction can be determined. After clinically diagnosing a specific upper airway disorder, spirometry is then not only used to objectively assess the functional limitation of the obstruction but can also be used to detect the

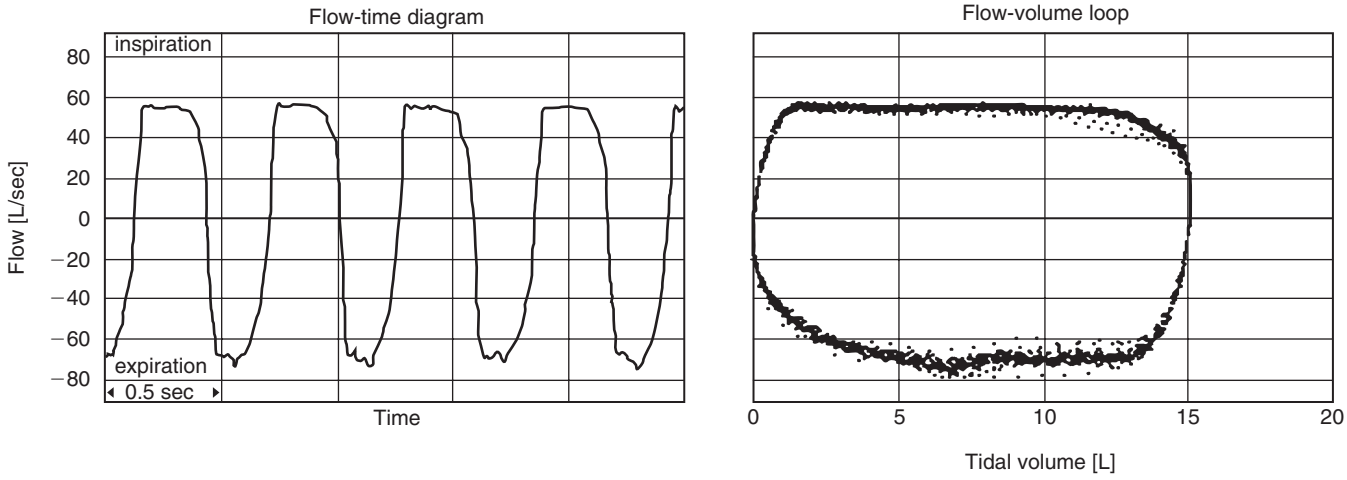


Figure 42-15. Representative flow-time diagram and tidal breathing flow-volume loop of a Warmblood horse with left recurrent laryngeal neuropathy. During the canter only minimal dynamic collapse of the left arytenoid was observed. The flow-volume loop *(right)* shows a typical flattening during inspiration *(top half of the loop)* as flow limitation occurs.

improvement of a surgical intervention in a follow-up spirometric analysis using the identical exercise protocol.

Examples of Airway Abnormalities Analyzed with Spirometry

Recurrent Laryngeal Neuropathy

In horses with grade 3 or 4 RLN with *mild collapse* of the airway, the inspiratory flow curve is characterized by a plateau phase, and peak values are limited between 28 and 60 L/sec (Figure 42-15), whereas at expiration, no limitation of airflow and peak flow values (up to 90 L/sec) occurs. To adapt to peak flow limitation while maintaining the tidal volumes necessary to meet the metabolic cost of the specific exercise intensity, the inspiratory time is prolonged. Inspiratory resistance typically increases so that the inspiratory time fraction is more than 50%.

Horses with *severe* dynamic collapse of the paralyzed arytenoid cartilage show an initial inspiratory flow peak with a subsequent characteristic drop in flow (Figure 42-16). At the canter, an affected horse changes its 1:1 locomotion-to-respiration coupling (LRC) intermittently or permanently to 2:1. When reassessing those patients after laryngoplasty combined with ventriculectomy, the most obvious changes can be observed in peak inspiratory flow (PIF) and minute ventilation (V_E). PIF increases between 30% to 70% and V_E up to 50%.

Dorsal Displacement of the Soft Palate

DDSP affects both inspiration and expiration, but the latter usually to a greater extent. DDSP causes a reduction of V_E and expiratory flow parameters, including peak expiratory flow (Figure 42-17).[25a,59]

Epiglottic Entrapment

An epiglottic entrapment has the potential to cause exercise intolerance in high-performance horses, even though some horses are capable of competing successfully with an entrapped epiglottis. The presence of severely inflamed and swollen entrapping tissue or the entrapment of the epiglottis in a semi-erected position exacerbates airway obstruction. In a case of intermittent epiglottic entrapment, inspiratory as well expiratory flow limitation was documented (Figure 42-18).[60]

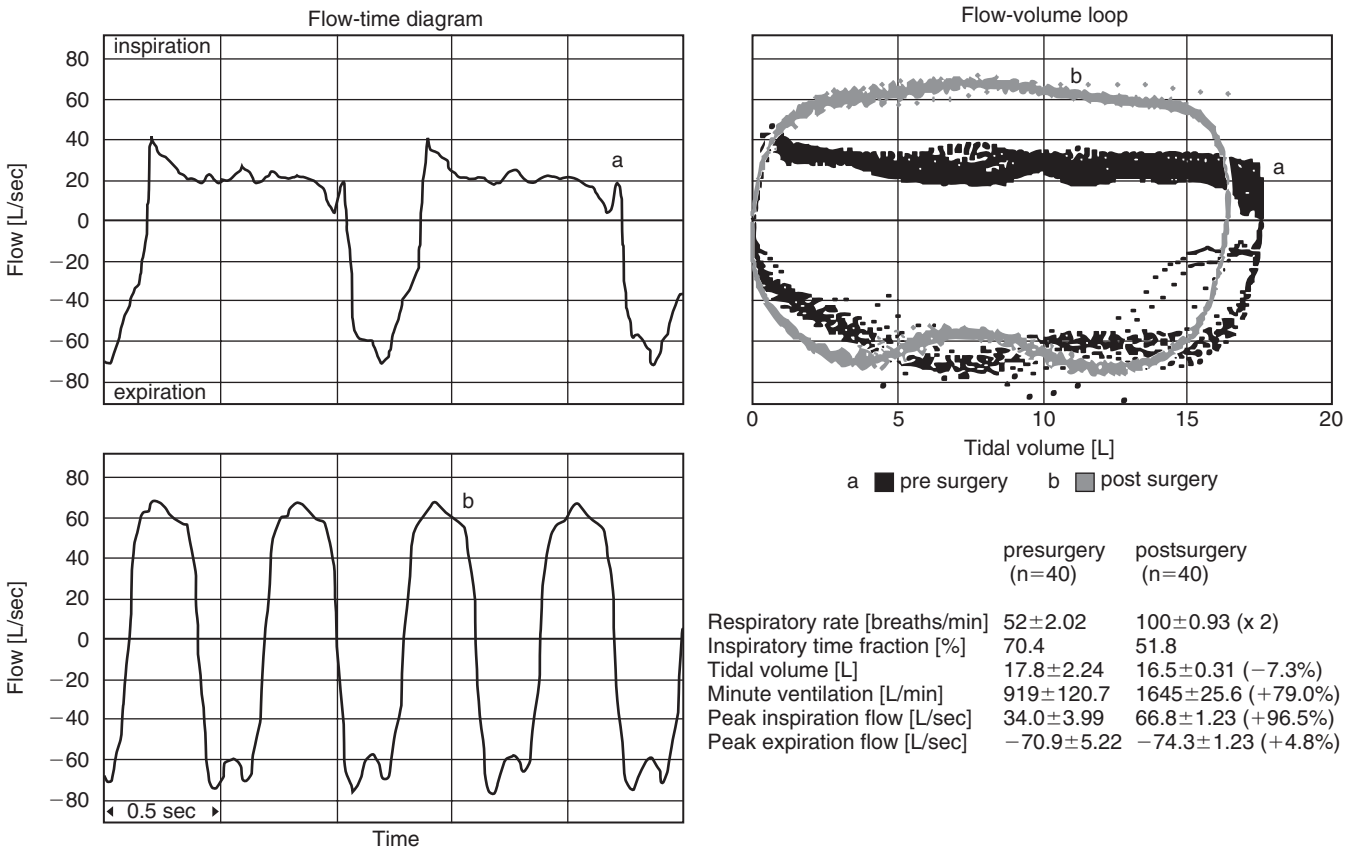
DIAGNOSTIC IMAGING

See also Section IX.

Ultrasonographic Examination of the Upper Airway

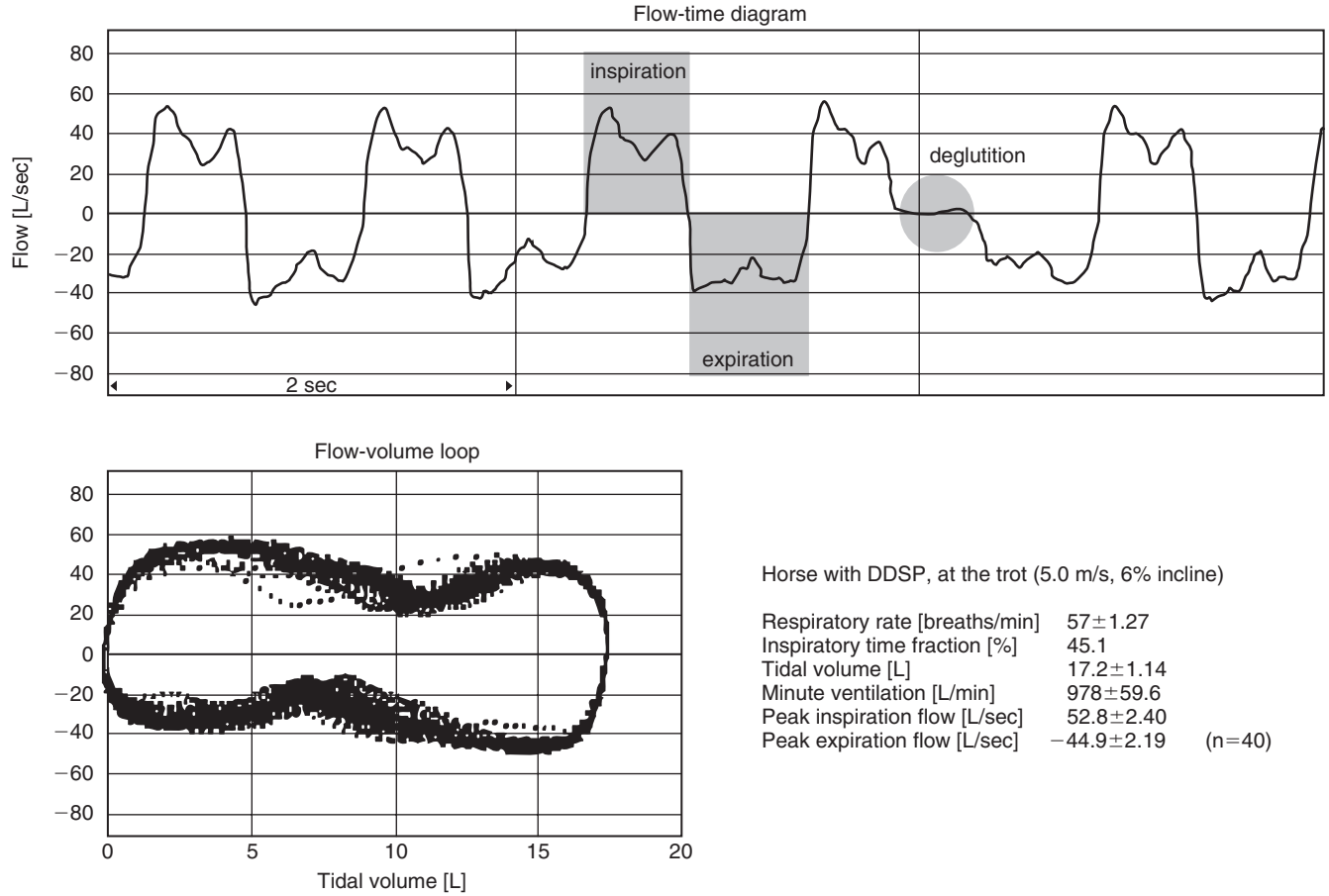
Heather J. Chalmers

Videoendoscopic examination at exercise is well established as the preferred means of evaluating the upper airway of horses of various disciplines. Ultrasonography has long been considered a key diagnostic tool for the equine respiratory tract, yet routine use of this modality has focused predominantly on the assessment of the lungs and pleural space.[61] More recently, the use of ultrasonography in the examination of the equine upper airway has been described as a useful adjunct diagnostic tool for the clinician.[62-64] Ultrasound examination of the larynx offers several advantages as it can be performed on the standing horse, offers both structural and functional information and allows the nonluminal side of the upper airway to be evaluated. Skillful laryngeal ultrasound can reveal important information about



	presurgery (n=40)	postsurgery (n=40)
Respiratory rate [breaths/min]	52±2.02	100±0.93 (x 2)
Inspiratory time fraction [%]	70.4	51.8
Tidal volume [L]	17.8±2.24	16.5±0.31 (−7.3%)
Minute ventilation [L/min]	919±120.7	1645±25.6 (+79.0%)
Peak inspiration flow [L/sec]	34.0±3.99	66.8±1.23 (+96.5%)
Peak expiration flow [L/sec]	−70.9±5.22	−74.3±1.23 (+4.8%)

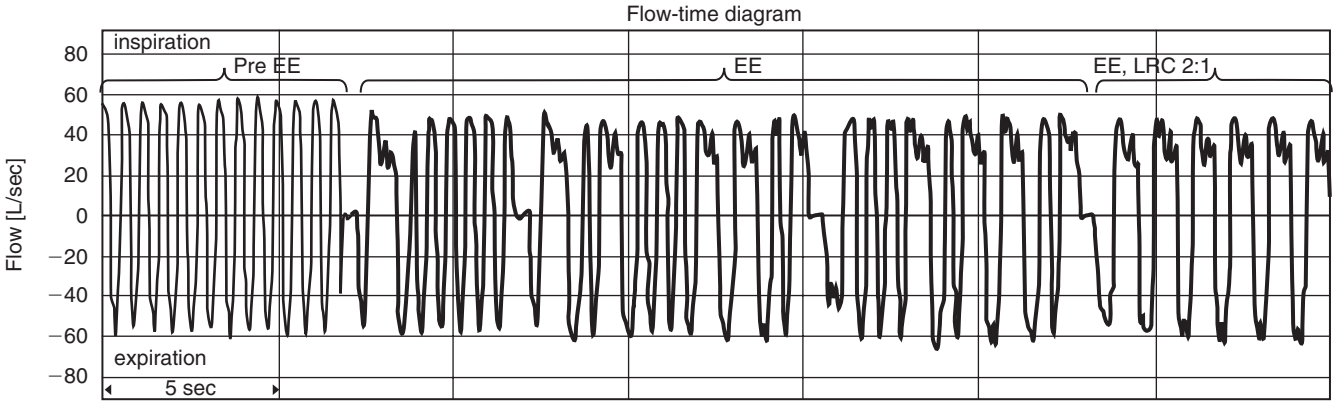
Figure 42-16. Flow-time diagram and tidal breathing flow-volume loop of a horse with *severe* collapse of the left arytenoid during exercise *(a).* The locomotion-to-respiration coupling has changed from 1:1 to 2:1 *(upper left graph)*, and the flow-volume loop shows initial inspiratory flows that decrease during peak inspiration. After surgery *(b),* the inspiratory flows are improved and the respiration-locomotion ratio has returned to 1:1.

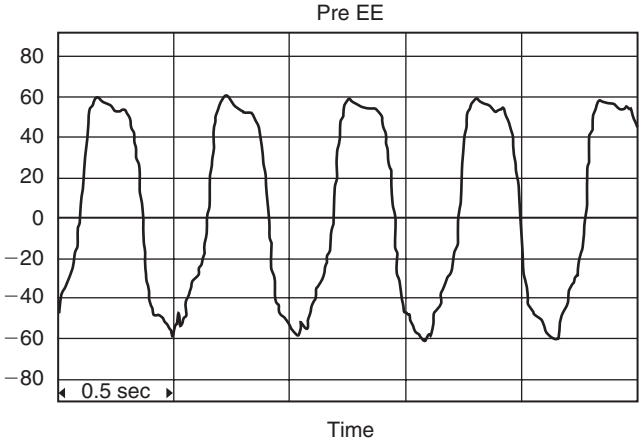


Horse with DDSP, at the trot (5.0 m/s, 6% incline)

Respiratory rate [breaths/min]	57±1.27	
Inspiratory time fraction [%]	45.1	
Tidal volume [L]	17.2±1.14	
Minute ventilation [L/min]	978±59.6	
Peak inspiration flow [L/sec]	52.8±2.40	
Peak expiration flow [L/sec]	−44.9±2.19	(n=40)

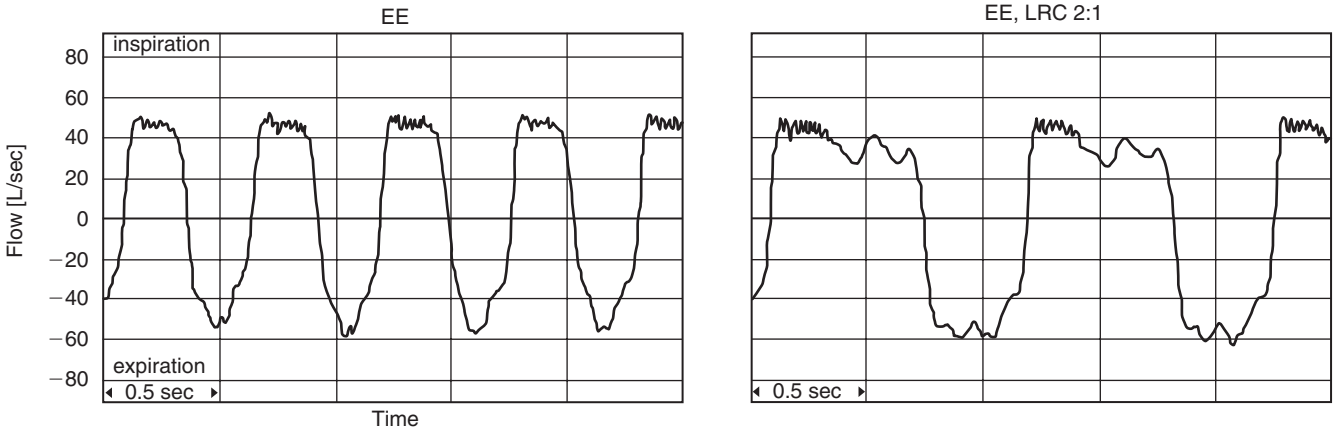
Figure 42-17. Flow-time diagram and tidal breathing flow-volume loops of a Standardbred trotter with a dorsally displaced soft palate. Expiratory and inspiratory flow patterns are altered and peak expiratory flows are reduced. Frequently zero-flow sections are observed, reflecting the repeated attempts of the horse to replace its epiglottis by swallowing. *DDSP,* Dorsal displacement of the soft palate.





A

	Pre EE
Respiratory rate [breaths/min]	110±0.71
Inspiratory time fraction [%]	48.8
Tidal volume [L]	12.2±0.14
Minute ventilation [L/min]	1348±18.9
Peak inspiration flow [L/sec]	58.7±0.87
Peak expiration flow [L/sec]	−59.1±1.92 (n=5)



	EE (n=5)	EE, LRC 2:1 (n=5)
Respiratory rate [breaths/min]	108±1.39	55±0.84 (x0.5)
Inspiratory time fraction [%]	50.4	59.4
Tidal volume [L]	11.1±0.35	23.0±0.52 (x2)
Minute ventilation [L/min]	1203±27.4	1261±18.2 (+4.8%)
Peak inspiration flow [L/sec]	49.6±0.53	47.4±0.52 (−4.4%)
Peak expiration flow [L/sec]	−56.9±1.86	−61.6±1.34 (+8.3%)

B

Figure 42-18. A *(top),* Flow-time diagram documenting the normal 1 : 1 pattern *(Pre-EE—left on top graph)* and the event of intermittent epiglottic entrapment *(EE—right on top graph); (bottom),* Slow-motion respiratory pattern of normal breathing *(left)* and actual flow data *(right).* **B,** Detailed comparison between pre-EE respiratory pattern *(top left)* and during EE *(top right)* as well as the actual flow data *(bottom right).* The disorder causes both inspiratory and expiratory airflow limitation. In this case, airway resistance exceeded a certain level, which forced the horse to give up intermittently its 1 : 1 locomotion-to-respiration coupling at the canter.

the location and extent of disease and may be used to corroborate or to question resting endoscopic findings.

Ultrasonography is especially useful in the evaluation of the structural status of tissues, making it highly complementary to the endoscopic evaluation in which the functional status is emphasized and the structural evaluation of tissues is limited to the luminal aspects only. Thus, ultrasonography and endoscopy should be considered complementary diagnostic tools that have the capability to augment the clinical data available for each case. Although laryngeal ultrasonography has not yet been compared to the results of over-ground endoscopy, ultrasonographic findings have been shown to have good agreement with treadmill video-endoscopic findings in horses with various upper airway problems.[62-65]

The upper airway structures that can be assessed with ultrasonography are limited in some locations by the presence of bone, mainly the hyoid apparatus and mandibles, and by the presence of gas within the airway lumen. When ultrasound waves travel through tissues and interact with a bone or gas interface, the waves are absorbed or reflected, which impairs imaging of deeper structures and results in an acoustic shadow.[66] Because of this restriction, and to optimize image acquisition, the use of established acoustic windows is recommended when evaluating the larynx with ultrasonography. Because the upper airway is complex, a thorough understanding of the regional anatomy is necessary to develop competency in this technique. The routine evaluation of the equine laryngeal region has been described using three ventral windows and one lateral window.[62] The structures evaluated at each acoustic window are summarized in Table 42-1. By performing a thorough and systematic evaluation, the operator can increase the information gained from each examination and become accustomed to the expected normal appearance at each site, thereby facilitating the recognition of abnormal findings and reducing the chances that lesions are missed. To date, the evaluation of the laryngeal and pharyngeal region with ultrasonography has been established as a diagnostic aid in several important conditions of the upper airway including DDSP, RLN, bilateral dynamic laryngeal collapse, arytenoid chondritis, and laryngeal masses and malformations.[62-65,67-70]

Dorsal Displacement of the Soft Palate

The ultrasonographic assessment of laryngohyoid position as a predictor of DDSP in horses has been reported.[63] The concept that the position of the larynx and hyoid apparatus could be an important factor in DDSP in horses originated from the finding that bilateral resection of the thyrohyoid muscles could cause DDSP in an experimental model.[59] The paired thyrohyoid muscles have their rostral insertion on the thyrohyoid bone and insert caudally on the lateral aspect of the thyroid cartilage lamina, effectively spanning the space between the larynx and the hyoid apparatus in a parasagittal manner.[71] Based on this anatomic location, the action of these muscles is reported to be to draw the larynx forward and draw the hyoid caudad.[71] The subsequent development of the laryngeal tie-forward procedure was aimed at mimicking the action of these muscles and has been shown to reduce the incidence of DDSP in clinical and experimental models.[59,72-74] These findings surrounding the role of the thyrohyoid muscles in DDSP led to the hypothesis that horses with DDSP could have a different relationship between the larynx and the hyoid apparatus, termed *laryngohyoid conformation*, than unaffected horses. This hypothesis was tested in a study of 56 racehorses undergoing treadmill evaluation for poor performance.[63] The difference in laryngohyoid conformation between affected and unaffected horses was assessed using ultrasonographic measurements taken percutaneously in the resting horse. In this population, a significant difference in laryngohyoid conformation was identified between affected and unaffected horses at the level of the basihyoid bone.[63] More specifically, when the depth of the basihyoid bone was measured at the base of the lingual process, the basihyoid location was significantly more shallow (ventral) in horses affected with DDSP compared

TABLE 42-1. Acoustic Windows Used in Routine Laryngeal Ultrasound Examination and Anatomic Structures Evaluated at Each Site

Acoustic Window (Anatomic Landmarks)	Structures Evaluated	Clinical Importance
Rostroventral (basihyoid bone)	Basihyoid bone including lingual process Base of tongue Ceratohyoid bones Mandibular lymph nodes	Basihyoid depth has been correlated to DDSP Postoperative assessment of laryngeal tie forward Ventral draining abscesses
Midventral (space between basihyoid and larynx)	Base of basihyoid bone Thyroid cartilage (ventral aspect) Strap muscles Thyrohyoid bones Thyrohyoid muscles	Relationship of larynx to hyoid apparatus Postoperative assessment
Caudoventral (cricothyroid notch)	Vocal folds Cricoid cartilage (ventral aspect) Cricothyroid muscle (ventral aspect) Rostral trachea	Vocal fold movement Cricothyroid muscle status
Lateral (right and left sides of larynx)	Cricoarytenoideus lateralis muscles (CAL) Arytenoid cartilage (caudal aspect) Cricoid cartilage (lateral aspect) Cricothyroid muscle (lateral aspect)	CAL muscle echogenicity in RLN Arytenoid movement Characterization of arytenoid lesions in chondritis

DDSP, Dorsal displacement of the soft palate; *RLN,* recurrent laryngeal neuropathy.

to unaffected horses (Figure 42-19). It is not known if this difference is related to the cause of DDSP or is a secondary effect of having DDSP. The reason for the observed differences in basihyoid position may relate to conformational differences and/or differences in strap muscle thickness between diseased and normal horses. The odds ratio (OR) of ultrasonographic assessment of basihyoid position for DDSP in the study population was approximately 17, meaning that for each 1 cm incremental increase in basihyoid depth the odds of DDSP decrease 17 fold.[63] As such, the basihyoid depth may be used in the resting horse as one possible indicator of DDSP; however, to use this criterion exclusively is not recommended. Rather, it is recommended to use the information gained through ultrasonography to ameliorate case selection for video endoscopy during exercise and to reduce the uncertainty about clinically suspected diagnoses. In addition to these diagnostic implications, the identification of an observable difference in laryngohyoid position between normal and affected horses at rest may have important pathophysiologic implications for DDSP, but these have not been fully explored to date.

Recurrent Laryngeal Neuropathy

Ultrasonography is reported as a convenient and noninvasive modality for assessing muscle tissue in normal and pathologic states.[75,76] RLN in horses is known to lead to changes in those muscles innervated by the left recurrent laryngeal nerve, including the *criroarytenoideus lateralis (CAL)* and the *cricoarytenoideus dorsalis (CAD)* muscles.[77,78] The effect of RLN in horses has been characterized as neurogenic atrophy of the muscles that it innervates, causing decreased movements of the arytenoid cartilage.[77] Histopatholigc abnormalities are reported to be present prior to clinical abnormalities, and the CAL muscle is affected first.[77,78] The use of ultrasonography to assess the CAL muscle for signs of neurogenic atrophy was originated to supplement resting endocscopic findings, especially for horses in which the resting grade does not correlate with the exercising grade. The hypothesis that increased muscle echogenicity would be present in horses with RLN was tested in 146 horses and compared with the clinical diagnostic standard of resting laryngoscopic examination.[79] In addition, 92 of these horses underwent upper airway videoendoscopy while exercising.[79] When compared with the resting laryngeal grading system, the test performance of the laryngeal ultrasound examination in the study population is as follows: sensitivity = 90.77% (95% confidence interval [CI], 80.97 to 95.90), specificity = 90% (CI, 81.4 to 95.3), positive predictive value = 88.06% (CI, 77.8 to 94.4), negative predictive value = 92.3% (CI, 84.1 to 96.6), odds ratio = 88.5 (95% CI, 27.43 to 301.94).[79] When compared with the exercising laryngeal grading system, the test performance is as follows: sensitivity = 91.9% (CI, 78.6 to 97.75), specificity = 96.3% (CI, 87.3 to 99.3), positive predictive value = 94.4% (CI, 81 to 99.0), negative predictive value = 94.5% (CI, 84.8 to 98.5), odds ratio = 244.16 (95% Sterne confidence intervals, 40.1 to 2039.1). Agreement between the testing methods was assessed using the kappa statistic; between resting laryngeal grade and exercising laryngeal grade the kappa is 0.7702 (95% CI, 0.636 to 0.904, $p < 0.001$). The kappa for CAL echogenicity and exercising laryngeal grade is 0.886 (95% CI, 78.8 to 98.3, $p < 0.001$). The findings to date demonstrate the capability of ultrasonography in assessing intrinsic laryngeal muscles for evidence of neurogenic atrophy and support the use of this technique to improve diagnostic accuracy in

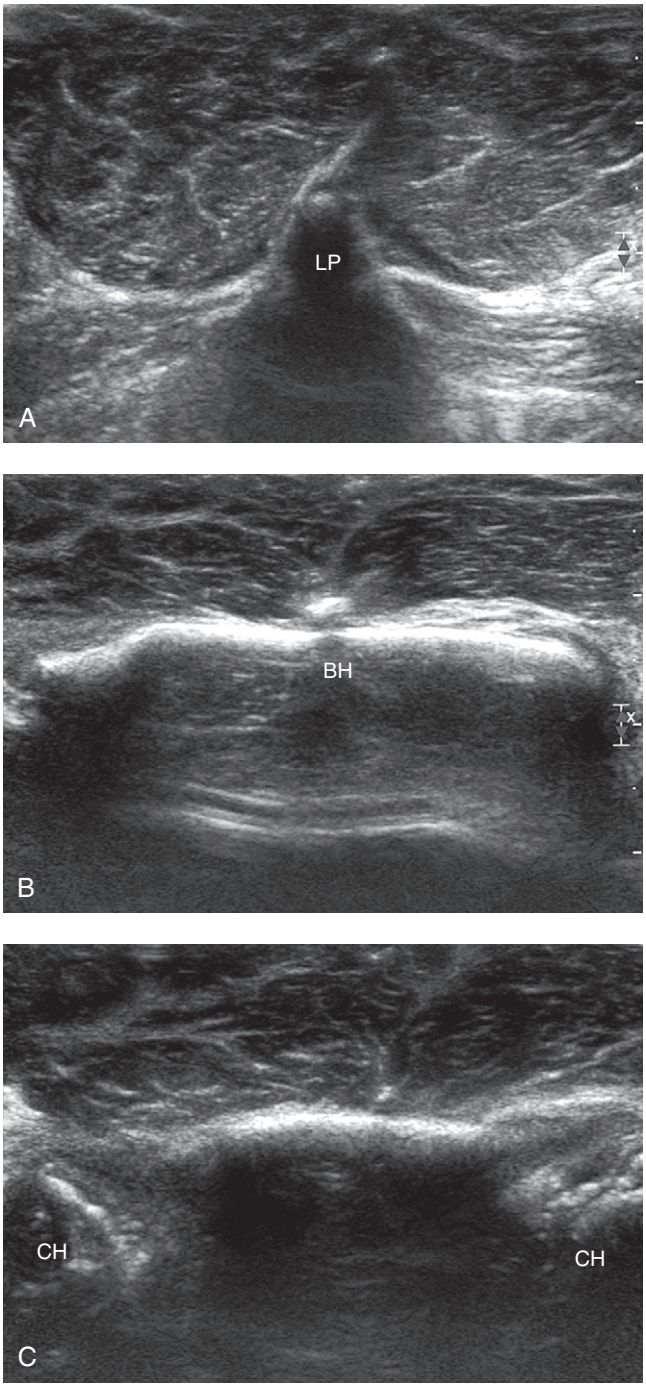


Figure 42-19. Composite images obtained from the rostroventral acoustic window at three different locations in the transverse plane using a 12.5 MHz linear transducer. From left to right the images are progressing from rostral **(A)** to caudal **(C)** within the rostroventral acoustic window. **A,** The lingual process *(LP)* is readily identified as a hyperechoic midline structure immediately dorsal (deep) to the paired *geniohyoideus* muscles. Commencing the exam at this site is recommended because the lingual process is easy to locate. **B,** From the level of the lingual process, moving the probe caudad to follow the lingual process along its entire length leads to the base of the basihyoid bone *(BH).* At this level, there is an association between the basihyoid bone depth and DDSP.[55] **C,** At the junction of the lingual process and base of the basihyoid bone, one can roll the probe slightly rostrad from a midline position to image the left and right ceratohyoid bones *(CH).* The ceratohyoid bones are paired, flat hyperechoic structures coursing dorsad (deep) in the image. Only the more ventral portion of the ceratohyoids is imaged; it is generally not possible to follow them to the level of the stylohyoid bones.

clinical cases (Figures 42-20 and 42-21).[64,79] Further, these findings suggest that ultrasonography may be superior to resting laryngeal grade in predicting the exercising status of the larynx.[79] Of particular interest, in horses in which the resting laryngeal grade does not appropriately predict the exercising laryngeal grade, ultrasonography provided a more accurate diagnosis prior to treadmill examination.[64,79]

Arytenoid Chondritis

Arytenoid chondritis or *chondropathy* is a term used to refer to a range of abnormalities of the arytenoid cartilage(s), which may be inflammatory, infectious, traumatic, or idiopathic in origin.[80-84] The spectrum of abnormalities encountered is broad and can include granuloma formation, draining tracts, distortion and displacement of the corniculate process, and laryngeal malfunction.[80-84] Regardless of the underlying cause, the extent of involvement of the arytenoid cartilages is variable and may include focal pedunculated arytenoid lesions or diffuse involvement of the entire cartilage with internal abscessation. Accurate assessment of disease location and severity has a key role in treatment planning and prognostication.[82-84] Ultrasonography is useful in these cases because it can assess the abaxial aspects and thickness of the arytenoid cartilages.[62] In addition, from the lateral acoustic window, the status of tissues adjacent to the arytnoid cartilage can be assessed to identify any involvement including the thyroid cartilage and the space between the thyroid and arytenoid cartilages.[62]

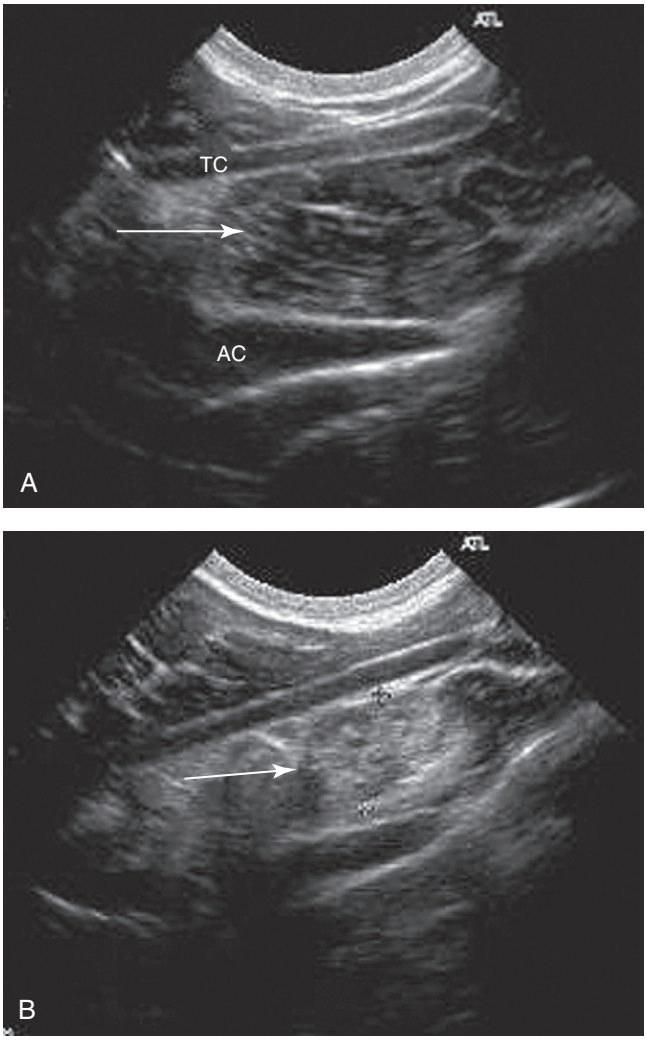


Figure 42-20. The normal right **(A)** and affected left **(B)** lateral acoustic windows from a 3-year-old Thoroughbred with grade 2-C recurrent laryngeal neuropathy obtained using an 8.5 MHz micro-convex probe. The probe is held in a rostrocaudal plane just caudal to the mandibles at a slightly ventral angle so the imaging plane is parallel with the line of the cervical vertebral column. The cricoarytenoideus lateralis *(white arrow)* muscle belly is imaged between the thyroid *(TC)* and arytenoid cartilages *(AC)* and has increased echogenicity with loss of normal muscular fiber pattern on the horses left side **(B)** compared to the normal right side **(A).**

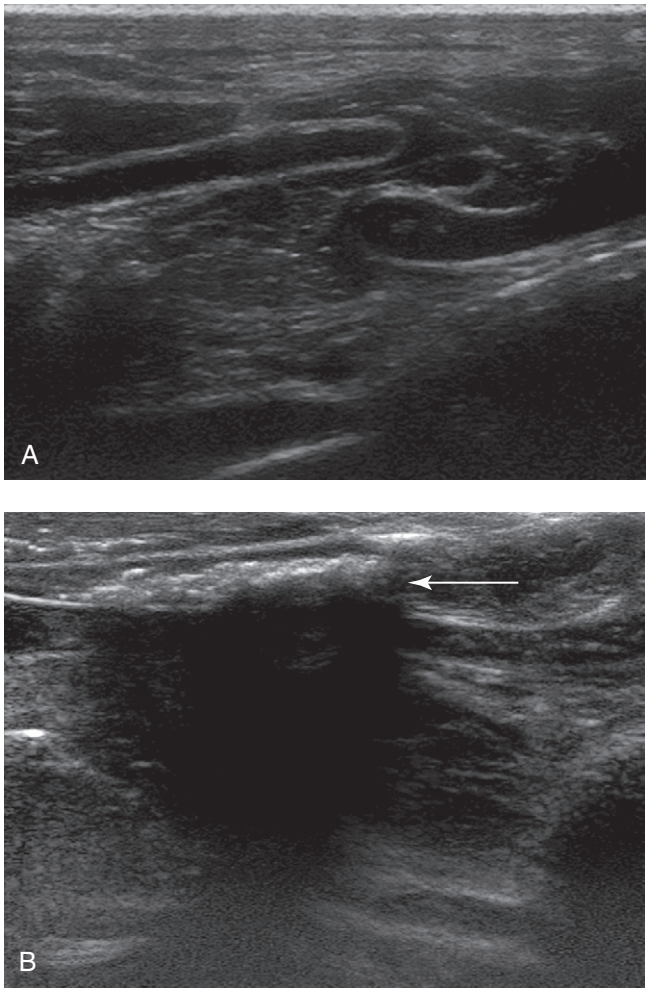


Figure 42-21. Composite image of the lateral acoustic window obtained using a 15 MHz linear array transducer from a yearling standardbred horse **(A)** and a 10-year-old Standardbred horse **(B).** In the yearling, the CAL muscle has normal appearance and the laryngeal cartilages are uniform and hypoehoic. In the older horse **(B)** disruption of the expected appearance of anechoic cartilage is seen with distinct mineralization of the thyroid cartilage *(arrow)* which casts and acoustic shadow over the CAL muscle, making it difficult to image. This type of mineralization, seen in older horses, can range from tiny pinpoint hyperechoic foci to more generalized mineralization that impedes the ultrasound examination as shown here.

Miscellaneous Conditions

The successful use of ultrasonography to evaluate various lesions of the larynx in horses has been reported, including lesions of neoplastic, traumatic, congenital, and infectious eitiology.[67-70] In my experience, routine examination is useful in identifying disease extent and location. Expanded imaging for conditions may include retropharyngeal and mandibular lymph nodes, and extension of the acoustic windows rostrally and caudally to fully trace diseased tissues and to follow draining tracts. Ultrasonography is particularly useful in identifying locations that may be amenable to fine needle aspiration or biopsy to facilitate diagnosis and to differentiate the underlying etiology. The use of ultrasonography as a diagnostic tool in congenital malformation of the larynx has been reported in five horses with fourth branchial arch defect (4-BAD).[70] In these cases, the absence of the cricothyroid articulation is described as a key diagnostic feature and was visible with both ultrasonography and magnetic resonance imaging.[70]

Ultrasound examination of the laryngeal and pharyngeal region is typically normal in dynamic conditions of the upper airway, such as nasopharyngeal collapse and aryepiglottic fold collapse. The use of ultrasonography to assess the larynx in bilateral dynamic laryngeal collapse in Norwegian trotting horses has been described as a useful adjunctive means of assessing the horses at rest.[65] In these patients, the ultrasound examination was performed in different head positions and an association was found between the change in apparent lumen width that occurred with poll flexion and bilateral dynamic laryngeal collapse.[65] Although the potential utility of laryngeal ultrasonography for other conditions of the upper airway is still being developed, the examination appears to be especially contributory in cases of laryngeal and perilaryngeal masses and malformations and when evaluating postoperative complications. Further, because the pathogenesis of most upper airway problems is unclear, the information gleaned from such investigations can contribute to understanding the mechanisms of these diseases. Laryngeal ultrasonography adds a different perspective (i.e., extraluminal) to the assessment of the structural status of a horse's larynx. Skillful ultrasonography of the equine upper airway can reveal important information about the location and extent of disease and may be used to corroborate or question resting endoscopic findings and select cases that would benefit most from exercising evaluation. Laryngeal ultrasonography and resting and high-speed video endoscopy should be seen as complementary, rather than competitive, modalities.

Radiography

Eric J. Parente

Although treadmill endoscopy is invaluable for evaluating the dynamics of the upper respiratory tract during exercise, other imaging techniques, such as radiography and computed tomography (CT), are just as essential in fully evaluating the upper respiratory tract. Radiography and CT are particularly valuable in the assessment of structural abnormalities in areas that are difficult to explore with the endoscope.

The larynx, pharynx, and guttural pouches can be clearly imaged with standard radiographic techniques. Computed radiography and digital radiography provide even greater detail and have the advantage of providing images can be manipulated. To acquire a lateral image of the laryngeal structures, the x-ray beam should be centered just rostral and dorsal to the angle of the mandible. A dorsoventral projection is usually not of benefit in evaluating these structures.

Air throughout the upper respiratory tract provides good contrast for structures within the pharynx and larynx. Any masses or swelling external to the lumen will lead to obvious compression of the pharyngeal vault. The epiglottis should appear as a thin structure on top of the soft palate. Subepiglottic or palatal lesions may cause dorsal displacement of the soft palate (Figure 42-22), but heavy sedation of the horse may also precipitate displacement of the palate, and this finding should not be overinterpreted. Measurements have been made of epiglottic length on lateral radiographs and correlated with dynamic upper respiratory dysfunction.[85] Clinicians depend more often on their endoscopic assessment of epiglottic length and apparent stiffness than on radiography. The arytenoids and the remainder of the laryngeal cartilages can be seen dorsal and caudal to the epiglottis. Mineralization of these structures may be associated with normal aging or with an inflammatory process, such as arytenoid chondritis.[86,87] Infrequently, abnormal air density may be seen around the larynx and could be consistent with severe laryngeal infection. The saccules should be seen as small air densities between the epiglottis and corniculate processes.

The guttural pouches are thin-walled, air-filled cavities that extend from the pharynx and project dorsal to the larynx. The guttural pouches are outpouchings of the eustachian tubes, and normally the stylohyoid bone is seen traversing through the guttural pouch. Enlarged retropharyngeal lymph nodes may lead to compression of the guttural pouch ventrally. Fluid associated with guttural pouch empyema or hemorrhage can typically be seen as increased soft tissue density or a fluid line. Temporohyoid disease may be seen as enlargement of the stylohyoid bone, but this condition is more reliably detected during the endoscopic examination.[88]

Radiography can be very beneficial in imaging most abnormalities within the sinus, but the technique does have limitations. Dorsoventral, lateral, and oblique projections are the minimum of radiographic views that should be taken to fully

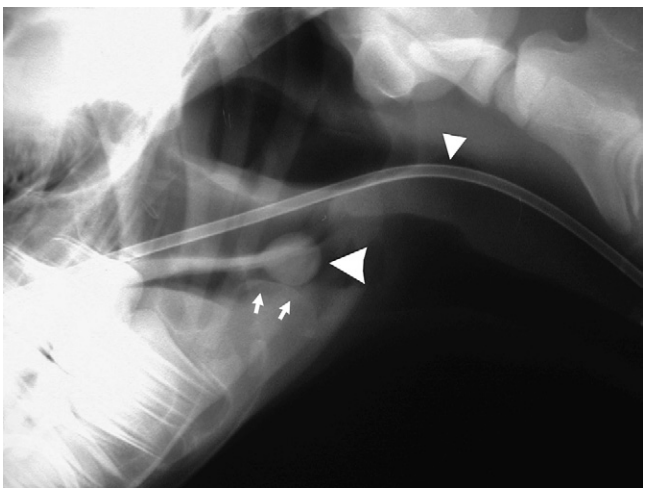


Figure 42-22. Lateral radiographic image of a foal with persistent dorsal displacement of the soft palate. The epiglottis *(small arrows)* is unable to assume its normal position because of a large cyst *(large arrowhead)* on the free edge of the palate. A feeding tube is in the esophagus *(small arrowhead).*

evaluate the sinuses. Although air density provides good contrast, superimposition of structures can make interpretation difficult, and familiarity with normal sinus architecture is essential. A fluid line can be seen in the lateral projection in many cases of sinusitis (Figure 42-23), but if the entire sinus is full, only increased soft tissue density will be seen. Often the borders of the frontal and maxillary sinuses can be discerned easily. The septum between the caudal and rostral maxillary sinuses can be best appreciated on a lateral radiograph, but sinusitis or neoplasia may result in significant distortion of normal anatomy. Contrast sinusography can be used to better help delineate abnormalities. Contrast material can be injected through standard portals for sinus centesis, and filling defects will define the extent of lesions.[89,90]

Sinusitis can be primary or secondary to another problem, such as tooth infection or neoplasia. It can be difficult to make this distinction with standard radiography, but this determination is essential in developing an appropriate treatment plan. If bony lysis is present on standard radiographs, the sinusitis is most likely secondary to some other, primary problem, but the absence of lysis is not definitive for a primary sinusitis. Sinuscopy can be used to visualize the internal aspects of the sinus, but it has limited application (see Chapter 43).[91] More sensitive imaging techniques are often of greater benefit.

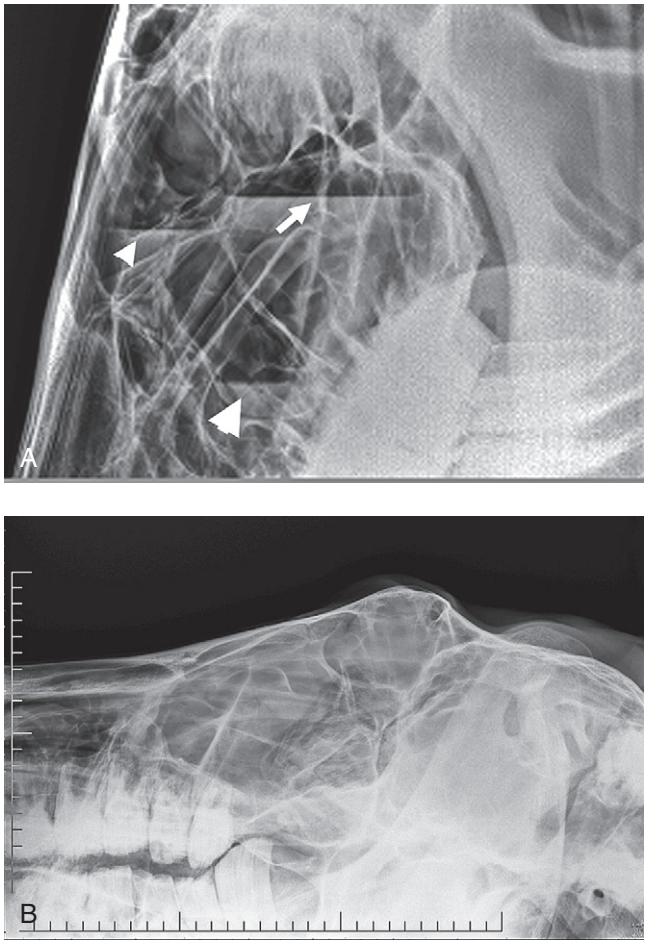


Figure 42-23. A, Lateral skull radiograph with multiple fluid lines. Rostral maxillary sinus *(large arrowhead),* caudal maxillary sinus *(arrow),* dorsal conchal sinus *(small arrowhead).* **B,** Oblique radiograph of the skull with complete opacity of sinus from chronic disease.

Scintigraphy

Eric J. Parente

Scintigraphy is an extremely sensitive technique for detecting bony remodeling. It can be used to evaluate the equine head reliably for the presence of dental disease, and it can be used to distinguish among dental disease, neoplasia, and primary sinusitis.[92] Bone phase images are acquired 3 to 4 hours after an intravenous injection of a radioisotope. The images are obtained by a gamma camera, and regions of interest are analyzed. Although scintigraphy can be used to determine the presence of dental disease, it may be difficult to discern the specific tooth that is involved with great certainty. Scintigraphy has a significant advantage over other imaging techniques because general anesthesia is not required. The images are acquired with the standing animal sedated, and multiple views can be taken to help minimize superimposition of structures. Many hospitals now have scintigraphic capability.

Computed Tomography

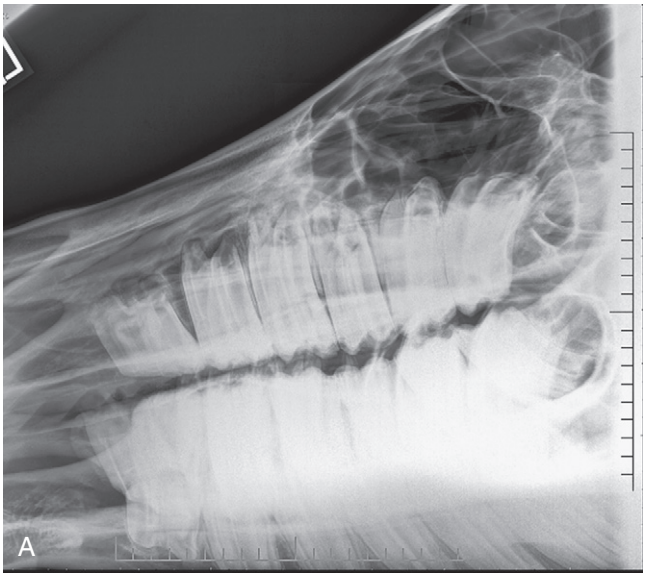
Caroline Tessier

Computed tomography (CT) has a distinct advantage over scintigraphy and standard radiography because highly detailed cross-sectional images of the nasal cavity and paranasal sinuses can be obtained. CT can discriminate tissues with only a 0.5% difference in density, and a 10% change in density is necessary to detect a radiographic difference.[93] The images are obtained using x-rays, but the x-ray tube and detectors encircle the patient, acquiring images from many angles. The resulting images are cross-sectional slices of a specified thickness (Figure 42-24). This eliminates the problem of superimposition encountered with conventional radiography, and it allows precise determination of an abnormality. By knowing the extent of a lesion in greater detail, a more precise surgical plan can be made. CT slices are always oriented perpendicular to the long axis of the table and body region in the gantry. The CT images can be reconstructed with computer software to provide images in other planes, but there is a significant loss of image quality. Imaging techniques[94] and normal CT anatomy of the equine head[95] have been described. CT imaging of the equine head can be performed in a relatively short time; therefore, surgical treatment can often be performed in the same anesthetic episode. It has proved to be a very valuable modality in imaging paranasal sinus lesions in horses[96,97] and is routinely used in some facilities in the preoperative planning of sinus surgery.

Magnetic Resonance Imaging

Caroline Tessier

Magnetic resonance imaging (MRI) enables the generation of high-resolution cross-sectional images of the head in horses. Although its use has become widely accepted for soft tissue lesions of the distal limb, MRI is now recognized also as a valuable tool to image the head of horses. It is generally thought to be a superior imaging technique for soft tissue, but MRI can be just as good at discerning bony abnormalities. The normal cross-sectional anatomy of the equine paranasal sinuses[98] as well as the pharynx and larynx[99] have been described. Distinct advantages of MRI over CT are that MRI can be multiplanar without loss of image quality and that MRI displays anatomy and pathology in the same examination. MRIs are usually



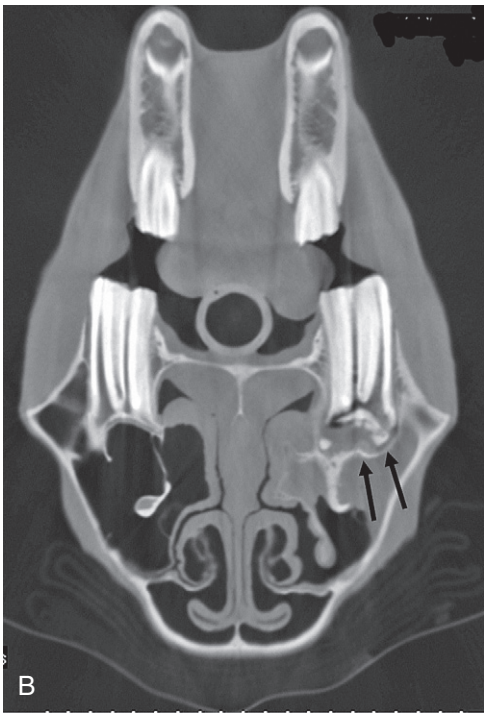


Figure 42-24. A, Lateral radiograph of a horse with purulent drainage from one side of the face and from the ipsilateral nostril. There is no bony lysis around any tooth roots to confirm a suspected diagnosis of tooth root infection. **B,** The cross-sectional CT image at the level of the first molar of the same horse in dorsal recumbency and under general anesthesia. The root structures on the left side are abnormal *(arrows)* and consistent with infection. (Courtesy Section of Veterinary Imaging, Cornell College of Veterinary Medicine.)

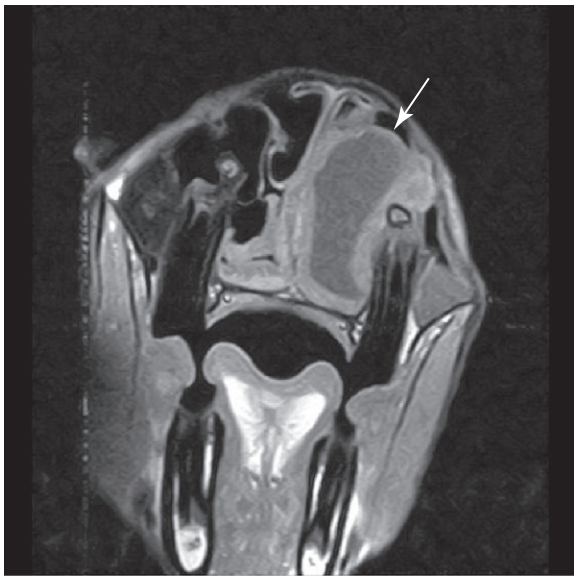


Figure 42-25. Transverse T1-weighted sequence at the level of the first molar of a horse with airway obstruction, facial deformity, and unilateral nasal discharge. There is a space-occupying lesion in the left middle meatus. The fluid is hypointense to the temporal muscles and surrounded by a wall *(arrow)*. The lesion was a paranasal sinus cyst. (Courtesy Division of Clinical Radiology, VetSuisse Faculty of Bern, Switzerland.)

Figure 42-26. Parasagittal T2-weighted sequence of a horse with airway obstruction and nasal discharge. Multiple cystic lesions are visible. Note also the presence of hyperintense signal in the maxillary sinus. This represented a secondary sinusitis due to airway obstruction caused by the cystic lesions. (Courtesy Division of Clinical Radiology, VetSuisse Faculty of Bern, Switzerland.)

acquired in transverse, dorsal, and sagittal planes. Slice number and slice thickness can vary according to the area of interest. The most useful sequences to image the equine head are transverse T1-weighted (pre- and postcontrast), transverse T2-weighted and proton density, sagittal T1-weighted (pre- and postcontrast), and dorsal T1-weighted sequences.[100] Imaging of the larynx and pharynx offers excellent three-dimensional display and good soft tissue discrimination. It is particularly helpful in preoperative imaging of complex laryngeal dysplasia.[101] MRI is also a valuable imaging modality in sinonasal disease, particularly in soft tissue lesions (Figure 42-25). The technique is extremely sensitive in detecting inflammatory changes, and fluids with different densities can be identified (Figure 42-26). MRI is the diagnostic modality of choice in patients with suspected neoplasia, as it can display very early the extension of lesions within surrounding tissues

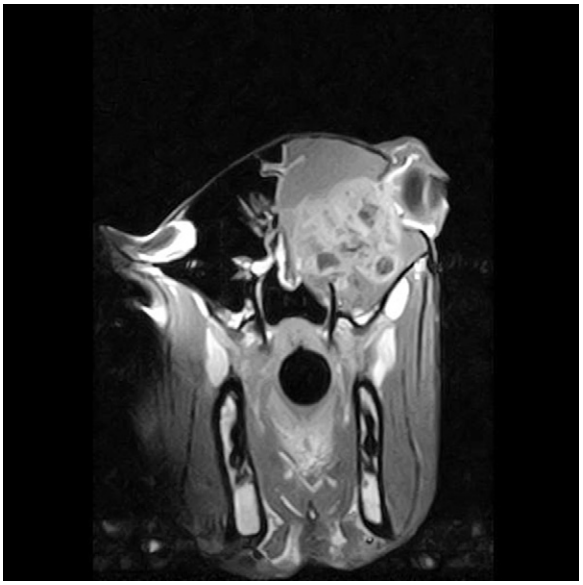


Figure 42-27. Transverse T1-weighted sequence at the level of the orbit of a horse with airway obstruction and facial deformity. There is a large inhomogeneous space-occupying lesion of mixed signal intensity surrounded by hypointense fluid. Destruction and infiltration of the orbital bones can be observed. Displacement of the conchae and ethmoid turbinates can be seen. The lesion was a sarcoma. (Courtesy Division of Clinical Radiology, VetSuisse Faculty of Bern, Switzerland.)

(Figure 42-27). However, the interpretation of images should be complemented by clinical findings. Furthermore, acquisition and interpretation of MRIs is lengthy, and a separate anesthetic episode might be required if surgical treatment is planned.

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CHAPTER 43

Nasal Passages and Paranasal Sinuses

Frank A. Nickels

FUNCTIONAL ANATOMY

External Nares

The external nares or nostrils in the horse are the openings into the nasal passages; they are widely placed and have an unusual shape.[1] The dorsal and lateral margins of the nares, the alae, are supported by the alar cartilages, which are attached to the rostral end of the nasal septum dorsally. The thick alar fold divides each naris into dorsal and ventral parts. The dorsal part leads into a blind sac called the nasal diverticulum, which occupies the nasoincisive notch formed by the incisive bone ventrally and the nasal bone dorsally. The ventral part of the naris leads directly into the nasal cavity.

Nasal Cavity

The nasal cavity is divided into equal halves by the nasal septum and the vomer bone. The reserve crowns of the upper cheek teeth and a portion of the paranasal sinus system occupy this area of the skull. Two major nasal conchae (thin scrolls of cartilage and bone) in each nasal cavity divide the nasal passage into the dorsal, middle, ventral, and common meatus. The dorsal concha extends from the cribriform plate of the ethmoid bone to the level of the 106/206 (second premolar in the upper arcade). The ventral concha extends from the level of the 111/211 (third molar) to the level of 106/206 (see Chapter 30).[2] Numerous small ethmoturbinates project into the caudal part of the nasal cavity from the lateral mass of the ethmoid bone.

Paranasal Sinuses

The horse's extensive paranasal sinus system consists of six pairs of sinuses: the dorsal (endoturbinate I), middle (endoturbinate II), and ventral nasal conchal sinuses; the sphenopalatine sinus; the frontal sinus; and the maxillary sinus (Figure 43-1). The major clinically significant sinuses are the frontal and maxillary sinuses. All the sinuses communicate with the nasal cavity directly (maxillary sinus) or indirectly (dorsal, middle, and ventral conchal sinuses; frontal sinus; and sphenopalatine sinuses) through the maxillary sinus. The paranasal sinuses are important clinically because they are susceptible to infections that extend from the nasal cavity or from the alveoli of the caudal upper cheek teeth (see Chapter 30).

Conchal Sinuses

Each conchal sinus is divided into a rostral and a caudal compartment by a septum. The caudal compartment of the dorsal concha forms the dorsal conchal sinus, which communicates with the frontal sinus, forming the conchofrontal sinus. The caudal compartment of the ventral concha forms the ventral conchal sinus, which communicates with the rostral maxillary sinus over the infraorbital canal through the conchomaxillary opening.[2]

Maxillary Sinus

The maxillary sinus is the largest of the sinuses. It is divided into rostral and caudal compartments by an oblique septum. The root of the maxillary first molar tooth (109/209) (see Figure 30-1) usually enters the rostral compartment, and the roots of the second (110/210) and third molars (111/211) usually enter the caudal compartment. The caudal maxillary sinus is partially divided by the infraorbital canal, over which it communicates with the sphenopalatine sinus. It communicates dorsally with the conchofrontal sinus via the frontomaxillary opening at the level of the osseous lacrimal canal and the medial wall of the orbit. It also communicates with the nasal cavity through a compressed nasomaxillary opening into the caudal aspect of the middle meatus. The rostral maxillary sinus is also divided by the infraorbital canal into medial and lateral compartments. The medial compartment of the rostral maxillary sinus communicates with the middle nasal meatus via a narrow slit, the nasomaxillary opening. The rostral compartment communicates with the ventral conchal sinus over the infraorbital canal through the conchomaxillary opening. The volume of the sinus continues to increase from birth through the life of the horse as the teeth continue to erupt and migrate forward. The maxillary sinus is more predisposed to disease than the other sinuses because of direct communication with the nasal cavity and its association with the dental alveoli.

Frontal Sinus

The frontal sinus occupies the dorsal part of the skull. It extends from a point midway between the infraorbital foramen and the medial canthus of the eye to a point midway between the caudal margins of the orbit in the mature horse. An extensive

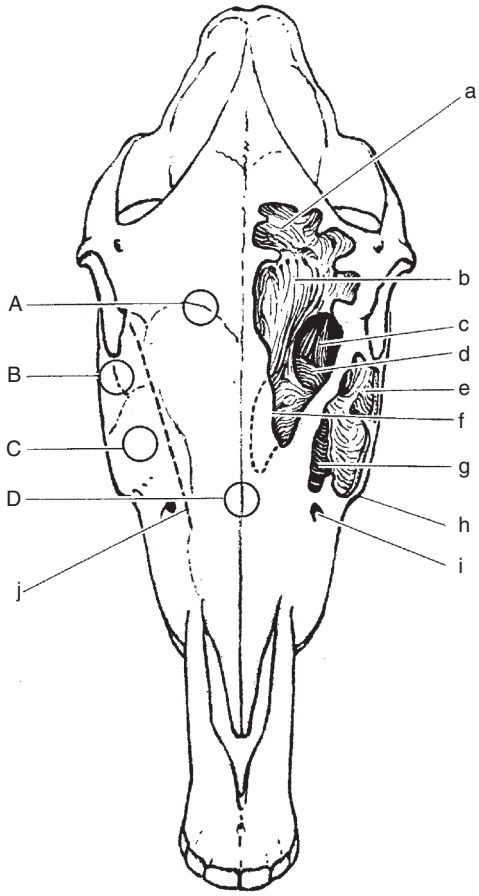

Figure 43-1. Diagram of the skull depicting the location of the equine paranasal sinuses and sites for trephination. *A,* A site for entering the frontal sinus. The sites for entering the caudal maxillary compartment *(B)* and the rostral maxillary compartment *(C)* are above the facial crest and below the nasolacrimal duct *(j).* The nasal bones start to diverge at *(D),* which is the site for exposing the nasal septum. (For a detailed description for entering the frontal sinus over the frontomaxillary opening, see "Sinoscopy.") The *dotted line* outlines the medial floor of the dorsal conchal sinus. *a,* Frontal sinus; *b,* ethmoid mass; *c,* frontomaxillary opening; *d,* dorsal bulla of ventral conchal sinus; *e,* caudal maxillary sinus; *f,* dorsal conchal sinus; *g,* rostral maxillary sinus; *h,* facial crest; *i,* infraorbital foramen; *j,* course of nasolacrimal duct.

communication exists between the rostromedial aspect of the frontal sinus and the dorsal conchal sinus; together they are referred to as the *conchofrontal sinus.*

Sphenoid and Palatine Sinuses

The sphenoid and palatine sinuses are usually contiguous in the horse, forming the sphenopalatine sinus. The sphenoid and palatine sinus compartments communicate under the ethmoidal labyrinth. The sphenopalatine sinus communicates freely with the caudal maxillary sinus.

DISEASES OF THE NARES

Diseases of the nares are relatively rare. The most common clinical signs are reduced airflow, nasal stertor, and occasionally facial distortion. The condition of the nares can usually be determined by observation and palpation.

Epidermal Inclusion Cysts (Atheromas)

These cysts, located in the dorsolateral aspects of the nasal diverticulum, were once thought to be sebaceous cysts and were referred to as atheromas.[3,4] A more recent report describing the histologic appearance found no remnants of sebaceous glands or cells in the cyst wall; thus, a more accurate and descriptive name, epidermal inclusion cysts, has been proposed.[5] These cysts are usually single, unilateral, spherical nodules that can vary from 3 cm to 5 cm in diameter. Epidermal inclusion cysts of the nasal diverticulum are easily identified by visual appearance and location. An accurate diagnosis can be confirmed by cytologic examination of a fine-needle aspirate.[5] Epidermal inclusion cysts are rarely associated with impaired athletic performance and need not be removed unless indicated for cosmetic reasons.

Surgical extirpation can be performed on a standing horse with sedation and local infiltration anesthesia. The skin of the external nares is prepared for aseptic surgery, as is the nasal diverticulum because of the possibility of inadvertent penetration into the diverticulum. The skin and subcutaneous tissue are incised over the lesion, and the cyst is removed by dissection. Care is taken not to rupture the cyst wall. Closure of the incision is routine, and the aftercare is minimal. An alternative method of removal of epidermal inclusion cysts has been described. The cyst is lanced into the nasal diverticulum through a stab incision, and the cyst lining is everted using a roaring burr. The lining is transected using scissors, and the wound is left to heal by second intention. Recurrence has not been reported.[6]

Redundant Alar Folds

The alar fold has been incriminated as a source of respiratory noise in the horse. To determine whether this is the source of abnormal noise or exercise intolerance, a large temporary mattress suture can be placed through the skin of each alar fold and tied over the bridge of the nose. Because the noise is apparent only at exercise, the horse should be exercised after suture placement to determine if the alar fold is the cause of the problem.

Treatment of flaccid or redundant alar folds consists of bilateral resection of the fold. This procedure can be performed through the external nares or by incising the lateral alae of the external nares.[7] Incising the alae facilitates resection of the alar fold, but the resulting scar may be undesirable in show horses.

The patient is anesthetized and positioned in either dorsal or lateral recumbency. Dorsal recumbency provides adequate access if the procedure is performed through the external nares. Lateral recumbency provides the best exposure to the alar fold if the alae of the external nares are incised. The resection involves incising the alar fold caudal to the alar cartilage on the lateral wall of the nasal cavity to the rostral end of the ventral concha (Figure 43-2). A second incision is directed caudad along the medial attachment of the alar fold to join the first incision. Approximately 2 cm of the rostral end of the ventral nasal concha is removed with the alar fold. Profuse hemorrhage may occur when the cartilage has been incised. A 22.5 cm curved Rochester-Carmalt forceps can be used as a guide for the incisions and to control hemorrhage (Figure 43-3). Because ligation of bleeders is almost impossible when the approach through the external nares is used, hemostasis is achieved by closure of the incision using a simple-continuous suture pattern with size

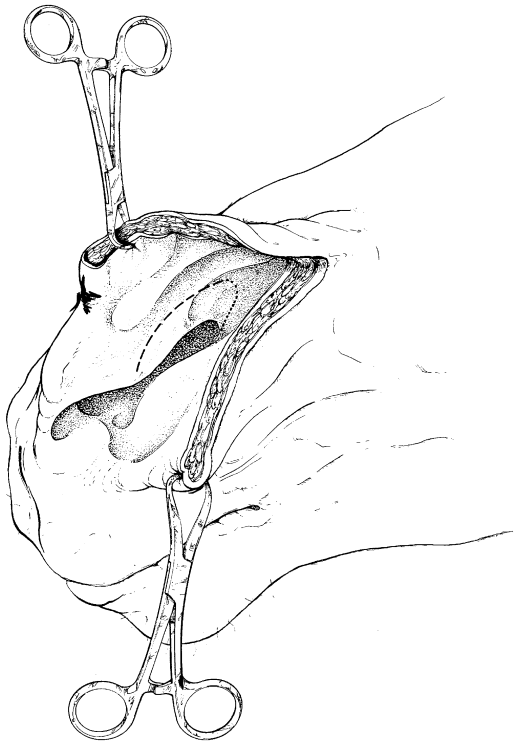

Figure 43-2. Resection of an alar fold. The *dashed line* represents the first incision, which extends caudad from the alar cartilage to the rostral end of the ventral concha. The *dotted line* represents the second incision.

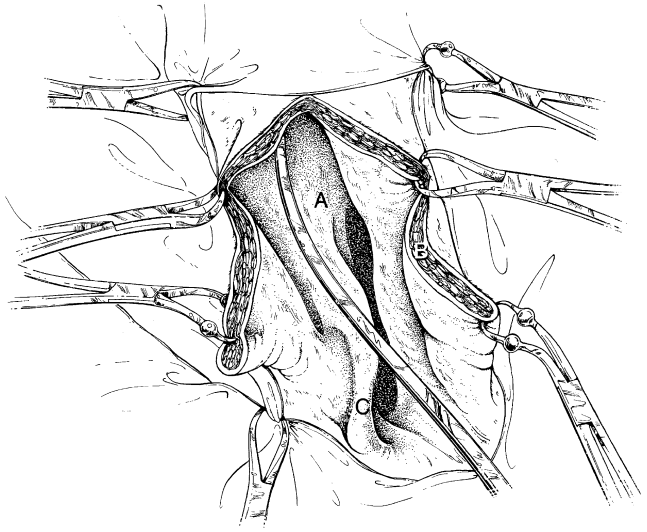

Figure 43-3. Resection of an alar fold. Long, curved forceps are used to control hemorrhage and act as a guide for incisions. *A,* Alar fold; *B,* incised edges of alae; *C,* alar cartilage.

0 absorbable suture material, commencing at the caudal limit. Healing is usually complete within 10 to 12 days, at which time the horse can return to work.

DISEASES OF THE NASAL CAVITY

The most common clinical signs of nasal cavity disease are nasal stertor and unilateral nasal discharge and can include a fetid odor and facial distortion. Endoscopy or radiography or both are usually required for diagnosis.

Facial Fractures

Facial fractures involving the paranasal sinuses and nasal cavities are common and result from direct trauma. Fractures of the paranasal sinuses and nasal cavity most commonly involve the nasal and frontal bones but may also involve the maxillae and lacrimal bones.

Diagnosis of fractures of the paranasal sinuses and nasal cavity is based on history, clinical examination, and radiography. Radiographs of the skull should always be taken to assess the severity of the fracture, but the extent and degree of displacement of the fracture fragments may be more readily determined by palpation. If available, computed tomography provides the best data for complete assessment of the involved structures.

Even though the skin may be intact, these fractures should be considered open, because penetration of the sinus and nasal mucosa usually occurs. Reconstructive surgery should be performed as soon as it is feasible, because primary open reduction provides the best cosmetic results. Some facial fractures of the paranasal sinuses and nasal cavity heal spontaneously; however, failure to treat them may result in chronic sinusitis, bone sequestra, nonhealing wounds, facial deformity, and secondary nasal septal thickening or necrosis.[4,8,9]

Many different reconstructive techniques have been described for facial fractures of the paranasal sinuses and nasal cavity.[9-13] Procedures are performed with the patient anesthetized and positioned in lateral recumbency. Large, slightly curved or S-shaped incisions extending beyond the margins of the fracture are used to expose the traumatized area. Attempts should be made to preserve the periosteal attachments to the fragments. The fracture site is thoroughly débrided and irrigated to remove blood clots and debris. Detached bone fragments devoid of periosteum can be thoroughly cleansed and replaced, but there is the possibility of eventual sequestration. If a paranasal sinus is involved, it should also be thoroughly lavaged to remove blood clots, small bone fragments, and other debris. Some small fractures may be elevated with a thin osteotome or a narrow periosteal elevator, whereas large fragments require other techniques to elevate them into position. The temporary use of orthopedic screws in the fragment and the use of holes drilled in solid bone adjacent to the fracture for insertion of a Langenbeck elevator have been described to assist in elevating the fragments into their normal anatomic position (Figure 43-4).[9,14,15] The use of suture material depends on the stability of the fractured fragments after reduction. Unstable fragments can be held in position using 20- to 22-gauge wire or size 1 monofilament absorbable sutures placed strategically through small holes drilled into the fragment and parent bone.

The incision is closed in two layers. The periosteum is re-apposed when possible using an absorbable suture. A simple-continuous intradermal suture pattern to reappose the skin edges has been reported to provide the best cosmetic results, but skin staples are faster and are also very cosmetic.[9] A pressure bandage placed over a nonadherent dressing is recommended for 3 to 4 days to protect the area and minimize postoperative swelling. Systemic antibiotic therapy should be continued for 5 to 6 days.

A serosanguineous nasal discharge may be present for 1 to 2 weeks postoperatively. If this drainage continues, however, it may indicate the presence of a sequestrum or sinusitis.[8] For additional information on facial fractures, see Chapter 102.

Nasal Septum Diseases

Diseases of the nasal septum are relatively rare and may cause thickening, malformation, or deviation of the septum. Specific diseases include cystic degeneration, malformation of normal tissue (hamartoma), abscess, traumatic thickening and necrosis secondary to septal fracture, longitudinal deviation of the incisive bones and nasal septum, and neoplasm.[4,14,16-20] All of these lesions may produce similar clinical signs, such as decreased airflow or complete unilateral obstruction, nasal stertor, nasal discharge, and occasionally facial distortion. Endoscopy, radiography, and computed tomography are useful to determine the exact site, extent, and nature of the involvement.

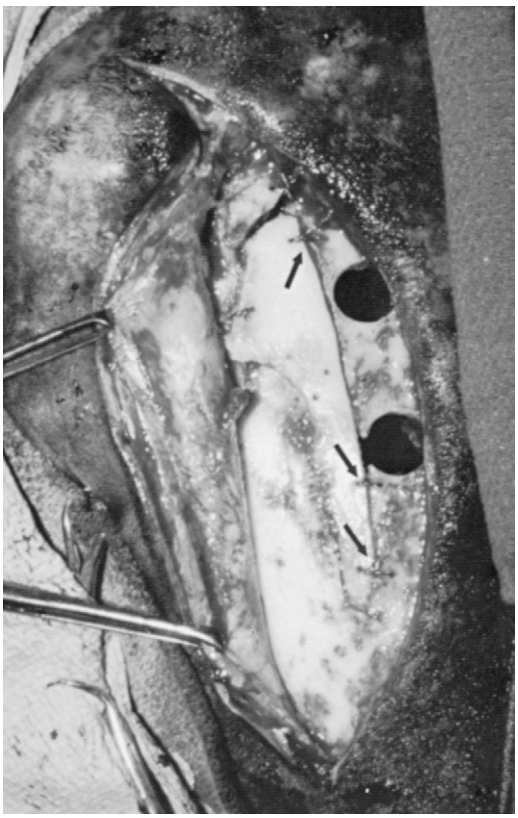

Figure 43-4. Fracture of the nasal and frontal bones. The holes in the parent bone adjacent to the fracture were made to insert a Langenbeck elevator to raise the fragment into its anatomic position. Interrupted stainless steel sutures were used to stabilize the fragment *(arrows)*.

Some of these conditions may improve with removal of the nasal septum. Resection of the nasal septum may involve removing a small portion or the majority of the septum. Incising the lateral alae of the nostril is the best approach for localized lesions of the rostral aspect of the septum, whereas a subtotal resection of the nasal septum or an alternative resection technique is used when the majority of the septum is involved.[16,21,22] Because of the likelihood of profuse hemorrhage during surgery, it is advisable to identify a suitable blood donor and collect 4 to 8 L of blood before surgery in case a blood transfusion becomes necessary. In addition, the administration of large volumes of intravenous fluids during surgery may be necessary to help alleviate hypotension. The most effective method of controlling hemorrhage is to use a nasal tampon after surgery.

The surgery is performed with the horse anesthetized and positioned in lateral recumbency. An airway should be established via a tracheotomy and the anesthetic delivered through it. The surgery for subtotal resection begins by making a 19-mm trephine hole on the bridge of the nose to gain access to the caudal portion of the septum. The center for this opening is located just rostral to the frontal sinuses where the nasal bones begin to diverge (see Figure 43-1, *D*). A curved incision is made through the skin and periosteum, and the periosteum is reflected to provide exposure for the trephine. The nasal septum is easily identified when the bone plug has been removed and the mucosa has been incised. Doyen intestinal forceps or other suitable straight forceps are placed vertically on the nasal septum down to the floor of the nasal cavity (Figure 43-5). The forceps act as a stop for the guarded chisel when severing the dorsal and ventral attachments of the nasal septum, and they act as a guide for making the caudal incision in the septum. The rostral division in the nasal septum is performed by making a curved incision with a scalpel, starting from the ventral aspect

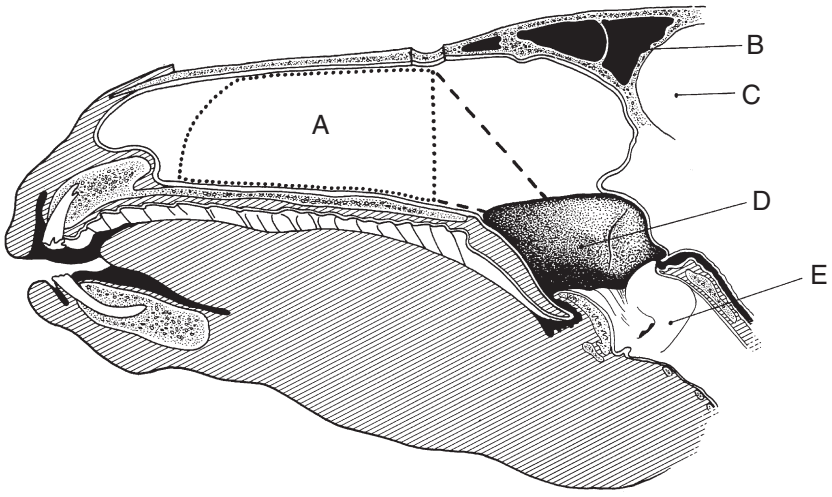

Figure 43-5. Resection of the nasal septum. The *dotted lines* outline the area of the septum removed in the subtotal resection. The *dashed lines* outline the caudal area of the septum removed in addition when a near-total nasal septal resection technique is used. Access to the caudal nasal septum is via a midline trephine opening. *A,* Nasal septum; *B,* frontal sinus; *C,* cranial cavity; *D,* nasopharynx; *E,* larynx.

of the septum and extending in a dorsocaudal direction, leaving at least 5 cm of the rostral septum to support the alar cartilages and external nares. A guarded chisel is used to incise the dorsal and ventral attachments of the septum caudal to the forceps. The caudal incision is made with a narrow osteotome immediately rostral to the Doyen forceps. The septum is then grasped through the external nares with heavy Vulsellum forceps and removed.

Obstetric wire can be used instead of a guarded chisel to incise the dorsal and ventral attachments of the nasal septum. This causes less trauma and hemorrhage. Another advantage of this technique is removal of more of the ventral aspect of the septum than with the guarded chisel. When using obstetric wire for a subtotal resection, the caudal incision is made before the incisions of the dorsal and ventral attachments. The obstetric wire is threaded through the trephine opening on either side of the septum to the external nares. To incise the ventral attachment, the obstetric wire has to be forced to the most ventral aspect of the caudal septal incision. The septal attachments are severed easily with obstetric wire, so care should be taken not to incise beyond the rostral incision of the septum. Digital palpation or visual inspection using a flexible endoscope can be used to determine if all of the ventral attachment of the nasal septum has been removed. Ferris-Smith rongeurs can be used to remove the remaining portions.

An alternative technique has been described to remove the remainder of the nasal septum (see Figure 43-5). The differences between this technique and the subtotal resection are as follows: the caudal incision is made at a 60-degree angle to the nasal bones in a dorsocaudoventral direction, the entire ventral septal attachment is incised up to the rostral incision, and the dorsal and ventral incisions are created by using obstetric wire. The obstetric wire is passed through the ventral meatus around the caudal aspect of the nasal septum and back through the opposite nasal passage to incise the ventral attachment. This is accomplished by passing the wire through the ventral nasal meatus into the nasal pharynx and retrieving the wire through the opposite side using a rat-tooth forceps passed through the biopsy channel in a flexible endoscope. Another instrument that can be used to retrieve the wire is a long flexible grasping forceps (see Chapter 45). For the dorsal incision, the obstetric wire is threaded through the trephine opening on either side of the septum to the external nares. The caudal incision is made at a 60-degree angle to the nasal bones using a long narrow osteotome or a grooved cutting instrument fashioned from a standard wide-tipped screwdriver.[16] The tip of the screwdriver is ground concave and the edges are rounded so that the cutting edge sits better on the septum, reducing trauma to the nasal conchae. The caudal incision can also be done with obstetric wire using a Chamber's catheter to guide the wire around the vomer as far caudad as possible. The wire is retrieved on the side of the vomer as previously with the ventral wire placement. The catheter is used to hold the wire in position while performing the caudal cut.[22]

Packing the nasal cavity with sterile cotton roll gauze controls hemorrhage. Excessive packing of the nasal cavity may cause the nasal tampon to extend into the nasopharynx, where it may be inhaled or swallowed. The end of the gauze should be secured to the skin of the nares to prevent inadvertent removal of the pack.The skin and periosteal incisions are closed in a routine manner. A tracheotomy tube is placed immediately after extubation.

Aftercare includes the use of systemic antibiotic therapy for 5 to 6 days. The nasal packing may be removed after 48 or 72 hours. The nasal cavity should be flushed daily with warm physiologic saline to remove dried blood, debris, and tissue fragments. Complete healing of the septal incisions occurs within approximately 4 to 6 weeks.

The prognosis for restoring normal function is guarded. Such complications as formation of excessive granulation tissue of the caudal septal stump or adhesions to the nasal conchae with subtotal resection may result in a persistent noise or exercise intolerance. Development of the technique that removes additional parts of the caudal septum has minimized these complications. Excellent cosmetic results can be expected except in foals younger than 6 months and in some Standardbreds, in which flattening of the bridge of the nose may occur even when adequate cartilage is left for support.[16]

Ethmoid Hematoma

Ethmoid hematomas are well-encapsulated masses originating in or around the ethmoid labyrinth or occasionally from the paranasal sinuses.[23,24] The cause is unknown, but hemorrhage occurs in the submucosa of an endoturbinate or a sinus, causing the mucosa to stretch and thicken, forming the capsule of the hematoma. An enlarging lesion of the ethmoid labyrinth can extend dorsally into the frontal sinus or ventrally into the sphenopalatine sinus by disrupting the tectorial plate. It may further extend into the maxillary sinus and nasal cavity as it enlarges.[23]

In gross appearance, an ethmoid hematoma has a smooth, glistening surface that may be mottled or green-tinged. Histologically, the lesions are very distinct. The capsule is composed of respiratory epithelium and fibrous tissue. The stroma contains blood, fibrous tissue, macrophages, and giant cells with deposits of hemosiderin and occasionally calcareous deposits.[25] Progressive ethmoid hematomas have been reported in horses ranging from less than 1 year to 20 years of age, with a mean of 9.9 years.[26] Although the condition occurs mostly in middle-aged male horses, it has also been described in young horses and in females; one report found no statistical difference between the occurrence in males and females.[23,26,27] Most cases have been reported in Thoroughbreds, but ethmoid hematomas also have been described in other breeds.[23,24,27-31]

The most consistent clinical sign is mild, intermittent, unilateral epistaxis that is usually spontaneous but may occur with exercise.[23,27] An abnormal respiratory noise may be heard when the hematoma extends into the nasal cavity or causes a distortion of the paranasal sinuses. Other possible clinical signs include malodorous breath, facial swelling, head shyness, and head shaking.[23] A tentative diagnosis can be made from the history, clinical signs, and endoscopic, radiographic, and computed tomographic findings. A definitive diagnosis is made by histologic examination of the tissue. Endoscopic findings early in the development of an ethmoid hematoma can include a trickle of blood from the ethmoidal meatus or middle meatus, and a discoloration or enlargement of the great ethmoturbinate.[23] Hence, small lesions of the sinuses may not be detected endoscopically.[32] However, as the condition progresses, a greenish yellow to purplish red mass may be seen obscuring the fundus of the nasal cavity (Figure 43-6) or the entire nasal passage.

The most characteristic radiographic abnormality is a smooth, discrete, rounded density in the frontal or maxillary sinus;

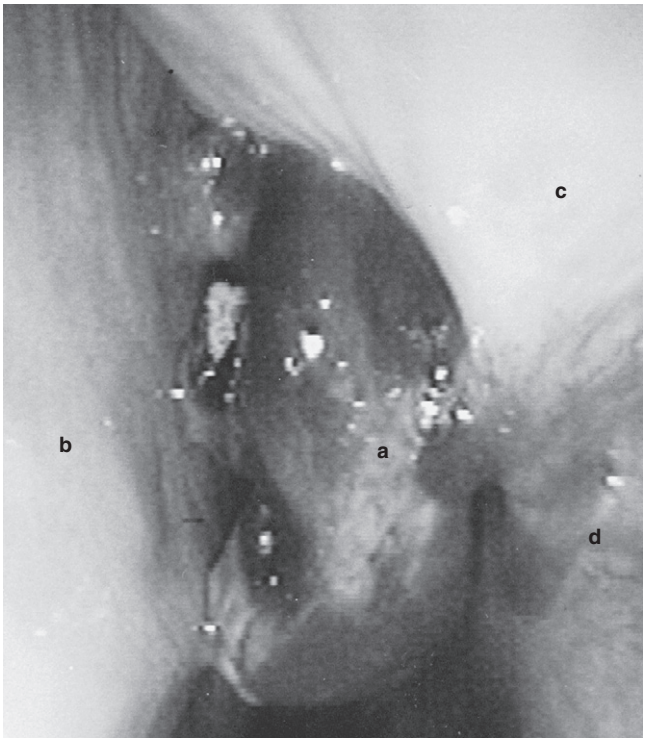

Figure 43-6. Endoscopic appearance of an ethmoid hematoma *(a)* in the left nasal fundus, located between the nasal septum *(b),* the dorsal concha *(c),* and the ventral concha *(d).*

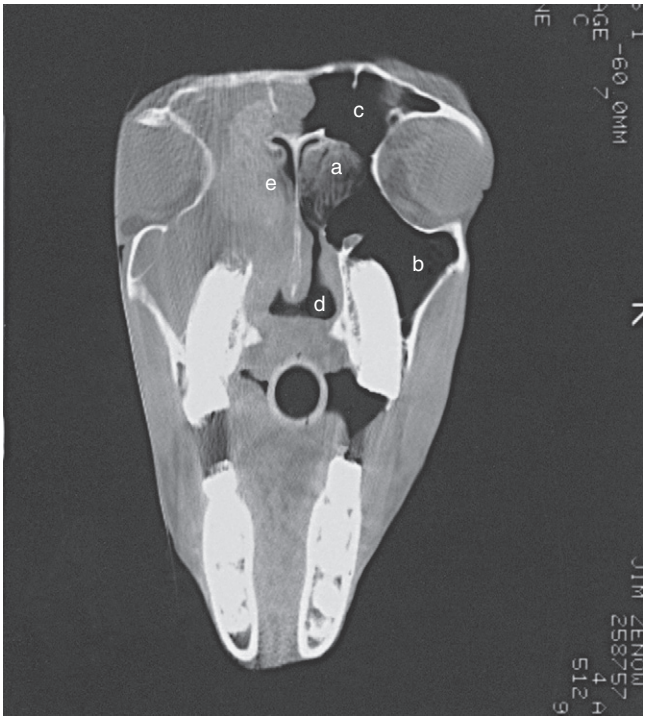

Figure 43-7. A computed tomographic image of a horse's head in a transverse plane at the level of the orbits, demonstrating the right normal ethmoidal labyrinth *(a),* caudal compartment of the maxillary sinus *(b),* frontal sinus *(c),* ventral nasal meatus *(d),* and a soft tissue density *(e)* in the frontal and maxillary sinuses on the affected left side. (The asymmetry of the scan is caused by the horse's lateral recumbency during imaging.)

however, radiographic changes may include fluid lines or diffuse opacities of the sinuses.[23,33] Computed tomography (CT) is useful to determine the exact location and extent of the hematoma (Figure 43-7), because it provides a cross-sectional view of the skull, which eliminates the superimposition of the paranasal sinuses and nasal cavity that occurs with the lateral radiographic projection.

The differential diagnosis includes other conditions that may result in persistent or intermittent epistaxis or blood-stained nasal discharge, such as ulcerative or fungal rhinitis; foreign body; ethmoidal neoplasia; mycosis or neoplasia of the guttural pouch; skull fracture; neoplasia, infection, or cyst of the paranasal sinuses; pulmonary abscess or neoplasm; and infectious pleuropneumonia.[33,34]

The goal of surgery is removal of the mass and destruction of its origin. The surgical approach for removal depends on mass size and location. Lesions limited to the fundus of the nasal cavity and less than 5 cm in diameter can be treated transendoscopically using a neodymium:yttrium-aluminum-garnet (Nd:YAG) laser in a standing patient or by intralesional injection of formalin (see later).[32,35,36]

Lesions that are large and extend into the nasal passage or sinuses are best approached via bone flap techniques, gaining access to the origin of the lesion with the horse standing or anesthetized in lateral recumbency. These techniques can be used to expose the maxillary sinus, frontal sinus, and nasal cavity.[28,30,36-38] One of these techniques, the frontonasal bone flap, gives improved access and has more versatility than other approaches, especially if there is involvement in both the nasal cavity and the frontal sinus.[30] It also allows the creation of a large endoscopic portal to the sinuses, which permits future evaluation for recurrence and treatment with the Nd:YAG laser in a standing patient (Figure 43-8).

Intraoperative hemorrhage is an expected complication from damage to the normal nasal or sinus mucosa, damage to the ethmoturbinates, or from the ethmoid hematoma itself. Various techniques have been described to provide hemostasis during surgery, such as bilateral carotid artery ligation, roll gauze packing, epinephrine-soaked gauze packing, vascular clips, lavage with sterile physiologic saline solution (cold or ice slush), and liquid nitrogen spray.

Cryosurgical removal of the lesion is reported to lower the incidence of recurrence and reduce hemorrhage, but care must be taken to protect the cribiform plate and avoid damage to normal structures.[23] The use of the Nd:YAG laser for removal of the lesion has been reported to decrease the amount of hemorrhage and the chance of recurrence.[32,35] My perference, if surgery is indicated, is extirpation of the lesion with radical débridement.

Thorough presurgical planning reduces delays and significant blood loss. In preparation, besides collecting 4 to 8 L of blood for a possible transfusion, it is advisable to administer at least 10 L of intravenous fluids immediately before the induction of anesthesia and to continue to administer fluids throughout the surgery, thereby alleviating hypotension associated with blood loss. The amount of hemorrhage is usually minimal until disruption of the base of the lesion or other highly vascular structures, such as the tectorial plate of the ethmoid labyrinth or the conchal wall of the conchofrontal sinus. Frequently, it is necessary to invade these structures to gain access to the base of the lesion.

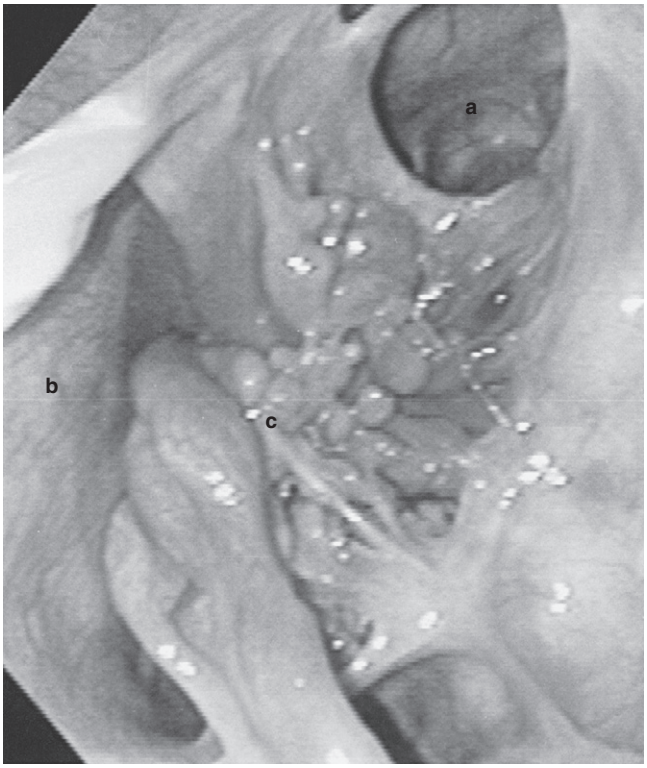

Figure 43-8. Endoscopic appearance of the nasal fundus 4 months after surgery, showing the permanent opening into the frontal sinus *(a)*, the nasal septum *(b)*, and the remains of the endoturbinates *(c)*.

Regardless of the removal technique, the most effective method for controlling hemorrhage is firm packing of the sinus or nasal cavity with sterile cotton roll gauze after surgery. The bone flap is closed in a routine manner. Before the flap is closed, a catheter is placed through the adjacent bone to simplify lavage of the sinus when the pack has been removed. To facilitate removal of the gauze, it can exit either from an aperture on the free edge of the bone flap or through the nasal cavity. The end of the gauze should be secured to the skin of the nares to prevent inadvertent removal of the pack. Administering a sedative and analgesic facilitates removal of the packing 48 to 72 hours after surgery.

Broad-spectrum systemic antibiotics are started before surgery and continued for 3 to 4 days. The use of an indwelling lavage system decreases labor and reduces the horse's resentment of the daily treatment. Lavage of the sinus is helpful in removing exudate, blood clots, and tissue debris, but its effectiveness depends on having adequate drainage through either the nasomaxillary opening or a surgically created opening into the nasal cavity (see later). The horse should be confined to stall rest with only hand-walking for at least 3 weeks. It is common to see fungal and necrotic tissue plaques develop on devitalized mucosal tissues for up to 60 days after surgery, but these usually resolve with no further treatment.

Prognosis is unfavorable without treatment because the lesion is progressive and eventually causes obstruction and dyspnea.[23] The chance of recurrence after routine surgical removal is relatively high (approximately 43%).[23,27] Clients should be advised of the importance of periodic endoscopic examinations after surgery to detect recurrence, so that prompt therapy can be instituted. Because bilateral involvement does occur, endoscopic examination of both nasal passages should always be performed. The use of the Nd:YAG laser on recurrent lesions shows promise in preventing the future recurrence, especially when access to the sinuses via the nasal passage has been provided.

Reported *ablation techniques* for the treatment of progressive ethmoid hematomas in a standing, sedated horse include transendoscopic use of the Nd:YAG laser, intralesional formaldehyde injection, and cryotherapy. The Nd:YAG laser is effective in the standing horse for lesions less than 5 cm in diameter when limited to the nasal fundus only.[35] Therefore, the technique is not applicable for lesions extending into the sinuses. Several laser applications are usually required to ablate the lesion successfully. Lesions are best photo-ablated using noncontact technique at 60 W of power. When a carbonized surface develops, at least a 7-day delay is recommended before the next treatment, to allow sloughing of the carbonized tissue.

Chemical ablation of ethmoid hematomas has been reported to be effective, inexpensive, and apparently safe.[36] Lesions are injected transendoscopically with a 4% formaldehyde solution. The solution is injected through a commercial polypropylene catheter with a retractable, swedged-on 23-gauge needle or a polypropylene tube inserted through the biopsy channel of the endoscope. The end of the tube is beveled to permit penetration of the mass. The volume of the injected solution depends on the size of the lesion. The mass is injected until it begins to distend and leakage of solution is noticed around the tube. The treatment is repeated at 3- to 4-week intervals until the lesion is obliterated or too small to inject. A range of 1 to 18 treatments with a mean of five injections has been reported.[36]

Complications included laminitis in one horse and the risk of nasal obstruction with treatment of bilateral lesions. In one report, a severe complication occurred using this technique: there was erosion and necrosis of the cribriform plate that may have allowed the formalin to reach the brain.[39]

Transendoscopic use of cryogen for the treatment of progressive ethmoid hematomas cannot be recommended because of potential damage to the endoscope, the lack of control of freezing depth, and damage to surrounding tissues with this direct evaporation technique.

DISEASES OF THE PARANASAL SINUSES

The primary clinical features of paranasal sinus disease are unilateral nasal discharge, facial swelling, and decreased nasal airflow. Occasionally seen are externally draining tracts, malodorous breath, ocular discharge, and stertor. The character of the discharge may be serous, purulent, mucopurulent, or serosanguineous, but hemorrhage usually indicates granulomatous or neoplastic lesions.

Diagnosis

A tentative diagnosis of paranasal sinus disease can be made from the history, clinical signs, and physical examination. The physical examination should include percussion of the paranasal sinuses and an oral examination. Endoscopy can be useful for determining the origin of a nasal discharge or evaluating the integrity of the nasal passage of the affected side. Radiography is the most useful diagnostic procedure to establish the location and extent of paranasal sinus disease.[8] Abnormal radiographic findings of the paranasal sinus include opacity, free fluid

accumulation (see Figure 42-23, *A*), dental abnormalities, and related bone pathology. Radiography is more reliable in revealing dental disease in the upper premolars rostral to the maxillary sinus than those located within the sinus.[33] Radiographic evidence of free fluid accumulation in the maxillary sinus is similar in frequency and quantity in both dental disease and primary sinusitis, but opacification of both the maxillary and the frontal sinus rarely occurs in dental disease, whereas it is common with primary sinusitis.

Sinocentesis

Sinocentesis is a useful diagnostic or therapeutic procedure for paranasal sinus disease. When it is performed as a diagnostic procedure, both compartments of the maxillary sinus should be sampled. The area is prepared for aseptic surgery. Anesthesia is provided by local infiltration of a 2% solution of lidocaine. The skin, subcutaneous tissue, and periosteum are readily desensitized by a small quantity of local anesthetic. After a stab incision is made through the skin and periosteum, a hole is made in the bone using a Steinmann pin or bone drill of slightly larger diameter than an indwelling plastic catheter or rigid catheter (such as a canine urinary catheter). If no sample is obtained, 20 to 30 mL of warm saline should be infused before subsequent aspiration, or the sinus may be lavaged with 0.5 L of saline and the nasal discharge examined for evidence of purulent exudate.

Sinoscopy

Sinoscopy of the paranasal sinuses has been reported to be a useful procedure for the examination, diagnosis, and treatment of some disorders of the paranasal sinuses.[40] It can be a valuable diagnostic aid when other noninvasive procedures are inconclusive. It is usually performed in the standing, conscious horse or under general anesthesia. The use of a rigid arthroscope was first reported[40] as means of examination of the paranasal sinusus but did not gain acceptance because of the inability to throughly examine the sinuses even with multiple portals. Inserting the arthroscope through one or two portals in the rostral maxillary sinus does not provide consistent access to the ventral conchal sinus or the rostral maxillary sinus. Direct access to the rostral maxillary sinus is especially difficult in horses younger than 6 years because the reserve crowns of the cheek teeth occupy most of the this sinus.

A flexible endoscope provides superior viewing and allows navigation around structures within the sinuses, facilitating identification of normal anatomy or pathology.[41,42] Inserting the endoscope through the frontal bone via a large trephine opening or a frontonasal bone flap provides direct access to the conchofrontal and caudal maxillary sinus and indirect access to the ventral conchal and rostral maxillary sinus by fenestrating the bulla of the ventral concha. If the bulla cannot be viewed through the portal in the frontal bone, a second portal is recommended into the caudal maxillary sinus.[43] The ventral conchal bulla can be fenestrated with either a Smith-Ferris arthroscopic ronguer or a Matthew aural (crocodile) forceps passed through the same trephine opening as the endoscope or the second portal in the caudal maxillary sinus.

The examination is performed with a 12-mm flexible videoendoscope using portals created with a 15-mm Galt trephine. Anatomic locations for these trephine sites in an adult horse are (1) conchofrontal sinus—60% of the distance from the midline to the medial canthus and 0.5 cm caudal to the medial canthus, (2) caudal maxillary sinus—2 cm rostral and 2 cm ventral to the medial canthus, and (3) rostral maxillary sinus—40% of the distance from the rostral end of the facial crest to the level of the medial canthus and 1 cm ventral to a line joining the infraorbital foramen and the medial canthus. The previously reported trephine site for the rostral maxillary sinus using 50% of the distance from the rostral end of the facial crest to the level of the medial canthus and 1 cm ventral to a line joining the infraorbital and the medial canthus has been shown to signficantly increase the risk of inadvertent entry into the caudal maxillary sinus than when using the more-rostral located site.[44]

Sinusitis

Sinusitis is the most commonly encountered disease of the paranasal sinuses. It may be classified as either primary or secondary and as either acute or chronic. Empyema refers to purulent exudate within the sinus and is not necessarily synonymous with chronic sinusitis. Primary bacterial sinusitis usually results from previous upper respiratory tract infections. *Streptococcus* species are the most commonly reported organisms of primary sinusitis.[14]

Primary Sinusitis

The principles of therapy for primary sinusitis are to provide adequate drainage and to use the appropriate systemic antimicrobial agents based on antibiotic culture results and sensitivity testing. Drainage may be enhanced by lavaging the sinus once or twice daily. Drainage and lavage are accomplished by either placing an indwelling catheter percutaneously into the maxillary sinus (see "Sinocentesis," earlier for the procedure) or by using a sterile 13-mm nasogastric tube through a trephine opening in the frontoconchal sinus. The skin over the site for entering the frontoconchal sinus is anesthetized with 2% mepivicaine (see later). A slightly curved incision is made through all layers of tissue to the bone (based axially). The skin incision is made 10 mm to 15 mm larger than the trephine opening into the sinus. This extra margin between the skin incision and edge of the bone provides support for the skin and periosteum on closure and reduces the risk of complication associated with skin closure. The skin and periosteum are reflected from the bone together, and a circular opening is made using a 19-mm Galt trephine. The disc of bone is discarded. The exudate is most efficiently removed by flushing the sinuses with a mild salt solution consisting of 35 g of salt per 4 L of water (1.2 ounces/gallon) using a sterile nasogastric tube and stomach pump.

Primary paranasal sinusitis usually resolves with systemic antibiotic therapy and lavage, but when the exudate becomes inspissated in the ventral conchal sinus, surgical treatment becomes necessary.[45,46] This condition should be suspected when a primary paranasal sinusitis does not resolve with systemic antibiotics and lavage. Other features of a ventral conchal sinusitis are as follows:

1. The radiographic presence of a soft tissue density over the roots of premolar 108/208 and molars 109/209 and molars 110/210.
2. Distortion of the sinus, seen as a narrowing of the nasal passage caused by an accumulation of inspissated exudate in the ventral conchal sinus

Surgical treatment includes removing the exudate from the ventral conchal sinus and providing additional drainage if necessary. The ventral conchal sinus can be accessed via a maxillary bone flap over the infraorbital canal or by trephining the conchofrontal sinus over the frontomaxillary opening in the standing, concious horse and fenestrating the ventral conchal bulla (see later). Flushing the rostral maxillary and ventral conchal sinus through the fenestrated bulla of the ventral conchal sinus is facilitated by attaching a bent Yankauer suction tip to a sterile, 13-mm nasogastric tube.

Secondary Sinusitis

Secondary sinusitis may be caused by dental disease, facial fractures, granulomatous lesions, or neoplasms. Identifying the cause of sinusitis radiographically can be very difficult with exudate in the sinus. Removal of the exudate via lavage through a trephine opening into the conchofrontal sinus enhances the radiographic and endoscopic examination of the sinuses. After the exudate is removed, the frontoconchal and caudal maxillary and indirectly the ventral conchal and rostral maxillary sinuses can be reexamined either radiographically or endoscopically.

The skin is closed temporarily with skin staples to allow daily flushing for 3 to 4 days. After the final flush, the skin is closed with four simple interrupted sutures to provide additional support to the skin staples. Dental disease is the most common cause of secondary sinusitis (see Chapter 30).[47] Secondary sinusitis is generally more difficult to treat and requires surgical intervention to remove the underlying cause (see "Surgical Approaches to the Paranasal Sinuses," later).

Paranasal Sinus Cysts

Occurrence of paranasal sinus cysts is well documented.[48-53] The exact etiology and pathogenesis are unknown. There is some evidence that these cysts may have a common origin with ethmoid hematomas,[49] but others did not find enough evidence to support this correlation.[50] The condition is usually seen in horses ranging in age from nursing foals to young adults, but it is also seen in adult horses. The most common clinical features are dyspnea, facial swelling, and nasal discharge. As the cysts expand, the pressure causes distortion of the ventral nasal concha, the normal internal structures of the sinus, and the maxilla, resulting in obstruction of the common nasal meatus and possibly deviation of the nasal septum and facial deformity. The cysts are typically filled with a yellow, viscous fluid unless they become secondarily infected. There have been no reports of spontaneous regression. Surgical management consists of removing the cyst lining. Removal of the lining from the wall of the sinus is performed by a combination of digital separation and traction. It may be difficult and impractical to remove all the tissue closely adherent to the infraorbital canal and alveoli. Exposure of the sinus is best provided by a frontonasal[30] or a modified frontonasal bone flap[54] technique. Although the prognosis is usually thought to be guarded because of the accompanying distortion of the ventral nasal concha and nasal septum, there are reports of regression of the nasal obstruction and facial deformity after surgery in young, growing horses.[51,53] A long-term retrospective study reported excellent results with complete resolution of the clinical signs without recurrence in 45 of 48 horses.[55]

Neoplasia

The most common tumor of the paranasal sinuses is squamous cell carcinoma. Other invasive tumors reported include spindle-shaped sarcoma; osteogenic sarcoma, lymphosarcoma, and a poorly differentiated carcinoma; ethmoid carcinoma; hemangiosarcoma; and adenocarcinoma.[20,56-58] Solid, noninvasive neoplasms (such as fibroma, osteoma, and ameloblastic odontoma) have been reported.[58-61] Useful clinical signs that may aid in differentiating neoplasia from other sinus diseases are malodorous breath without evidence of dental disease and radiographic evidence of widespread bone destruction. The prognosis is generally unfavorable unless the neoplasm is a solid noninvasive tumor, because the majority are malignant.[58]

SURGICAL APPROACHES TO THE PARANASAL SINUSES

Surgical access to the paranasal sinuses can be obtained by either trephination or bone flap techniques.[30] The bone flap techniques are superior to trephination because they provide better exposure, visualization, and access for surgical manipulation within the sinuses, and they eliminate the need for multiple trephine openings. The bone flap technique was first described for exploration of the maxillary sinus, but the technique can also be used for the frontal sinus and nasal cavity. The frontonasal bone flap is much more versatile than the maxillary bone flap because it allows thorough exploration of most of the paranasal sinuses including the ventral conchal and rostral maxillary sinuses and better exposure for surgical manipulations.[30] A modified frontonasal bone flap has been described in the standing horse as an alternative to the traditional frontonasal bone flap approach.[54] It was found to be practical, provide adequate exposure and reduce surgery time.

Trephination

Trephination is the traditional approach for surgical access to the paranasal sinuses. It is useful for diagnostic and therapeutic access to the sinuses. The common Galt trephines range in size from 6.4 mm to 25 mm. The size of the trephine selected depends on the intented purpose of the opening. A large trephine (19 mm or 25 mm) opening through the frontal bone over a frontomaxillary opening has become very useful. It provides better exposure for direct and indirect (via fenestration of the bulla of the ventral conchal sinus) endoscopic examination and treatment of the sinus. The trephine opening can be enlarged even more with a curved-jaw Stille Luer ronguer if slight modification in the opening is needed. When enlarging the trephine opening, it is important to leave a 10-mm to 15-mm margin between the skin incision and the bone for skin closure. The approximate sites for trephination of the frontal, caudal, and rostral maxillary sinuses are shown in Figure 43-1 (also see "Sinoscopy," earlier). The trephination sites usually heal within 3 to 4 weeks with minimal blemish. The trephine opening for the so-called "modified frontonasal bone flap technique" (which in fact is a trephine opening) is centered 5 cm axial to the nasal lacriminal duct, 4 cm lateral to the nasal midline, and 2 cm below a horizontal line between the medial canthi.[54] The nasal lacriminal duct can be identified by a line from the medial canthus to the incisive notch. A curved incision is made through the skin and periosteum and reflected from the

bone as one. A 5-cm skull trephine is used to make the opening into the conchofrontal sinus. As with other trephinations, the bone disc is discarded. As mentioned before, the skin flap should be larger than the size of the trephine hole in the bone. Leaving a 10-mm to 15-mm margin of skin is even more important for skin closure and cosmetic results using this technique than when using small trephine openings.

The age of the horse should be considered when selecting the site for trephination of the maxillary sinus. The alveoli of the caudal three cheek teeth form the ventrolateral wall of the maxiallry sinus. In horses 3 years old and younger, the alveoli lie immediately under the osseous infraorbital canal. As the horse ages and the teeth advance, the sinus becomes larger. The trephination sites usually heal within 3 to 4 weeks with minimal blemish.

Bone Flap Approaches

For the *maxillary bone flap* (Figure 43-9), the *rostral* margin is a line drawn from the rostral end of the facial crest to the infraorbital foramen; the *dorsal* margin is a line from the infraorbital foramen to the medial canthus of the eye, the *caudal* margin is a line (parallel to the rostral margin) from the medial canthus of the eye to the caudal aspect of the facial crest, and the *ventral* margin is the facial crest. These boundaries provide maximal exposure of the maxillary sinus while protecting the vulnerable infraorbital canal and nasolacrimal duct. The osteotomy for the ventral margin of the flap should be located just dorsal to the facial crest to prevent injury to ventral border of the flap. Initiating the ventral osteotomy from the caudal aspect also allows safer flap creation because there is more distance to the underlying tooth roots than at the rostral aspect.

Alternatively, for the *frontonasal bone flap* (Figure 43-10), the *caudal* margin is a perpendicular line from the dorsal midline to a point midway between the supraorbital foramen and the medial canthus of the eye, the *lateral* margin begins at the caudal margin 2 to 2.5 cm medial to the medial canthus of the eye and extends to a point approximately two thirds the distance from the medial canthus of the eye to the infraorbital foramen, and the *rostral* margin is a perpendicular line from the dorsal midline to the rostral extension of the lateral margin. The estimated course of the nasolacrimal duct is a line from the medial canthus of the eye to the nasoincisive notch. In some horses, the rostral portion of the lateral margin has to be angled toward the midline to avoid the duct (see Figure 43-10).

When the boundaries of the area to be explored are determined, a skin incision is made along the rostral, ventral or lateral, and caudal borders. The incision is continued through the subcutaneous tissue to the periosteum, which is exposed and incised. The periosteum is reflected from the site of the proposed osteotomy incision approximately 5 mm. The osteotomy can be performed using an oscillating bone saw, a pneumatic drill with a tapered burr, or an osteotome and mallet. The osteotomies should be beveled so that when the bone flap is replaced it will provide a more secure closure. When the osteotomy has been completed on all three sides, the bone flap can be slowly elevated until it fractures along the fourth (or dorsal) side of the rectangle beneath intact tissue. As the bone flap is being elevated for the maxillary approach, the septum between the compartments should be severed using a long, thin

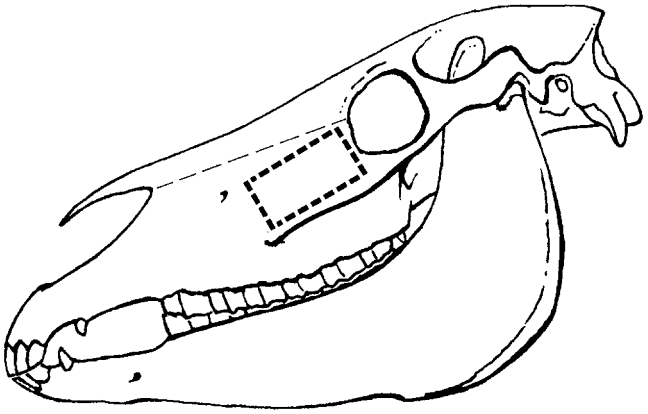

Figure 43-9. Diagram of the skull demonstrating the site for the bone flap technique for exposing the maxillary sinus. The boundaries outlined by *bold dashed lines* provide maximal exposure of the maxillary sinus while protecting the nasolacrimal duct and infraorbital canal. The *long dashed line* from the medial canthus to the incisive notch depicts the approximate course of the nasolacrimal duct. (For a description of the margins of the maxillary bone flap, see "Surgical Approaches to the Paranasal Sinuses.")

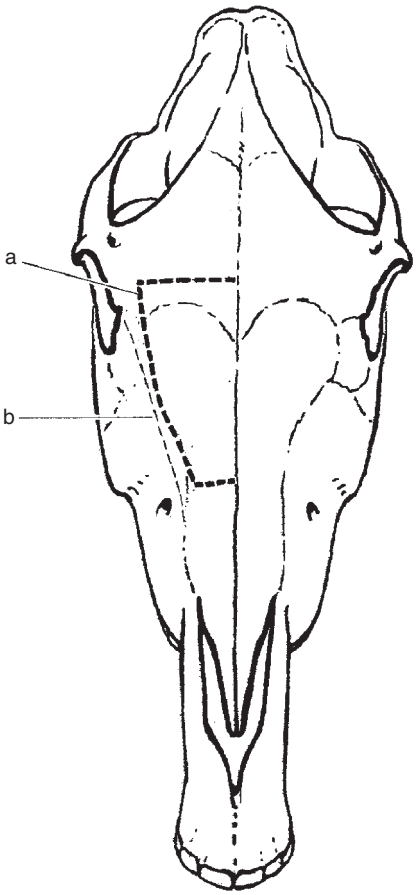

Figure 43-10. Diagram of the skull demonstrating the site for the frontonasal bone flap technique for exposing the caudal aspect of the nasal cavity, dorsal conchal sinus, and frontal sinus *(a)*. The *long dashed line (b)* from the medial canthus to the incisive notch depicts the approximate course of the nasal lacrimal duct. (For a description of the margins of the frontonasal bone flap, see "Surgical Approaches to the Paranasal Sinuses.")

osteotome. The fracture can be controlled by steady, even pressure and the bone flap completely elevated to expose the area.

Before closure, an indwelling lavage system is usually placed through the adjacent bone or a corner of the flap and secured to the skin to facilitate daily lavage. Closure is initiated by pressing the bone flap into its original position and by closing the periosteum with 2-0 absorbable suture using a simple-continuous pattern. A simple-interrupted stainless steel or monofilament nonabsorbable suture placed at each corner of the flap through small drill holes in the respective facial bones occasionally is necessary to secure the flap to the parent bone, but these are frequent sites of fistula formation and should be avoided when possible. The subcutaneous tissue and skin are closed in routine manner. The skin incision usually heals rapidly with minimal scarring.

Frequently, creating a surgical opening into the nasal cavity is necessary to improve drainage. Identifying such a site in the conchofrontal sinus (Figure 43-11) can be aided by passing a mare urinary catheter caudad in the dorsal meatus and feeling the catheter tip through the thin conchal portion of the sinus. The conchal wall is perforated using a curved forceps. The catheter is passed through the opening to aid in the placement of a Seton drain threaded through the nasal cavity from the sinus. The site for establishing communication between the rostral maxillary sinus and the nasal cavity is located dorsally over the infraorbital canal in young horses and ventral to the canal in older horses. Caution should be used to prevent damage to the infraorbital canal. The use of a Seton drain helps maintain the patency of the newly created opening to the nasal cavity; a mushroom catheter is usually used instead of gauze or Penrose tubing with the bone flap technique. After the Seton drain has been removed, the newly created opening might remain permanently open or it might close naturally; therefore, the Seton drain should not be removed until the underlying disease is resolved.

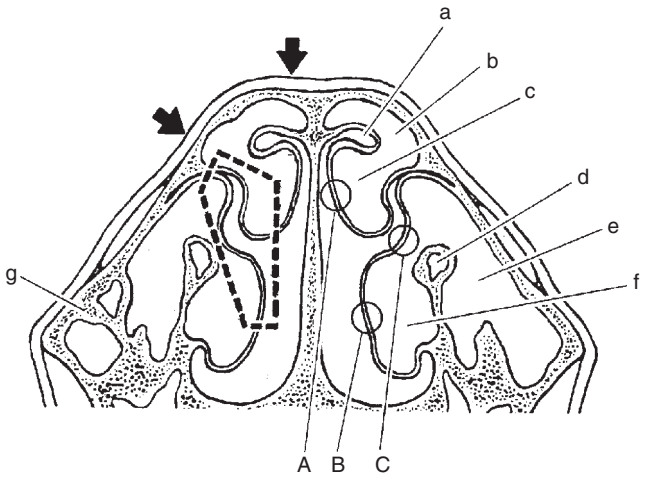

Figure 43-11. Sites for establishing drainage from the paranasal sinuses into the nasal cavity (cross-section of the skull at the level of the first molar). The conchofrontal sinus may be fenestrated at *A* or *B* to establish drainage into the nasal cavity. Fenestration of the ventromedial wall of the dorsal conchal sinus *(C)* creates ventral drainage of the ventral concha sinus into the nasal cavity. This diagram of the skull also demonstrates the medial and lateral edges of the frontonasal bone flap *(arrows)* at this level. The structures within the *dashed lines* have to be removed to provide access to the dorsal conchal and maxillary sinuses and the nasal passage. *a*, Dorsal meatus; *b*, frontal sinus; *c*, dorsal conchal sinus; *d*, infraorbital canal; *e*, rostral maxillary sinus; *f*, ventral conchal sinus; *g*, oblique septum between the rostral and caudal compartments of the maxillary sinus.

Gaining Surgical Access to the Ventral Conchal Sinuses

The ventral conchal sinus can be entered over the osseous infraorbital canal through the conchomaxillary opening in young horses or by penetrating the thin bony plate (see Figure 43-11) below the osseous infraorbital canal in older horses. After the inspissated exudate is removed, the nasomaxillary opening should be assessed for patency by flushing the sinus with saline and observing for nasal outflow. The medial or ventromedial wall of the sinus can be fenestrated to create an opening into the nasal passage if the nasomaxillary opening is not functional. Removal of inspissated exudate from the ventral conchal sinus can also be achieved in the standing horse via a 19-mm or 25-mm trephine opening (a larger trephine opening is better for surgeons with large fingers) into the conchofrontal sinus (see Figure 43-1).[45] The bulla of the ventral conchal sinus is identified by palpating ventral and slightly rostral to the frontomaxillary opening (see Figure 43-1). The bulla can be fenestrated with an index finger, and the thin bony fragments and mucosa are removed with either a Ferris-Smith arthroscope ronguer or a Matthews aural (crocodile) forceps. The exudate is removed by inserting a Yankaue suction tip attached to a sterile, 13-mm (outside diameter) nasogastric tube into the ventral conchal sinus and flushing with a mild salt solution (35 g of salt per 4 L of water) with a sterile stomach pump.

One of the most important aspects of the treatment of sinusitis is lavage of the sinus to remove exudate, blood, blood clots, or tissue debris. The effectiveness of the lavage depends on adequate drainage, either through a patent nasomaxillary opening or through surgically created openings into the nasal cavity. The frequency and volume are probably more important than the type of lavage solution. Sterile physiologic saline or a mild salt solution is as effective as antiseptic solutions in most cases and reduces irritation of the mucous membranes. The use of an indwelling lavage system decreases labor and reduces the horse's resentment of daily lavage. Placing a sterile tube through a small trephine opening and pumping a mild salt solution through a sterile pump is a very simple method of flushing the sinuses but does not deliver volume or pressure obtained by flushing with a sterile nasogastric tube and pump (see treatment modalities under "Sinusitis," earlier).

The use of systemic antibiotic therapy depends on the primary disease and should be based on bacterial cultures and antibiotic sensitivity results. Feeding the horse on the floor enhances drainage and reduces contamination by hay and dust. The use of a sterile dressing or protective hood made from stockinette for a few days reduces contamination and protects the surgical sites from self-inflicted trauma.

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CHAPTER 44

Pharynx

Norm G. Ducharme

ANATOMY AND PHYSIOLOGY

The pharynx is a musculomembranous tubular structure unsupported by bone or cartilaginous matrix. It has different roles during breathing, deglutition, and vocalization. The pharynx is composed of the nasopharynx, oropharynx, and laryngopharynx. The pharynx extends from the caudal end of the nasal cavity/oral cavity to the larynx and is divided by the soft palate to form the nasopharynx dorsally and the oropharynx ventrally.[1] Starting at the base of the epiglottis and extending caudad to the arch of the cricoid cartilage is the laryngopharynx. During respiration, the caudal free margin of the soft palate intimately contacts the subepiglottic tissue at the base of the equine larynx (Figure 44-1) and prevents communication between the oropharynx and nasopharynx. The nasopharynx is attached to the pterygoid, palatine, and hyoid bones and to the laryngeal, cricoid, and thyroid cartilages by muscles that cause dilation and constriction of the nasopharynx.[2] In describing the anatomical basis of the respiratory function of the nasopharynx, the oropharynx has been largely ignored in horses. The anatomy of the oropharynx is likely to become more important as our understanding of the disease grows as was evidenced from the role of the hypoglossal nerve on stability of the soft palate at exercise.[3]

Although the nasopharynx is not directly supported by cartilage or bone, it must withstand large changes in intraluminal pressures (from 24 to 50 cm H_2O) that occur at varying airflow velocities (up to 90 L/sec), with minimal changes in diameter. It does this through muscular contraction, whose neural (i.e., sensory drive) inputs are not well understood in horses. Several groups of muscles alter the size and configuration of the nasopharynx and oropharynx, including the muscles that move the tongue, insert on the hyoid apparatus and larynx, and regulate soft palate position.[4-10] Other muscles alter the position of the hyoid apparatus. specifically, ventral displacement of the basihyoid, which increases nasopharyngeal size and stability by increasing the diameter or decreasing the compliance of the nasopharynx.[5,6] The relevant musculature can be classified as intrinsic or extrinsic to the nasopharynx.[11] The intrinsic muscles, which include those of the soft palate and nasopharyngeal musculature, insert on the mucosa or the confined walls and roof of the nasopharynx. The extrinsic muscles include those of the larynx and the hyoid apparatus, which affect either or both the position of the basihyoid bone or the larynx, and probably affect the morphology of the nasopharynx by changes to the oropharynx.

Intrinsic Musculature

The intrinsic musculature contributes to the stability of the nasopharynx mainly by timely muscular contractions that tense and dilate the pharyngeal walls (Figure 44-2). The floor of the nasopharynx is formed by the soft palate extending caudad from the hard palate to the base of the larynx. The soft palate consists of oral mucous membrane that contains the palatine glands and their ductile openings, palatine aponeurosis, palatinus and palatopharyngeus muscles, and nasopharyngeal mucous membrane.[2] The palatine aponeurosis is formed by expansion of the tendon of the tensor veli palatini muscle.[2,10] This aponeurosis attaches to the caudal margin of the hard palate. It is thick cranially and very thin caudally, where the soft palate is more muscular. The caudal free margin of the soft

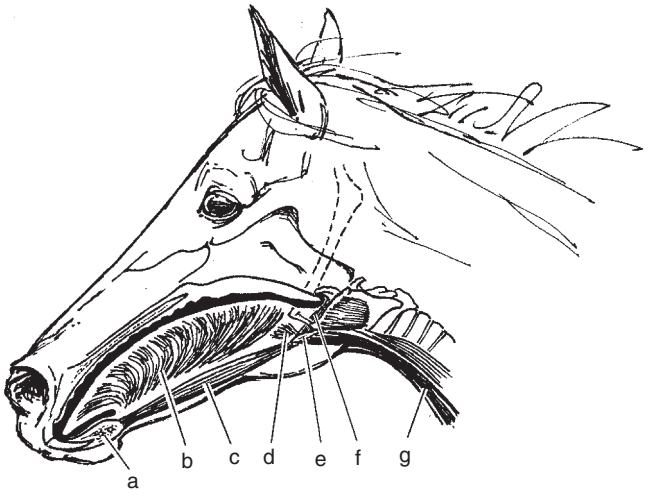

Figure 44-1. The extrinsic structures relevant to equine nasopharynx stability, showing the definition of the nasopharynx and relative relationships of the hyoid apparatus, larynx, and extrinsic muscular attachments. *a,* Mandible; *b,* genioglossus muscle; *c,* geniohyoideus muscle; *d,* styloglossus; *e,* hyoglossus muscle; *f,* basihyoid bone; *g,* hyoepiglotticus muscle; *h,* thyrohyoideus; *i,* sternohyoideus muscle.

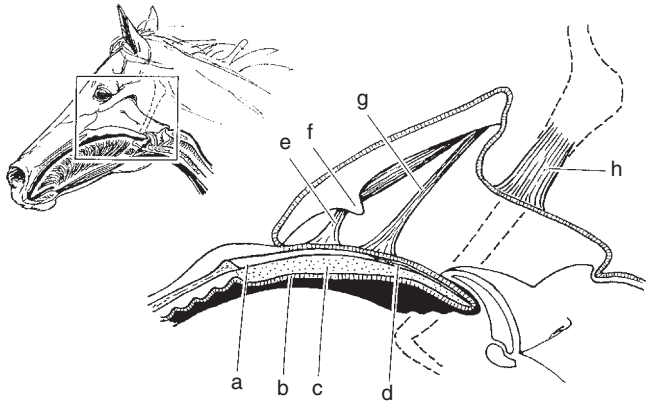

Figure 44-2. The intrinsic structures of the nasopharynx. The nasopharynx forms the passageway that transfers airflow from the caudal aspect of the nasal cavity to the larynx. The floor of the nasopharynx is formed by the soft palate and its relevant structures (*a,* palatine aponeurosis; *b,* oral mucosa; *c,* glandular layer; *d,* palatinus muscle) and is normally in a subepiglottic position during respiration. The hamulus *(f)* of the pterygoid bone, and the tensor veli palatini *(e),* levator veli palatini *(g),* and palatopharyngeus (not shown) muscles are underneath the mucosa of the lateral walls of the nasopharynx. The stylopharyngeus *(h)* with the palatopharyngeus muscles add to the stability of the roof of the nasopharynx.

palate continues dorsad on either side of the larynx, forming the lateral pillars of the soft palate. Muscle fibers of the palatinus muscle course beneath the nasopharyngeal mucosa and extend along the pillars of the soft palate. These pillars unite dorsally, forming the palatopharyngeal arch.[2]

The position of the soft palate is partially determined by the coordinated function of four muscles (see Figure 44-2, and Figure 44-3): the tensor veli palatini, levator veli palatini, palatinus, and palatopharyngeus muscles. The tensor veli palatini muscle is innervated by the mandibular branch of the trigeminal nerve, and the other three are all innervated by the pharyngeal branch of the vagus nerve.[2,7,10] The levator veli palatini muscle arises from the muscular process of the petrous part of the temporal bone and the lateral lamina of the auditory tube and passes along the lateral wall of the nasopharynx to insert within the soft palate dorsal to the glandular layer.[2] This muscle elevates the soft palate during swallowing, closes the nasopharynx, and facilitates oral ventilation in nonobligate nasal breathers. The palatinus muscle consists of a paired fusiform muscle that originates at the caudal aspect of the palatine aponeurosis and courses through the middle of the soft palate, just beneath the nasal mucosa, to ramify in the caudal free margin of the soft palate. The palatopharyngeus muscle arises from the palatine aponeurosis (lateral to the palatinus muscle attachment) and from the palatine and pterygoid bones.[2] The fibers continue caudad on the lateral wall of the pharynx and partially insert into the upper edge of the thyroid cartilage. The remainder of the muscle continues dorsad and inserts at the median fibrous raphe. The palatinus and palatopharyngeus muscles shorten the soft palate and depress it toward the tongue.[4] The tensor veli palatini muscle is a fusiform muscle that originates at the muscular process of the petrous part of the temporal bone, pterygoid bone, and lateral lamina of the auditory tube and travels rostroventrad along the lateral wall of the nasopharynx lateral to the levator veli palatini.[2] The tendon courses around the hamulus of the pterygoid bone and ramifies in the palatine aponeurosis. This muscle tenses the rostral aspect of the soft palate by using the hamulus as a pulley and retracts the soft palate away from the dorsal pharyngeal wall, expanding the nasopharynx and slightly depressing it ventrad during inspiration.[10]

The final intrinsic muscle of the nasopharynx is the stylopharyngeus, which is divided into two parts.[8,12] The rostral stylopharyngeus muscle originates from the medial surface of the rostral end of the stylohyoid bone and inserts on the pharyngeal raphe. It is a pharyngeal constrictor that is not known to be stimulated during exercise, so it will not be discussed further. The caudal stylopharyngeus muscle is a pharyngeal dilator that originates from the medial aspect of the caudal third of the stylohyoid bone and courses ventrally and rostrally to attach on the dorsolateral wall of the pharynx (see Figure 41-5).[8,12] It is innervated by the glossopharyngeal nerve and is responsible for tension on the roof of the nasopharynx (Figure 44-4) that helps it resist collapsing with inspiratory pressure.

The blood supply to the soft palate is derived from the linguofacial trunk, and maxillary artery and venous drainage occurs via the accompanying veins.[2] The lymph vessels drain toward the retropharyngeal lymph nodes. The afferent innervation from branches of the trigeminal, glossopharyngeal, and vagus nerves arises from pressure, mechanical, and temperature receptors lining the mucous membrane of the nasopharynx, including the soft palate.

Extrinsic Musculature

The extrinsic muscles contribute to the respiratory patency of the nasopharynx by indirectly increasing its diameter through change in size of the oropharynx or position of the larynx[13,14] and by a mechanism not yet understood, it increases the stability of the soft palate at exercise. The potential roles of these extrinsic muscles have been identified in horses when interfering with their function, results in dorsal displacement of the

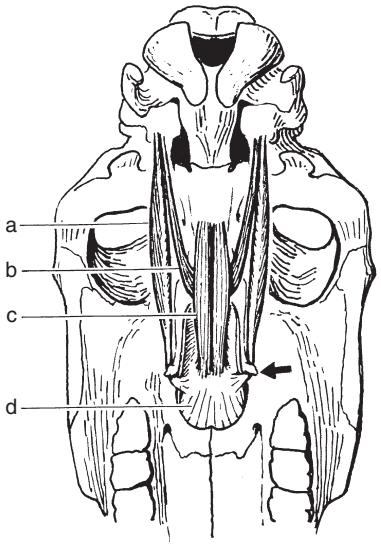

Figure 44-3. The intrinsic structures of the nasopharynx viewed from the ventral aspect of the nasopharynx, showing the muscles of the soft palate, the palatine aponeurosis *(d),* and the hamulus of the pterygoid bone *(arrow). a,* Tensor veli palatini muscle; *b,* levator veli palatini muscle; *c,* palatinus muscle.

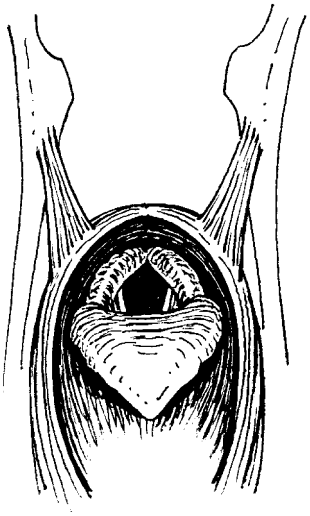

Figure 44-4. The intrinsic muscles that form the wall and roof of the nasopharynx. Note that contractions of the stylopharyngeus caudalis support the roof of the nasopharynx.

soft palate (DDSP) during exercise.[3,15] They are the left and right geniohyoideus, thyrohyoideus, genioglossus, hyoglossus, styloglossus, sternohyoideus and sternothyroideus muscles.

The genioglossus muscle is a fan-shaped extrinsic tongue muscle that originates within the median plane of the tongue and attaches to the oral surface of the mandible, caudal to the symphysis (see Figure 44-1).[2] Contraction of the genioglossus muscle protracts the tongue and pulls the basihyoid bone rostrally. In horses, this activity may be reproduced by the tongue tie,[3] which has been shown to be useful in treatment of DDSP in a recent case-control study.[16] Although this treatment is logical, experimental studies failed to elucidate the anatomic mechanism of action as computed tomographic imaging showed no measurable improvement in nasopharyngeal diameter by using a tongue-tie in normal horses.[17]

In humans, contraction of muscles that insert on the hyoid arch increases upper airway size and stability by increasing the diameter and stiffness of the nasopharynx.[5,6] The rostrohyoid and caudohyoid muscle groups exert rostral and caudal forces, respectively, on the hyoid apparatus. The geniohyoideus is a rostrohyoid muscle that originates, in conjunction with the aforementioned genioglossus, on the medial surface of the mandible near the symphysis and inserts on the basihyoid bone. Its action draws the hyoid apparatus rostrally and protudes the tongue.[2] The styloglossus muscle lies on the lateral aspect of the tongue, which originates on the lateral aspect of the stylohyoid bone and inserts on the tip of the tongue, and its function is to retract the tongue (i.e., retruder). The hyoglossus muscle is located medial to the styloglossus muscle, originates on the hyoid bones (lingual process and stylohyoid and thyrohyoid bone), and inserts on the median plane of the dorsum of the tongue. Its action is to retract (i.e., retruder) and depress the base of the tongue.[2] Because local anesthesia of the hypoglossal nerve at the mid level of the ceratohyoid bone interferes with the action of the geniohyoideus and genioglossus muscles (protuders of the tongue) at the same time as that of the styloglossus and hyoglossus (retruders), the exact function of each muscle is not yet known.[3] The sternohyoideus and sternothyroideus muscles are caudohyoid muscles that originate on the sternal manubrium and extend cranially.[2,18] The sternothyroideus inserts on the caudal abaxial aspect of the thyroid cartilage, and the sternohyoideus muscle inserts on the basihyoid bone and lingual process of the hyoid apparatus. Contraction of these muscles results in caudal traction on the hyoid apparatus and larynx. In dogs and humans, muscles that put rostral traction on the basihyoid bone work in a coordinated fashion with muscles that apply caudal traction, and the sum of these vector forces is a net ventral displacement of the basihyoid bone.[5] This motion increases the angle at the ceratohyoid–stylohyoid joint, increasing the dorsoventral dimension of the nasopharynx. In doing so, the lateral walls of the nasopharynx expand slightly and become taut.[5] Although this has not been demonstrated conclusively in horses, dysfunction of the strap muscles (sternohyoideus and sternothyroideus) in horses has been shown experimentally to increase upper airway pressure at exercise.[17] This suggests that nasopharyngeal impedance is increased by dysfunction of these extrinsic muscles.

Another extrinsic muscle known to increase the patency of the nasopharynx is the hyoepiglotticus muscle (see Figure 44-1), which attaches to the hyoid bone at the base of the epiglottis and, during its contraction, pulls the epiglottis ventrad toward the base of the tongue, thereby increasing the ventral dimension of the rima glottidis.[2,9] The hyoepiglotticus muscle is the only muscle that inserts on the epiglottis.

Finally, the thyrohyoideus muscle, which extends from the lateral lamina of the thyroid cartilage to the caudal aspect of the thyrohyoid bone (see Figure 44-1) was thought to move the larynx rostrad only during deglutition.[2] However, resection of this muscle disrupts the normal stability of the nasopharynx during exercise, resulting in DDSP.[15] Therefore, the mechanism of action of the thyrohyoideus muscle is to enhance soft palate stability during exercise by moving the larynx rostrad so the thyroid cartilage rests more dorsal and rostral in relation to the basihyoid.[15]

The hypoglossal nerve innervates the following muscles: geniohyoideus (main branch), genioglossus (medial branch), styloglossus, hyoglossus (lateral branch), and hyoepiglotticus muscles (main branch).[2,3] The sternohyoideus and sternothyroideus muscles receive motor innervation from branches of the first and second cervical nerves.[2] Although it has been reported that the thyrohyoid branch of the hypoglossal nerve is responsible for innervation of the thyrohyoideus muscle, one investigation suggests that the pharyngeal branch of the vagus is responsible for this innervation (in rabbits).[19] Arterial blood supply to the pharynx is provided by the common and external carotid arteries and the linguofacial trunk.[2] Venous drainage is provided by the accompanying veins.[2] Lymph vessels drain toward the retropharyngeal and cranial cervical lymph nodes.[2] The trigeminal, vagus, and glossopharyngeal nerves provide afferent sensory innervation to the nasopharyngeal mucosa.

ANATOMIC DISORDERS OF THE NASOPHARYNX

Nasopharyngeal Cicatrix

Nasopharyngeal cicatrix was a condition first reported as being more commonly seen in aged female horses, but more recent data suggest only a slight predisposition for mares (60% of 87 horses).[20,21] Affected animals range in age from 5 to 29 years.[20,21] This anomaly is seen almost exclusively in hot climates of the United States (mostly in eastern and southern Texas, with occasional reports from Mississippi, Louisiana, Oklahoma, and Florida) in horses kept on pasture. An environmental allergen is thought to lead to nasopharyngeal inflammation and subsequent damage to the nasopharyngeal and laryngeal mucosa and submucosa. Secondary healing results in a web of nasopharyngeal scarring that reduces the diameter of the nasopharynx, or restricts its function when severe (Figure 44-5).[20,21] In nearly 95% of reported cases, deformed epiglottic, arytenoid, or medial cartilage of the guttural pouch opening is seen (Figure 44-6).[20,21] Because of the larger cross-sectional area of the nasopharynx in comparison to the rima glottidis and extrathoracic trachea, only severe scarring of the nasopharynx causes functional abnormalities. The degree of involvement of other structures determines clinical signs.

Clinical signs for this condition include upper respiratory noise and exercise intolerance, with dysphagia rarely seen. The airway sounds may be variable, depending on whether nasopharyngeal scarring results in DDSP or arytenoid chondritis. Involvement of one or both of the arytenoid cartilages is the most common reason for exercise intolerance and respiratory noise. The degree of performance interference depends on the athletic demands and degree of ventilation interference.

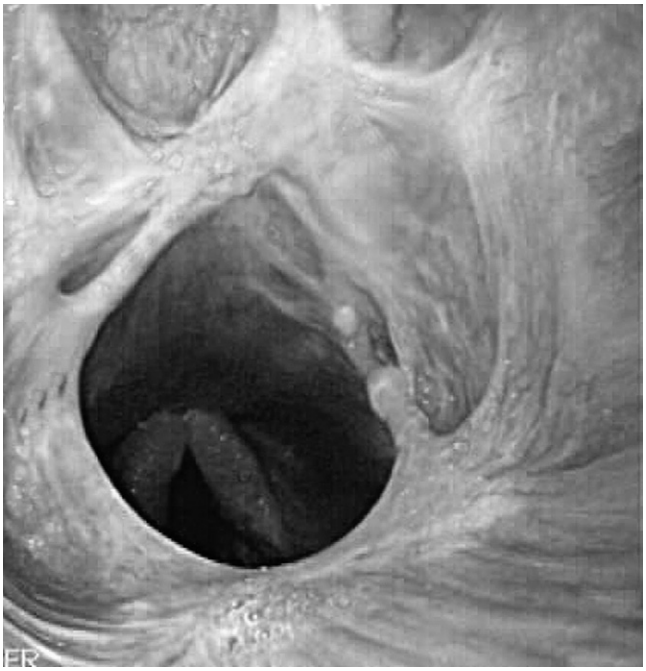

Figure 44-5. Endoscopic photograph of a nasopharyngeal cicatrix in a horse, characterized by scar formation across the floor of the nasopharynx. (Courtesy P. Rakestraw, Texas A&M University.)

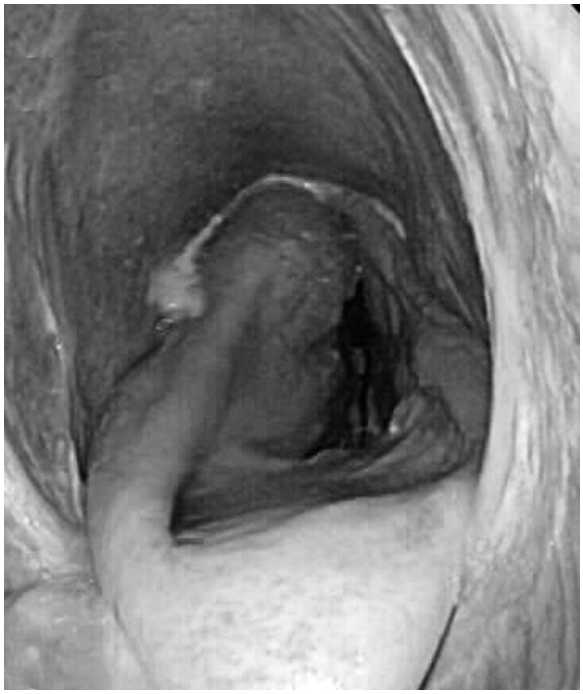

Figure 44-6. Endoscopic photograph of a nasopharyngeal cicatrix affecting the larynx and epiglottic cartilage. Note the deformation of right arytenoid cartilage, as well as scar formation across the floor of the nasopharynx. (Courtesy P. Rakestraw, Texas A&M University.)

The diagnosis is made by endoscopic examination. In the earliest stages, hyperemic mucosa is observed; there may also be yellow or discolored areas (plaques) on the pharyngeal walls, even in areas where a cicatrix does not form (Figure 44-7). The cicatrix can be limited to a local area or involve the entire

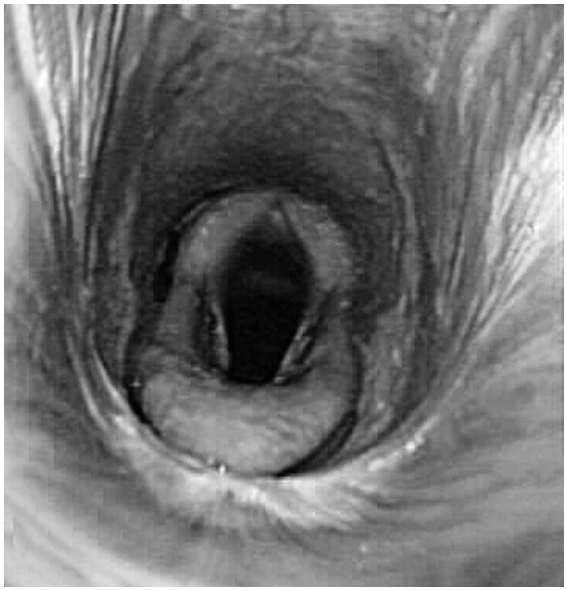

Figure 44-7. Endoscopic view of early nasopharyngeal inflammation, a precursor of nasopharyngeal cicatrix. (Courtesy P. Rakestraw, Texas A&M University.)

circumference of the pharynx, and it usually includes one or more transverse bands of tissue located between the guttural pouch opening and the larynx (see Figure 44-5). In 20% of the cases reported, one of the guttural pouch openings was involved in the cicatrix.[20] Because this type of cicatrix is generally associated with epiglottic and arytenoid chondropathy (see Figure 44-6), the anatomic integrity of these structures should be assessed as well as the position of the soft palate.

As soon as the diagnosis is made, the horse should be removed from the pasture and anti-inflammatory therapy initiated.[21] Horses housed in a dry lot or box stall do not seem to be affected by this disease. If the cicatrix causes nasopharyngeal obstruction, it can be transected in two or three places under videoendoscopic guidance with the horse standing, using a diode or other appropriate laser; however the cicatrix reforms unless the horse is moved to a different environment.[22] Although nasopharyngeal stenting has been reported, its use does not seem to have significant value.[21,23] Partial arytenoidectomy in this heavily scarred environment leads to an inappropriate diameter of the rima glottidis during postoperative healing and, therefore, should not be performed. If airway obstruction is associated with chondritis, a permanent tracheostomy[22] (as described in Chapter 47) should be performed. Very good long-term results was reported after permanent tracheostomy for treatment of this condition in 59 horses.[22]

Nasopharyngeal Masses

Nasopharyngeal masses occur rarely in horses. When masses are present in the nasopharynx, they usually are extensions of paranasal sinus, guttural pouch, or oropharyngeal diseases. However, primary masses involving the nasopharynx can include benign lesions (such as fungal granulomas and cysts) and neoplastic lesions (such as lymphosarcoma and squamous cell carcinoma).[24-26] Depending on the size and location of these masses, they may result in DDSP, dysphagia, or airflow obstruction. Therefore, clinical signs may include exercise intolerance,

respiratory noise, nasal drainage, and weight loss. If the mass extends into the rostral nasopharynx or nasal cavity, uneven airflow can be detected at the nostril, and mucopurulent drainage may be present. Occasionally, pedunculated cysts or polyps originate near the guttural pouch opening, obstructing the nasopharynx.

The diagnosis of nasopharyngeal masses is made by endoscopic examination on recognition of these clinical signs (Figure 44-8). A thorough evaluation of adjacent structures (nasal cavity, paranasal sinuses, and guttural pouches) is imperative. Radiography and/or general anesthesia followed by oral manual or oral endoscopic examination may help identify masses extending into the oropharynx. A biopsy sample of the mass can be taken by sedating the horse and using uterine biopsy forceps. Two or three samples can be taken with a small amount of local hemorrhage. A biopsy of the mass yields the critical information necessary for appropriately managing and treating the lesion.

Treatment is based on the extent of the lesion and the biopsy results. Surgical access to the nasopharynx is limited, and three main options are available: videoendoscopy-assisted laser resection, intralesional injection of a necrosing pharmacologic agent (10% formaldehyde), and surgical attention through an oral approach or pharyngotomy.

Videoendoscopy-Assisted Laser Resection

Videoendoscopy-assisted laser resection is performed as follows. The average 450-kg horse is sedated with a solution of detomidine (6 mg) and butorphanol (4 mg). One should err toward a smaller dosage of butorphanol, because some horses using this medication experience "head tics" that interfere with the surgical procedure. The videoendoscope is passed into the right nostril, and the mass is desensitized by applying 50 to 100 mL of a topical anesthetic solution (2% lidocaine or mepivacaine hydrochloride) and 10 mL of vasoconstrictive agent (0.15% phenylephrine) through the biopsy channel of the videoendoscope. The videoendoscope is withdrawn to the nasal cavity near the dorsal meatus, and local anesthetic is applied to desensitize this area and thus facilitate forceps manipulation. The videoendoscope is passed through the left nostril, and the bronchoesophagoscopic forceps are passed into the right nostril to grasp or otherwise facilitate access to the base of the mass. Using a diode (12 to 15 watts) or neodymium : yttrium-aluminum-garnet (Nd : YAG, 30 to 35 watts) laser, the base of the mass is incised. (Note: Incision is facilitated when traction is applied to the mass.) A large mass that cannot be removed nasally should be removed in smaller sections. Some masses must be reduced by intra-lesional injection (see later) weeks prior to laser resection. Preoperatively, the horses are administered dexamethasone (0.02 mg/lb) and phenylbutazone (2 mg/lb) intravenously. Postoperatively, the steroids are continued for one or more days and the nonsteroidal anti-inflammatory drug (NSAID) for 3 to 5 days. In addition, 20 mL of throat flush solution (composed of 250 mL of glycerin, 250 mL of 90% dimethyl sulfoxide (DMSO), 500 mL of nitrofurazone, and 50 mL of prednisolone 25 mg/mL) is administered twice a day using a 12 French red rubber feeding tube and alternating nostrils between treatments.

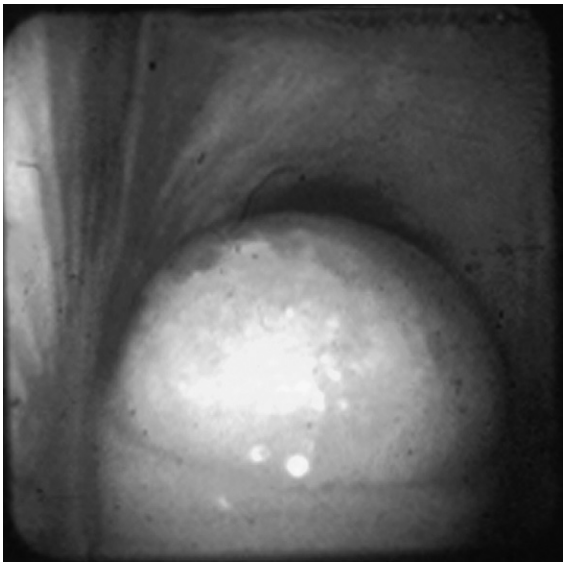

Figure 44-8. Endoscopic view of a pharyngeal mass in a 20-year-old horse. The mass originated at the root of the tongue. (Courtesy J. Stick, Michigan State University.)

Intralesional Injection

Treatments by intralesional injection to induce chemical necrosis of the mass, or local treatment with antimicrobials or antifungal agents, can be used if mass resection is not possible. A polyethylene catheter is passed through the endoscope biopsy channel until it emerges from the biopsy port of the endoscope. A 16-gauge needle is attached to the tubing by removing the needle hub and securing the needle 5 to 7 mm into the tubing. The needle should fit tightly so that it will not dislodge during the injection. The horse is sedated before the injection as described earlier for the laser procedure. Most masses, other than abscesses, are treated with repeated intralesional injection of 10% formaldehyde (10 to 20 mL). The masses are reinjected at 3- to 4-week intervals until resolution or until the size has been reduced. After each treatment, the horse receives a 3- to 5-day course of phenylbutazone (1 mg/lb PO twice a day) or a similar NSAID.

There is little information in the literature to substantiate the optimal treatment of fungal granulomas, such as are seen in coccidioidomycosis.[26,27] Typically they are treated with weekly intralesional injections of amphotericin B (100 mg of a 100 mg/mL preparation) combined with 3 mL of a 90% solution of medical-grade DMSO. More recently, multimodal treatment has been reported.[27] The combined modalities were oral administration of fluconazole (14 mg/kg loading dose, followed by a dosage of 5 mg/kg, PO daily until resolution) and organic ethylenediamine dihydriodide (30 mg of the 4.57% dextrose powdered formulation per kilogram [13.6 mg/lb] PO daily for 4 weeks) and intermittent intralesional injections with fluconazole, amphotericin B (50 mg), and formalin (as described earlier). In addition, potassium iodide (125 mL of a 20% sodium iodide solution IV, once a day for 3 days, followed by 30 g PO once a day for 30 days) is given after clinical remission or until signs of iodine toxicity (dry skin) are noted.

SURGICAL APPROACHES TO THE NASOPHARYNX OR OROPHARYNX

The nasopharynx is best approached through nasal videoendoscopy, although limited exposure can be obtained by a

pharyngotomy with rostral retraction of the caudal free edge of the soft palate. The oropharynx also can be approached orally (see "Tension Palatoplasty" and "Thermal Palatoplasty," later), orally after a mandibular symphysiotomy (see "Surgical Approaches" under "Cleft Palate," later), and through a laryngotomy via incision of the cricothyroid ligament (see "Staphylectomy," later).

Approaches to the oropharynx are performed with the horse under general anesthesia, and the surgical procedures are best performed using long instruments. The pharyngotomy allows access to the caudal third of the soft palate for cleft repair or removal of associated cysts or masses. Likewise, a pharyngotomy provides access to remove a subepiglottic mass or cyst, but a laryngotomy approach has less morbidity, making it preferable. The major disadvantages of the pharyngotomy approach are its limited exposure and the possibility of damaging the hyoepiglotticus muscle (see Figure 44-1) or its innervation. This can result in significant disability, as epiglottic retroversion during exercise is the consequence of neuromuscular damage to the hyoepiglotticus muscle[28] (see Chapter 45). Therefore, the pharyngotomy approach should be reserved for conditions that cannot be approached through the oral route or a laryngotomy and for revision surgery where repeat mandibular symphysiotomy is not indicated.

With the horse under general anesthesia and in dorsal recumbency, the ventral aspect of the basihyoid bone and thyroid cartilage is palpated on the ventral midline (Figure 44-9). The skin incision is extended from the rostral aspect of the thyroid cartilage forward toward the basihyoid bone. The sternohyoideus muscles are separated bluntly on the ventral midline, and the incision is extended to the loose fascia between the thyroid cartilage and the basihyoid bone (Figure 44-10). The hyoepiglotticus muscle is enclosed in elastic fascia (hyoepiglotticus ligament) deep to the loose fascia. The left and right hyoepiglotticus muscles are separated on the midine, and the incision is extended through multiple layers of loose fascia until the oropharyngeal mucosa is reached. The latter is picked up with a rat's tooth forceps and opened with curved scissors. Further exposure is obtained by splitting the basihyoid longitudinally with an osteotome. Malleable or Langenbeck retractors are needed to retract each side of the incision laterad and the root of the tongue rostrad.

The incision is closed using No. 0 poliglecaprone (Monocryl, Ethicon) in a simple-interrupted-continuous pattern on the oropharyngeal mucosa. A few simple-interrupted sutures are used to reappose the loose areolar tissue ventral to the oropharyngeal mucosa. The basihyoid (if it was split) is reapposed using a simple No. 2 steel suture. The sternohyoideus muscle is reapposed using a No. 0 polyglactin 910 or No. 0 Monocryl in a simple-continuous pattern. The subcutaneous tissue and skin are closed only over the split basihyoid bone. Complete closure is not recommended because of the clean-contaminated nature of the incision.

FUNCTIONAL DISORDERS OF THE NASOPHARYNX

Rostral Pharyngeal Collapse

Rostral pharyngeal collapse (i.e., fluttering of the rostral aspect of the soft palate) has been observed during treadmill videoendoscopy and was once thought to be a precursor to DDSP. This condition has been reproduced experimentally by bilateral transection of the tendon of the tensor veli palatini muscle.[10] In this experimental model, rostral instability of the soft palate caused inspiratory obstruction, but DDSP did not occur. The rostral

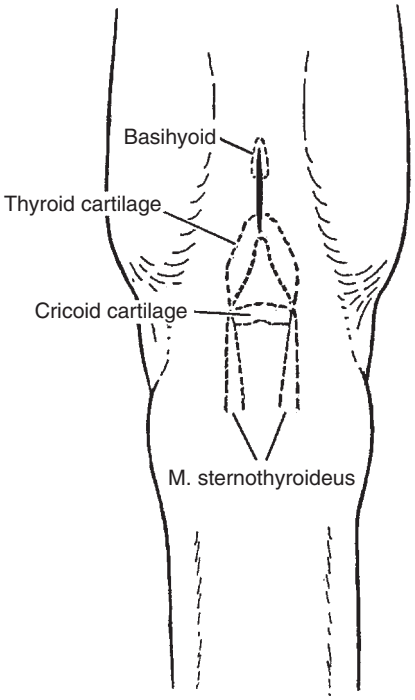

Figure 44-9. Pharyngotomy. Schematic shows the view of the ventral cervical area of a horse in dorsal recumbency and illustrates the landmarks for a pharyngotomy. The *bold line* represents the line of incision extending from the *basihyoid* to the *thyroid cartilage*.

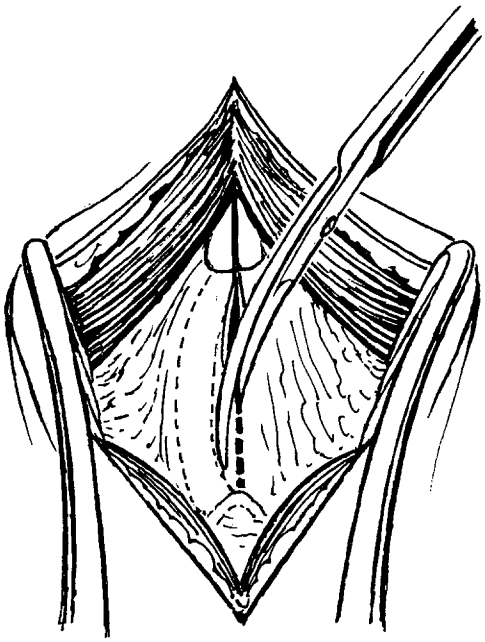

Figure 44-10. Pharyngotomy. The sternohyoideus muscles have been bluntly separated, allowing exposure to the underlying fascia. The basihyoid has been split longitudinally with an osteotome (an optional step), and the superficial fascia is being incised with curved Mayo scissors, exposing the underlying hyoepiglotticus muscle.

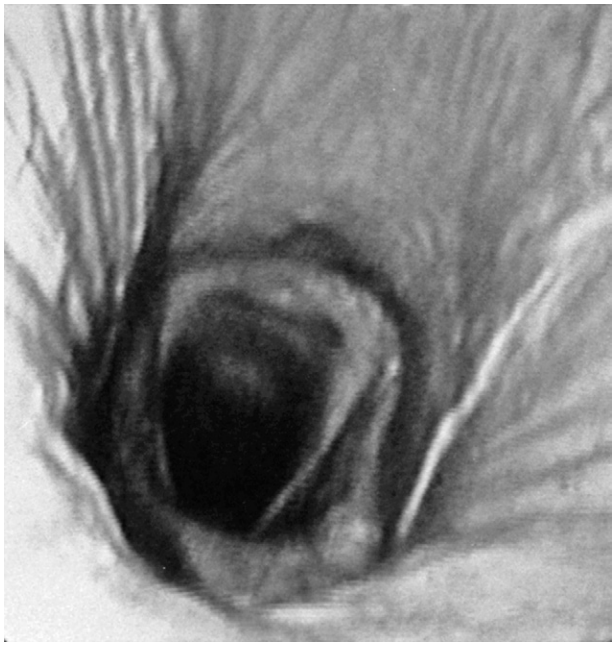

Figure 44-11. Endoscopic photograph of the collapse of the rostral portion of the soft palate in a horse exercising on a treadmill.

Figure 44-12. The rostral aspect of the soft palate after being treated with a laser to induce local fibrosis. This 3-year-old Standardbred filly had rostral nasopharyngeal collapse during exercise.

aspect of the soft palate billows dorsad during inspiration, since it is unable to resist nasopharyngeal pressures (Figure 44-11). It is displaced ventrad during exhalation, so it interferes only with ventilation during inhalation.

Horses with rostral pharyngeal collapse are presented with a history of upper respiratory noise caused by billowing of the rostral soft palate termed *rostral palatal instability (RPI)*. The diagnosis can be obtained only by videoendoscopic examination of nasopharyngeal function while the horse is exercising. The clinical significance of this condition as it relates to athletic performance is unknown, but it is not often performance limiting because of the minor obstruction observed. However, if the entire soft palate billows up and down, a condition known as *palatal instability (PI)* may exist. This may be a precursor of DDSP; therefore, evaluating and perhaps treating the horse for DDSP should be considered if PI is diagnosed.

Medical treatment consists of reducing any nasopharyngeal inflammation with the use of local and systemic anti-inflammatory agents. For an average 450-kg horse, I have used either systemic dexamethasone (30 mg IV or PO daily for 3 days, followed by 20 mg IV or PO daily for 3 days, then 10 mg IV or PO daily for 3 days, and finally 10 mg IV or PO every other day for three treatments) or aerosol fluticasone (3 mg q 12, or 2200 µg 10 puffs once daily) and topical anti-inflammatory solution containing glycerin, DMSO, dexamethasone, and nitrofurazone. I administer 20 mL of a throat flush solution twice a day (composition described earlier). In addition, I empirically recommend a dropped or figure-of-eight nose band to prevent airflow into the oropharynx.

There is no proven surgical treatment, but decreasing the compliance of the rostral aspect of the soft palate is logical.[29-31] However, the methodology to permanently create a sustained decrease in palatal compliance is not known. Indeed, both laser-induced fibrosis[32] and injection of a sclerosing agent (sodium tetradecyl sulfate)[33] failed to show a sustainable biomechanical change in the caudal aspect of the soft palate. Because the rostral aspect of the soft palate is thicker than the soft palate, more-intense fibrosing modalities are used. At this time, it is unknown if these more-aggressive treatment methods used to induce permanent fibrosis on the rostral aspect of the soft palate are effective. With this caveat, I have used laser thermoplasty via nasal approach (diode laser [20 watts] with a contact fiber of 600 µm and pulse duration of 3 seconds) (Figure 44-12) to topically "laser" the soft palate.[29] In addition, it seems rational to consider two other treatments, tension and thermal palatoplasty, which were orginally designed to decrease compliance of the rostral soft palate because their authors hypothesized that this condition was a precursor to DDSP. These two treatments are both performed through an oral approach.

Thermal Palatoplasty

The horse is placed under general anesthesia using an intravenous agent, and a mouth gag is placed. The tongue is pulled rostrad and the head is elevated to facilitate an oral view of the proximal aspect of the ventral soft palate (appropriate lighting is required).[30] A metal instrument is used to protect the tongue during the procedure. Custom-built "irons" are made of two 1-cm–diameter, 50-cm–long steel rods, with a 4- to 6-cm length welded on one end to form a T. After heating the T-shaped metal instrument, the ventral surface of the soft palate (oropharynx) is cauterized, from the hard palate extending caudad to beyond the palatoglossal arch (Figure 44-13, *A* and *B*). Cauterization obliterates the two parasagittal longitudinal folds of the soft palate (see Figure 44-13, *B*), and this results in a scar (see Figure 44-13, *C*), which is thought to decrease the compliance of the soft palate.

Tension Palatoplasty

After general anesthesia induction and placement of a mouth gag, an elliptical incision is made through the oral mucosa,

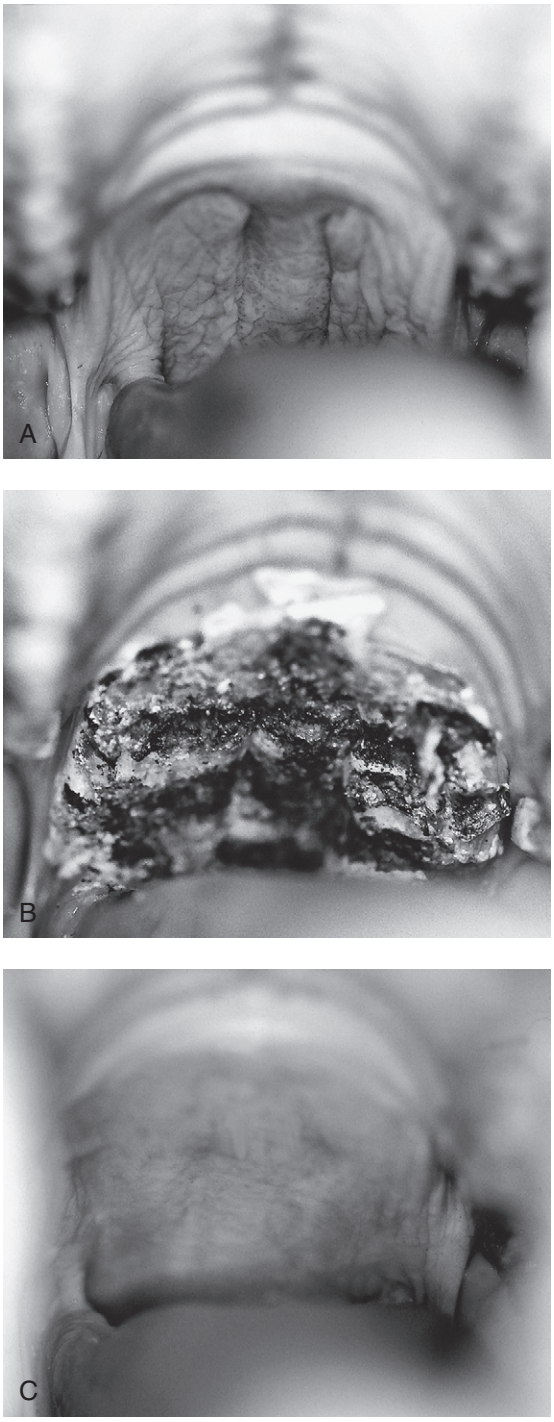

Figure 44-13. Thermal rostral palatoplasty. **A,** Rostral aspect of the soft palate viewed through the oropharynx immediately before surgery. Note parasagittal longitudinal folds on the oropharyngeal mucosa of the rostral aspect of the soft palate. **B,** Appearance of the soft palate immediately after cauterizing. **C,** Appearance of the soft palate 3 months after treatment. (Courtesy R. Ordidge, Rainbow Equine Clinic, UK.)

starting immediately caudal to the caudal edge of the hard palate. An elliptical section of approximately two thirds of the rostral soft palate mucosa and submucosa is excised (9 to 12 cm long by 1 to 2 cm wide) using long-handled forceps and curved Metzenbaum scissors (Figure 44-14).[31] The mucosal and submucosal edges are reapposed using No. 0 Vicryl or Monocryl in

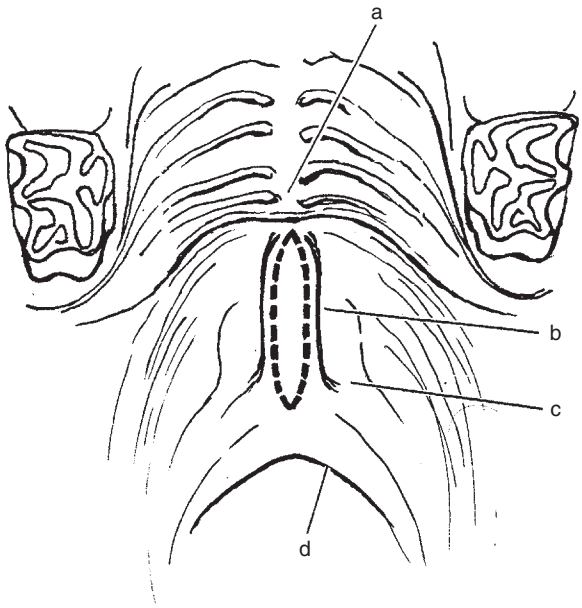

Figure 44-14. Tension rostral palatoplasty. Schematic drawing shows the location of the elliptical incision *(dotted line)* on the ventral and rostral aspect of the soft palate as viewed though the oropharynx. This incision leads to resection of the oropharyngeal mucosa and glandular portion of the soft palate in that specific location. *a,* Hard palate; *b,* isthmus faucium; *c,* sagittal fold; *d,* caudal border of the soft palate.

a simple-interrupted pattern. The procedure can be repeated in 4 weeks (a procedure termed *maximum tension palatoplasty*) to further increase the tension in the soft palate.

Postoperatively, horses are fed mash and wet hay for 3 to 7 days before returning to work. They are administered phenylbutazone for 3 to 5 days and a trimethoprim and sulfonamide combination for 5 to 7 days. Walking exercise is resumed after 2 days (walking and trotting only), and training can resume in 4 weeks. The degree of postoperative pain is reportedly minimal after thermal palatoplasty (2 to 3 days) compared with tension palatoplasty (up to 7 days in some cases).

Prognosis is related to the degree of collapse. A guarded prognosis should be given because the current knowledge of this condition is limited.

Dorsal/Lateral Nasopharyngeal Collapse

Some horses with exercise intolerance and upper respiratory noise are observed to have (1) unilateral or bilateral ventral displacement of the roof of the nasopharynx (Figure 44-15) or (2) medial displacement of the lateral walls of the nasopharynx, or both (Figure 44-16).[34,35] A certain degree of collapse is normal in horses during exercise. For instance, the roof of the nasopharynx does normally displace ventrally at the end of expiration during exercise.[36] At rest, the pressure in the guttural pouches, which form the roof of the nasopharynx, is in phase with nasopharyngeal pressure, and the pressure in the guttural pouches is subatmospheric when no airflow is present at rest.[36] During exercise, guttural pouch pressure does not stay in phase with nasopharyngeal pressure. Nasopharyngeal peak expiratory pressure is reached earlier than in the guttural pouch during the expiratory cycle, so peak expiratory pressure in the guttural pouch lags behind pressure changes in the nasopharynx.[36]

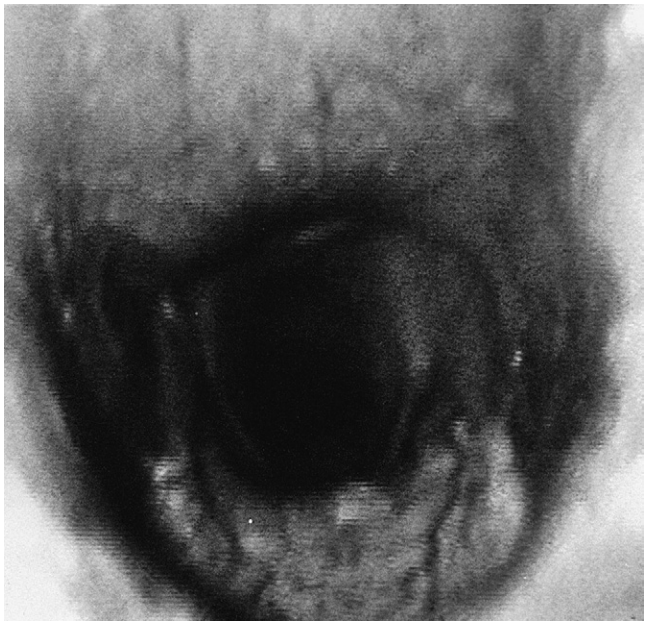

Figure 44-15. Video endoscopic image of the nasopharynx of an exercising horse, demonstrating partial collapse of the dorsal pharyngeal wall. The corniculate processes of the arytenoid cartilages are obstructed from view by the ventral displacement of the roof of the nasopharynx.

Figure 44-16. Endoscopic photograph of medial collapse of the lateral walls of the nasopharynx during treadmill exercise. Note the lateromedial reduction of the nasopharynx.

Therefore, the roof of the nasopharynx normally collapses at the end of expiration, but this should not extend beyond the ventral surface of the fully abducted arytenoid cartilages. It is known from experimental studies that dysfunction of the stylopharyngeus caudalis muscle (see Figure 44-4) results in collapse of the roof of the nasopharynx.[8] Presumably, intraluminal collapse of the lateral walls of the nasopharynx occurs because of dysfunction of the palatopharyngeus muscles. Dysfunction of these muscles may be associated with severe inflammation or neuritis of the pharyngeal branch of the vagal or glossopharyngeal nerve. Alternatively, dorsal and lateral nasopharyngeal collapse may be the result of a sensory dysfunction that prevents appropriate reflex contraction of the intrinsic musculature of the nasopharynx. Guttural pouch distention leads to ipsilateral nasopharyngeal roof collapse. Other systemic diseases, like a hypertrophic periodic paralysis (HYPP) episode, botulism, or equine protozoal myelitis, could be the cause. Finally, nasal obstruction may lead to a more negative inhaling airway pressure, overwhelming the muscular activity of the intrinsic nasopharyngeal musculature.

In the clinical disease, collapse of the nasopharynx occurs during inhalation, causing a greater reduction in the diameter of the lumen of the nasopharynx than in the cross-sectional diameter of the rima glottidis. In my experience, this condition is most commonly seen in 2- and 3-year-old racehorses. Clinical signs of dynamic dorsal pharyngeal collapse include inspiratory upper respiratory noise and exercise intolerance.[34,35] Diagnosis can be suspected by endoscopic examination during nasal occlusion (without sedation). The guttural pouches and both nasal cavities should also be examined, looking for the primary cause of this disease. The diagnosis can only be made accurately, by endoscopic examination during exercise (treadmill or overground).

Treatment should be directed toward resolving the primary condition (e.g., guttural pouch disease, nasal obstruction, systemic disease). In young horses, an anti-inflammatory protocol (as described under "Rostral Pharyngeal Collapse," earlier) should be considered. Affected 2-year-old horses can be allowed to mature until their 3-year season. Some experimental treatments are under investigation, but there is no stabilizing treatment available yet.

Dorsal Displacement of the Soft Palate

Intermittent DDSP is a performance-limiting upper airway condition in horses that was identified in 1.3% of a population of 479 horses examined endoscopically at rest.[37] However, the prevalence of this condition is probably higher, because palate displacement is a dynamic condition that occurs most frequently during intense exercise, making diagnosis at rest imprecise. The prevalence of this condition is probably closer to 10% to 20% of 2- to 3-year-old racehorses.

The horse is an obligate nasal breather, perhaps to allow the olfactory senses to function during deglutition. The normal epiglottis is positioned dorsal to the soft palate and contacts the caudal free margin, forming a tight seal around the base of the soft palate. The pillars of the soft palate converge dorsad, forming the palatopharyngeal arch. When the soft palate displaces dorsad, the epiglottis cannot be seen in the nasopharynx and is positioned in the oropharynx (Figure 44-17). The caudal free margin of the soft palate billows across the rima glottidis during exhalation, creating airway obstruction (see Figure 44-17). Upper airway pressure measurements made in horses clinically affected with DDSP during treadmill exercise indicate that DDSP is an expiratory obstruction.[38] This observation was confirmed by measuring upper airway mechanics in horses in which DDSP was induced by bilaterally blocking the pharyngeal branch of the vagus nerve. Tracheal expiratory pressure and

impedance were increased, minute ventilation was reduced, and horses were more hypoxic and hypercarbic than controls.[37]

From a clinical perspective, DDSP usually interferes only with ventilation during exercise, producing most frequenly, but not always, an upper respiratory noise (approximately 20% to 30% are "silent displacers"). In some cases, there is a digestive disturbance that leads to feed, water, and saliva contamination, predominantly of the upper airways. It is important to identify this digestive disturbance for the following reasons. Horses with DDSP are usually presented with a respiratory deficit that only occurs during intense exercise. This presentation carries a reasonable prognosis, and treatment varies depending on the specific cause. However, true DDSP with dysphagia generally indicates a more-advanced deficit of the palatinus/palatopharyngeus muscles (or its innervation) or an anatomic deficit (such as cleft palate or an acquired deficit of the caudal free edge of the soft palate—that is, staphylectomy or loss of epiglottic cartilage secondary to septic epiglottis or surgical trauma) or a mechanical deficit as after placement of laryngeal prosthesis that allows feed contamination of the nasopharynx from the oropharynx. This should be differentiated from aspiration of feed material into the trachea from laryngeal disease (or its treatment), which can lead to DDSP, presumably by inducing ventral and caudal laryngeal movement-displacement. Equine clinicians should carefully attempt to identify the cause of feed contamination of the upper airway seen in association with DDSP; applying routine treatment of intermittent DDSP to a truly dysphagic horse because of palatimus neuromuscular damage or mechanical deficit after laryngoplasty are not successful and may worsen the aspiration.

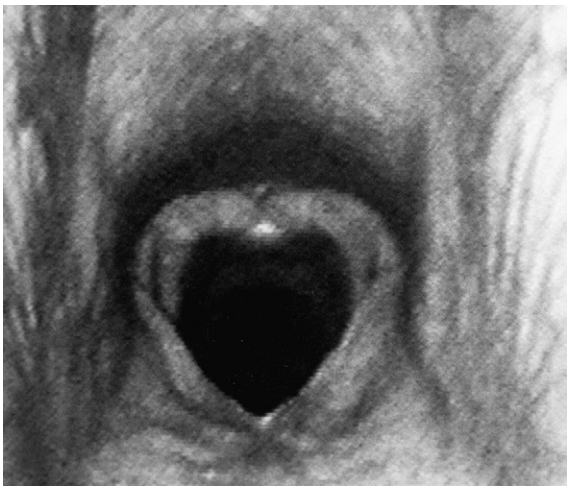

Figure 44-17. Videoendoscopy of the rima glottidis of a horse experiencing dorsal displacement of the soft palate during exercise. The palate moves ventrad relieving obstruction of the the rima glottidis during inhalation, but billows dorsad as air escapes into the oropharynx during exhalation.

Observational Studies on the Etiopathogenesis of Dorsal Displacement of the Soft Palate

The early hypotheses for the occurrence of DDSP was paralysis of the palate muscles[39] followed by elongation of the soft palate[40]; those hypotheses have not been supported by modern investigations. From a clinical perspective, anectodal observation (not evidence-based data) suggests mechanical factors to be associated with the the etiopathogenesis of DDSP. For example, cysts on the caudal free edge of the soft palate have been observed to interfere with the normal subepiglottic position of the soft palate. This seems to be a mechanical effect, since removing the cyst results in immediate postoperative correction of the DDSP. Other lesions that mechanically interfere with the junction of the caudal free edge of the soft palate and subepiglottic tissue are also believed to result in DDSP. They include subepiglottic or palatal granuloma (Figure 44-18), masses, and cysts (see Figure 45-27). The current understanding is that these masses predispose to DDSP, either by mechanically interfering with the seal between the caudal free edge of the soft palate and subepiglottic tissue or by causing irritation or pain that stimulates DDSP.

Epiglottic hypoplasia or deformation has been implicated as a cause of DDSP in horses.[41,42] It was thought that inadequate length or rigidity of an epiglottis might make it unable to "hold" the soft palate in a subepiglottic position.[41,42] Equine practitioners have noted that some horses with intermittent DDSP have

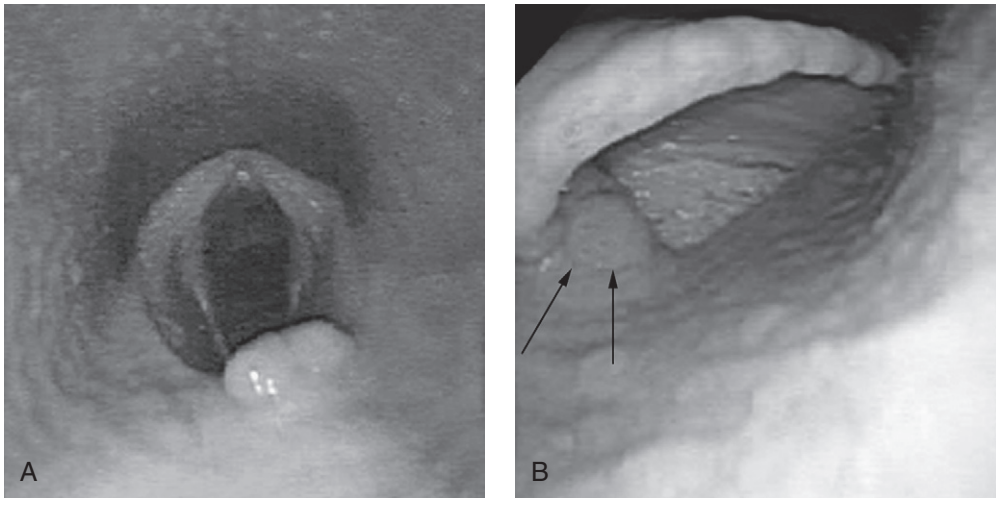

Figure 44-18. A, Palatal granuloma in a 2-year-old Thoroughbred with dorsal displacement of the soft palate. **B,** Appearance of the granuloma dorsal to the palate and ventral to the epiglottic cartilage *(arrows)* with the soft palate replaced.

what appears to be a hypoplastic or flaccid epiglottis. There has a been a clinical association between the endoscopic appearance of short or flaccid epiglottic cartilages in yearlings and decreases in racing performance later.[43] However, the appearance of a flaccid epiglottic cartilage is probably a positional change. I have noted that in horses experiencing DDSP, a progression of morphologic changes in the epiglottis frequently occur prior to DDSP (as observed endoscopically during high-speed exercise). First, caudal retraction of the larynx leads to an increase in "apparent flaccidity" of the epiglottic cartilage by the soft palate being lifted against the ventral surface of the epiglottic cartilage generally accompanied by billowing of the soft palate (termed *palatal instability*) eventually followed by DDSP. A swallow seems to reset the larynx forward and the correct subepiglottic position of the soft palate. This progression of events is repeated a few times before persistent DDSP is seen. The importance of swallowing in the occurrence of DDSP was highlighted by the recent report indicating that during exercise, horses with DDSP swallow more often in the minute immediately prior to displacement than those horses that do not experience DDSP, supporting the hypothesis that horses with DDSP are trying to stabilize an unstable palate by swallowing.[44]

Therefore, a flaccid epiglottis is probably a result rather than a cause of the DDSP. Indeed, the role or importance of an abnormal epiglottis as the initiating factor in DDSP is questionable, because DDSP does not occur despite the fact that epiglottic cartilage is not present to "hold the palate down" when epiglottic retroversion is experimentally induced.[45] Therefore, I no longer believe that epiglottic augmentation (i.e., stiffening) should be used routinely to treat horses with intermittent DDSP.

Other factors implicated in the pathogenesis of DDSP include caudal retraction of the tongue, which was hypothesized to lead to caudal retraction of the larynx.[46] If the tongue is retracted, the base of the tongue may "push" the soft palate dorsad, inducing DDSP. This leads to the belief that a tongue-tie pulls the tongue out of the mouth and prevents this occurrence. However, experimental investigations failed to identify a morphologic or physiologic effect from a tongue-tie.[17,47,48] Despite this, clinical studies support the use of a tongue-tie. One report showed that DDSP was reversed in two out of six horses by using a tongue-tie.[49] Two cohort studies (evidence–based level II-2) have reported a positive preventative effect of a tongue-tie on the occurrence of DDSP.[16,50]

The clinical association of opening the mouth or swallowing during exercise and the induction of DDSP has led to the following hypothesis. Opening the mouth lets air enter the oropharynx, disturbing the stabilizing effect of the subatmospheric pressure on the ventral surface of the soft palate.[46] This has led to the use of a dropped or figure-of-eight nose band to prevent DDSP during exercise. There are no experimental data investigating this hypothesis.

Most recently, three studies reinforce the possiblity that the position of the larynx and hyoid bone are relevant to the occurrence of DDSP. First, it was found that horses with DDSP at exercise have a more ventral position of their basihyoid bone.[51] In parallel with this finding, horses that have a more dorsal position of their basihyoid bone after laryngeal tie-forward surgery have a better postoperative outcome.[52] Finally, horses with permanent DDSP, a presumbly more severe form of DDSP, had a more caudal position of their larynx compared to horses with intermittent DDSP.[53] These static findings of the position of the basihyoid bone may be associated with the observation that a strong pull or "hold" of the drivers on the lines during exercise can lead to DDSP during exercise, perhaps by influencing the position of the basihyoid bone or larynx.[54]

Experimental Studies on the Etiopathogenesis of DDSP

Many studies described earlier have led to the identification of intrinsic and extrinsic factors responsible for the stability of the nasopharynx at exercise, and their anatomical relevance has been described. Functionally, there are three experimental models that result in DDSP in horses. Model 1, *blockade of the pharyngeal branch of the vagus nerve model*, signified that the palatinus and platopharyngeus muscles are important in stabilising the soft palate at rest and at exercise.[7] Deficits of the palatinus muscle function also lead to feed and water contamination of the upper airway.[7] The remaining two experimental models differ from model 1 because DDSP occurs only at exercise. Model 2, *thyrohyoideus muscle model,*[15] indicated that the actions of these muscles decrease the collapsibility of the nasopharynx at exercise; this model supports the beneficial effect of swallowing on palatal stability. Model 3, *blockade of the hypoglossal nerve model*, showed a stabilizing effect on the nasopharynx.[3] The exact mode of action of the affected muscles in the latter two models is unknown, but it is hypothesized that they prevent caudal and ventral movement of the larynx during exercise, a phenomemom that is corrected (at least temporarly) by swallowing.

Summary of the State of the Art on the Etiopathogenesis of DDSP

Structural causes of DDSP, such as palatal cyst, subepiglottic masses, epiglottic entrapment, subepiglottic cyst, epiglottic deformity, and so on, contribute to a small percentage of the cases of DDSP. In the majority of the cases, DDSP is a functional deficit where inappropriate intrinsic muscular contraction results in an increased compliance of the soft palate, which in turn leads to PI and eventually DDSP. A dorsal position of the larynx and basihyoid bone confers stability to the soft palate during exercise, and through a mechanism not yet understood; swallowing can correct DDSP or temporarily decrease the collapsibility of the soft palate into the airway.

History and Clinical Signs

Racehorses with intermittent DDSP generally have a history of exercise intolerance and may make a gurgling or vibrating noise during exhalation.[46] The exercise intolerance is most commonly an acute drop of performance that may be described by the trainer or owner as "choking down"or "swallowing the tongue." Concurrent with these signs is open-mouth breathing, because airflow is directed through the mouth during exhalation. Horses used for show or pleasure riding may exhibit only the respiratory noise described, and the signs are usually exacerbated with head flexion.

Diagnosis

It is now well established that the resting endoscopic examination has a poor predictive value in determining the occurrence of DDSP during exercise.[55-57] Therefore, the accuracy of an endoscopic diagnosis of DDSP in nonexercising horses is poor and

leads to a misdiagnosis 35% of the time.[56] The gold standard for diagnosis of intermittent DDSP is based on a history of poor performance associated with respiratory noise, physical examination, and endoscopic examination at rest and during exercise either on a treadmill or over ground. It should be noted that both treadmill and overground endoscopic examinations in noncompeting horses is likely to miss the occurrence of DDSP in a small percentage of horses. In addition, unlike for the diagnosis of laryngeal hemiplegia, where both techniques are comparable, the treadmill exam has a higher diagnostic rate of DDSP.[58] In my opinion this is because treadmill exercise protocols are well established and exercise protocols for overground exams are not fully established, plus the intensity of exercise during the overground exam is negatively influenced by the trainer's limitation on the exercise intensity). A complete physical examination is important to rule out other concomitant causes of exercise intolerance, such as pulmonary disease, cardiac abnormalities, lameness, and neurologic disease.

Should an endoscopic exam during exercise not be available, an endoscopic examination immediately after cessation of exercise is preferrable. Here, the observation of DDSP would support the diagnosis. If only a resting exam is available, surrogate evidence (but not pathognomonic) of DDSP is an ulcer on the caudal edge of the soft palate (Figure 44-19) and brusing on the nasopharynx (Figure 44-20). To further assess the soft palate and subepiglottic area, displacement can be induced in most horses by introducing the endoscope into the larynx and proximal trachea and inducing a gag reflex. After withdrawing the endoscope, one can see the free edge of the soft palate. A more complete exam is done by sedating the horse and applying 50 to 100 mL of lidocaine hydrochloride on the soft palate and epiglottis. Then, using an appropriate instrument (equine laryngeal forceps), the epiglottic cartilage can be elevated to look for subepiglottic abnormalties.

Treatment

I make treatment recommendations using evidence-based data, when available. There is a paucity of data available and the data are at best only at level II-b, so be aware that some of the following recommendations are based on weaker evidence.

Treatment should be initially directed at modifying or eliminating recognized factors associated with the occurrence of DDSP. If structural abnormalities (cysts, granuloma, entrapment, epiglottis, etc.) are found, they should be adressed. If no structural abnormalities are present, nonsurgical management should be used first.

NONSURGICAL MANAGEMENT OF DDSP

Both nasopharyngeal and guttural pouch inflammation can lead to dysfunction of the pharyngeal branch of the vagus nerve[7] and thus interfere with palatinus and palatopharyngeus muscle tone, leading to PI and/or DDSP. Indeed, horses with intermittent DDSP have an elevated prevalence of upper respiratory inflammatory diseases, such as pharyngitis, that may affect the function of the pharyngeal branch of the vagus nerve.[7,59] If upper respiratory tract inflammation is diagnosed, treatment should include judicious use of systemic anti-inflammatory medication (dexamethasone as described earlier or aerosol fluticasone 3 mg every 12 hours, or 2200 µg 10 puffs once daily)[60] and topical anti-inflammatory solution containing glycerin, DMSO, dexamethasone, and nitrofurazone.

In addition to considering inflammatory conditions, one should take into account that unfit and immature horses should be conditioned and re-evaluated before surgical intervention is considered. Tack changes may be suggested to alter (i.e., elevate) the horse's head position, and a figure-of-eight noseband can be used to keep the horse from opening its mouth and allowing airflow into its oropharynx. The bit may be changed; a bit that secures the tongue (such as a W bit, a spoon bit, or a "Serena

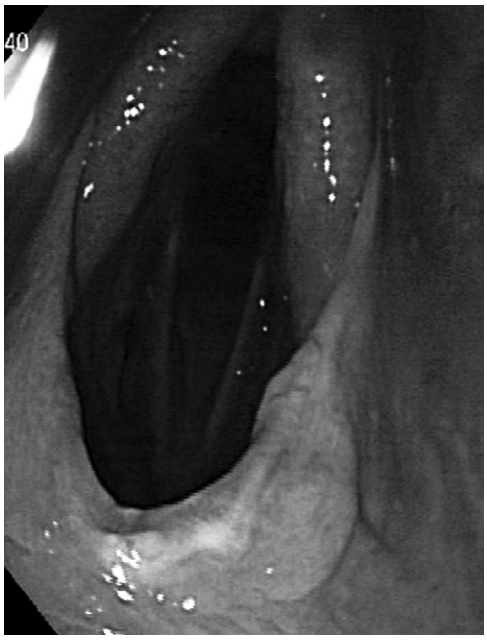

Figure 44-19. Ulcer on the free edge of the soft palate in a 3-year-old Standardbred. This finding supports a diagnosis of DDSP and can also be seen if a subepilgottic mass or lesion is present.

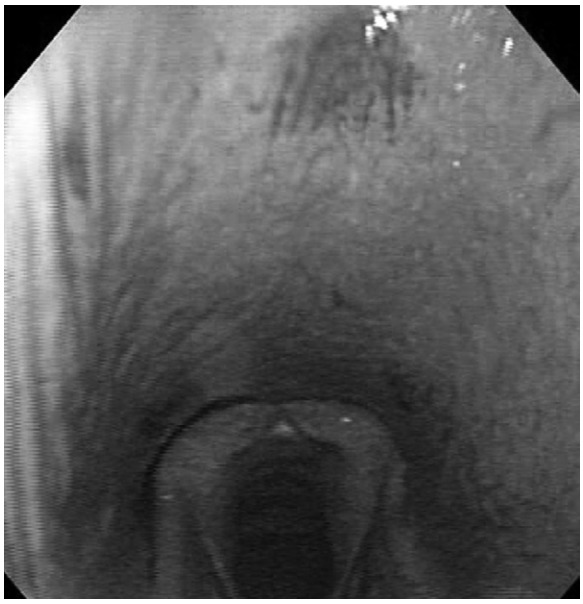

Figure 44-20. Bruising of nasopharynx approximately12 hours following racing in a 7-year-old Standardbred gelding. This supports a diagnosis of DDSP during racing.

song" bit) and/or is designed to restrict caudal movement of the tongue should be used in addition to a tongue-tie.

Tongue-tie use has been popular for many years, and now there are some evidence based data to support its use.[16,4 9,50] Initially the hypothesis was that it prevented "swallowing of the tongue," but most likely it prevents the root of the tongue from applying dorsal pressure on the ventral surface of the soft palate or prevents the basihyoid or larynx from moving caudad. In one study, two out of six horses with naturally occuring disease were improved with a tongue-tie, as evidenced by treadmill videoendoscopy.[49] Additionally, the tongue-tie was part of the medical treatment of horses with naturally occuring disease in two studies, which reported a 53% to 61% success rate.[16,50] Compared to this positive result, others failed to detect a mechanical effect (a change of nasopharyngeal diameter or upper airway patency) when the tongue-tie was used in normal horses.[47,48] A throat-support device (Throat Support Device*) that positions the larynx and basihyoid more dorsad and rostrad similar to the tie-forward surgery (see later) has been shown to prevent DDSP at exercise in experimentally created DDSP[61] and is frequently used in race horses to control DDSP or as a diagnostic tool in horses suspected to have DDSP.

The data are insufficient to draw clear conclusions on all the nonsurgical treatments. However, this type of treatment should be used first when anatomic anomalies are not present. Reports show a 53% to 61% success rate when nonsurgical treatments were used alone, a figure similar to the outcome seen after many surgical treatment techniques.[62,63] If the horse fails to respond to medical or nonsurgical treatment, surgical intervention should then be considered.

SURGICAL OPTIONS

Many surgical options are available. Tension and thermal palatoplasty, described previously for treatment of rostal palate instability, have been shown recently to be ineffective for treatment of DDSP and should no longer be performed for this condition.[16,64] In addition, no technique has been shown to be effective in stiffening the soft palate, so laser staphylectomy or injection of sclerosing agent should be abandoned.[31,32]

The treatments should be directed to the recognized cause of the disease when it can be identified. Structural or anatomic abnormalities involving the nasopharynx or subepiglottic and epiglottic cartilage (such as bacterial or viral epiglottitis, cyst, granuloma, aryepiglottic entrapment) should be addressed medically or surgically through a nasal, oral, or laryngotomy approach. Recent evidence-based data (level II-b) suggest that at present the best treatment is a laryngeal tie-forward combined with a partial bilateral resection of the thyrohyoideus tendon/ muscle.[52] Despite these findings, other surgical treatments are indicated under certain conditions and are discussed later.

Staphylectomy

Staphylectomy, or partial soft palate resection, originally was described as a treatment for an excessively long soft palate.[40] This condition does not exist, except perhaps in neonatal foals,[65] so staphylectomy should also be discontinued as a routine treatment of DDSP. When a staphylectomy is performed one should be aware that resection of more than 0.75 cm (¼ inch) of the soft palate may disturb the seal between the oropharynx and the nasopharynx and allow the passage of water and saliva into the nasopharynx. Staphylectomy should be reserved to resect a granuloma or cyst from the caudal free edge of the soft palate or for treatment of permanent DDSP as an additional step following a laryngeal tie-forward procedure.

For treatment of palatal cysts or granulomas, staphylectomy is performed with the horse under general anesthesia and positioned in dorsal recumbency with the head and neck extended and prepared for a laryngotomy.[62] A 10- to 12-cm (4- to 5-inch) skin incision is made along the midline, centered over the cricothyroid space. The sternohyoideus muscles are divided bluntly using a curved Mayo or Metzenbaum scissors. A self-retaining retractor, such as a Weitlaner or a Hobday, is placed between the separated sternohyoideus muscles, exposing the cricothyroid membrane. The cricothyroid membrane is sharply incised (along with the underlying laryngeal mucosa) with a scalpel along the midline, from the cricoid cartilage to the junction of the thyroid cartilages (Figure 44-21). A small blood vessel is usually also incised at the level of the caudal two thirds of the membrane. The vessel is ligated or cauterized. The self-retaining retractor is subsequently placed within the cricothyroid space. The caudal free margin of the soft palate rostral to the incision is identified. If the horse was intubated nasotracheally, the soft

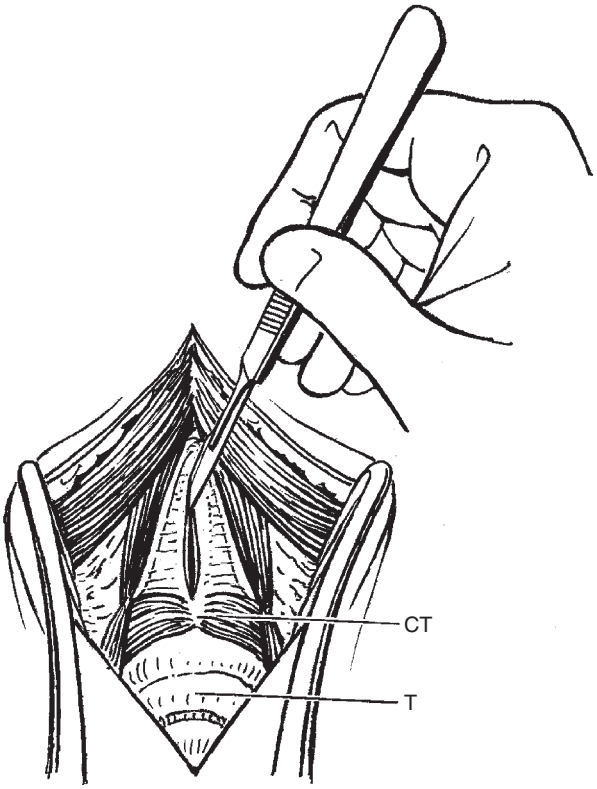

Figure 44-21. Schematic showing view of the ventral cervical area of a horse in dorsal recumbency and illustrating the landmarks for a laryngotomy. The sternohyoideus muscles have been bluntly separated on the ventral midline, and the cricothyroid membrane and underlying laryngeal mucosa is sharply incised from the cricoid rostrally to the thyroid cartilage. *T,* Tracheal ring; *CT,* cricothyroid muscle overlying the ventral aspect of the cricoid cartilage.

*Conflict-of-interest disclosure: The author and Cornell University both benefit through patent royalties and have equity interest with the company marketing this product.

palate may not be displaced, and the caudal edge of the soft palate may have to be freed from beneath the epiglottis using a pair of sponge forceps. If the horse was intubated orally, the palate will be displaced and the endotracheal tube should be retracted at this time.

The mass or cyst is identified, the mucosa of the caudal free margin of the soft palate is grasped with an Allis tissue forceps on the midline, and a second pair of forceps is used to grasp the left and right mucosa 2 to 2.5 cm lateral to the midline. The caudal free margin of the soft palate containing the mass or cyst is resected using a curved Satinsky thoracic scissors. A crescent-shaped 3- to 4-cm–long piece of mucosa that is less than 1 cm wide on the midline and tapered toward both ends should be resected.

The laryngotomy may be left to heal by second intention, or preferably the cricothyroid membrane is reapposed using No. 0 polyglactin 910 suture material in a simple-continuous pattern. Some surgeons close all layers of the laryngotomy, but this step increases morbidity unnecessarily. Postoperatively, the horse should wear a muzzle for several hours. Systemic antibiotic therapy is continued for 7 days, and anti-inflammatory medication is continued for 3 days. The laryngotomy site should be cleaned twice daily until it is healed (approximately 3 weeks). The horse can resume training 2 to 3 weeks later.

In the treatment of permanent DDSP, the gap between the dorsal aspect of the epiglottis and the overlying soft palate is eliminated by the tie-forward surgery (Figure 44-22, *A* and *B*). When needed, a laser-assisted staphylectomy follows the tie-forward procedure to permit the epiglottis to be repositioned dorsal to the soft palate after a swallow. The goal is to remove the least amount of soft palate needed to allow the epiglottis to remain positioned dorsal to the soft palate without loss of laryngopalatal seal. This is assessed by correcting the displacement with a laryngeal forceps and observing the redundant palate underneath the epiglottis, which needs to be trimmed. It is imperative that the site of intended resection is marked prior to using the laser, because the soft palate is so elastic that inadvertent excessive resection could occur (Figure 44-23, *A*). The edge of the soft palate is then lifted and the soft palate is resected (Figure 44-23, *B*).

Preoperatively, the horse is treated with NSAIDs and broad-spectrum antibiotics. Complications of staphylectomy are rare but can occur if too large a section of the soft palate is resected. These horses may be dysphagic, leading to signs of aspiration that include coughing, expulsion of feed material through the nose, and pneumonia.

Standard myectomy

Standard myectomy (a partial sternohyoideus and sternothyroideus, with or without omohyoideus resection) is performed to reduce caudad retraction of the larynx.[46,62,63,66] Sections of the sternothyroideus and sternohyoideus muscles are removed with the horse standing as the procedure was first described. If the plan is to also remove a section of omohyoideus muscle, the procedure should be performed under general anesthesia with the horse positioned in dorsal recumbency and the head and neck extended. This is because a more-extensive dissection is required when the omohyoideus muscle is resected.

The horse should be treated preoperatively with NSAIDs and broad-spectrum antibiotics. It is restrained in a set of stocks, a stanchion, or a stall doorway. Tranquilization may be necessary or useful. (Alternatively, the horse is anesthetized and placed in dorsal recumbency.) The hair is clipped from the ventral surface of the neck, and the skin is aseptically prepared. Local anesthetic is infiltrated along the midline at the junction of the proximal third and the middle third of the neck.

A 10-cm ventral midline incision is made through the skin, continuing through the cutaneus colli muscles. The paired sternohyoideus muscles are identified. Using curved forceps, the sternohyoideus and sternothyroideus muscles are undermined. The sternothyroideus muscle is positioned caudolateral to the sternohyoideus muscle at this level of the neck. The muscles are elevated through the incision and clamped with a Rochester-Carmalt forceps at the proximal and distal extent of the

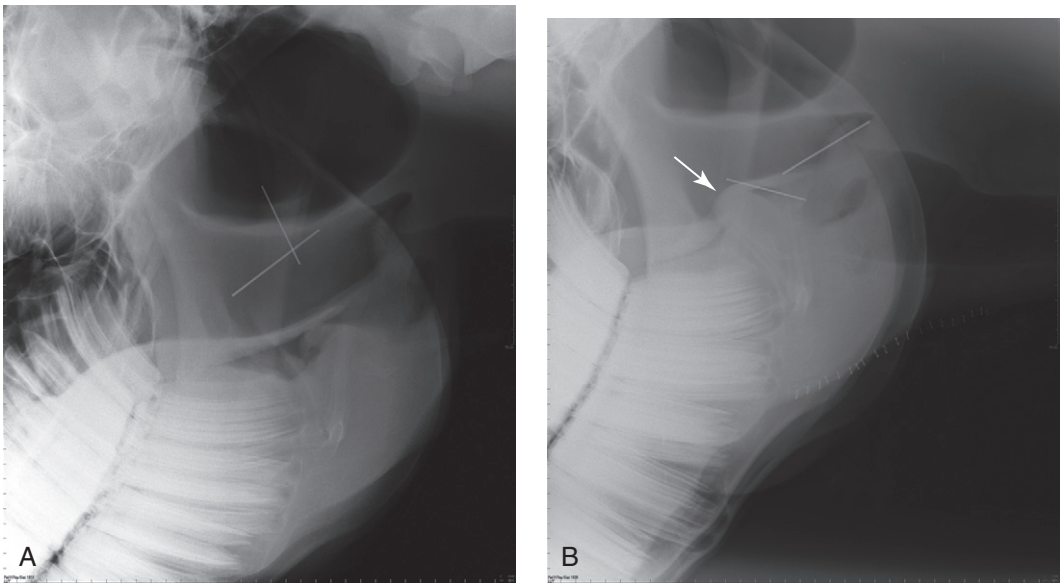

Figure 44-22. Lateral radiograph of a 4-year-old Thoroughbred gelding with a permanent displacement of the soft palate prior to surgery **(A)** and after laryngeal tie-forward **(B).** Note the air in the mouth and the ventral position of epiglottis prior to surgery. After surgery **(B)** the epiglottis is bulging *(arrow)* against the soft palate. Linear white densities are 5-cm (2-inch) pins used to measure magnification of radiogaphs.

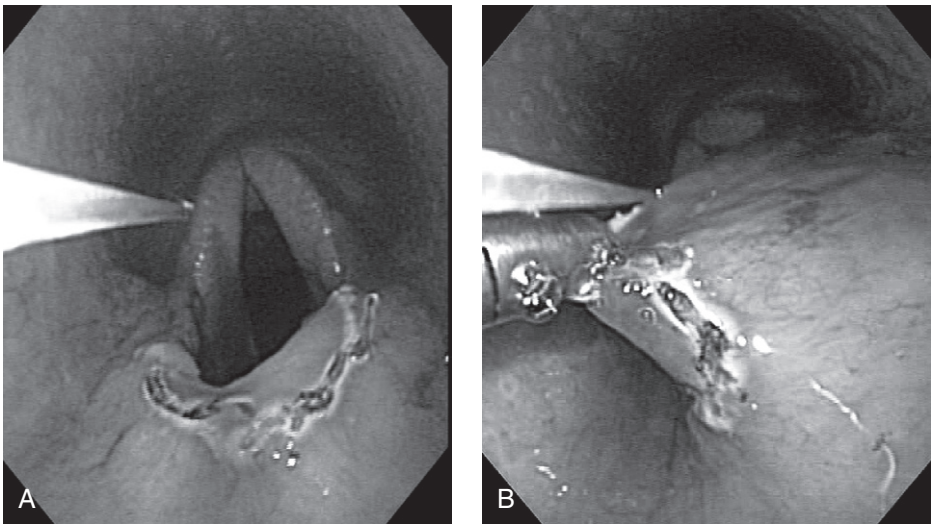

Figure 44-23. Intraoperative laser staphylectomy in a 4-year-old Thoroughbred colt with permanent DDSP after first having performed a laryngeal tie-forward. **A,** the intended line of resection has been marked using a diode laser. **B,** The left edge of the soft palate is grasped with a laryngeal forceps and the marked line of resection is cut.

incision. The muscle bellies are sharply transected between the forceps, removing a 6- to 8-cm–long section of muscle. The muscle tissue that was removed should be inspected to ensure that sections of both sternohyoid muscles and the smaller sternothyroid muscles were indeed removed. Previously, the omohyoideus muscle was sometime removed; however, significant dead space was created, leading to a higher rate of incisional complications, and therefore resection of the omohyoideus is no longer recommended.

The subcutaneous and skin layers are closed routinely. If a Penrose drain is used, it is placed alongside the ventral aspect of the trachea and tunneled through a stab incision distal to the surgical incision. A firm bandage is applied around the neck and may be removed along with the drain 24 hours later.

NSAIDs can be continued for 3 days, and antibiotics should be continued for 5 to 7 days. The horse is kept in a stall with daily hand-walking for 2 weeks. Training can be resumed 2 weeks after surgery, when the sutures are removed. Complications are usually related to the incision and include incisional seromas and infections. No long-term complications are notable except for the cosmetic defect associated with the lack of muscle tissue at the surgical site. Uncontrolled reports indicate a success rate of 58% to 71% for treating DDSP after this procedure.[67] However, standard myectomy has lost some popularity for treatment of DDSP.

Minimally invasive myectomy

Minimally invasive myectomy (partial sternothyroidectomy or the Llewellyn procedure) is rapidly performed and has minimal morbidity. It is often performed as a first line of surgical treatment because it does not require an operating theater, and it is simpler than the standard myectomy and thus is the field procedure of choice.

Partial sternothyroideus myectomy and tenectomy is performed with the horse under general anesthesia.[68] A 5- to 7-cm ventral midline skin incision is made centered on the cricoid cartilage. The subcutaneous tissue is incised, and the sternohyoideus muscles are divided bluntly using curved Mayo or Metzenbaum scissors. The blunt dissection is continued dorsal to the sternohyoideus muscle, exposing the caudolateral border of the thyroid cartilage. The tendon of insertion of the sternothyroideus muscle at the thyroid cartilage is identified, undermined, and elevated. The tendon is transected 1 cm caudal to its attachment to avoid the caudal laryngeal artery, taking care to avoid damaging the cricothyroid muscle. Damage to the cricothyroid muscles may result in vocal cord collapse.[69] Using one index finger, the sternothyroideus muscle is freed from the surrounding fascia and transected more proximally, thus removing a 3-cm section of muscle. This same procedure is performed on the contralateral sternothyroideus tendon. The incision is closed by reapposing the sternohyoideus muscles, and the skin is closed in a routine manner.

Postoperatively, NSAIDs are given for 3 to 7 days. Training often is resumed 2 to 3 days postoperatively in Standardbreds and 2 to 3 weeks postoperatively in Thoroughbreds. The reported success rate for this procedure is 58% to 70%.[62,63,68]

Laryngeal tie-forward

The laryngeal tie-forward is performed with the horse anesthetized and in dorsal recumbency.[15,52,70] The principle of the procedure is to replace the action of the thyrohoideus muscles bilaterally by sutures placed between the thyroid cartilage and the basihyoid bone (Figure 44-24). The ventral cervical and intermandibular areas extending 10 cm rostral to the basihyoid bone are prepared aseptically. A ventral skin incision is made starting 1 cm caudal to the cricoid cartilage and extending 2 cm rostral to the caudal aspect of the basihyoid bone. The sternohyoideus muscle is separated on the midline and bluntly dissected free of the dorsolateral aspect of the larynx lateral to the thyrohyoideus muscles. The sutures are first passed through the thyroid cartilage. A No. 5 USP polyblend suture (Fiberwire) is passed twice into the right lamina of the thyroid cartilage ventral to the insertion of the sternothyroid tendon (Figure 44-25). Alternatively a metal buttress is placed on the medial side of the thyroid cartilage to minimize cutting of the thyroid cartilage with sutures.[71] The junction of the basihyoid and lingual process is identified with a Crile forceps after limited blunt dissection, and a wire passer is placed under the hyoid

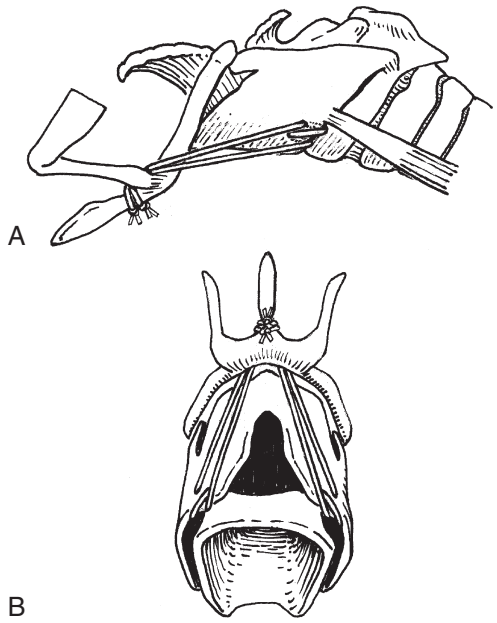

Figure 44-24. Schematic drawing showing the principle of the laryngeal tie-forward procedure. **A,** Lateral view: Note that the sutures are placed from the basihyoid into the lateral and caudal aspects of the lamina of the thyroid cartilage. The suture is passed twice through the thyroid cartilage and the most dorsal bite is immediately ventral to the tendon of the sternothyroid muscle. **B,** Ventral view: The sutures course on the dorsal surface of the basihyoid bone and are tied with a slip knot on the ventral aspect of the junction of basihyoid bone and lingual process.

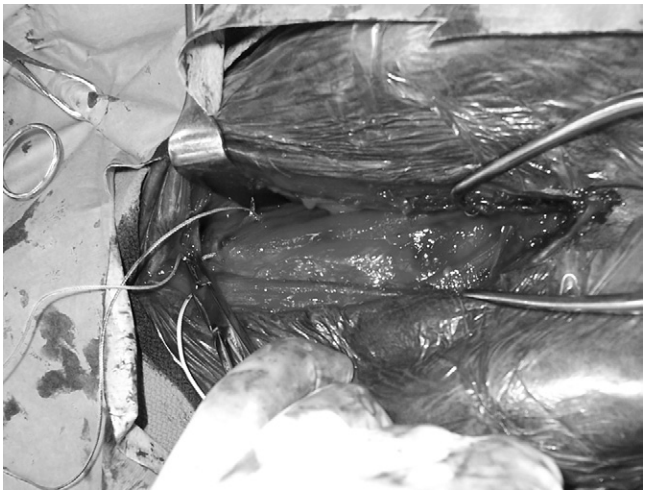

Figure 44-25. Intraoperative view of the laryngeal tie-forward procedure. Surgical view showing one suture passed twice (approximately 1-cm bites) through the lamina of the thyroid cartilage with the most dorsal suture entering ventral to the tendon of the ST tendon. This gives a dorsal (leader) and ventral (trailer) end of the sutures that will be passed around the basihyoid bone. Orientation: caudal to the left, rostral to the right.

bone immediately lateral to the lingual process. The wire passer courses over the dorsal aspect of the basihyoid bone and exits on the midline at the caudal aspect of the basihyoid bone (Figure 44-26). After the needle has been cut, the dorsal (leader) suture and the ventral (trailer) suture of the

Figure 44-26. Intraoperative view of the laryngeal tie-forward procedure. Surgical view showing a wire passer in place where it enters immediately lateral to the lingual process in the corner *(white arrows)* of the junction of the basihyoid and lingual process. The wire passer courses over the dorsal aspect of the basihyoid bone and exits on the midline at the caudal aspect of the basihyoid bone *(black arrow).* Orientation: caudal *(bottom),* rostral *(top).*

contralateral side are passed into the wire passer and retrieved. The procedure is repeated on the other side (Figure 44-27) such that the dorsal (leader) and ventral (trailer) sutures of each side can be tied over the ventral aspect of the basihyoid. A bilateral partial sternothyroidectomy is performed at this time. The sutures on each side are then tied so the rostral aspect of the thyroid cartilage is located immediately dorsal and 0.1 to 1.5 cm rostral to the caudal border of the basihyoid bone. Closure is obtained by reapposing the sternohyoideus muscles with No. 0 poliglecaprone (Monocryl, Ethicon) in a simple-continuous pattern. The loose fascia overlying the larynx is incorporated into that closure; this is an important step to prevent postoperative seromas. The subcutaneous tissues and skin are then closed in a routine manner.

Postoperatively the horses are fed and watered at shoulder height to reduce stress on the sutures. They are administered NSAIDs for 3 to 5 days. Horses are maintained in a box stall for 2 weeks with daily handwalking. Training often is resumed 2 to 3 days after this rest period.

Assessment of postoperative success is obtained by endoscopy where the epiglottis is located more forward and dorsally, often not contacting the soft palate (Figure 44-28). Radiographs could be taken with the head in an extended position,[72] and after surgery one should be able to observe (and measure)[52] a forward and dorsal position of the tip of the epiglottis and larynx on the image (Figure 44-29, *A* and *B*). Success rate of 20% greater than that reported for partial sternothyroideus myectomy and tenectomy (strap muscle resection) is expected.[52,70]

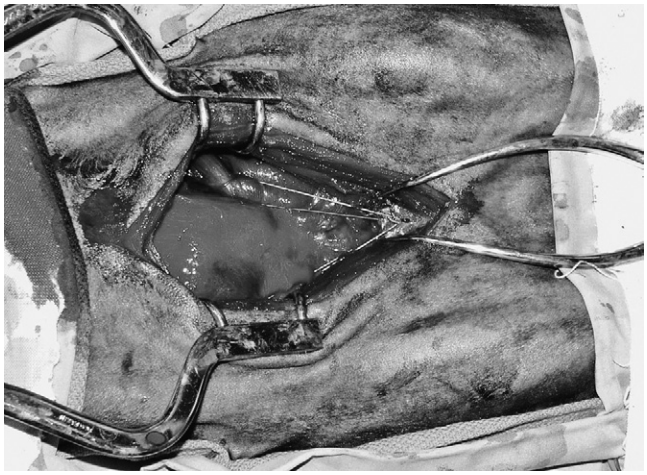

Figure 44-27. Intraoperative view of the laryngeal tie-forward procedure, after the sutures have been placed so that the dorsal suture (leader) has been passed ipsilateral to the lingual process and the ventral (trailer) end is passed on the contralateral side. Orientation: caudal *(bottom),* rostral *(top).*

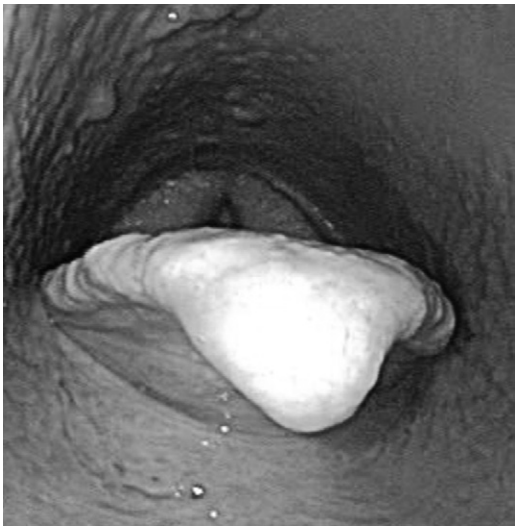

Figure 44-28. Postoperative view of larynx in a 3-year-old Standardbred filly. Note that the epiglottis is elevated and not contacting the soft palate.

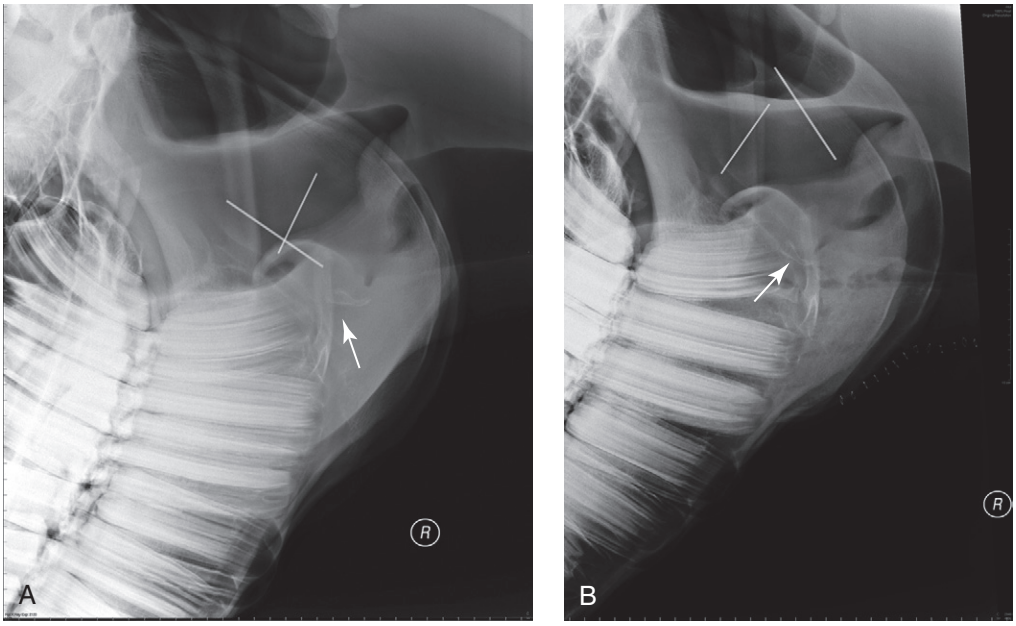

Figure 44-29. Lateral radiograph of a 3-year-old Thoroughbred filly with a intermittent displacement of the soft palate prior to surgery **(A)** and after laryngeal tie-forward **(B).** Note the tip of the epiglottis is more rostral and dorsal after surgery. Also the ossification at the base of the thyroid cartilage *(white arrow)* is more rostral and dorsal after surgery. Linear white densities are 5-cm pins used to measure magnification of radiographs.

NASOPHARYNGEAL DISORDERS OF FOALS

Choanal Atresia

Early in embryonic development, a membrane separates the oral and the nasal cavities.[73] Choanal atresia, a rare upper respiratory malformation in horses, is a congenital abnormality associated with failure to resorb this bucconasal membrane during embryonic development.[74-76] Because horses are obligate nasal breathers, bilateral choanal atresia usually results in the foal's death unless an emergency tracheostomy is performed at birth.[74] When the atresia occurs unilaterally, foals exhibit loud respiratory noise and exercise intolerance, and the nasopharynx can be observed through one nostril, whereas a membrane obstructs the caudal nasal passage of the other nostril. These horses are usually asymptomatic at rest; however, asymmetry of airflow from the nostrils can be detected. The diagnosis is made by endoscopic examination, skull radiography, contrast radiography, and computed tomography.[73-77]

Although my experience is very limited, foals with unilateral choanal atresia should be allowed to grow untreated until 1 year of age, for the following reasons. The larger nasal cavity facilitates surgical exposure with less likelihood of postoperative fibrosis, reduced diameter of the choana, or, even worse, complete closure. In addition, the need for a postoperative nasal stent is avoided. Finally, the larger size of the airway allows the procedure to be performed less invasively under endoscopic control with the animal standing.

Three treatment approaches have been used. In the first, the yearling is sedated, usually with a combination of detomidine and butorphanol, and topical anesthesia with phenylephrine (2% lidocaine or mepivacaine hydrochloride and 10 mL of 0.15% phenylephrine) is applied to the membrane and nasal cavity. The endoscope is placed in the affected nostril and the outline of the choanal membrane is identified using equine laryngeal forceps. Using a 600-µm laser fiber placed in the biopsy channel of the videoendoscope and a diode laser at 15 watts (pulse duration, 3 seconds), selected vessels are cauterized by placing the laser tip adjacent to but not on the vessel itself. Then the membrane is incised, creating two lines of incision perpendicular to each other that cross at the center of the choanal membrane. This cross incision is made because the membrane (even though it appears thin) is vascular, and local pressure to stop the bleeding may be needed at times. When excessive bleeding occurs, a nasotracheal tube is passed through the membrane into the nasopharynx, where the cuff is inflated to apply pressure that promotes hemostasis at the surgical site. After a few minutes, the tube is removed and each flap is resected; the aforementioned forceps may be used to apply tension on the flaps as needed. Postoperatively, the animal receives a 5-day course of phenylbutazone (1 mg/lb PO twice a day) or a similar NSAID.

The second treatment approach is used when the choanal membrane is osseous or the condition is bilateral, and a nasal flap giving access to the caudal aspect of the nasal passage is required. The choanal membrane (bone) is resected, as well as part of the nasal septum, using a nasal bone flap. The foal is anesthetized and placed in lateral recumbency, and an endotracheal tube is placed through a tracheostomy. A C-shaped incision (for unilateral lesions) or an S-shaped incision (for bilateral lesions) is made over the nasal bones starting at the level of the medial canthus of the eye and extending rostrad to approximately the middle of the nasal cavity, which is near the level of the infraorbital foramen. The periosteum is incised on the midline, extending laterally to an area near the nasomaxillary suture, taking care to stay at least 1 cm medial to the infraorbital canal. A nasal bone flap is created along the line of the periosteal incision by placing an osteotome at a 45-degree angle to create a ridge for the bone flap to rest on at the end of surgery. The nasal bone flap is made in the shape of the periosteal incision. The procedure must be performed bilaterally if the atresia is bilateral. The nasal mucosa is excised, and the choanal membrane is identified and resected, along with the caudal nasal septum if necessary. The area is packed with sterile rolled gauze, which exits the nostril and is sutured to the false nostril.

If it is necessary to operate on a young foal, the third approach can be used. The membrane is resected through a laryngotomy after placing the endoscope through the nares to illuminate the persistent membrane. My preference is to incise the membrane using endoscopic control and to place a stent in the nostril to prevent stricture.[75] In my limited experience, Nd:YAG laser dissection of the membrane has been unrewarding because profuse hemorrhage associated with recumbency and general anesthesia rapidly obscures the endoscopic view, preventing the application of local pressure in a small foal.[75] The membrane is incised best using laparoscopic scissors with unipolar or bipolar cautery under endoscopic control while the foal is under general anesthesia. A nasotracheal tube is placed through the surgically created fenestration and sutured to the false nostrils. The stents are removed 14 days postoperatively. Appropriate antibiotic therapy is used.

Because of the rarity of this condition, the prognosis is not well known, and persistence of airway obstruction is a possible complication. The heritability of this condition is also unknown.

Nasopharyngeal Dysfunction

Nasopharyngeal dysfunction, characterized by respiratory distress, respiratory stridor, and dysphasia, can occur in foals during the first month of life. Endoscopic examination may reveal severe nasopharyngeal edema, laryngeal edema, milk pooling in the nasopharynx, and persistent DDSP.

The etiology of this obstructive syndrome is unknown. However, on the basis of information obtained from neonatal laboratory animals and human infants, nasopharyngeal dysfunction in neonatal foals may be related to immaturity of the neuromuscular reflexes and defense mechanisms that support the nasopharynx and larynx.[78] Specifically, newborns have a smaller number of pressure and flow receptors in the upper airways than adults, and these receptors discharge at a slower rate.[78] Some of the sensory and motor nerves that supply the larynx and nasopharynx are demyelinated at birth, and they change and mature morphologically until 12 months of age.[78] Perhaps this immature neural circuit causes muscular dysfunction and incoordination in the nasopharynx that results in dysphagia and nasopharyngeal collapse, although this is only speculation.

A thorough physical examination, an endoscopic examination, and radiography of the thorax and pharyngeal region should be performed when evaluating a foal with nasopharyngeal dysfunction. While examining the foal, precautions should be taken to provide it with a patent airway if the examination induces respiratory distress. Appropriate blood work, including serum chemistry, complete blood count, serum immunoglobulin G (IgG) levels, and an arterial blood gas analysis, should be performed.

Treatment may begin by securing a patent airway for the foal. Respiratory distress may be sufficiently severe that an emergency tracheotomy is performed immediately. If the foal has persistent DDSP, a laryngotomy followed by placement of a tracheostomy tube usually corrects this problem. Rarely, a staphylectomy may be needed to correct this problem.[65] However, surgical intervention is not necessary in many foals. It is best to support the foal medically for 1 month to see if the problem resolves. Clearly, surveillance and prophylaxis for possible aspiration pneumonia should be strongly considered. White muscle disease should be identified and treated, because this may also cause nasopharyngeal dysfunction. If edema is present, anti-inflammatory medication may be useful in improving function. If the foal is dysphagic, enteral feeding can be performed through a nasogastric feeding tube, or parenteral feeding can be initiated.

Most often, these foals recover from nasopharyngeal dysfunction in 10 to 30 days. The nasopharyngeal edema resolves and dysphasia dissipates. Long-term survival rates and effects of this episode on future athletic performance are unknown.

Cleft Palate

Cleft palate (palatoschisis) is a rare congenital defect in foals.[79] The cleft is caused by an interruption in embryologic closure that occurs along the midline in a rostrad-to-caudad direction of the palatal folds and may, therefore, involve portions of the soft palate or of the hard and soft palate.[79] Defects in the lips and maxilla that frequently occur with cleft palate in people have never been reported in horses. The exact etiology of this defect is unknown, but genetics, nutrition, teratogens, and traumatic mechanical factors that could affect palatal fold closure during the 47th day of gestation are possible causes.[79]

Clinical signs of a cleft palate include milk draining from the foal's nose after nursing, coughing, and signs of aspiration.[79-82] The cleft may be observed during an oral examination or diagnosed by digital palpation, or both. Endoscopic examination, using a pediatric endoscope, is helpful in making the definitive diagnosis and assessing the extent of the defect. A careful endoscopic examination is necessary because the field of view with a pediatric endoscope is small. A small but clinically significant cleft may be obscured by epiglottic cartilage or saliva. It is helpful to make the foal swallow a few times to ensure that the palate is intact. The margins of the soft palate will be visible, and the epiglottis will be positioned in the oropharynx. Once a cleft palate has been diagnosed, the foal should be examined for other congenital defects.

If the cleft is small (about one third of the soft palate) and tracheal aspiration is minimal, it may be better to delay surgery while closely monitoring the foal so that aspiration pneumonia does not go undetected. Weaning the foal early seems to decrease tracheal aspiration, as feed reaches the trachea less readily than milk. The advantage of delaying surgery is that a larger oropharynx facilitates surgical manipulation and more precise surgical repair. If the cleft is large or tracheal aspiration of milk is significant, treatment options should be restricted to either surgical repair of the cleft palate or euthanasia. Allowing foals to continue with the cleft palate is a poor choice, because of their resultant poor quality of life and morbidity from chronic aspiration pneumonia. Surgical repair of a cleft palate is considered a salvage procedure, and it is fraught with complications.[80-84] Palatoplasty usually results in some degree of failure because of complete or partial dehiscence of the repair.[83] Reoperation is frequently needed and is often unsuccessful.[80] Pneumonia can be a serious, even life-ending, complication despite attempts to repair the cleft, because of the initial aspiration of milk or feed material.

The prognosis is affected by the size of the defect, the length of the soft palate involved, involvement of the hard palate, and pneumonia.[80,82] Repair of asymmetric defects should not be attempted, because there will not be sufficient tissue available for reconstruction.[83] Some authors recommend that if more than 20% of the soft palate tissue is missing, repair should not be attempted.[83] Cleft palate defects that occur on the midline with minimal tissue missing have a successful prognosis of 50%.[83] If the hard palate is involved, the chance of success, defined as an animal that can eat and grow normally, drops significantly. Little information is available on whether the respiratory function of the soft palate during strenuous exercise can be restored (i.e., there are no reports about the prognosis for athletic endeavors).

Surgical Approaches

Preoperative care includes broad-spectrum antibiotic coverage and plasma transfusion if the IgG level is inadequate. If the foal already has pneumonia and is aspirating feed material, the surgery should be postponed, systemic antibiotics continued, and the foal fed enterally through a nasogastric feeding tube or parenterally.

Surgical approaches to the palate include a transoral approach, laryngotomy, pharyngotomy, and mandibular symphysiotomy.[81,83-86] Pharyngotomy with splitting of the basihyoid bone is used to repair a cleft involving the caudal third of the soft palate (see previous description of pharyngotomy under "Surgical Approaches to the Nasopharynx or Oropharynx" and Figures 44-9 and 44-10).[81] Mandibular symphysiotomy provides the best exposure to the hard and soft palate (Figure 44-30).[86] To perform a palatoplasty using the mandibular symphysiotomy approach, the foal is placed under anesthesia and positioned in dorsal recumbency. A tracheotomy is performed, and the endotracheal tube is placed in the trachea (see Figure 44-30, *A*). Hair is clipped from the lower lip to the proximal trachea, and the skin is prepared for aseptic surgery. A skin incision is made from the hyoid to the lower lip (see Figure 44-30, *B*). The skin and gingiva over the mandibular symphysis are completely incised, and the mandibular symphysis is severed using a scalpel blade (in a neonate), an osteotome, or an oscillating bone saw. It is preferable to avoid incising the lip to minimize postoperative dehiscence and discomfort.[80] This is achieved by making a transverse incision at the base of the lip so the lip can first be moved caudad to give access to the symphysis. After splitting the symphysis, the lip is moved orally. The mandible spread achieved is slightly less after splitting the mandibular symphysis compared with splitting the lower lip, but it reduces morbidity. Surgical dissection subsequently proceeds along the right ramus of the mandible, and the attachments of the mylohyoid, geniohyoid, and genioglossus muscles are transected, exposing the buccal mucosa. The buccal mucosa is sharply incised, allowing the rami of the mandibles to be retracted (see Figure 44-30, *C* and *D*). A thin malleable retractor may be inserted through the laryngotomy over the base of the tongue to push the tongue out of the surgical field. Moist towels should be used to retract the mandibles.

After the cleft is repaired, the mandibles are apposed and stabilized with a single cancellous screw or a Steinmann pin supplemented by a hemicerclage, and the central incisors are wired together. The buccal mucosa is closed, and the geniohyoid and mylohyoid muscle layers are apposed and reattached. The lip is reconstructed by closing the gingiva, muscle layer, and skin (see Figure 44-30, *I*). The laryngotomy is left open, and the skin incision from the mandibular symphysis to the laryngotomy is closed with sutures interrupted by 2-cm gaps for drainage.[86]

A successful two-step repair of a cleft palate through a purely transoral approach was reported in a 4-week-old colt.[87] Because the instruments were too short, the procedure had to be stopped 4 cm short of the caudal rim, and it was completed 4 weeks later, after the initial repair had healed. In this procedure, a special mouth speculum with a long, narrow extension into the mouth was used to elevate the tongue out of the surgical field.

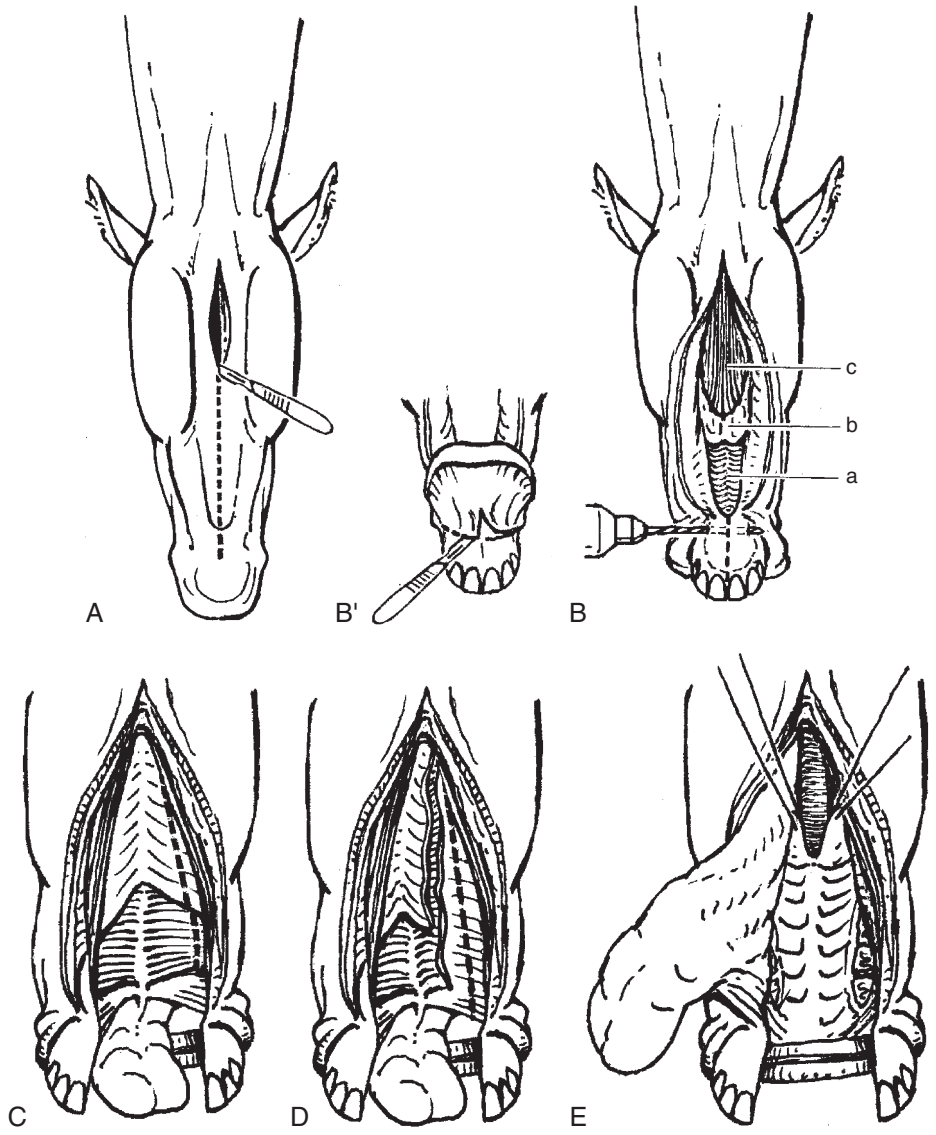

Figure 44-30. A and **B,** A ventral midline incision is made from the angle of the mandible to the lip. Before splitting the symphysis, a hole is prepared to facilitate realignment of the bones during closure. The lip has been moved orally to avoid morbidity associated with incising it. *a,* Mylohyoideus; *b,* mandibular lymph nodes; *c,* sternohyoideus and omohyoideus muscles. **C,** The mandibles are spread, and the rostral oral mucosa is incised. The mylohyoid and geniohyoideus muscles are incised near their tendinous origin. They should be cut with enough tissue left to suture them together for closure. **D,** The exposed ventral surface of the oral mucosa is tensed by further spreading of the mandibles. It is incised as far caudad as possible. Care is taken to avoid the sublingual salivary gland near the mandible and the lingual nerve near the tongue. **E,** The edges of the incision are covered with moistened sponges or towels and spread as far as possible. The nasotracheal tube is visible through the cleft. Two stay sutures are placed at the caudal corners of the cleft.

Illumination of the surgical field was provided by a pediatric endoscope, which was introduced through one of the nasal passages.[87]

Soft Palate Repair

Graffe was the first surgeon to attempt surgical closure of cleft palate in humans in 1816, but he reported poor results.[88] Little progress was made until Langenbeck (1961) described a new technique to close a palatal cleft using two bridge flaps. The technique was further modified in 1967, when Kriens suggested that the main surgical problem with palate repair was caused by the misoriented muscles in the soft palate, which, instead of building a velopharyngeal sphincter mechanism, have an insertion in the hard palate.[89] To solve this problem, the faulty origin of the palatopharyngeus and levator veli palatini muscles was resected from the hard palate and subsequently included in the cleft repair, which resulted in a more anatomically correct reconstruction of the soft palate. The interposition of the palatal muscles between the oral and nasal mucosa led to good functional results in humans.[89]

The soft palate is identified, and the cleft is evaluated. A surgical light is directed into the incision. If a pharyngotomy was used for access, the videoendoscope is placed orally. If a mandibular symphysiotomy was chosen, the endoscope can be positioned through a laryngotomy to improve visibility at the caudal

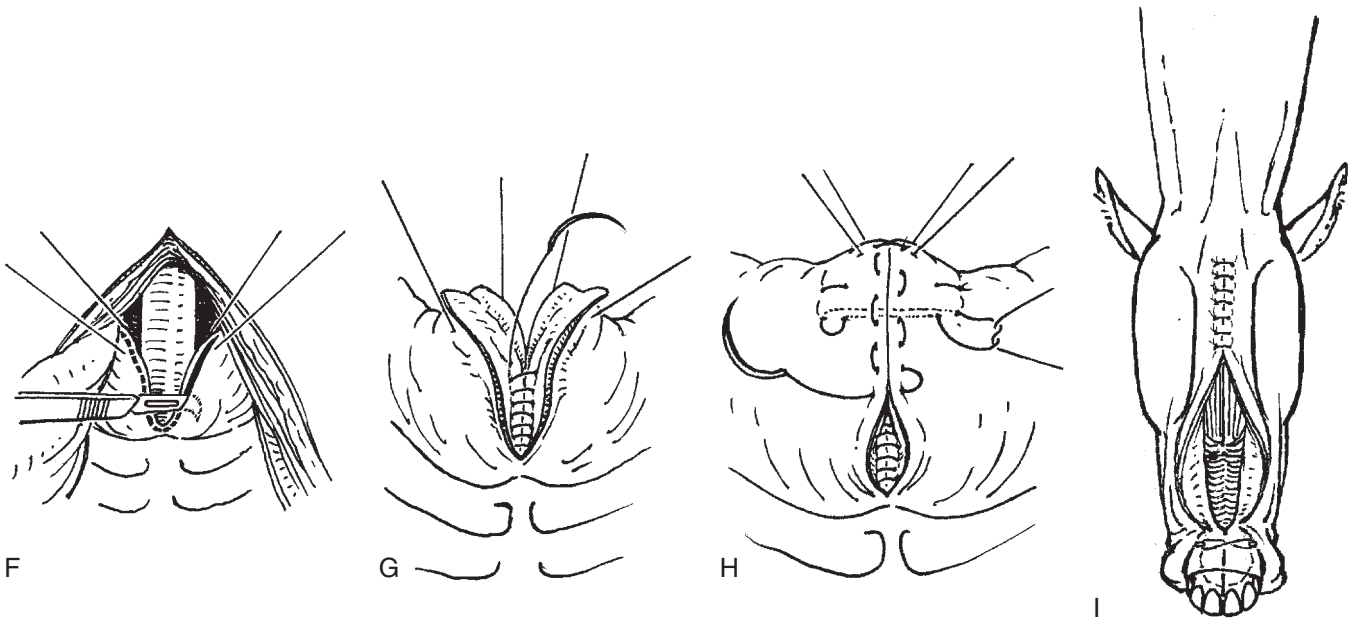

Figure 44-30, cont'd **F,** Tensing the edges of the cleft with the stay suture, a No.12 Bard-Parker blade is used to split the thickness of the tissue. The stay sutures can be held by an instrument inserted through a laryngotomy. **G,** A simple-continuous (or a Lembert) pattern is placed in the nasal side of the split edge in a rostral-to-caudal direction. **H,** A continuous horizontal mattress pattern is placed in the oral side of the split edge. Two to four widely spaced interrupted vertical mattress sutures are placed to reduce tension on the primary suture line. **I,** Soft tissues are apposed and a lag screw or pin with figure-of-eight wire is placed using the previously drilled hole. A cerclage wire around the rostral mandible just caudal to the erupted incisors augments stability.

margin of the soft palate. A stay suture is passed through the soft palate at the caudal extent of the cleft, and these stay sutures are secured at the laryngotomy site to tense the edges of the defect (see Figure 44-30, *E*). A thin strip of mucosa is excised from each edge of the cleft, and a narrow incision is extended into the palate using a No. 12 hooked blade (see Figure 44-30, *F*). The nasal mucosa is closed first, beginning at the caudal free margin of the soft palate, using 2-0 absorbable suture material in a simple-continuous pattern (see Figure 44-30, *G*). The oral mucosa and muscular layers are closed together using horizontal mattress sutures of absorbable material or silk (see Figure 44-30, *H*). Silk has excellent handling characteristics and good knot security.[79] The oral mucosa is closed with absorbable material in a simple-continuous pattern.

Tension on the repair can lead to dehiscence; therefore, tension-relieving incisions are made parallel to the closure plane in the lateral mucosa axial to the molar teeth. Alternatively, widely spaced interrupted vertical mattress sutures can be placed in the palate to relieve the tension. The tendon of the tensor veli palatini muscle can be transected bilaterally or osteotomy of the hamulus of the pterygoid can be performed to decrease tension applied to the caudal half of the soft palate by the tensor veli palatini muscle. These latter procedures should be used with caution, as they can lead to instability of the rostral aspect of the soft palate[10] and may interfere with respiratory soundness at exercise.

Because it is difficult to repair the caudal end of the soft palate, a combined transoral and laryngotomy approach is an alternative to provide good apposition and reconstruction of the cleft palate. To provide better access to the caudal aspect of the soft palate, the thyroid cartilage may be split sagittally (Figure 44-31). The surgical repair of the soft palate is identical to the one previously described. At the end of the procedure, the thyroid cartilage is sutured with three single interrupted sutures of No. 2 monofilament nylon.[88] The incision of the cricothyroid membrane is closed in a simple-continuous pattern with 2-0 polyglactin 910 suture material. The subcutaneous tissues and skin are left to heal by second intention.

Figure 44-31. Laryngotomy approach for repair of the caudal aspect of a cleft palate. The two parts of the cleft palate are retracted with Allis forceps. Good visibility of the cleft palate was achieved by splitting the thyroid cartilage longitudinally. (Courtesy J. Auer, University of Zurich, Switzerland.)

Hard Palate Repair

Repair of hard palate defects is performed using mucoperiosteal flaps.[90] Hemorrhage can be controlled by injecting small volumes of 2% lidocaine with epinephrine along the incision lines in the hard palate. A mucoperiosteal incision is made parallel to the maxilla from the junction of the hard and soft palate to a few centimeters beyond the defect. The entire width of the mucoperiosteum is elevated to the edge of the defect using a periosteal elevator. Care is taken to preserve the palatine artery, which can be seen as it emerges from the palatine foramina at the caudolateral aspect of the hard palate at the level of the second molar tooth. As the flap is elevated, the palatine artery stretches, and the entire flap is moved axially. The rostral (rostral hard palate) and caudal (junction of the hard and soft palates) attachments of the mucoperiosteal flap are preserved. The nasal mucosa of the flap is closed in a simple-continuous pattern using absorbable suture material, and the mucoperiosteal flaps are sutured together with horizontal mattress sutures. The defects along the lateral aspect of each mucoperiosteal flap heal by second intention.

Postoperative Care

If only the soft palate was repaired, the foal is allowed to nurse. If the soft palate defect was extensive or the hard palate was repaired, or both, the foal should be fed enterally through a nasogastric feeding tube or parenterally for 7 to 10 days. Broad-spectrum antibiotics should be continued for 5 days unless the foal has pneumonia, in which case antibiotics are continued until the pneumonia resolves. The repair may be assessed by careful oral examination (by looking in the mouth, not by digital palpation), but endoscopic examination is not recommended during the early postoperative period.

Dehiscence is the most likely and most severe complication. Generally, dehiscence is apparent within the first 7 to 14 days and is evidenced by nasal reflux of food and coughing. Other complications include pneumonia, osteomyelitis of the mandible, salivary fistulas, and incisional infections.[82]

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CHAPTER
45 Larynx

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ANATOMY

The larynx is a conduit between the pharynx and trachea that contributes to the functions of breathing, vocalization, and deglutition. The architecture of the larynx comprises several individual cartilages and muscles with the lumen being lined by a mucous membrane composed of stratified squamous and pseudostratified columnar ciliated epithelium.[1]

The cricoid, thyroid, and epiglottic cartilages are unpaired, whereas the arytenoid cartilages and attached corniculate processes are paired. The signet ring–shaped cricoid cartilage is positioned rostral to the first tracheal ring and connected to the trachea by the cricotracheal membrane.[1] The thyroid cartilage is the largest of the laryngeal cartilages and is situated rostral to the cricoid cartilage. The arytenoid cartilages are positioned on either side of the cricoid cartilage, being conjoined at the cricoarytenoid articulations. These articulations are diarthrodial joints that allow the arytenoid cartilages to rotate dorsolaterally during abduction, and axially during adduction.[1] Each arytenoid cartilage has a corniculate process forming part of the dorsolateral border of the rima glottidis, a cuneate process and a muscular process that serves as the insertion for the cricoarytenoideus dorsalis muscle. The arytenoid cartilages are the most dynamic of the laryngeal cartilages. The articulations with the cricoid cartilage allow complete closure of the glottis during swallowing (adduction) and maximal opening (abduction) during exercise (Figure 45-1). Any pathologic process that changes the normal function of one or both of the arytenoid cartilages can have an impact on the athletic ability of a horse. Treatment of arytenoid cartilage disease therefore becomes essential for horses undertaking high-intensity exercise. The body or laminar portion of the arytenoids consists of hyaline cartilage, which is subject to ossification with age or trauma. However, the apex of each arytenoid (the corniculate process, which curves upward and backward, forming a pitcher-shaped lip from which the cartilages derive their name) is made of elastic cartilage.

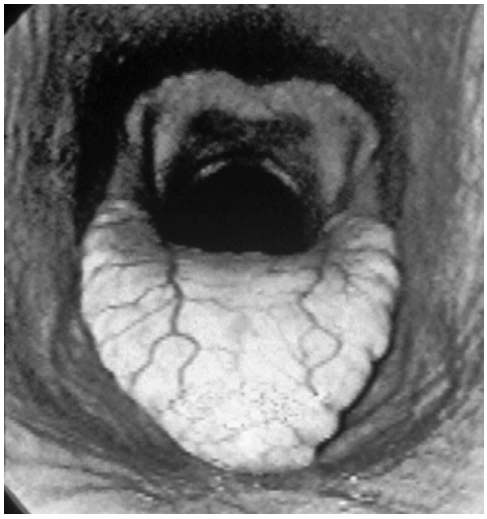


Figure 45-1. Endoscopic image of the normal equine larynx during inhalation, showing maximal abduction of the arytenoid cartilages and a normal epiglottis.

The epiglottis rests on the dorsal surface of the body of the thyroid cartilage and is held there by the thyroepiglottic ligaments. Unlike the cricoid and thyroid cartilages, which consist of hyaline cartilage, the epiglottis is made up of elastic cartilage and has the shape of an oblanceolate leaf.[1]

Movement of the laryngeal cartilages in relation to each other is achieved by intrinsic laryngeal muscles, whereas movement or stabilisation of the larynx as a whole results from extrinsic laryngeal muscles. Contraction of the intrinsic laryngeal muscles produces changes in the caliber of the rima glottidis first by abducting or adducting the corniculate processes of the arytenoid cartilages and second by increasing or decreasing tension of the vocal folds, all combining to alter airway resistance during respiration. The intrinsic muscles include the paired cricoarytenoideus dorsalis (CAD), thyroarytenoideus (ventricularis and vocalis), cricothyroideus and cricoarytenoideus lateralis, plus the unpaired transverse arytenoideus muscle.[2] The CAD muscle is the major abductor muscle that widens the laryngeal aperture by abducting the corniculate process of the arytenoid cartilage and mechanically tensing the vocal folds.[2,3] Contraction of the arytenoid transversus muscle produces arytenoid cartilage adduction by drawing the dorsoaxial margins of the arytenoid cartilages together.[1] The thyroarytenoideus, arytenoideus transversus, and cricoarytenoideus lateralis muscles adduct the corniculate processes of the arytenoid cartilages, narrowing the rima glottidis and protecting the lower airway during swallowing.[1,3] The cricothyroideus muscle tenses the vocal folds during vocalization but receives efferent motor innervation from the external branch of the cranial laryngeal nerve, a branch of the vagus nerve, whereas all other intrinsic laryngeal muscles receive motor innervation from the recurrent laryngeal branch of the vagus nerve.[1,2] The extrinsic laryngeal muscles of the larynx include the thyrohyoideus, hyoepiglotticus, cricopharyngeus, thyropharyngeus and sternothyroideus muscles and are involved in stabilizing the larynx and pharynx during exercise.[1]

The mucosa of the larynx is closely adhered to the cartilages and contains many different types of afferent receptors. The mucous membrane covering the epiglottic cartilage reflects off the lateral border of the epiglottis and blends with the mucous membrane covering the corniculate processes of the arytenoid cartilages, forming the aryepiglottic folds.[1] The mucous membrane lines the vocal ligaments and lateral ventricles and forms the vocal folds and the laryngeal saccules. These saccules are about 2.5 cm (1 inch) deep with a capacity of 5 to 6 mL.[1] They extend between the medial surface of the thyroid cartilage and the ventricularis and vocalis muscles. The laryngeal mucosa contains sensory mechanoreceptors for the detection of different stimuli, including transmural pressure changes, airflow, temperature, and laryngeal motion.[4] These mechanoreceptors

receive afferent neural supply from the internal branch of the cranial laryngeal nerve, a branch of the vagus nerve.[4] This rich sensory nerve supply is the source of many respiratory reflexes that influence upper airway patency and breathing patterns.[4,5]

The larynx receives arterial blood supply from the caudal laryngeal artery and branches of the ascending pharyngeal arteries. Venous drainage is provided by the caudal laryngeal and ascending pharyngeal veins, which flow to the external jugular vein via the thyroid vein. The lymphatic chains that serve the laryngeal area include retropharyngeal, cranial, and deep cervical lymph centers.[1]

RECURRENT LARYNGEAL NEUROPATHY (LARYNGEAL HEMIPLEGIA)

Etiology and Incidence

Although horses can develop unilateral or bilateral paralysis of the CAD muscle leading to dysfunction of the associated arytenoid cartilage,[6-9] unilateral left-sided laryngeal paralysis is most commonly encountered (Figure 45-2). Because the muscle paralysis results from progressive loss of large myelinated axons in the left recurrent laryngeal nerve,[6] recurrent laryngeal neuropathy (RLN) is now considered the most appropriate descriptive term for this condition. Although a genetic basis to the condition is most likely,[9-11] in the majority of affected horses, a precise etiology is rarely evident, and the term *idiopathic laryngeal hemiplegia* has commonly been applied as a synonym. Neurogenic atrophy of the intrinsic laryngeal musculature results in progressive loss of both abductor and adductor arytenoid function.[6] Because of this progressive rather than immediate loss of muscle function, varying degrees of abnormal movements of the arytenoid cartilage(s), depending on presence of CAD muscle paresis or paralysis, is frequently observed endoscopically, which strengthens the use of RLN as the most appropriate diagnostic term. RLN occurs in horses from a few months to 10 years of age and older, with large-breed horses (such as Thoroughbreds and draught breeds) more commonly affected than small-breed horses or ponies.[12,13] The incidence is highest in young horses, often diagnosed before they have started any type of training, or in 2- and 3-year-olds that are racing or are in race training.[13-15] The prevalence of RLN varies between breeds, with the largest population studied being the Thoroughbred, where between 2.6%[15] and 8%[14] of horses are reported to be affected. However, in the heavy draught breeds, an incidence of up to 35% has been reported.[16] Following the introduction of pre- and post-sale endoscopy at Thoroughbred yearling sales, the incidence of RLN has been widely documented.[13-15,17] Although the incidence of complete laryngeal hemiplegia was 2.75% in a study of yearling Thoroughbreds, the overall incidence in the entire yearling Thoroughbred population is thought to be higher.[15,18] In a study of 427 Thoroughbred sale yearlings, 64% had asynchronous arytenoid cartilage movements and 25% had laryngeal asymmetry that was significantly associated with decreased racing performance.[17]

In normal horses during sustained high-intensity exercise, the rima glottidis of the larynx dilates fully to maximize airflow. Full arytenoid cartilage abduction is sustained, despite increasing negative inspiratory pressure that occurs as exercise intensity increases. Horses with RLN cannot achieve maximal abduction of the affected arytenoid cartilage, and as negative inspiratory pressure increases, the rima glottidis progressively reduces in size (Figure 45-3).[19] Subsequently, hypoxemia, hypercarbia, and metabolic acidosis develop more rapidly than in normal horses with the same workload, causing early musculoskeletal fatigue and poor performance.[20,21]

A specific etiology can be identified in some horses with acquired unilateral or bilateral laryngeal paralysis. The recurrent laryngeal nerve can be damaged as a result of perivascular jugular vein injection, guttural pouch mycosis, trauma from injuries or surgical procedures of the neck, strangles abscessation of the head and neck, and impingement by neoplasms of the neck or chest.[9] Organophosphate toxicity, plant poisoning, hepatic encephalopathy, lead toxicity, and central nervous system diseases have also been shown to cause laryngeal paralysis.[22,23] A study of 375 cases of laryngeal hemiplegia in a mixed-breed population of horses indicated 94% (351) were idiopathic in origin.[24] However, in the 6% of nonidiopathic cases of recurrent laryngeal neuropathy, over half were bilaterally affected.

Diagnosis

A diagnosis of RLN can be suspected on the basis of history and a physical examination. Horses with RLN commonly demonstrate exercise intolerance with resultant poor performance during high-speed exercise and produce an abnormal

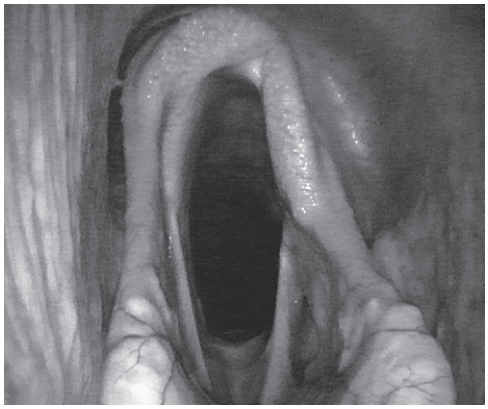


Figure 45-2. Endoscopic image of the larynx of a horse at rest with grade 4 recurrent laryngeal neuropathy. (Courtesy M. Weishaupt, Zurich.)

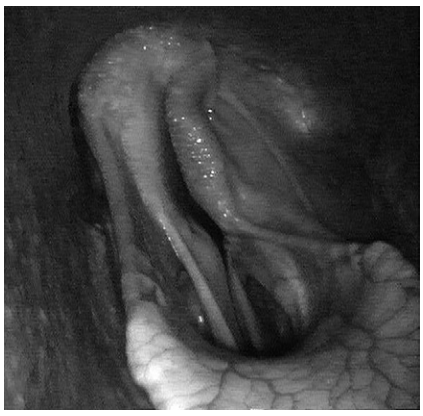


Figure 45-3. Endoscopic image of the larynx of a horse during high-speed exercise with grade 4 recurrent laryngeal neuropathy, showing complete collapse of the left arytenoid cartilage and vocal fold on inspiration. (Courtesy M. Weishaupt, Zurich.)

inspiratory respiratory noise during exercise described as a nonvibratory, single-tone whistle that has resulted in the colloquial term *roaring*. This noise is the result of turbulence created by a narrowed rima glottidis as air passes over the affected vocal cord and ventricle, which acts as a resonator.[23] Physical examination should include palpation of the neck and larynx because horses with significant RLN have palpable atrophy of the left CAD muscle, which manifestes as a percutaneous prominence of the muscular process of the arytenoid cartilage.[18] The larynx should also be palpated for congenital malformation and evidence of arytenoid chondritis.[22,25] If there is suspicion of previous laryngeal surgery, clipping of the hair is advised to allow visual confirmation of palpable laryngotomy or laryngoplasty scars. Ultrasound examination can identify arytenoid cartilage movements but patient cooperation is essential for successful interpretation of this diagnostic mode.[26] (Additional information can be found in Chapter 42.)

A definitive diagnosis of RLN is made on endoscopic examination when there is partial or complete loss of abductor function on the affected side of the larynx (see Figure 45-2). Because the various descriptions of the loss of abductor function (hemiplegia, paresis, complete or incomplete paralysis) can lead to confusion, a variety of grading systems have been developed over the past 30 years by several surgeons to describe arytenoid cartilage movements as assessed endoscopically in the resting horse.[27] Currently a seven-grade system (Grades 1 though 4 with subgrades) (Table 45-1) has been adopted by many surgeons to establish international consistency in reporting this condition.[28] This grading system should make possible a better description of the endoscopic apperance of laryngeal movements and more accurate reporting of treatment outcomes.

To aid assessment of arytenoid cartilage movements, both the swallow reflex and nasal occlusion can be helpful. In the normal horse a momentary full abduction of the arytenoid cartilages can be stimulated by inducing swallowing. Prolonging nasal occlusion for several attempted breaths can also induce maximal abduction of arytenoid cartilages in the normal horse.[29] Trackside endoscopy immediately after strenuous exercise has been used to help establish a diagnosis of recurrent laryngeal neuropathy, but it is not a reliable technique. If doubt exists about the diagnosis and surgical intervention is planned, treadmill videoendoscopy or "over-ground" videoendoscopy[30] are now the "gold standards" for accurate disagnosis (Chapter 42). A video image can be recorded and reviewed in slow motion to accurately observe dynamic collapse of the affected arytenoid cartilage and ipsilateral vocal cord.[31] When high-speed videoendoscopy is not avaibale, momentary full abduction of the arytenoid cartilages after swallowing is thought to be more reliable than nasal occlusion at predicting which horses would demonstrate arytenoid cartilage collapse during treadmill endoscopy at speed. In many horses that cannot achieve full abduction during nasal occlusion the arytenoids will collapse at speed, but not all will. However, horses that showed full symmetrical abduction after swallowing all maintained full

TABLE 45-1. Subjective Grading System of Laryngeal Function Assessed Endoscopically in the Standing Horse[28]

Grade	Description	Subgrade	Description
I	All arytenoid cartilage movements are synchronous and symmetrical, and full arytenoid cartilage abduction can be achieved and maintained.		
II	Arytenoid cartilage movements are asynchronous and/or larynx is asymmetric at times, but full arytenoid cartilage abduction can be achieved and maintained.	A	Transient asynchrony, flutter, or delayed movements are seen.
		B	There is asymmetry of the rima glottis much of he time owing to reduced mobility of the affected arytenoid cartilage and vocal fold, but there are occasions, typically after swallowing or nasal occlusion, when full symmetrical abduction is achieved and maintained.
III	Arytenoid cartilage movements are asynchronous and/or asymmetric. Full arytenoid cartilage abduction cannot be achieved and maintained.	A	There is asymmetry of the rima glottis much of the time owing to reduced mobility of the affected arytenoid cartilage and vocal fold, but there are occasions, typically after swallowing or nasal occlusion, when full symmetrical abduction is achieved *but not* maintained.
		B	There is obvious arytenoid abductor muscle deficit and arytenoid cartilage asymmetry. Full abduction is never achieved.
		C	There is marked but not total arytenoid abductor muscle deficit and arytenoid cartilage aymmetry with little arytenoid cartilage movement. Full abduction is never achieved.
IV	Complete immobility of the arytenoid cartialge and vocal fold.		

abduction at speed.[32] It is well established in horses standing at rest, without clinical evidence of upper respiratory obstruction, that the arytenoid cartilages can be positionned asymmetrically and/or have asynchronous movements. Therefore, it is important to acknowledge that this asymmetry and/or asynchrony may or may not predict failure of arytenoid function during athletic exercise.[17] Whereas the clinical significance of these variations in arytenoid cartilage movement has been controversial, it is generally accepted that as arytenoid cartilage abduction decreases, athletic perfromance reduces and therefore it is an undesirable finding during endoscopy of the equine athlete. Furthermore, it is recognized that horses with RLN, even after treatment with prosthetic laryngoplasty or a nerve muscle pedicle graft, often do not achieve full restoration of athletic performance.[33,34]

Therefore, interpretation and significance of altered laryngeal cartilage movements identified at endoscopy falls to the veterinarian, who must make an educated prediction of how these altered movements will affect the horse's performance. Hence the veterinarian has to determine how severely affected is the larynx and answer the question: "Will these altered laryngeal movements progress to complete laryngeal paralysis?" In 1982, a study of 168 horses over several years demonstrated there was no apparent progression from asynchronous arytenoid cartilage movements to complete paralysis.[35] This led the investigators to conclude that such movements were normal. Subsequently, a four-point grading system describing arytenoid cartilage movements was developed to predict present and future arytenoid cartilage dysfunction based on resting endoscopic evaluation. Although clinical and experimental studies suggested RLN may have been progressive, no factual evidence was available in the literature. In 1997, when 109 young Thoroughbred and Standardbred horses were examined 16 months apart, 12% of horses progressed from normal laryngeal function to that indicative of RLN.[36] In 2002, another study supported the progressive nature of RLN.[37] The long-term histories and clinical findings of 351 horses suffering from RLN were examined for evidence of progression of laryngeal asymmetry. Fifty-two out of 351 cases (15%) had evidence of progressive laryngeal dysfunction over a median period of 12 months. A more recent study has also highlighted the progressive nature of the disease with successive treadmill examinations over time.[38] The fact that 67% of foals with marked arytenoid dysfunction can show improved laryngeal function 12 months later[39] supports the statement that endoscopic findings on a given day may not apply to the future, and periodic re-examination is justified. As well as RLN being progressive, many adult horses have an acute onset of exercise intolerance and loud inspiratory noise.[39] The progression of RLN is assumed to be slow and insidious; however, clinical signs may only become apparent when sufficient muscle atrophy of the cricoarytenoideus dorsalis muscle occurs, permitting complete arytenoid collapse during exercise. This manifestation could result in a history of acute onset. However, rapid progression in as little as 6 weeks has been reported.[37]

Variation of arytenoid cartilage movements in an individual horse has been reported to be common when the horse has multiple endoscopic examinations.[17] This variation, whether examinations are days or even hours apart, is thought to be caused by intra-assessor variation or laryngeal muscular fatigue from multiple reexaminations. Using the seven-grade system, a high degree of reliability in assessing laryngeal movements has been shown, but variation can be seen in the same horse evaluated endoscopically on different days.[40] Differences in clincial and endoscopic findings between veterinarians who perform prepurchase and postpurchase examinations, weeks rather than hours apart, may in some cases simply reflect clinical progression of recurrent laryngeal neuropathy rather than intra-assessor variation.

Treatment

General Considerations

Surgical treatments available for RLN include prosthetic laryngoplasty, ventriculectomy (sacculectomy), ventriculocordectomy, reinnervation of the CAD muscle, and occasionally arytenoidectomy.[31] The choice of surgery is usually based on the presenting complaint, the age and use of the horse and the grade of arytenoid cartilage movement present. When considering this decision, the surgeon should recognize that some horses with RLN can tolerate and work to capacity despite partial upper airway obstruction, especially when high-speed exercise is not expected of the horse. The decision to operate on the upper airway should be based on clear evidence that it is essential to allow the horse to perform to the owner's requirements because complications can be frequent and sometimes devastating. Therefore, treatment of RLN should be reserved for horses in which upper respiratory tract obstruction is reducing performance.

Horses that work and race at speed for farther than 800 meters ($\frac{1}{2}$ mile) are significantly affected by obstructions of the upper airway.[33] At these speeds and distances, reduction in the laryngeal aperture by 50% increases the work of breathing 16-fold. Therefore, some form of surgical intervention to eliminate this obstruction is warranted.[35] The most common technique is the prosthetic laryngoplasty. Frequently, ventriculectomy or ventriculocordectomy is performed with laryngoplasty, but neither prolongs the efficacy of the surgery or improves upper airway flow mechanics during high-speed exercise.[41,42] However, these procedures are often used along with laryngoplasty and laryngeal reinnervation because they reduce noise, and the scar can assist with airflow mechanics. In show and non–racehorses, the respiratory noise caused by RLN is an important clinical problem because being "unsound of wind" may result in judging penalties. Therefore, the primary goal of surgery in these horses is often to eliminate the respiratory noise. A study in 2003 demonstrated that bilateral ventriculocordectomy in grade 4 hemiplegic horses effectively reduced inspiratory noise by 90 days after surgery.[43] Conversely, it was shown that a unnilateral laser vocal cordectomy did not effectively reduce the noise associated with grade 4 laryngeal hemiplegia by 120 days after surgery.[44] Both of these techniques can modestly improve airway flow mechanics and improvement in exercise performance and noise reduction has been identified with ventriculocordectomy.[45]

Clinical experience suggests that bilateral standing ventriculectomy has a very similar result in draft breeds, decreasing airway noise while improving air flow mechanics. Although laryngoplasty reduces respiratory noise in horses with laryngeal hemiplegia, ventriculocordectomy is more efficacious in reducing airway noise in the normal hearing range.[46] A further advantage of ventriculocordectomy over laryngoplasty is that postoperative complications after laryngoplasty are more prevalent and severe. These can include dysphagia, bilateral nasal discharge of feed, water, and saliva, aspiration pneumonia,

chronic coughing, wound infection, prosthesis failure and chondritis.[33] Because many horses still make varying levels of noise during exercise following laryngoplasty, sound production should not be used as an indicator of surgical success in horses where restoration of upper airway flow mechanics is the primary goal of surgery.[46] There is a poor correlation between the sound indices and degree of airway obstruction as measured by inspiratory pressure. A strong positive correlation exists in the degree of arytenoid abduction and sound indices. It would be intuitive that greater arytenoid abduction would be correlated with less respiratory noise, but in fact the opposite was true.[46]

Occasionally, partial arytenoidectomy (the muscular process is left *in situ*) is chosen to treat laryngeal hemiplegia when there is a congenital malformation of the cartilages or when the laryngoplasty technique has failed because of fractures in the cartilage. This technique has been shown to improve airway mechanics to a level similar to laryngoplasty,[47] and Thoroughbred horses have returned to successful careers.[48] The nerve muscle pedicle graft is suitable for horses of all breeds and ages but most commonly is used in young horses, where return to athletic function is not expected before 4 months after surgery. Although successful reinnervation is possible in grade 4 recurrent laryngeal neuropathy, horses with grade 3 arytenoid carilage movements are thought to be better candidates because less muscle atrophy is present in the latter.

In general, before laryngeal surgery, horses are fasted for 8 to 12 hours. Preoperative and perioperative nonsteroidal antiinflammatory medication (phenylbutazone) and broad-spectrum antibiotics are indicated for these surgeries. Disruption of the protective laryngeal mucosa via laryngotomy or cordectomy/ventriculocordectomy can justify prolonged perioperative antibiotics for up to 2 to 3 days because these major laryngeal surgeries induce dead space associated with the approach, increasing the risk of infection from bacteria originating in the lumen of the airway. The anesthetist should be aware that some horses with a longstanding unilateral arytenoid paralysis can be difficult to intubate with a standard-size tube, and a smaller-diameter tube should be available. The smaller tube also allows easier rotation and manipulation of the larynx intraoperatively. Prosthetic laryngoplasty is performed with the horse in lateral recumbency, affected side up. In unique situations, the horse may be first placed in dorsal recumbency to allow visual and digital inspection of the arytenoid cartilages through a laryngotomy. This approach might be taken when endoscopic observations suggest the presence of subtle arytenoid chondritis or when there has been previous laryngeal surgery. If the cartilage is diseased or has lost flexibility and cannot be pushed into an abducted position, the surgeon may elect to proceed directly to an arytenoidectomy rather than perform a laryngoplasty.

Prosthetic Laryngoplasty

Prosthetic laryngoplasty, commonly referred to as a *tie-back*, involves placing a prosthesis (usually a nonabsorbable suture) between the cricoid and arytenoid cartilage (Figure 45-4, *A* and *B*).[49,50] The goal of laryngoplasty is to achieve some degree of permanent abduction of the affected arytenoid cartilage (see Figure 45-4). The ideal result for the horse is an arytenoid cartilage abducted sufficiently to allow adequate airflow during exercise but not allow entry of saliva, food, or water into the laryngeal or tracheal lumen during swallowing. The art of performing this surgery is finding that balance.

Following induction of general anesthesia and placement of the horse in lateral recumbency, the head and neck are extended moderately for best surgical exposure. A videoendoscope can be secured transnasally at this time to allow intraoperative viewing of the larynx by the surgeon. The surgical site is routinely prepared for aseptic surgery and draped, and a 10- to 12-cm (4- to 5-inch) skin incision is made ventral and parallel to the linguofacial vein, extending caudally from a point 4 cm (1½ inch) cranial to the ramus of the mandible. Blunt and sharp dissection with Metzenbaum scissors separates the linguofacial vein from the lateral margin of the omohyoideus muscle along the length of the incision. Often it is necessary to ligate and divide a branch of the linguofacial vein that enters the omohyoideus muscle. Elevation of the linguofacial vein with an Allis tissue forceps attached to perivascular tissues at the middle of the incision allows a natural cleavage plane between the sternocephalicus and cricothyroideus muscles to become evident, which can be digitally opened and enlarged. This exposes the lateral and dorsal aspects of the larynx. A 6- to 8-cm wide malleable retractor placed under the linguofacial vein and sternocephalicus muscle is used to expose and maintain acess to the larynx. The muscular process of the arytenoid cartilage lies beneath the cranial portion of the cricopharyngeus muscle and can be exposed by sharply separating the cricopharyngeus and thyropharyngeus muscles along the junction of their aponeuroses. Alternatively, a plane of dissection can be created off the back edge of the cricopharyngeus muscle, under the vascular plexus that lies over the CAD muscle. Rostral retraction of the cricopharyngeus muscle often exposes almost the entire CAD muscle and muscular process without interference from the cricopharyngeus muscle. The esophagus, thyroid gland, laryngeal and thyroid vessels, and ventral branch of the first cervical and cranial laryngeal nerves should be avoided during site preparation and suture placement.[49]

The choice of prosthetic suture is often based on surgeon preference. Materials that have been used include braided polyester, with (No. 5 Ticron) or without silicone coating, 6-mm surgical stainless steel wire, braided lycra, and nylon. The prosthetic suture is first placed through the cricoid cartilage. The use of two sutures is common and has been shown *in vitro* to be mechanically superior to one[51] and also results in a larger cross-sectional area of the rima glottidis.[52] We prefer the large, swaged-on, reverse-cutting needle on the Ticron, but the suture can be threaded onto a No. 3 Martin uterine reverse-cutting needle and the ends tagged with a small hemostat. (This type of needle is less likely to break off in the laryngeal cartilages than a cutting needle, and it pierces cartilage more easily than a trochar-point needle.) Using the left index finger as a guide, the needle is "walked off" the caudal edge of the cricoid cartilage 2 to 3 mm lateral to the dorsal midline until the point slips under the cartilage. (There is a palpable notch in the cricoid cartilage at this site, but its location can be quite variable.) The needle is advanced in a cranial direction while avoiding penetration into the lumen of the larynx. Then the needle is rotated to penetrate the cricoid cartilage 2 to 3 cm cranial to its caudal border and 1 cm lateral to the dorsal ridge (Figure 45-5, *A*). Before drawing the needle through the cartilage and overlying CAD muscle, inspection of the laryngeal lumen via videoendoscope can ascertain if mucosal penetration has occurred. If the needle has penetrated the larynx, it should be

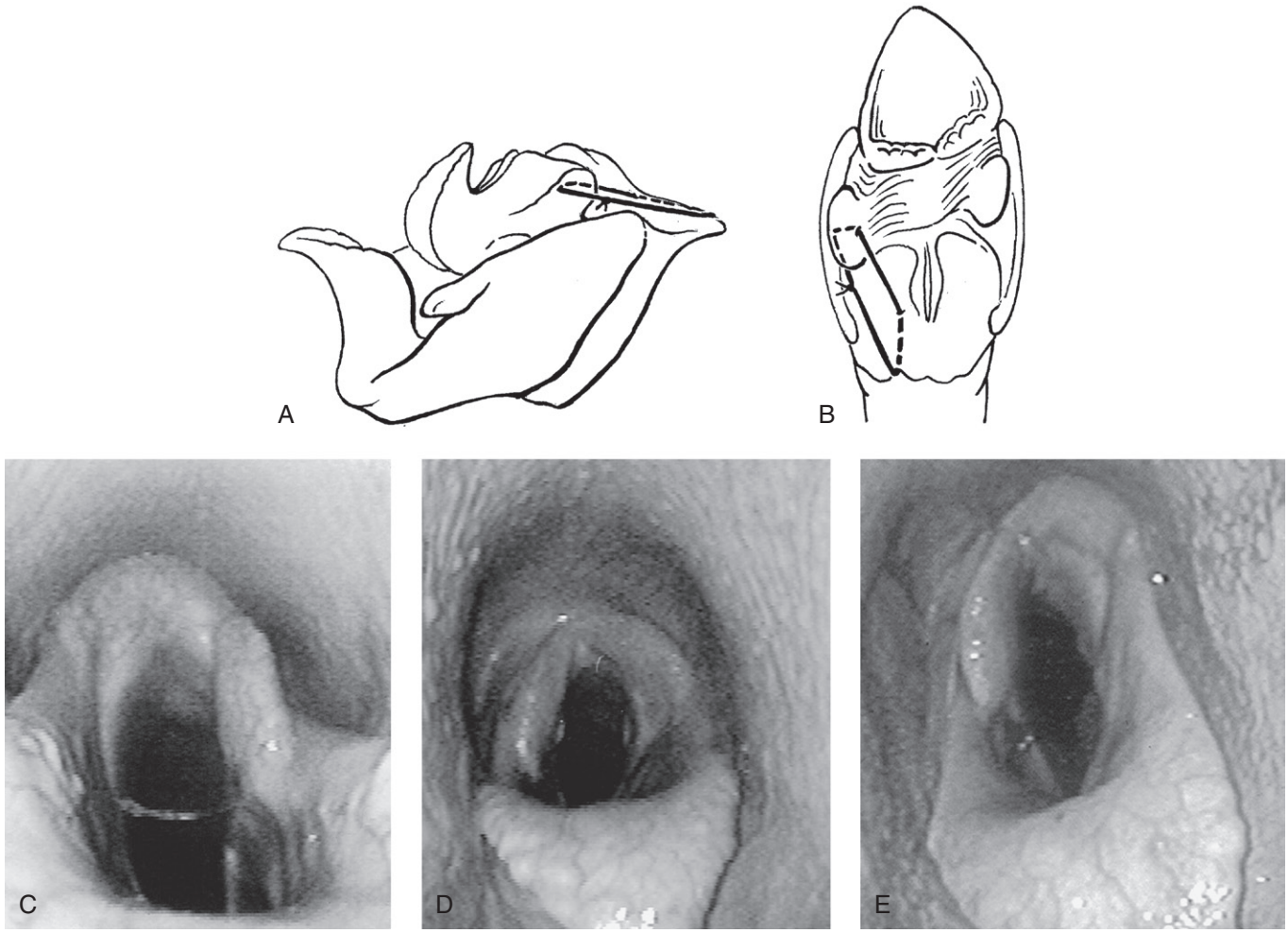


Figure 45-4. Illustration of the correct placement of the laryngoplasty suture and the resultant effects on arytenoid position. **A,** Lateral view of single prosthetic suture in place and tied. **B,** Dorsal view of the larynx, showing configuration of the laryngoplasty suture. **C,** Endoscopic image of a larynx with grade 4 recurrent laryngeal neuropathy before laryngoplasty. **D,** Endoscopic appearance of the larynx after laryngoplasty with the left arytenoid cartilage in moderate abduction (Dixon grade 2).[56] **E,** Endoscopic image of an overabduction of the left arytenoid cartilage owing to excessive tension of the prosthetic suture (Dixon grade 1).[56]

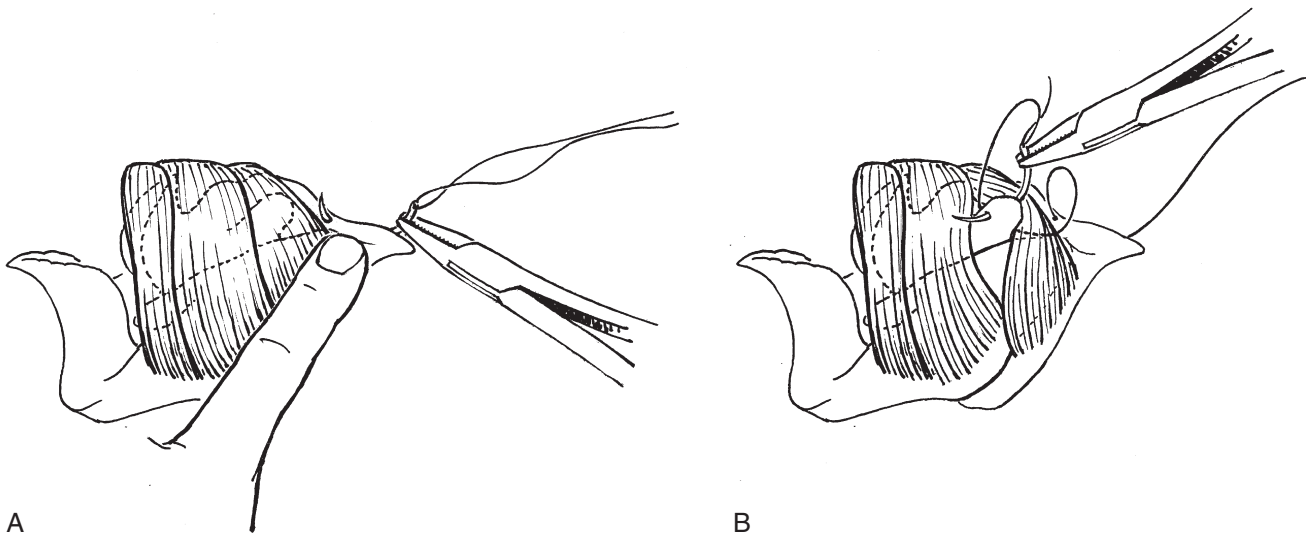


Figure 45-5. Schematic illustration of the laryngoplasty technique. **A,** Placement of the prosthesis behind the caudal border of the cricoid cartilage and exiting through the dorsal portion of the cartilage. **B,** Placement of the prosthesis through the muscular process of the arytenoid cartilage from a caudomedial to craniolateral direction.

backed out, the incision should be lavaged with saline, and the process should be started again with a new needle. When the needle has been drawn through the cartilage and out of the incision, the needle is cut off and the suture ends are tagged with a small hemostat. The second suture is usually placed 10 mm lateral to the first using the same technique. If the cricopharyngeus muscle has not been retracted forward, a large hemostat is passed under the cranial aspect of the cricopharyngeus muscle to bring both ends of the first and second sutured craniad toward the muscular process of the cricoid cartilage.

Placement of the suture through the muscular process can be achieved by a variety of techniques. Use of heavy needle drivers and a reverse cutting No. 6 Mayo needle or No. 6 Martin uterine needle is common. The use of a 3-mm bone trocar to create a tunnel for the suture has been reported to reduce fissure formation and risk of cartilage failure from suture pullout.[53] A 12- to 16-gauge hypodermic needle can also be used to create the tunnel through the muscular process. The prosthesis can be placed through a loop of No. 1 stainless steel wire that is passed through the tunnel to facilatate prosthesis placement through the muscular process. Before needle placement, some surgeons transect the tendon of insertion of the CAD muscle onto the muscular process and open the cricoarytenoideus articulation. After it is open, the joint can be curetted to induce fibrosis and ankylosis to try to reduce the loss of abduction frequently observed in the early postoperative period.[54] To enhance this even further, injection of polymethylmethacrylate has been advocated as a better method of joint ankylosis.[55] Removal of the attachment of the cricoarytenoideus muscle also reduces the opportunity for cycling of the prosthesis in horses with grade 3 RLN, which is thought to increase the early postoperative loss of arytenoid abuction. The shape of the muscular process varies among horses, and cutting the tendon of insertion can help identify these variations. To the inexperienced surgeon, this may help, via palpation, avoid placing the suture along a less than ideal pathway through the muscular process. Typically the needle or trocar is passed through the muscular process from the caudomedial aspect of the process toward the craniolateral aspect (Figure 45-5, *B*). The needle can also be placed in a caudal to cranial (sagittal) direction.[56]

After removal of the needle, firm tension is placed on the cranial and caudal ends of the suture to remove any slack, ensuring the prosthesis is tight against the larynx. If a trocar is being used, both sutures can pass through a single tunnel; otherwise, the second suture is placed approximately 5 mm more caudad in the muscular process. Engaging the spine of the muscular process rather than the tip is essential to achieve maximal biomechanical advantage and to avoid early pullthrough of the prosthesis. The trailing ends of the prosthetic sutures can be drawn under the cricopharyngeus muscle if necessary with a hemostat, and the sutures are tied (see Figure 45-4). Care must be taken to ensure that each trailing suture end is matched to its leading end and that both sutures are placed before one is tied (to avoid cutting the tied suture). When tying sutures, direct vision of the larynx via videoendoscopy is advised to avoid excessive or inadequate abduction of the arytenoid cartilage. The degree of abduction observed intraoperatively by videoendoscope sometimes does not match the immediate postoperative view in the standing horse, but surgical experience can reduce postoperative disappointment. After the prosthetic suture or wire is tied or tightnened, all retractors should be removed to allow the larynx to assume a normal, nonrotated central position to allow accurate assessment of the degree of abduction. When using suture as the prosthesis, leaving the cut ends 1.5 to 2 cm long can allow the knot to be undone and retied if repeat laryngoplasty is performed within the first week of the original surgery (see "Repeat Laryngoplasty," later). Maximal abduction, referred to as grade 1,[56] is not necessary to achieve success (see Figure 45-4, *E*). Abduction to achieve approximately 88% of maximal rima glottidis cross-sectional area (Dixon grade 2) is sufficient to allow adequate airflow[57] (see Figure 45-4, *D*). As a general guide, when the curvature of the corniculate cartilage comes into contact with the wall of the pharynx, this is generally the ideal degree of "pull out." When the sutures have been tied, the thyropharyngeus and cricopharyngeus muscles can be reapposed with simple continuous 2-0 absorable suture, if they were separated, followed by apposition of the fascia adjacent to the linguofacial vein to the omohyoideus muscle with simple-interrupted or continuous sutures of 2-0 synthetic absorbable suture material. The skin is closed with staples or 2-0 nonabsorbable monofilament suture material. A stent bandage sutured over the incision can help protect the incision during recovery and minimize swelling postoperatively. At the conclusion of the laryngoplasty surgery, many surgeons routinely place the horse into dorsal recumbency and perform a bilateral ventriculectomy via laryngotomy (described later).

Varations of the laryngoplasty technique include using one prosthetic suture only or using a crochet-style hook rather than a loop of wire to pull the leading edge of the prosthesis through the muscular process.[49,50] A novel technique to refine the tension applied to create arytenoid abduction involves using a tension and crimping device to adjust and secure the prosthetic suture (Securos Equine Tie-Back System).[58] In an *in vitro* study, an increase in holding strength of a double loop fixation of the suture through the muscular process was shown.[59] Additonally, the use of a Titanium Corckscrew as suture anchor in the muscular process of the arytenoid cartilage and a stress-reducing washer at the cricoid[60] showed significantly higher forces to failure compared with the the original laryngeoplasty technique.[49]

Although individual surgeons often introduce variations in the technique or suture material, the following principles should be observed:

- Tissue trauma associated with the initial blunt dissection over the larynx should be minimized to prevent damage to adjacent structures (particularly nerves) and to reduce the possibility of postoperative dysphagia.[61]
- The prosthetic suture should be placed securely in the cricoid cartilage and the muscular process to minimize cartilage failure.
- Placement of one suture dorsally and one about 10 mm laterally in the cricoid cartilage is important for achieving adequate abduction. Placement of the suture too far laterally results in inadequate abduction. The ideal position is between 10 and 30 degrees from zero (zero degrees is defined as a line through the muscular process of the left arytenoid cartilage and parallel to the wing of the thyroid cartilage when viewed dorsally).[62]
- The suture must not penetrate the lumen of the larynx. Placement of the suture in the cricoid cartilage is the most likely time when the lumen can be penetrated.

Videoinspection intraoperatively allows early identification of this problem.

POSTOPERATIVE MANAGEMENT

Following prosthetic laryngoplasty, confinement to a stall or a walk-in, walk-out yard is recommended for 30 days. Routinely, feed and water are placed at ground level to reduce laryngeal and upper tracheal contamination. This method of feeding should be encouraged long term. Hand-walking is allowed for exercise. During the second postoperative week, the swelling in the laryngoplasty incision area subsides. During the fifth and sixth postoperative weeks (30 to 45 days after surgery), the horse is exercised lightly or turned out in a small paddock or round pen. After this, training is resumed. The owner should be advised to feed hay from the ground and that the horse may develop a chronic cough associated with eating.

COMPLICATIONS

Intraoperative complications

Intraoperative complications include hemorrhage from deep in the surgical site, needle breakage, perforation of the laryngeal mucosa, and prosthetic suture "cut-through" of either the cricoid cartilage or muscular process of the arytenoid cartilage. Significant hemorrhage can arise from the plexus of laryngeal vessels that are inadvertently punctured as the needle is passed through the cricoid cartilage and CAD muscle. Temporary packing with sponges often slows bleeding and allows the placement of the prosthetic suture through the muscular process. Hemorrhage usually stops or reduces significantly when the suture is tightened and tied. Therefore, the surgery should be hastened to accomplish tying of the suture as soon as possible.

Breakage of the needle when passing through cartilage can occur and all reasonable effort should be made to retrieve the broken portion. If unsuccessful because the needle is embedded in cartilage or buried in the adjacent soft tissues and has not penetrated the lumen of the larynx, the needle is left because extensive dissection increases the risk of postoperative dysphagia. The needle is clearly visible with radiography of the horse's larynx, and owners should be informed of the complication.

When the prosthetic suture is tightened, suture pullout from either cartilage is a serious problem. The suture must be replaced, avoiding the damaged portion of the cartilage and using less tension. In this instance, a second suture is always used to secure the arytenoid cartilage in an abducted position. Damage to the muscular process from suture pull out can necessiatate placing the new suture at 90 degrees to the original and farther down the spine of the muscular process to minimize the opportunity of a repeat complication. Perforation of the laryngeal mucosa is most likely to occur when placing the needle under the caudal border of the cricoid cartilage. If this goes unrecognized until after recovery and the suture is seen during postoperative endoscopy, infection of the suture and possibly the cartilage can be expected unless the suture is removed. Intraoperative videoscopic examination immediately after both sutures have been passed through the cricoid cartilage will prevent this complication.

Postoperative complications

Complications in the first 2 weeks postoperatively related to the surgical procedure (as distinct from general anesthesia complications) include seroma, wound infection, wound dehiscence, dysphagia, coughing (often, but not always) associated with eating and mild to excessive loss of abduction. Excessive abduction (see Figure 45-4, *E*) may necessitate a repeat laryngoplasty. Chronic complications include persistent coughing; chronic airway contamination with feed, saliva, and water; chronic tracheitis and bronchitis; lung abscess formation; pneumonia; chondritis with formation of a luminal suture sinus; isolated inflammation and granuloma formation on the corniculate process of the arytenoid cartilage; perilaryngeal abscess formation; suture pullout; progressive loss of abduction; and persistent nasal discharge of feed and saliva, both of which could require repeat laryngoplasty.

After surgery, large seromas can develop at the laryngoplasty site, and an endoscopic examination may show collapse of the pharyngeal vault, especially on the side where surgery has been performed. Provided the horse is eating well and remains afebrile (temperature less than 38.5° C [101° F]) and the swollen area is relatively pain free, warm compresses and anti-inflammatory drugs are usually all that is required to resolve the the seroma. However if the horse develops a fever, systemic antibiotic treatment is warranted. Drainage of the laryngoplasty incision is indicated if medical treatment fails to resolve the swelling. The prosthetic suture rarely becomes infected; however, if this occurs, chronic drainage of purulent material may occur for weeks or even months. Because antimicrobial therapy may resolve the infection, removal of the prosthetic suture is often not necessary and should be delayed for about 4 months, because early removal will result in failure of arytenoid cartilage abduction.

Following laryngoplasty, many horses experience some degree of coughing and dysphagia. Dysphagia can be related to excessive and prolonged retraction of the cricopharyngeus and thyropharyngeus muscles because these are involved with esophageal sphincter function. Initially, pain may result in a reluctance to swallow. In the first few days after surgery, saliva, water, and food material may enter the trachea when the horse eats and drinks, resulting in significant coughing. Intensity of coughing often reduces over 7 to 10 days, although many horses have some degree of residual coughing when eating. The incidence of coughing in the immediate postoperative period has been reported to be as high as 43%.[34,56,61,63] Persistent coughing is most likely the result of continued contamination of saliva, water, or food material into the trachea. This pharyngeal dysfunction resulting in laryngeal and tracheal contamination may have a neurologic origin owing to surgical manipulation or it may be related to overabduction of the arytenoid cartilage.[61] Chronic coughing has been reported in up to 14% of horses more than 6 months after surgery with many of them having concurrent pulmonary disease.[56] Chronic airway contamination causes chronic coughing and, in some horses, repeated episodes of tracheal, bronchial, or lung infection when they resume training. In rare cases, bronchopneumonia may be the long-term result. The diagnosis of airway contamination is easily made by endoscopic examination. Removal of the prosthesis may resolve the contamination problem and even the chronic cough in some horses. Time may allow this problem to resolve in others.

Prosthetic suture pullout resulting in failure of laryngoplasty has been proposed to be more likely in yearlings or 2-year-old horses, although experimentally there is no significant effect of age on the *in vitro* cartilage retention of the prosthesis.[64] Most commonly, suture pullout occurs at the muscular process.[64,65] Partial muscular process cartilage failure is suggested as the

likely cause of postoperative "relaxation" of the suture, with some loss of abduction.[64] This is a well-recognized phenomenon that occurs during the first 30 days after surgery.[46,56] One retrospective clinical study revealed a much higher rate of successful outcome after prosthetic laryngoplasty in horses that were 2 years old or younger than in horses 3 years old or older.[63] Therefore there is no hard evidence to exclude young horses (younger than 2 years) from laryngoplasty treatment, and they are at no greater risk for laryngoplasty failure than older horses.[34,63,64]

Repeat laryngoplasty may be necessary for loss of abduction or when overabduction results in persistent coughing and aspiration of feed. The requirement for repeat surgery has been reported to be as high as 10% for loss of abduction and up to 7% for management of overabduction.[56] Timing of repeat laryngoplasty should considered carefully in the first 10 days after surgery. Undergoing two general anesthesias in close proximity can increase the risk of intestinal disorders, including cecal impaction.[66] The surgical approach mimics the original incision, but care must be taken not to penetrate the linguofacial vein during the dissection between the vein and the omohyoideus muscle as a result of fibrosis. Reoperation weeks or months later presents dense scar tissue, making it difficult to expose the cricoid cartilage and the muscular process of the arytenoid cartilage. Dissection through this scar tissue either digitally or with scissors can cause hemorrhage that will fill the surgical field. Temporary pressure from gauze sponges packed into the incision is often necessary to reduce hemorrhage, but patience may be required and intraoperative suction is essential.

The goal of reoperation in the early postoperative period (first 14 days) is to tighten or loosen the prostheis by twisting if wire was used, to retie the suture if there has not been pullout from either cartilage, or to extract the original laryngoplasty sutures and replace them with another suture (or sutures) that achieves more ideal arytenoid cartilage abduction. If a surgery report from the original surgery is not available, the surgeon should be aware of the possibility of more than one prosthetic suture. After the first 4 weeks, the suture is frequently buried in scar tissue, but the knot can usually be found caudal to the muscular process. The suture is cut beside the knot and removed, although it may break and fragments can be left behind. The scar tissue along the lateral and caudal surfaces of the muscular process must be incised with Metzenbaum scissors and digital guidance to mobilize the arytenoid cartilage. A laryngoplasty suture is subsequently placed and tied ideally under endoscopic observation (Figure 45-6).[22,31,67] If the cartilage is elevated away from the endotracheal tube, it is likely that the goal of improving abduction has been achieved. If not, it is likely that the reoperation attempt will fail. With this intraoperative knowledge, the surgeon can decide whether to place another suture to try to achieve greater abduction or whether to advise of the need for arytenoidectomy.[22]

If overabduction after laryngoplasty produces excessive and persistent airway contamination, the original sutures can be removed. A new prosthesis can be placed that fixes the arytenoid cartilage with less abduction than the original suture if the reoperation is performed within 2 weeks of the initial surgery. When reoperation takes place more than 120 days after the first surgery, removal of the sutures without replacement may be all that is necessary. Minimal dissection around the muscular process during suture removal often leaves sufficient scar tissue to maintain moderate arytenoid cartilage abduction.

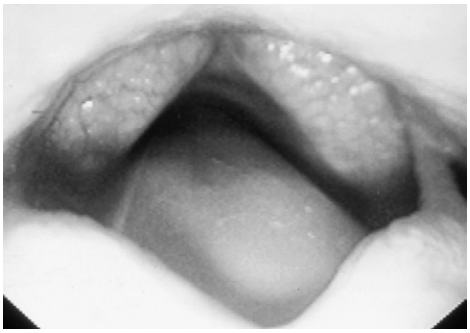


Figure 45-6. Endoscopic appearance of the arytenoid cartilages during laryngoplasty. The endotracheal tube has been positioned in the rima glottidis through the mouth, and the displaced palate is seen ventrally. The space between the endotracheal tube and the left arytenoid cartilage indicates that the prosthetic suture tension has produced adequate abduction.

For horses with chronic tracheal contamination with feed, water, and saliva and the resultant range of respiratory tract disease from tracheitis to pulmonary abscessation or pneumonia, removal of the suture is warranted. However chronicity of lung pathology and effect on future athletic performance should be considered and discussed with the owners prior to surgery, as should the increased risk of anesthesia-related complications.

The presence of a draining sinus into the laryngeal lumen usually represents chronic infection of the suture, and in some cases the suture can be seen hanging into the airway. Although resolution of infection has been reported without removal of the suture,[56] some cases require removal of the prosthesis to resolve the luminal sinus and associated infection.

PROGNOSIS

The reported success of laryngoplasty varies widely because of the different criteria used to measure success and the intended use of the horse. Although reported success rates have ranged from 5% to 90%,[18,31,34,61,68-70] it is realistic to expect that between 50% and 70% of racehorses treated with a laryngoplasty will have improved racing performance after surgery.[22,34,63] The outcome will be more successful in horses that are not intended to race after surgery.[63] In a retrospective performance analysis after prosthetic laryngoplasty, the degree of arytenoid abduction achieved did not necessarily correlate to a successful surgical outcome.[63] An *in vitro* analysis has identified that an airway with an opening of 88% of the maximal rima glottidis cross section, allowed what was interpreted as adequate airflow for exercise.[57] Clinically, even in horses with grade 3 abduction following laryngoplasty, satisfactory performance is achievable.[70] Noise production should not be used as a measure of airway obstruction.[46] However, laryngoplasty should be viewed as a salvage procedure in racehorses, because even the horse with near maximal abduction of the arytenoid cartilage will have some flow limitations at racing speeds.[41]

Ventriculectomy (Sacculectomy)

Ventriculectomy (unilateral or bilateral) refers to the removal of the mucosal lining of the laryngeal ventricle located caudal to the vocal fold.[31] The surgery is usually performed to eliminate

noise and can have some beneficial effects on performance. Because ventriculectomy does not produce abduction of the arytenoid cartilage, it is not recommended as a sole procedure for racing horses affected with advanced RLN.[31] However soft tissue collapse during exercise is decreased, and the procedure can be successful when performed on hunters, show jumpers, and eventing-type horses. In draft-type horses, complication rates of laryngoplasty (e.g., failure, myopathy, neuropathy, anesthetic problems, coughing) can be as high as 25%; therefore, bilateral ventriculectomy, either by surgical (via laryngotomy) or laser excision, in the standing horse is an excellent alternative.[71,72] Surgical ventriculectomy is performed via a laryngotomy (see later) under general anesthesia, frequently at the conclusion of a laryngoplasty, but it can also be done as a standing procedure with the horse in stocks with the head elevated and sedated with loading doses of detomidine (4 mg IV) and butorphanol (10 mg IV). Following local anesthesia of the skin and subcutaneous tissues, a detomidine drip using 14 mg added to 250 mL of saline given to effect (approximately 2 drops per second for 15 minutes, then 1 drop per second for 15 minutes and then to effect) is used to maintain sedation during the procedure. For additional information, review Chapter 22.

If surgery is performed under inhalation general anesthesia, and after the laryngotomy has been performed, the endotracheal tube can be removed or left in place while the ventriculectomy is being performed. Use of a smaller endotracheal tube often negates the need for tube removal. A burr (e.g., Blatternburg burr) is introduced into the ventricle to its maximum depth and twisted to engage the mucosa in the projections on the burr (Figure 45-7, *A* and *B*). Occasionally, the ventricle is so large that the surgeon needs to press on the laminar portion of the arytenoid cartilage to enable the burr to engage the mucosa of these large saccules at their apex. The burr is subsequently withdrawn slowly from the ventricle, everting the attached saccule. Swallowing often occurs during this procedure in the standing horse and helps evert the saccule. A large forceps (Carmalt, Lahey Gall) is placed across the everted saccule between the head of the burr and the vocal fold (Figure 45-7, *C*). With traction on the clamp, a second forceps is placed behind it. By applying digital pressure on the opening of the ventricle, the entire saccule is everted and subsequently excised with Metzenbaum scissors (Figure 45-7, *D*). The same procedure is repeated on the opposite ventricle if bilateral ventriculectomy is indicated

POSTOPERATIVE MANAGEMENT

Topical administration of a pharyngeal medication with a mixture of nitrofurazone (Furacin) or pediatric trimethoprim sulfa (oral preparation), dimethyl sulfoxide, glycerin, and prednisolone can be beneficial. A No. 10 French catheter is advanced along the floor of the nasal passage into the nasal pharynx, and 10 to 15 mL of the solution is sprayed slowly through this catheter at 12-hour intervals for 7 days. Use of corticosteroids either in conjunction with phenylbutazone or alone are often advocated, especially with laser surgery of the upper respiratory tract. Regimens used include dexamethasone powder (0.022 mg/kg) given orally once daily in the morning feed for 3 consecutive days, day 4 is skipped, and the same dosage is then given on the fifth postoperative day. If a longer course of corticosteroid therapy is deemed necessary, 0.9 mg/kg of prednisone is given orally, starting the day after surgery, once daily in the morning feed for 7 days. The same dosage is then given every other day for three treatments. The dosage is subsequently reduced to 0.45 mg/kg, given every other day for three treatments. An individual horse's risk of cortisone-induced or -related laminitis should be given prior to administration. The horse is restricted to hand-walking exercise for the first week after surgery and then endoscopically assessed to determine if the horse can return to paddock exercise or if additional rest and anti-inflammatory medication are necessary. The horse should not be returned to training for 60 days.

Ventriculocordectomy

Ventriculocordectomy refers to the removal of the mucosal lining of the laryngeal ventricle as described earlier, as well as the removal of a crescent-shaped wedge of tissue from the leading edge of the vocal fold. Again, this technique does not result in abduction of the arytenoid cartilage, so its use as a sole procedure for most racing horses that have RLN and that run over long distances is not recommended. However, its use to reduce noise in sport and draught horses has been established.[43,71] There is also evidence that ventriculocordectomy can allow improved athletic performance in a subset of horses with RLN.[45,73] Suitable candidates for ventriculocordectomy include:

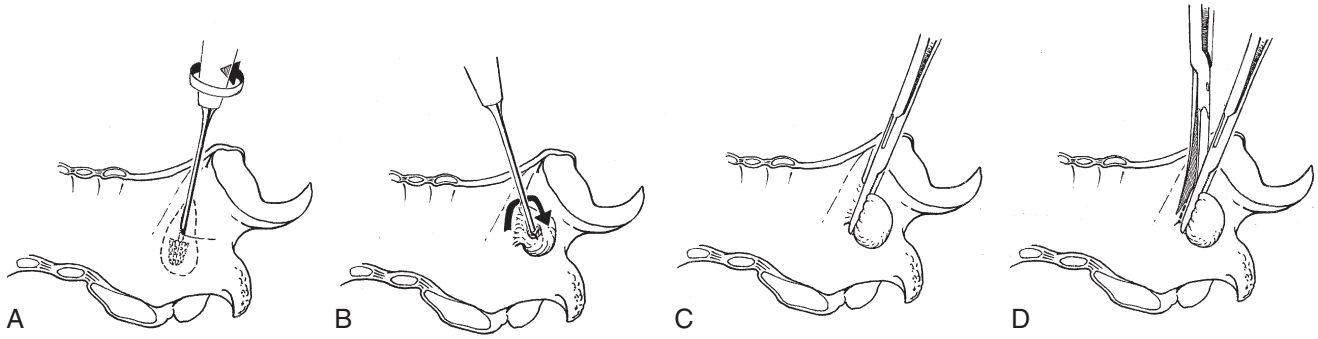


Figure 45-7. Schematic illustration of the ventriculectomy technique with the horse in dorsal recumbency. **A,** The "roaring" burr is placed into the laryngeal ventricle and rotated so that the head of the burr engages the mucosa of the laryngeal saccule; **B,** When the saccule is firmly engaged, it is everted into the lumen of the larynx by steadily pulling on the burr; **C,** A large hemostat is placed across the saccule immediately adjacent to the vocal fold, and the burr is removed; **D,** The saccule is completely excised using Metzenbaum scissors adjacent to the hemostat.

- Sport horses with grade 4 laryngeal movements where the primary complaint is respiratory noise, and exercise intolerance is not a feature (hunters, eventers, draught breeds, and other show horses)
- Race horses with grade 3 laryngeal movements that do not experience arytenoid collapse during high-speed exercise but do experience vocal fold collapse identified with exercising videoendoscopy.
- Race horses that have had a laryngoplasty and still experience vocal fold collapse during high-speed exercise. Caution must be used in performing a ventriculocordectomy in racehorses that have had a laryngoplasty where noise is a complaint because noise cannot be correlated with upper airway obstruction.[46] Again, the use of exercising videoendoscopy is recommended in such horses.

Surgical ventriculocordectomy is performed via laryngotomy (see later). Following the ventriculectomy as described earlier, a 2-cm long and 2- to 3-mm wide, crescent-shaped wedge of tissue is removed from the leading edge of the vocal fold. If the procedure is performed standing, the incisions are left to heal without suturing. If the horse is under general anesthesia, using a continuous suture pattern of 2-0 polydioxanone, the outside (abaxial edge of the vocal fold) and inside (axial) border of the ventricle are apposed. Suturing limits hemorrhage at the time of surgery while lessening cicatrix formation and redundant tissue folds during healing of the surgery site, leaving a smooth surface over the ventral half of the rima glottidis. The procedure is repeated on the other side.

Endoscopically guided laser-assisted ventriculocordectomy can be performed in the standing sedated horse.[72] Commonly a diode laser is used and surgery is performed via manipulation of the vocal fold by bronchoesophageal grasping forceps and a modified Blatternburg burr, which can be passed along the nasal passage.[73-75] Preoperative and postoperative care is the same as that given for ventriculectomy via laryngotomy except wound care is not required.

Laryngotomy

The ventral laryngeal region is clipped, aseptically prepared, and draped (if under general anesthesia) for surgery. The surgery can be performed under inhalation or intravenous anesthesia. A 10- to 15-cm (4- to 6-inch) incision is made along the ventral midline from just rostral to the palpable keel of the thyroid cartilage caudad to just beyond the cricoid cartilage. Careful dissection of subcutaneous tissues allows identification of the septum of the paired sternothyrohyoideus muscles, which are separated along the full length of the incision using Metzenbaum scissors. Blunt Weitlaner retractors are positioned between the paired muscle bellies to expose the ventral aspect of the larynx. Usually there is some loose adventitia overlying the larynx, and this is separated to expose the cricothyroid membrane. Careful attention to hemostasis at this stage of the surgery will maintain a blood-free approach. The cricothyroid membrane and underlying mucosa is incised along the midline with either a No. 10 or No. 11 scalpel blade. If an endotracheal tube is present, care should be taken to avoid penetrating tube or cuff. The membrane is incised from the cricoid to the anterior limit of the thyroid cartilage V. The previously placed Weitlaner retractors can be repositioned in the laryngotomy to maintain good access to the laryngeal lumen. For ventriculectomy and most laryngeal lumen procedures, incising the cricothyroid membrane will provide adequate exposure; however, in some cases (arytenoidectomy), transecting the cricoid cartilage and partially splitting the keel of the thyroid cartilage is acceptable to achieve better surgical exposure.

At the conclusion of the laryngotomy, the surgeon has two choices: to leave the entire incision open or to close the cricothyroid membrane with simple interupted absorbable sutures. Complete closure of the laryngotomy incision is not recommended because up to 52% of horses have been reported to have complications associated with the closure, including subcutaneous emphysema and incisional infection.[76] For horses under genreal anesthesia, a 15-cm (6 inch) sterile roll of gauze can be sutured over the laryngotomy site as a stent bandage for recovery. This helps prevent contamination of the surgical site if head collars are used during recovery and also prevents widespread disemination of blood and mucus throughout the recovery room, which is associated with forceful respiratory efforts that can occur during recovery. The stent is removed immediately when the horse is standing.

POSTOPERATIVE MANAGEMENT

Use of perioperative antibiotics for 48 to 72 hours is recommended any time the respiratory tract is invaded. Anti-inflammatory therapy using either phenylbutazone (4.4 mg/kg, IV) or flunixin meglumine (1.1 mg/kg, IV) should also be given immediately before surgery. Continued phenylbutazone therapy for 7 days postoperatively is recommended (2.2 mg/kg PO, twice a day for 7 days).

Management of the laryngotomy incision initially involves twice-daily cleansing of the skin around the wound and also circumfrentially inside the incision using moist gauze swabs to remove accumulated mucus and exudate. Petroleum jelly should be applied to the surrounding skin to prevent scalding from the exudate that often remains for up to 3 weeks. Under normal circumstances, laryngotomy incisions heal within 21 days and as time progresses, cleaning can be reduced to once daily. The horse is restricted to hand-walking exercise for the first week after surgery and then is endoscopically assessed to determine if the horse can return to paddock exercise or if additional rest and anti-inflammatory medication are necessary. The horse should not be returned to training for 60 days.

COMPLICATIONS

Complications following laryngotomy are rare. Excessive retraction during surgery can cause bruising and trauma to the sternothyrohyiodeus muscle. Necrosis and local infection can follow several days later, resulting in malodorous and excessive wound discharge. Resection of necrotic tissue results in a rapid resolution. Splitting the septum of the sternothyrohyoideus accurately and careful surgical technique will help avoid this complication. Clostridial infection at the laryngotomy site can occur within a 24-hour period after surgery, producing tremendous swelling of the head and neck and resulting in a massive tissue slough with potentially a fatal outcome. The use of perioperative penicillin may prevent such an infection, and if it does occur, early recognition and prompt treatment with intravenous penicillin is critical. Occasionally, excessive granulation tissue forms in the ventricles after sacculectomy but usually resolves with time and the application of anti-inflammatory sprays.[43,77]

Laryngeal Reinnervation

Although any horse with RLN is a candidate for laryngeal reinnervation, younger horses and those with grade 3 laryngeal movements are the ideal candidates for this surgical procedure.[78] The technique involves implanting a nerve–muscle pedicle (NMP) graft into the affected CAD muscle. The first cervical nerve and omohyoideus muscle are used for the NMP graft because the omohyoideus muscle is an accessory muscle of respiration (active on inspiration) and the first cervical nerve is in close proximity to the larynx.

Patient preparation and initial surgical approach is similar to laryngoplasty. Antimicrobial and anti-inflammatory medications are instituted as for other laryngeal surgeries. Surgical instruments should include very small atraumatic thumb forceps, a spay hook, and small blunt Weitlaner self-retaining retractors in addition to the instruments used for laryngeal surgery.

For left RLN, the horse is placed in right lateral recumbency under general anesthesia, and the head and neck are positioned for as for laryngoplasty. A 12-cm (5-inch) linear incision is made along the ventral border of the linguofacial vein, which is carefully separated from the omohyoideus muscle. The first cervical nerve usually lies under a branch of the linguofacial vein that is positioned midway between the rostral and caudal aspects of the incision. The ventral branch of the first cervical nerve emerges through the alar foramen of the atlas and descends over the cricopharyngeal muscle caudal to the larynx before dividing, usually, into a cranial, middle and caudal branch and entering the omohyoideus muscle (Figure 45-8, *A*). This nerve is commonly encountered during dissection for laryngoplasty and can be traced from the atlas to the omohyoideus muscle. The main body of the first cervical nerve is identified, and ventral retraction of the omohyoideus muscle is achieved with Allis tissue forceps. Insertions of the first cervical nerve can be found in the dorsal aspect of the muscle belly (see Figure 45-8, *B*). Meticulous dissection is necessary to expose the fine branches of the first cervical nerve as they enter the omohyoideus muscle. The middle branch of the first cervical nerve often divides into smaller branches and can be traced to a point of muscle entry, allowing creation of two or three nerve-muscle pedicles. The main branch of the first cervical nerve is gently elevated using a spay or nerve hook and is freed from its fascial attachments on the lateral aspect of the larynx. Before creating the muscle pedicles, mepivacaine hydrochloride is used to bathe the area to prevent excessive muscle contraction from occurring during harvesting of the pedicles. Nerve branches that do not penerate the omohyoideus muscle and continue to run over the muscle surface are transected to be used as nerve implants. The NMP grafts are created with fine tenotomy or iris scissors and are about 3 mm^3.

The larynx is subsequently rotated laterally by placing a spay hook through a window created by separating the aponeuroses of the cricopharyngeus and thyropharyngeus muscles and applying traction on the wing of the thyroid cartilage. Exposure of the CAD muscle can either be via the window created between the cricopharyngeus and thyropharyngeus muscles or by blunt dissection off the back edge of the cricopharyngeus muscle and under the vascular plexus overlying the CAD muscle. The window over the CAD muscle can be maintained using a Weitlaner retractor (Figure 45-8, *C*). Longitudinal separation of the CAD muscle fibers, using fine vascular forceps, allows pockets to be created for the pedicles to be placed. Each nerve-muscle

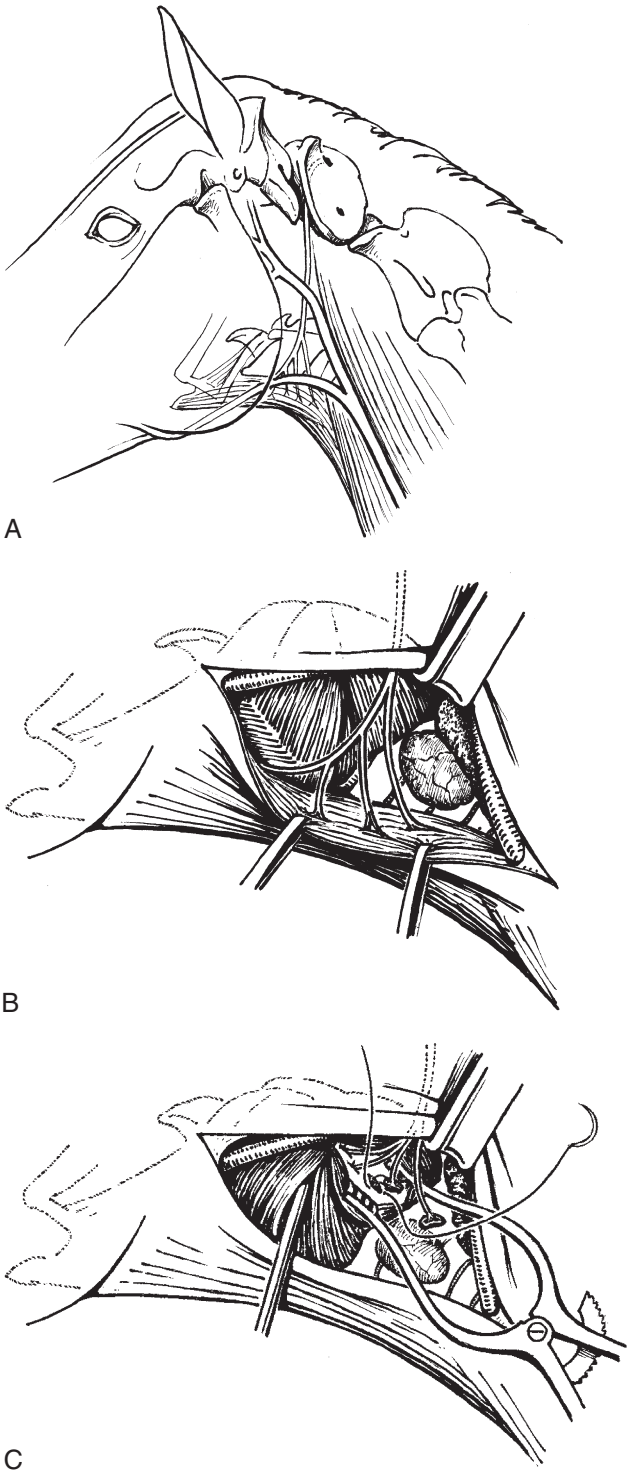


Figure 45-8. Schematic illustration of the neuromuscular pedicle graft technique. **A,** The origin and insertion of the left first cervical nerve into the omohyoideus muscle is shown. This nerve and muscle serve as the neuromuscular pedicle graft donors for treatment of recurrent laryngeal neuropathy. **B,** The first cervical nerve and future pedicle grafts of the omohyoideus muscle, which has been retracted with Allis tissue forceps. **C,** Weitlaner retractors positioned on the edge of the cricopharyngeus muscle and dorsal laryngeal adventitia to expose the atrophied cricoarytenoideus dorsalis (CAD) muscle and allow implantation of the neuromuscular pedicle grafts.

pedicle or nerve branch is sutured into a pocket using 4-0 polydioxanone (see Figure 45-8, *C*). With the mapping of the motor endplates in the CAD muscle, nerve muscle pedicles and nerve implants are now concentrated more along the junction of the dorsal and lateral bellies, concentrating in the caudal third of the muscle, rather than the more dorsal approach originally used.[79] Closure of the incision is routine; however, care should be taken to ensure leaking lymphatic vessels are ligated to help prevent seroma formation.

The horse is confined to a stall for 2 to 3 weeks after surgery until sutures are removed and inflammation has subsided. Perioperative antibiotic therapy is maintained for 3 days and anti-inflammatory medications for 7 days after surgery. After stall confinement, the horse receives paddock turnout for 14 weeks. At this stage (16 weeks after surgery), the horse is put into training. This time frame is based on information from studies on dogs, people, and horses, which indicate that the earliest time to see clinical evidence of reinnervation is around 12 weeks following surgery.[80-83] When the horse is returned to exercise, episodes of fast exercise should be introduced as early and as frequently as possible. Because the omohyoideus muscle is an accessory muscle of respiration, considerable respiratory effort must be undertaken to activate the first cervical nerve and therefore the CAD muscle. Swimming is also considered good exercise because of the head elevation and neck extension.

After 6 weeks of training the horse should have an endoscopic assessment of the larynx. At rest, the left arytenoid cartilage usually appears as it did before surgery. This is because the first cervical nerve is inactive at rest; thus, there is no depolarization of the nerve and no CAD muscle contraction. Two diagnostic reflexes have been developed to stimulate contraction of the omohyoideus muscle and the newly innervated CAD muscle.[78] The first involves stretching the horse's head and neck upward as high as possible while closely observing the larynx through the endoscope. If reinnervation has occurred, there is often a spontaneous flicker or single abduction of the left arytenoid cartilage. The second reflex involves pulling back rapidly with a finger or with the bit, resulting in sudden pressure on the commissure of the horse's lips. Again, a sudden abduction of the left arytenoid cartilage occurs if reinnervation has been successful. This reflex can be stimulated from the left or right side of the head.

Any abduction, no matter how small, of the left arytenoid cartilage indicates that there has been reinnervation of the CAD muscle. When this has been identified, the recommendation to the trainer is to continue training toward a return to racing. If there is no evidence of movement at the first revisit, the horse is turned out again for another 8 weeks, receives 6 weeks of training and is re-examined. Horses can take up to 12 months to show evidence of successful reinnervation.[81] Clinical experience suggests that if there is no arytenoid abduction as a result of reinnervation 9 months after surgery, there is only a small chance of improvement from that point. Reinnervation probably occurs in nearly all patients by 4 to 5 months.

The best evidence of successful reinnervation in an individual horse is derived from a treadmill endoscopic or upper airway flow mechanics study. In treadmill studies, we have found that horses with movement of the left arytenoid cartilage that is visible at rest after laryngeal reinnervation maintained arytenoid abduction during vigorous exercise, but often not maximal.[82] Inability to demonstrate effective arytenoid cartilage movement in the standing horse is an indication that the horse will most likely have dynamic collapse during exercise.

COMPLICATIONS

Complications associated with laryngeal reinnervation are few when compared with prosthetic laryngoplasty. The most frequent complication is seroma formation 3 to 5 days after surgery. The large potential dead space that exists after the nerve graft procedure lends itself to the formation of a seroma. In our opinion, many of the seromas that form are caused by leaking lymphatic vessels that are inadvertently cut during surgery. Large lymph vessels travel along the lateral border of the omohyoideus muscle and can be transected during the surgical approach. Many of these seromas resolve without intervention; however, some require open drainage and daily lavage. Horses have been placed on procaine penicillin therapy until the open tract discontinues draining. Some sinus tracts have become infected, and these have been treated with antibiotics selected from culture and sensitivity results. Because of the ventral position of the incision, the seroma drains easily after it is opened and requires minimal nursing care apart from daily lavage and attention to the skin around the incision. Horses that have suffered a postoperative incisional infection have gone on to have successful reinnervation; thus it would seem that this complication does not necessarily compromise success of the laryngeal reinnervation graft.[78]

PROGNOSIS

From experimental and clinical work, it is clear that reinnervation of the CAD muscle following an NMP graft can take between 6 and 12 months depending on the amount of CAD muscle atrophy.[82-84] Therefore, horses with grade 3 laryngeal movements will respond sooner (in 4 to 6 months) than those that have grade 4 movements (complete paralysis).[84] As a result of this long time period, horses best suited for the transplantation technique are those whose immediate return to performance is not necessary, such as yearling or early 2-year-old racehorses and pleasure horses. Horses that have had previous laryngoplasty for the treatment of RLN are not candidates for this surgery because of disruption of the first cervical nerve during that surgery or trauma and resultant fibrosis of the CAD muscle. To date, Ian C. Fulton has operated on 235 horses. A group of 165 racing Thoroughbreds have been followed to the end of their racing careers. This group of horses were classified at the time of surgery as raced (94) and unraced (71). Unraced horses had an average time of 13.5 months from surgery to race one with a range of 7 to 28 months. Fifty-one of the 71 horses started a race and 63% of these won at least one race. On average, these horses had 14.6 starts.[85] In the raced group of horses, the average time to race one was 8 months. Sixty percent of horses won at least one start and 52% of horses won more prize money in total after surgery. However only 39% earned more money per start after surgery and only 45% had an improved performance ranking.[83] Based on follow-up endoscopy of these horses, 80% were considered to have a successful reinnervation because of the presence of flickering abductory movements of the arytenoid cartilage observed at rest. The inability of the technique to provide a higher success at the track may relate to either insufficient muscle regeneration to withstand racing or, in the unraced group, a natural inability to perform. Laryngeal reinnervation has allowed horses to compete successfully, and improve laryngeal function with time. Current success rate is on

a parity to that of prosthetic laryngoplasty. For young horses that have never been raced and horses of high value because of their breeding potential, the NMP graft can be offered as a viable treatment for RLN. If the result of laryngeal reinervation is unsatisfactory, a laryngoplasty may still be performed.

LARYNGEAL DISORDERS OTHER THAN RECURRENT LARYNGEAL NEUROPATHY

A number of laryngeal conditions that can manifest with clincial symptoms similar to RLN but are separate entities from idiopathic left-sided RLN deserve discussion. They include axial deviation of the vocal fold (collapse into airway),[86] bilateral laryngeal hemiplegia, fourth branchial arch defects, right-sided RLN, collapse of the apex of the corniculate process of the arytenoid cartilage, and axial deviation of the aryepiglottic folds.

Vocal Fold Collapse

Vocal fold collapse can only be observed during high-speed exercise using videoendoscopy (Figure 45-9). Stabilization of the vocal cord occurs when the cricothyroid muscle, which is innervated by a branch of the cranial laygeal nerve, contracts.[86] Occasionally, axial deviation of the fold occurs in horses with grade 3 laryngeal movements (see Table 45-1). Rarely, it is observed in some horses after laryngoplasty when a concurrent ventriculectomy has not been performed. Clinical signs, especially noise production and history, are similar to those of RLN. Treatment for this condition is bilateral ventriculocordectomy (see earlier).

Bilateral Laryngeal Paralysis

Bilateral laryngeal paralysis has been associated with organophosphate toxicity; central nervous system diseases, such as equine protozoal myeloencephalitis (EPM)[7]; hepatic dysfunction; hepatic encephalopathy[87]; temporarily following general anesthesia[88]; associated with lead toxicity[89]; and has been observed in horses with Australian stringhalt (Ian Fulton's personal experience). Horses with bilateral arytenoid cartilage paralysis show severe exercise intolerance with only minimal exercise or stress. Those horses in severe distress make a loud inspiratory noise even at rest. Horses may die of asphyxiation if a tracheostomy is not performed as an emergency procedure. Although both arytenoid cartilages are affected, the degree of paresis may differ between sides. During the endoscopic examination both guttural pouches should be examined and if the horse shows signs of ataxia, a cerebrospinal fluid tap should be performed for EPM testing.

Horses with bilateral laryngeal paralysis that is associated with EPM can show improved laryngeal function after appropriate medical treatment for this condition. Horses that are in danger of asphyxiation require a temporary tracheostomy (Chapter 47). In nonresponsive cases, unilateral laryngoplasty can be performed on the more severely affected side to salvage the horse for nonperformance activities. However, airway contamination with feed, water, and saliva can be severe because adductor function on the contralateral side may be compromised. Therefore, some estimate of adductor function should be made before unilateral laryngoplasty is undertaken. Because bilateral laryngoplasty is not recommended, a permanent tracheostomy should be considered (Chapter 47).

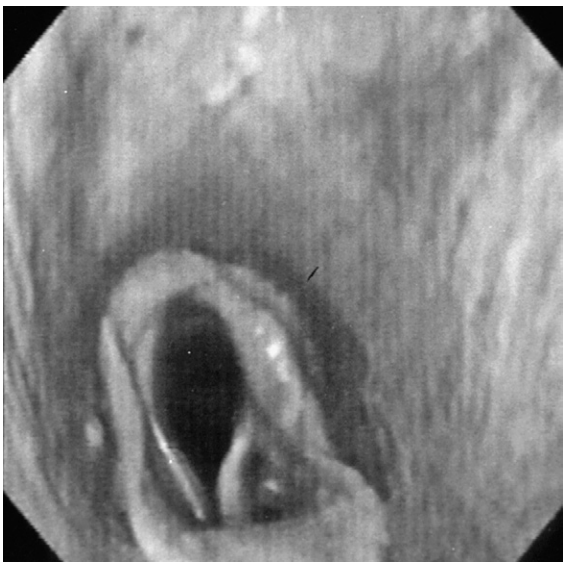


Figure 45-9. Videoendoscopic image (freeze-frame) of the larynx of a horse during exercise, demonstrating left vocal fold collapse.

Fourth Branchial Arch Defects

In the horse, the lateral wings of the thyroid cartilage, cricoarytenoid articulation, and the cricothyroid, thyropharyngeus, and cricopharyngeus muscles are all derived from the embryological fourth branchial arch. Abnormal development of one or more of these structures, unilaterally or bilaterally, produces a syndrome defined as fourth branchial arch defects, commonly abbreviated as 4-BAD syndrome.[90] The term *cricopharyngeal-laryngeal dysplasia* has also been used to describe this syndrome.[91] Although this group of congenital laryngeal deformities has an incidence of 0.02%[92] and has been sparsely reported, [91,93-95] clinicians need to be aware of them to avoid incorrect diagnoses of laryngeal disorders. The diagnosis of 4-BAD syndrome has been made in ponies, Warmblood, draught, and Thoroughbred horses.

The most common presentation of 4-BAD syndrome is exercise-induced respiratory noise secondary to dynamic collapse of soft tissues or cartilaginous laryngeal tissues. Although the CAD muscle is normal in these horses, the abnormal cartilage skeleton renders CAD contraction ineffective. The right side of the larynx is most frequently involved, and bilateral involvement is more common than left-sided involvment. Horses with a severe deformity can show respiratory incompetence, dysphagia, and repeated colic because of a deformity and dysfunction of the cranial esophageal sphincter muscles (cricopharyngeus and thyropharyngeus).

Although endoscopy is the cornerstone of identifying the cause of abnormal respiratory noise, 4-BAD syndrome can be detected with laryngeal palpation. Most affected horses have an obvious palpable deficit of the thyroid wing on one or both sides, or they have an easily palapted gap between the cricoid and thyroid cartilages because of the absence or atrophy of the cricothyroid muscle. Ultrasound or, if available, magnetic resonance imaging of the laryngeal cartilages can provide accurate detail of cartilage anatomy and support palapation findings.[26,96] Endoscopic findings must be assessed carefully, because

unilateral 4-BAD can appear endoscopically like RLN, and bilateral 4-BAD can mimic bilateral laryngeal paralysis. The descriptive diagnosis *rostral displacment of the palatopharyngeal arch* is commonly used to report the abnormal endoscopic findings when the caudal margin of the ostium intrapharyngeum is displaced rostral to the dorsal tips of the corniculate processes of the arytenoid cartilages (Figure 45-10). If laryngeal endoscopy indicates right-sided RLN, 4-BAD should be on the top of the differential diagnosis until proved otherwise. Obvious rostral displacment of the palatopharyngeal arch is not present in all cases of 4-BAD, but it is exacerbated during intense exercise as observed during high-speed video endoscopy. Even though it is not considered mandatory, radiography of the larynx can support a diagnosis of 4-BAD because of air in the upper esophagus will be contiguous with air in the pharynx as a result of dysfunction of the esophageal sphincter. The palatopharyngeal arch may also be seen as a soft tissue mass rostral to the corniculate process.

Although some horses have been operated on following misdiagnosis RLN, and arytenoidectomy has been attempted, in general, there is currently no accepted surgical solution for horses with 4-BAD defects. Some horses can perform athletically, but usually not to their full potential. The tie-forward procedure has been suggested as a possible treatment when dynamic collapse of the palatopharyngeal arch is identified with high-speed videoendoscopy without significant collapse of the arytenoid cartilages.

Right-Sided Laryngeal Hemiplegia

Right-sided recurrent laryngeal neuropathy is rare in horses and an etiology should be sought because the idiopathic form of RLN does not usually affect the right CAD muscle to a level resulting in marked paresis or paralysis. If none of the previously mentioned causes can be detected, the surgeon should suspect 4-BAD in young horses,[25] and palpation of the muscular process of the right arytenoid cartilage and thyroid cartilage is indicated. Ultrasonography should also be employed to assess laryngeal cartilage and muscle anatomy. In a series of horses treated for right-sided laryngeal hemiplegia, 7 of 11 Thoroughbreds had congenital malformation of the laryngeal cartilages resulting in a high percentage of failure of a laryngoplasty in this group of horses. As a consequence of this, partial arytenoidectomy may be selected as a treatment, but as previously mentioned, high-speed videoendoscopy is recommended prior to advising surgery. Although spontaneous recovery from right-sided laryngeal hemiplegia has been reported, it would not be an expected outcome in a 1- to 2-year-old Thoroughbred. A poor prognosis for athletic function should be given to the owner.

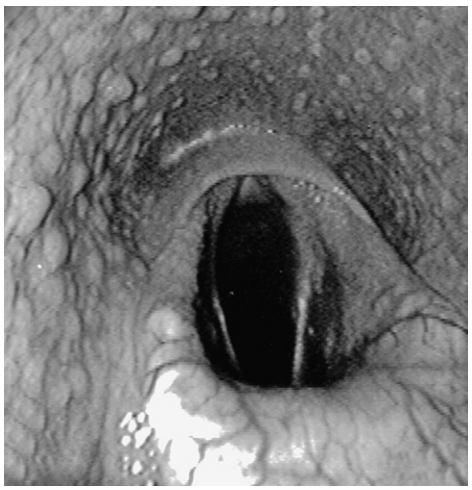


Figure 45-10. Videoendoscopic image of the larynx showing rostral displacement of the palatopharyngeal arch in a case of fourth branchial arch defect. The palatopharyngeal tissue is covering the apical portions of the corniculate processes, and both arytenoid cartilages show very limited abduction.

Collapse of the Apex of the Corniculate Process

Collapse of the apex of the corniculate process of the arytenoid cartilage under the contralateral corniculate process has been reported as a finding on high-speed treadmill videoendoscopy[97,98] and during resting endoscopy after swallowing or nasal occlusion.[98] Initially a high prevalance of the left corniculate process being involved led to the suggestion that muscle atrophy or dysfunction of the transverse arytenoideus muscle, as a component of RLN, could be the cause of this condition. However postmortem studies have not found any pathology in these muscles but rather a significant widening of the transverse arytenoid ligament that normally keeps the tips of the corniculate cartilages in close apposition. Currently, no reported treatment is available for this condition, and further postmortem studies will be necessary to elucidate the anatomic pathology involved.

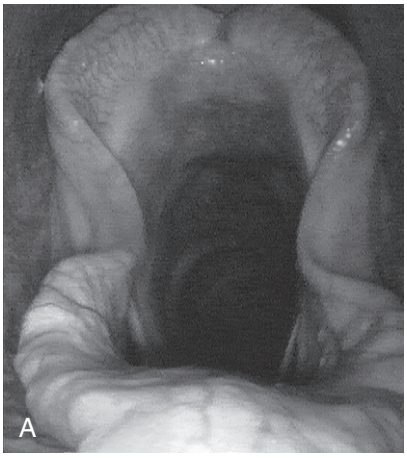
AXIAL DEVIATION OF THE ARYEPIGLOTTIC FOLDS

Diagnosis

Axial deviation of the aryepiglottic folds occurs during maximal exertion and causes dynamic partial upper respiratory tract obstruction. A diagnosis cannot be made during resting endoscopic evaluation but requires videoendoscopic examination during high-speed exercise (Figure 45-11, *A*). In a study of 15 affected horses, 80% were racing Thoroughbreds, 13% were racing Standardbreds, and 7% were racing Arabians. Abnormal respiratory tract noise and exercise intolerance were the most common clinical manifestations.[99] The condition is almost always bilateral, but unilateral involvement, if present, has been reported to be right-sided.[100]

Treatment

Transendoscopic laser surgery using an Nd:YAG or a diode laser with a 600-µm sculpted tip fiber in contact fashion can be performed on the conscious, standing horse or with the horse anesthetized. The tissue that is collapsing into the airway during exercise is grasped and placed under tension with 600-mm bronchoesophagoscopic grasping forceps, and an approximately 2 cm-wide triangle is excised transendoscopically.[99,101] Alternatively, this surgery can be performed through a laryngotomy in the anesthetized horse. Allis tissue forceps can be used to grasp the tissue, and Metzenbaum scissors are used to trim the redundant tissue (see Figure 45-11, *B*). No suturing is necessary.



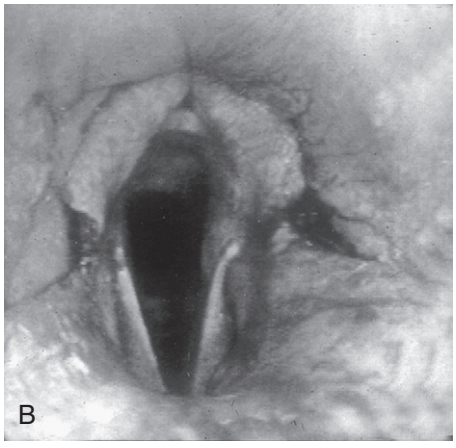


Figure 45-11. A, Videoendoscopic appearance of axial deviation of the membranous portions of the aryepiglottic folds during high-speed treadmill exercise; **B,** Immediate postoperative endoscopic appearance of the membranous portions of the aryepiglottic fold after excision through a laryngotomy. (**A,** Courtesy M. Weishaupt, Zurich.)

ARYTENOID CHONDROPATHY

Definition and Etiopathogenesis

Disease of one or both arytenoid cartilages resulting in abnormal enlargement is referred to as *arytenoid chondropathy.* Pathologic changes identified in affected cartilages include chondritis, chondrosis, chondroma formation, dysplasia, necrosis, perichondritis, granulating laryngitis, and abscessation.[102] The precise etiopathogenesis has not been determined, but it has been proposed that mucosal injury is followed by ascending infection and subsequent inflammation of the underlying cartilage[103-109] This can result in superficial chondritis with formation of granulation tissue and a fistulous tract, or if deeper infection occurs, the development of an abscess. Arytenoid cartilage mucosal injury could have multiple and coexisting causes. Respiratory tract infection is likely very significant and can cause mucositis and mucosal injury. Similarly, trauma-induced mucosal injury from endoscopic examination, from previous laryngeal surgery, concussion from the opposing cartilage, or from inhaled or swallowed foreign objects can occur. Inadvertent trauma of the arytenoid cartilages by nasogastric intubation can result in arytenoid inflammation, but it is unlikely to cause mucosal ulceration leading to chondropathy.[105] A hereditary component has also been suggested in Thoroughbreds.[109] The inflammatory processes involve not only the laryngeal mucosal surface and the arytenoid cartilage(s) but also periarytenoid tissues and dorsal muscular structures[102,110,111] The end result is usually fibrous tissue lamination, necrosis, deposition of poor-quality cartilage matrix, and mineralization of the laminar portion of primarily the body of the arytenoid cartilage.[102,112] Frequently, the affected cartilage has reduced mobility. Partial or complete failure of abduction is caused by the laterally positioned thyroid cartilage physically restricting movement, and/or impaired function of the cricoartyenoid joint because of extension of the inflammatory process to affect the joint or the dorsal cricoarytenoid muscle. The net outcome is a reduced rima glottidis aperture and airflow compromise.[103]

History and Clinical Signs

Arytenoid chondropathy can be acute, but it is more commonly chronic and affects all breeds and ages of horses, causing respiratory obstruction and exercise intolerance. Because of the different respiratory demands required by working and nonworking horses, the clinical presentations are different for each.

In working horses, typically young racing horses, signs are not detected at rest but occur during strenuous exercise, where even small changes in the cross-sectional area of the rima glottidis caused by the chondropathy manifest as respiratory stridor and exercise intolerance.[110,113] The disease can be asymptomatic, but generally there is a slow progression of clinical signs. It is important to note that the history for racehorses with the disease, particularly where cartilage enlargment is not severe, is very similar to that of RLN. Therefore, a careful clinical and endoscopic evaluation is required to differentiate the two conditions.[102,114] In contrast, because of the reduced respiratory demand in nonworking horses, the effects of arytenoid chondropathy are not recognized until there is marked airway narrowing and chronic disease is present. Therefore, these horses commonly present with obvious respiratory noise or dyspnea during mild exercise or at rest.[110] Typically, affected horses are older and have an insidious onset of clinical signs with gradual worsening over months to years. However, in some horses with advanced unilateral or bilateral chronic chondropathy, life-threatening respiratory obstruction occurs, necessitating emergency tracheotomy. Acute inflammatory chondropathy results from generalized laryngitis (perichondral submucosal edema), causing a markedly reduced diameter of the rima glottidis and therefore stridor or dyspnea at rest. Acute chondropathy is more common in older nonworking horses, where clinical signs can be rapid in onset and progress quickly, and depression, fever, and leuckocytosis may be present.[110] In these horses, this acute form of the disease may be an intermittent or severe manifestation of chronic inflammatory chondropathy. Acute disease has also been observed in yearling Thoroughbreds, associated with severe upper respiratory tract infections, including strangles, but respiratory noise at rest is uncommon in our experience.

Diagnosis

A diagnosis of arytenoid chondropathy can usually be made during endoscopic examination of the larynx. On initial observation, RLN might be suspected because the arytenoid cartilage is in an adducted position and shows little movement. However, the affected cartilage is usually mishapen or distorted (Figure

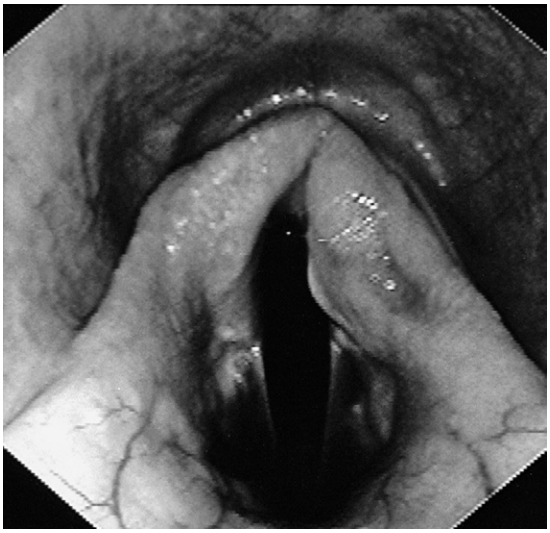


Figure 45-12. Videoendoscopic appearance of the rima glottis of a horse with arytenoid chondritis, showing distortion of the left arytenoid cartilage. The corniculate cartilage is in an abnormally adducted position, often making this condition look like recurrent laryngeal neuropathy or hemiparesis early in the disease process.

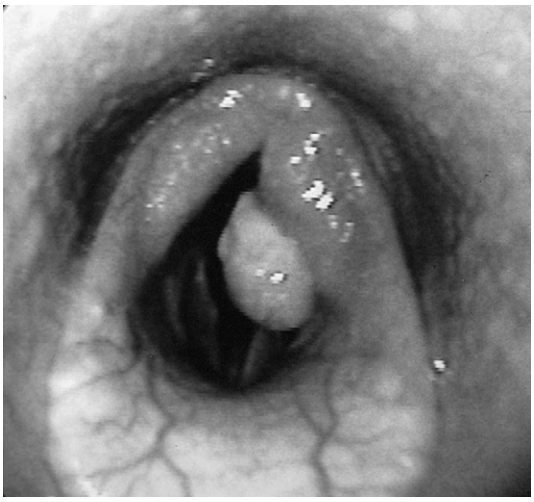


Figure 45-13. Endoscopic appearance of arytenoid chondropathy, showing incomplete abduction of the left arytenoid cartilage and granulation tissue on the medial surface of the body of the arytenoid at the entrance to a fistula.

45-12). One of the endoscopic hallmarks of arytenoid chondritis is the presence of intraluminal projections of granulation tissue (Figure 45-13). A fistula or sinus tract in the center of the granulation tissue communicating with the body of the arytenoid cartilage is frequently present. A contact ulcer or projection of granulation tissue is often present on the opposing cartilage ("kissing lesion"). The palatopharyngeal arch on the affected side is often more prominent (see Figure 45-12). Because significant RLN of the right side of the larynx is rare, arytenoid chondropathy should always be a primary consideration in the differential when impaired abductor function of the right arytenoid cartilage is noted endoscopically. In acute chondropathy, the predominant endoscopic feature is mucosal and submucosal perichondrial edema and perilaryngeal swelling with intense hyperemia. The rima glottidis diameter can be markedly reduced, often to a slit-like opening. The degree of underlying cartilage pathology may be highly variable and difficult to determine until medical therapy resolves the edema.[110]

Careful palpation of the larynx with attention to comparison of the percutaneous prominence of the muscular processes on either side of the larynx is important. In cases of significant RLN, the left muscular process is more obvious. In contrast, thickening around the muscular process can be palpated in cases of arytenoid chondropathy and might be the only diagnostic finding. In general, the larynx may feel less resiliant to digital palpation, and firm manual pressure may cause stidor or dsypnea.[110,115] Ultrasonography may be useful in further defining the disease. It can help in detecting abaxial arytenoid cartilage lesions (including abscessation) and in determining if the disease is confined to the arytenoid cartilages or extends into the periarytenoid tissues.[26] Radiographic features of arytenoid chondropathy include abnormal patterns of cartilage mineralization, increased density of the arytenoid cartilages, abnormal contour or size of the corniculate process, obliteration of the laryngeal ventricle, and laryngeal masses.[110] Some mineralization in the larynx occurs with aging in horses, particularly of the thyroid and subsequently the dorsal arch of the cricoid cartilage. Extensive mineralization of the larynx is a poor prognostic sign for successful surgical treatment.[102,110]

Treatment

Management of arytenoid chondropathy depends on the stage of the disease (acute or chronic), the degree of airway dysfunction, and whether athletic activity is required. In the acute form, horses may be presented in respiratory distress (stertor) with laryngeal edema. Medical therapy with broad-spectrum antibiotics, NSAIDs, and/or steroids and pharyngeal throat sprays often resolves the condition, but occasionally an emergency tracheotomy is required. A 7- to 10-day course of treatment is prescribed with endoscopic review. In some animals, the chondropathy regresses and arytenoid cartilage function returns to normal or near normal. In other animals, chronic chondropathy ensues, but if airway obstruction is not severe, it is possible for some athletic animals to continue to compete. Because arytenoid chondropathy is frequently progressive, repeated endoscopic monitoring is recommended, especially if respiratory noise increases.

In cases where rima glottidis size is reduced because of granulation tissue or cartilage abscessation but arytenoid function is acceptable, local excision and drainage using laser therapy or local curretage either in the standing horse or under general anesthesia via larygotomy can be attempted (see later). However, if cartilage function is significantly imparied and/or the laminar portion of the arytenoid cartilage is thickened sufficiently (chronic form), surgical removal of the affected arytenoid cartilage is usually necessary. This condition is most often unilateral and therefore treated with unilateral arytenoidectomy. If the condition is bilateral, unilateral arytenoidectomy of the most affected cartilage may be sufficient. Other surgeons believe bilateral arytenoidectomy is inevitable and prefer a single-stage procedure.[110] Bilateral arytenoidectomy is considered a salvage procedure. Permanent tracheostomy is a valid procedure for bilateral disease in nonathletic animals and has been used with good results in Thoroughbred broodmares with the disease (see Chapter 47).[110]

Arytenoidectomy

Prior to surgery, preparation of the patient should include perioperative broad-spectrum antimicrobial therapy as well as anti-inflammatory medication. The skin should be clipped and prepared for a laryngotomy approach (see earlier), as should the mid-neck region in preparation for placement of an endotracheal tube. If the horse is dyspneic or has such severe disease that tracheal intubation would be difficult or impossible, the tube can be placed before induction of general anesthesia using local anesthetic under sedation. Otherwise a mid-cervcial tracheotomy (see Chapter 47) is performed with the horse positioned in dorsal recumbency. It is important that the tracheotomy be made sufficiently distal so that during recovery the tracheotomy site does not become obstructed. It is helpful to flex the poll toward the chest during this procedure so that the skin and soft tissues of the ventral neck are in a similar position and tension to those in the standing horse.

Two methods of arytenoidectomy have been described: subtotal, in which the muscular process and rim of the corniculate process are left intact; and partial, in which only the muscular process is left intact (Figure 45-14).[116,117] The merits of subtotal and partial arytenoidectomy have been evaluated subjectively on the basis of the assessment of performance and postoperative complications in a series of retrospective studies.[102,116,118] However, in a quantitative evaluation in 1990, it was demonstrated that subtotal arytenoidectomy failed to improve upper airway function in horses exercising on a high-speed treadmill with surgically induced left laryngeal hemiplegia.[119] The effect of partial arytenoidectomy on upper airway function was evaluated by tidal breathing flow-volume analysis, measurement of upper airway impedance, and videoendoscopic examination of the larynx in strenuously exercising horses with surgically induced left laryngeal hemiplegia in 1994.[120] This study showed that partial arytenoidectomy improved upper airway function in exercising horses with surgically induced left laryngeal hemiplegia and was superior to subtotal arytenoidectomy as a treatment for both failed laryngoplasty and arytenoid chondropathy.

A number of methods and modifications for performing partial arytenoidectomy have been reported.[47,117,118,120-123] Controversy exists over whether the mucosa on the axial surface of the arytenoid cartilage should be retained and sutured closed for primary wound healing or whether complete resection of the cartilage with attached mucosa gives the best results.[48,123] We recommend combining a partial arytenoidectomy with a ventriculocordectomy, removing all devitalized and redundant mucosa and suturing the incision so that all excised mucosal edges are apposed and the luminal surface is as smooth as possible. Any redundant tissue left in the airway creates turbulence and noise and may interfere with airway function. This tissue should be removed at 30 days after surgery via laser ablation before cicatrix formation and mineralization make it impossible. Ventral drainage is achieved by leaving the ventriculocordectomy site open, and the most rostral and abaxial mucosa covering the corniculate cartilage should be retained and not sutured under excessive caudal tension because obliteration of the pyriform recess is likely to result in problems with tracheal aspiration and coughing postoperatively.[118,121,122] This is supported by the observation that injection of the rostral mucosa of the repair (with Teflon) in cases of chronic aspiration and coughing has temporarily resolved the problem.[124] Tidal breathing flow-volume loop, respiratory mechanics, and blood gas analysis show that partial arytenoidectomy does not completely restore the upper airway to normal but that the procedure is a viable treatment option for arytenoid cartilage malfunction in the horse.[47,120]

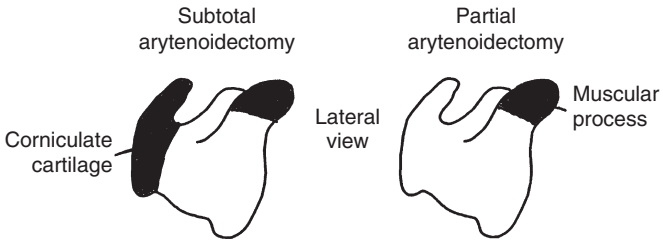


Figure 45-14. Schematic representation of a lateral view of the arytenoid cartilage, demonstrating the portions of the cartilage that are removed *(unshaded)* during subtotal and partial arytenoidectomy.

PARTIAL ARYTENOIDECTOMY

Horses are placed in dorsal recumbency under general anesthesia with an endotracheal tube placed through a tracheotomy incision (Figure 45-15). An endoscope placed via the nostril with the tip positioned in the dorsal pharynx is useful for illumination of the surgical site, as is a headlamp. Suction is critical for removing blood in the surgical field, and gauze soaked in epinephrine solution (1 : 10 000) is helpful to reduce hemorrhage. A ventral laryngotomy is performed (see earlier) and self-retaining retractors are used to allow access to the lumen of the larynx. For increased exposure, the cricoid cartilage can be split longitudinally, as can the body of the thyroid, if needed. On entry to the larynx, granulomatous masses are often observed on the affected arytenoid cartilage (Figure 45-16). A submucosal injection of epinephrine helps to elevate the mucosal membrane and to control bleeding. Using a No. 15 Bard Parker scalpel, a dorsally based U-shaped mucosal flap is made overlying the arytenoid cartilage (Figure 45-17). The mucosal incision starts at the apex of the corniculate cartilage 2 mm caudal to the rostral surface and extends ventrad then caudad and finally dorsad around the outline of the arytenoid cartilage. This forms

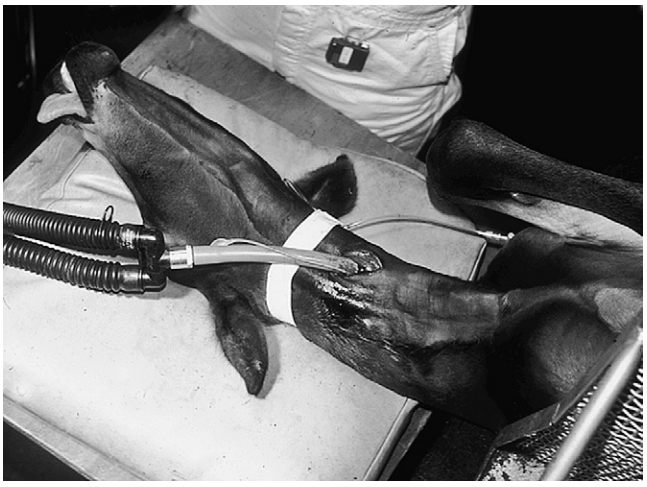


Figure 45-15. Preparation of a foal for arytenoidectomy shows an endotracheal tube placed through a tracheotomy incision located distal to the larynx. The tube can be placed in the standing patient just prior to general anestheia if required.

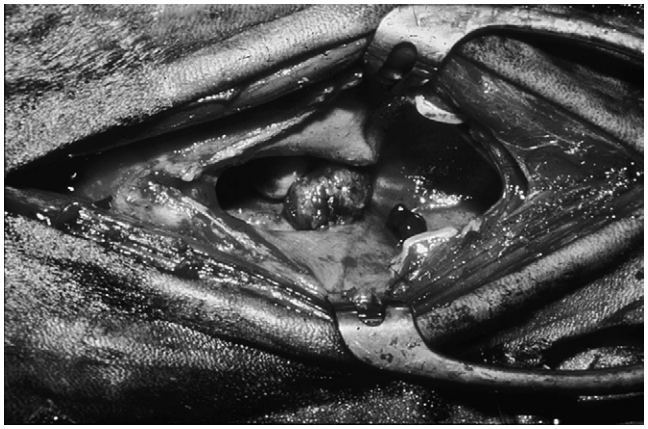


Figure 45-16. A laryngotomy allows full vision of the granulomatous masses observed endoscopically on the arytenoid cartilage with chondropathy. Self-retaining retractors provide good access for arytenoidectomy.

a three-sided U-shaped incision. The mucosa on the luminal side of the arytenoid cartilage is then elevated starting caudad and working rostrad over the corniculate portion. A scalpel is used to sharply undermine the mucosal tissue initially, and then the dissection is completed with a periosteal elevator, leaving the dorsal mucosa attached. Next, the lateral surface of the arytenoid carilage is freed from its soft tissue attachments using blunt dissection, staying close to the cartilage, and the remaining mucosa over the rostral and lateral aspect of the corniculate cartilage is separated using sharp and blunt disection. The arytenoid cartilage is elevated rostrally using Allis tissue forceps or a towel clamp, and the muscular process is transected using heavy Mayo scissors or a scalpel. Any diseased or irregular cartilage attached to the muscular process dorsally is removed with rongeurs. A ventriculocordectomy is subsequently performed by incising the mucosa around the oriface of the ventricle and removing the entire saccule followed by resection of the cord. During closure, the surgeon should take care to smooth the airway as much as possible by removing all redundant tissue. The rostral and lateral corniculate mucosa is retracted slightly caudad and sutured with minimal tension to the leading rostral edge of the mucosal flap using interupted or continuous No. 2-0 absorbable suture. Obliteration of the pyriform recess should be avoided by not pulling the leading edge of this tissue caudal to the palatopharyngeal arch. This can be confirmed by an assistant examining the rima glottidis via videoenoscopy intraoperatively. The remainder of the mucosal flap is sutured to the adjacent larygeal mucosa similarly but leaving the ventral part of the incision open for drainage. The ventriculocordectomy incisions are also not sutured. Large mucosal defects are left unsutured to heal by second intention, and the corniculate mucosa is sutured laterally to the remaining soft tissues. If the cricoid cartilage was divided, the two sides are brought into close apposition by suturing the soft tissues immediately ventral to the cartilage ends using No. 0 absorbable suture in an interupted or cruciate pattern. The sutures should not be placed through the cartilage to prevent chondroma formation. The laryngotomy is left unsutured because of the possibility of laryngeal edema and subsequent airway obstruction. A tracheostomy tube is secured in place to ensure a patent airway during recovery.

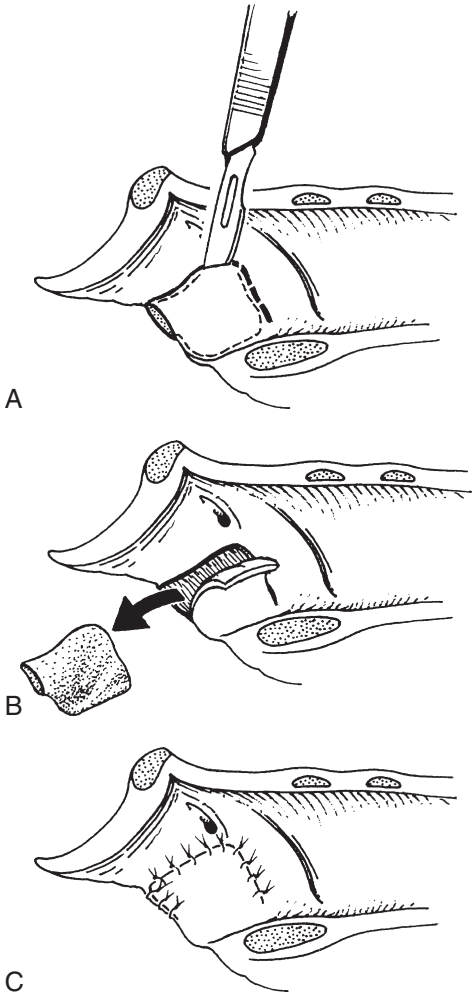


Figure 45-17. Schematic illustration of partial arytenoidectomy technique. **A,** Beginning at the apex of the corniculate cartilage and 2 mm caudal to the rostral surface, a mucosal incision is made that extends ventrad then caudad and finally dorsad around the outline of the arytenoid cartilage *(dotted line)*. **B,** The mucosa is firstly elevated from the arytenoid cartilage using a scalpel then a periosteal elevator, which exposes the laminar portion of this cartilage. The cartilage is then freed from its deep attachments using blunt disecction, staying close to the cartilage. The muscular process is transected with heavy scissors or a scalpel and the dissected cartilage is removed. Then the ventricle and vocal fold are removed. **C,** All remaining mucosal incisions are apposed with simple continuous or interrupted sutures of 2-0 polydioxanone, taking care to remove any redundant tissue during closure. The ventral part of the incision is not sutured to allow postoperative drainage.

Antimicrobial agents, such as penicillin and gentamicin, as well as anti-inflammatory drugs, such as phenylbutazone, are administered before surgery and continued for at least 3 days after surgery. Food is withheld for 24 hours, at which time the larynx is examined endoscopically and airway patency is assessed by plugging the tracheostomy tube together with the laryngotomy wound and listening for respiratory stridor. If there is little postoperative edema or swelling and the airway is patent, the tracheostomy tube is removed; otherwise the tube is removed and cleaned twice daily before eventual removal. The horse is reintroduced to food 24 hours after surgery. At that time, wet hay only should be fed until the horse is able to swallow without coughing, followed by the reintroduction of

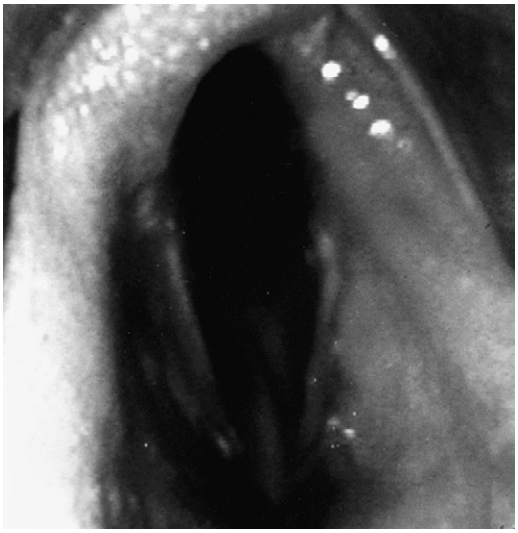


Figure 45-18. Endoscopic view of the rima glottidis of a horse 4 weeks after left partial arytenoidectomy and ventriculocordectomy. Note that all redundant tissue has been removed from this airway.

dry feedstuffs. The horse is stall-rested for 4 to 6 weeks prior to complete healing, which usually occurs within 8 to 10 weeks. Postoperative endoscopic evaluations should reveal a hemilarynx devoid of vocal folds, ventricle, and corniculate cartilage (Figure 45-18).

A modification of the earlier technique has been described experimentally and has been used clinically.[47] A flap of mucosa on the luminal or medial side of the arytenoid cartilage is not elevated and retained for suturing; instead, the arytenoid cartilage is removed in two pieces with accompanying mucosa attached, one with the body of the arytenoid and the other with the corniculate process. The rostral and lateral mucosa overlying the corniculate process is retained and together with the contiguous aryepiglottic fold is drawn caudad over the defect left by artenoid cartilage removal and sutured with 3-0 absorbable suture material in a continuous pattern, leaving only the ventral 5 mm of the repair open to drain at the site of the ventriculocordectomy. The proposed benefits of this modification include an easier dissecction, shorter surgery time, and a reduction in the amount of soft tissue lining the airway, which could be subjected to dynamic collapse during fast exercise.[125] Experimental and clinical studies did not show an increased incidence of postoperative dysphagia or coughing using this modification. Improvements in airway mechanics similar to prosthetic laryngoplasty are achieved, and in clinical cases 62% of Thoroughbred horses raced successfully after the treatment of unilateral arytenoid chondropathy.[126]

Complications

Dyspnea and dysphagia are the most important complications. Dyspnea occurs as a result of edema or hemorrhage trapped under the sutured mucosal flap. Therefore, the tracheostomy tube should be left in place until airway patency is confirmed. Antimicrobial and anti-inflammatory medications are continued until laryngeal swelling and associated dyspnea are resolved, and then the tracheostomy tube is removed. Dysphagia with regurgitation of food and water through the nares has been reported in up to 36% of horses after arytenoidectomy.[118] However, significant dysphagia was not encountered in more recent reports.[47,48,120,123,126] It has been proposed that atraumatic transection of the body of the arytenoid from the muscular process and of the soft tissue attachments on the lateral surface of the arytenoid cartilage may reduce problems with deglutition after surgery.[48] Maintaining sufficient rostral mucosa especially of the ventral part of the repair is also suggested to reduce aspiration.[127] Coughing during eating is not uncommon and may continue for some months. Transection of the interarytenoid ligament in the dorsal part of the larynx is to be avoided as this has been associated with significant coughing.[118] Owners are encouraged to feed the animal on the ground, and pasture feeding may be beneficial. Although current techniques have significantly reduced morbidity associated with dysphagia and coughing, it is likely that many horses aspirate feed material and respiratory secretions into the distal part of the respiratory tract to some degree. One study showed horses with induced RLN subjected to a partial arytenoidectomy had significant increases in neutrophils and macrophages containing intracellular bacteria as well as an increase in the number of lymphocytes in transbronchial fluid aspirates taken 30 minutes after strenuous exercise.[47]

The formation of intralaryngeal granulation tissue was identified 1 month after surgery in 17% of horses treated with partial arytenoidecotomy,[48] presumably because of wound dehiscence (typically at the dorsal aspect of the rostral part of the incision). The granulation tissue and any excess mucosa should be excised using a transendoscopic laser at this time. Advanced larygeal cartilage mineralization rendering horses useless for work was reported in 5% of horses treated with a partial arytenoidectomy.[102]

Mucosal Disease and Granulomas of the Arytenoid Cartilages

Mucosal disease of the arytenoid cartilages in young Thoroughbred horses in Australia and New Zealand has been described.[105,108] Bilateral kissing lesions are found on or near the vocal processes just proximal to the attachment of the vocal cords (Figure 45-19). The etiology is uncertain; therefore, the condition has been termed *idiopathic mucosal lesions.*[105] These lesions manifest as small erosions or ulcers or, alternatively, raised areas of epithelial injury. They are often hyperemic and can have small, slightly purulent centers. The incidence of mucosal lesions in two surveys of Thoroughbred yearlings presented to public auction sales (5629 horses) was 0.6% and the incidence of arytenoid chondritis was 0.21%.[105,108] In a population of 774 racing Thoroughbreds, 2.4% had mucosal lesions.[128] These lesions do not affect subsequent racing performance, and many heal uneventfully with or without medical therapy.[105,128] However, treatment is recommended, because in one report in 10% of affected horses lesions progressed to become granulomas and 5% to arytenoid chondritis.[108] Broad-spectrum antibiotics, NSAIDs, and pharyngeal throat washes are recommended for 10 to 14 days.

Occasionally, particularly in Thoroughbred and Standardbred racehorses, a mass projecting from the medial surface of the arytenoid cartilage at its junction with the corniculate cartilage just dorsal to the insertion of the vocal cord is seen on endoscopic examination (see Figure 45-13). These lesions are commonly referred to as "chondromas"; however, histologic

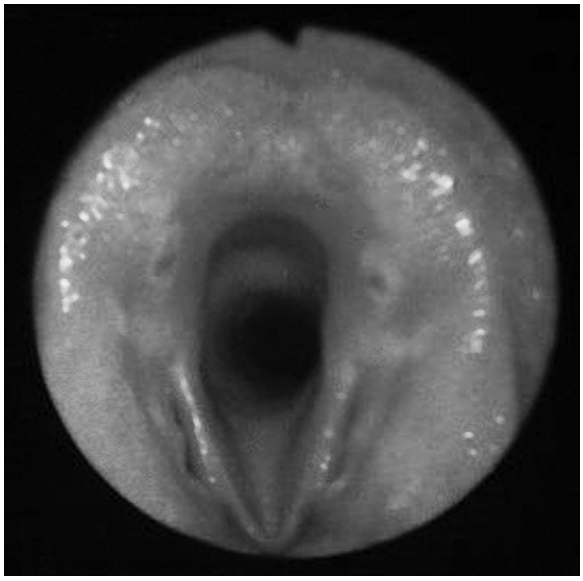


Figure 45-19. Endoscopic appearance of bilateral mucosal ulcerations on the axial surface of the arytenoid cartilages.

examination of the excised tissue invariably reveals granulation tissue.[129] An ulcerated mucosal defect is often seen on the medial surface of the opposing arytenoid where the granulation tissue rubs during laryngeal adduction. In addition, abscessation of some affected cartilages may be present. If abductor function of the affected cartilage is normal or only slightly reduced, the mass should be removed to improve airflow, prevent advancement of chondropathy, and protect the opposite cartilage from contact injury. Excision of the granulation tissue does not alter any preexisting underlying cartilage abnormalities, but it is carried out in an attempt to allow the mucosal defect to epithelialize and to prevent contact injury to the opposing arytenoid. This technique may be temporarily successful when combined with antibiotic and anti-inflammatory therapy in some performance horses and many sedentary patients. However, when dealing with an equine athlete, arytenoidectomy is usually required eventually. Options for treatment include laryngotomy followed by scalpel or laser excision and focal curretage under general anesthesia or transendoscopic laser excision in the standing sedated patient. Drainage of purulent exudate can be achieved at the same time.[130]

Treatment

The horse is sedated and the lesion and surrounding tissue are transendoscopically topically anesthetized. Using an Nd:YAG laser, a 2.2 mm-outer-diameter laser fiber with a chisel probe attached or an 800 µm sculpted fiber is introduced through the endoscope biopsy channel. A diode laser with a sculpted fiber can also be used. When using the chisel probe, the bevelled surface is oriented parallel to the medial mucosal surface of the arytenoid. Beginning at the perimeter of the mass, a dissection plane is established between the granulation tissue and the underlying cartilage. The mass is completely freed up except for small remaining tags of attachment, and it is retrieved with 600-mm–long bronchoesophagoscopic grasping forceps. Any remaining small tags of loose tissue are subsequently débrided with the laser and removed with the forceps. Usually, a crater defect has been created with smooth mucosal margins that are firmly attached to underlying cartilage.

Gaining access to the axial part of the cartilage to completely débride the lesion can be difficult. Placing the endoscope up the nostril opposite the affected cartilage can help. A more direct approach can be made by introducing a laser fiber through a 5 mm × 6.5 mm trocar inserted through a stab incision through the cricothyroid membrane.[130] The lesion can also be explored by passing a 2-mm flexible stainless steel probe through the trocar. A small curette passed through the trocar can also be used to manually débride deeper parts of the tract. In some horses with abscessation of the underlying cartilage, drainage may be achieved with this method.

Exercise is restricted until the defect heals. Systemic NSAIDs and a pharyngeal spray containing anti-inflammatory and antimicrobial medications may be useful in reducing inflammation. Regrowth of the granulation tissue can occur in 16% of horses requiring a second procedure,[129] but in some cases the regrowth regresses with medical therapy and time.

Prognosis

The prognosis following treatment for arytenoid chondropathy depends on the nature and extent of the disease and the respiratory demands placed on the patient.[48,102,110,123,126] If treatment is instituted early, the condition is unilateral, and arytenoid function is minimally affected, medical management can halt progression, and in one study palliative treatment allowed some horses to race for 1 to 2 years.[102] The likelihood of Thoroughbred horses with idiopathic arytenoid mucosal lesions racing more than three times is not different from unaffected control animals.[105] Excision of intralaryngeal granulation tissue in nonracehorses has an excellent prognosis. A group of five racehorses with only slight cartilage thickening and no other larygneal pathology, all returned to racing three or more times with only one having reduced performance after treatment. However, if moderate cartilage thickening is present or concurrent laryngeal pathology exists, only 50% of treated horses returned to racing and usually at a lower level of performance.[129]

Partial arytenoidectomy is very effective in restoring rima glottidis diameter, but prognosis depends on the respiratory demands for exercise. Six of eight horses (75%) with occupations other than racing were useful after partial arytenoidectomy was performed for unilateral chondropathy (5), bilateral chondropathy (1), or failed laryngoplasty (2).[102] In general, the prognosis in nonworking horses treated with partial arytenoidectomy is favorable but is reduced in cases of advanced unilateral chondropathy accompanied by complicating lesions, such as contralateral laryngeal paralysis, periarytenoid inflammation, and pharyngeal cicatrices. Even horses suffering from bilateral arytenoidectomy may have a better prognosis for survival compared to those with advanced unilateral chondropathy and accompanying lesions. In these horses, a permanent tracheotomy might be a better treatment option.[110]

The prognosis for athletic activity after partial arytenoidectomy in working horses, primarily racing Thoroughbreds, has been reported to be between 45% and 80%.[48,102,123,126] Comparison between studies is difficult because of differences in sample size, inclusion criteria, surgical technique, and measures of success.

However, in racing Thoroughbreds it is likely that partial arytenoidectomy for the treatment of arytenoid chondropathy will allow two thirds or more of treated horses to train, race, and earn money. A shortened athletic career is likely, and although there is some evidence that shows a reduced performance after surgery, other studies show similar postoperative earnings per start and similar earnings compared to age- and sex-matched controls. Bilateral partial arytenoidectomy for bilateral arytenoid chondropathy is a salvage procedure and carries a poor prognosis for continued racing (22%).[102] In addition, it is clear that horses treated with partial arytenoidectomy for arytenoid chondropathy still have some limitation to airflow when exercising at racing speed.[47,120] Dynamic collapse of unsupported soft tissue on the left side of the larynx following partial arytenoidecotomy is the likely cause.

Finally, different techniques or modifications for partial arytenoidectomy have been described but not critically compared. Partial arytenoidectomy with or without primary mucosal closure can be performed. Proposed benefits of primary mucosal closure include a faster healing time, reduction in postoperative excessive granulation tissue production, and an ability to streamline or smooth the luminal surface of the wound.[47,48,102,120-122] However, in advanced or extensive chondropathy cases only partial mucosal closure may be possible, and suturing diseased mucosa under tension is not advised. Some surgeons elect to perform all partial arytenoidectomies without mucosal closure citing quicker surgery time, prevention of hematoma formation and no suture dehiscence problems.[123] Healing time in one study of unsutured partial arytenoidectomies was only 8 weeks,[123] similar to that reported for partial arytenoidecotomy using primary mucosal closure. However, the number of horses racing three or more times after surgery was only 55% compared to 63% of horses treated with primary mucosal closure in another report.[48]

EPIGLOTTIS

The epiglottis is an isosceles triangle–shaped structure with the tip projecting rostrad from the base of the larynx. During normal breathing, the epiglottis is positioned above the soft palate with the caudal free margin of the palate interfacing with the epiglottic base. The epiglottis is covered on its dorsal surface by a tightly adherent mucous membrane, whereas the ventral surface has a loosely attached, somewhat elastic mucosa. This loosely folded subepiglottic mucosa allows unrestricted dorsal and caudal movement of the epiglottis during swallowing. The normal epiglottis has a broad base and is narrow at the tip with a convex dorsal surface projecting slightly above but parallel to the soft palate. The lateral margins of the epiglottis have a crenate or scalloped appearance. A normal vascular pattern is present on the dorsal surface with a main vessel on either side that courses from the base toward the tip, parallel to and approximately 8 to 10 mm in from the margin (see Figure 45-1). From these two major vessels, small perpendicular branches are seen coursing toward the epiglottic margins.

Epiglottic Entrapment

The aryepiglottic folds are thickened bands of mucous membrane that attach along the entire free edge of the epiglottis, blending in with the tightly adherent dorsal epiglottic mucosa and the mucosa covering the corniculate process of each arytenoid cartilage.[131] The loose subepiglottic mucosa compresses in an "accordion" fashion when the epiglottis is in the normal resting position. Epiglottic entrapment occurs when the loose subepiglottic mucous membrane becomes positioned above the dorsal epiglottic surface and then appears continuous with the aryepiglottic folds. Epiglottic entrapment is a common cause of abnormal respiratory noise and often, but not always, exercise intolerance, particularly in Thoroughbred and Standardbred racehorses.[131-137] Other, less frequent complaints associated with epiglottic entrapment are coughing and nasal exudate, sometimes after drinking. Epiglottic entrapment is occasionally an incidental endoscopic finding.

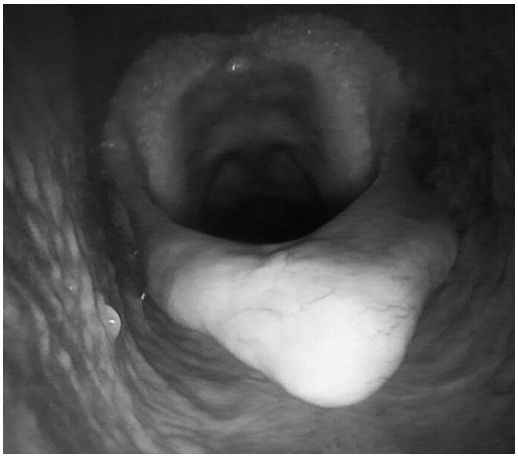


Figure 45-20. Endoscopic appearance of epiglottic entrapment showing a thin entrapping membrane where the outline of the epiglottis is still visible. (Courtesy M. Weishaupt, Zurich.)

Diagnosis

On endoscopic examination, the general shape of the epiglottis is still visible, and in the majority of horses it is positioned above the soft palate (Figure 45-20). However as the epiglottis is covered with a fold of mucosa, the crenate margins of the epiglottis and the dorsal epiglottic vessels are obscured. The entrapping membrane may be intermittently relieved when the horse swallows, but in most cases it is persistent (Figure 45-21). The appearance of the entrapping membrane endoscopically varies and can be classified as being thin or thick, narrow (less than half the length of the epiglottis) or wide (greater than half the length of the epiglottis), and ulcerated or nonulcerated (Figure 45-22). Mucosal ulceration can vary from minimal to extensive, with variable amounts of exposed granulation tissue and pale fibrous cicatrix formation around the perimeter of the ulcer (Figure 45-23). Most epiglottic entrapments (97%) are persistent, most (98%) are thick, most (97%) are wide, and 45% are ulcerated.[132]

Horses with epiglottic entrapment may also suffer from varying degrees of epiglottic hypoplasia and/or flaccidity. On endoscopic examination, 31% to 36% of horses with entrapment appear to have epiglottic hypoplasia.[132,136,137] A lateral laryngeal radiograph allows accurate measurement of thyroepiglottic length and information about epiglottic thickness and contour. In one study of 9 Thoroughbreds with epiglottic entrapment, the thyroepiglottic length was 6.59 ± 0.33 cm,[138] and in another study of 35 Thoroughbreds it was 7.28 ±

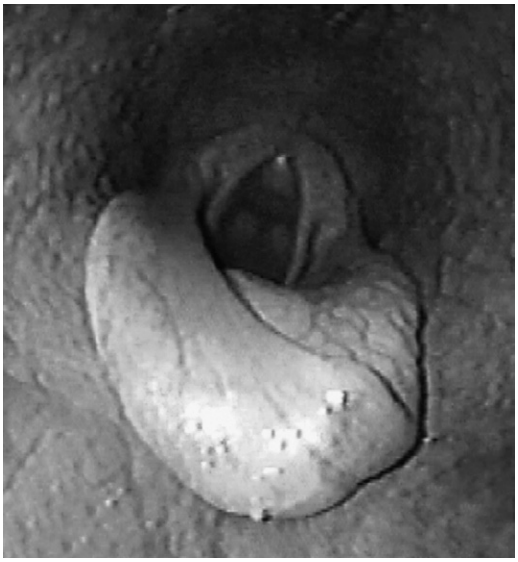


Figure 45-21. Endoscopic appearance of epiglottic entrapment where the aryepiglottic fold partially entraps the epiglottis and is intermittently relieved by swallowing. (Courtesy J.T. Robertson, Ohio.)

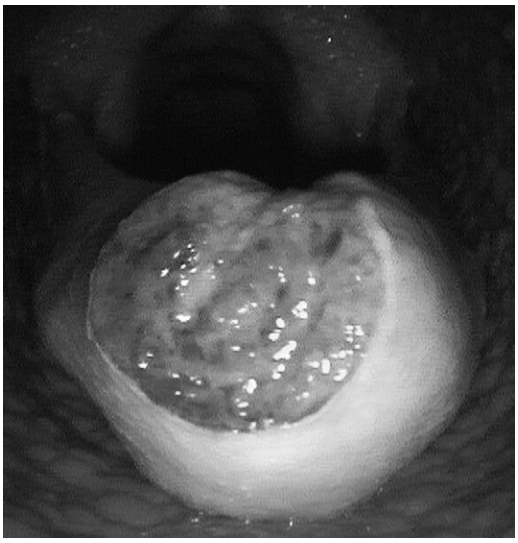


Figure 45-22. Ulcerated aryepiglottic fold entrapment in a Thoroughbred racehorse. (Courtesy M. Weishaupt, Zurich.)

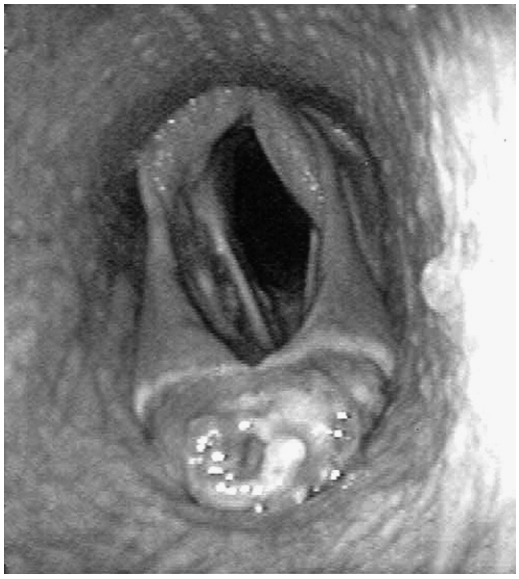


Figure 45-23. Endoscopic appearance of epiglottic entrapment with severe mucosal ulceration, showing exposed granulation tissue and pale fibrinous connective tissue proliferation around the perimeter of the ulcer.

0.67 cm.[139] These measurements were significantly smaller than those found in normal Thoroughbreds without entrapment, which in two studies was 8.76 ± 0.38 cm and 8.56 ± 0.29 cm.[1,139] The thyroepiglottic length in 44 Standardbreds with entrapment was 7.21 ± 0.62 cm, also significantly less than the length of 8.74 ± 0.38 cm found in normal Standardbreds.[139] Among Standardbred and Thoroughbred horses with epiglottic entrapment, those with apparent epiglottic hypoplasia had significantly shorter thyroepiglottic lengths than horses with entrapment and a normally shaped epiglottis.[139]

Treatment

Surgical correction of epiglottic entrapment has been described using transnasal endoscopically guided contact Nd:YAG or diode laser axial division, using transnasal or transoral axial division using a curved bistoury, using transendoscopic electrosurgical axial division, and by surgical excision through a laryngotomy or a pharyngotomy.[100,131-140] Conventional side-to-side excision of the aryepiglottic and subepiglottic folds performed through a laryngotomy commonly resulted in dorsal displacement of the soft palate.[131,137] Therefore, tissue-sparing techniques that preserve aryepiglottic fold mucosa and minimize scarring are preferred, especially with the knowledge that epiglottic hypoplasia is present in many horses with epiglottic entrapment. Axial midline division of the entrapping membrane is the technique of choice in most horses. This procedure allows the membrane to retract and heal in a normal subepiglottic position, often without removing any tissue. The membrane can be divided with an Nd:YAG, a holmium:YAG, or a diode laser; a curved bistoury transnasally or transorally; or electrocautery to achieve the same effect.

Laser surgery can be safely performed transendoscopically in the standing sedated horse on an outpatient basis with the entrapping membranes topically anesthetized. Using the Nd:YAG laser as an example, a 600-µm sculpted fiber can be used to incise the membrane from caudad to rostrad in contact fashion using approximately 12 W of power. Alternatively, a contact chisel probe attached to a 2.2-mm outer-diameter fiber can be used to divide the membranes from rostrad to caudad with 15 W of power (additional information on laser surgery can be found in Chapter 15). Using these two techniques, the re-entrapment rate in a series of more than 500 cases was approximately 4%, and between 10% and 15% of horses experienced dorsal displacement of the soft palate. Extreme care must be taken to avoid thermal trauma to the epiglottis. Using a transnasally placed bronchoesophageal grasping forceps to elevate the entrapping membrane away from the epiglottis can help avoid laser trauma to the epiglottis. To reduce the opportunity for re-entrapment, some surgeons also remove two small triangles of the mucosal fold from either side after the intial

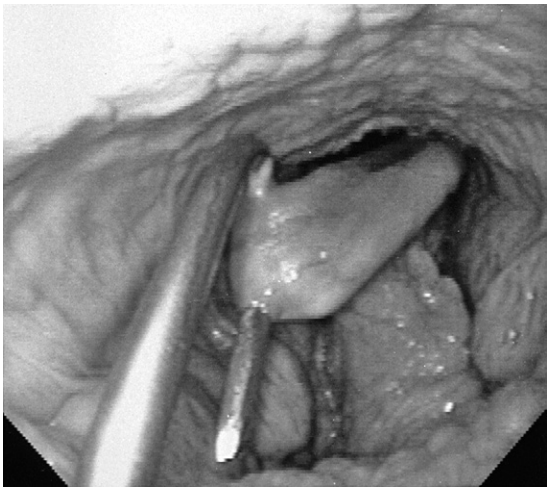


Figure 45-24. Endoscopic appearance of a curved bistoury axially dividing an entrapping membrane via an oral approach. Note the glossoepiglottic fold ventral to the epiglottis.

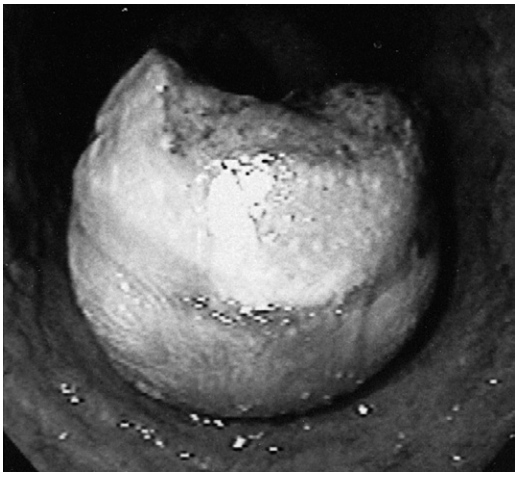


Figure 45-25. Endoscopic appearance of a thick, ulcerated epiglottic entrapment in a Thoroughbred racehorse. Surgical correction was achieved by excising the central third of the aryepiglottic fold through a laryngotomy.

axial split. The bronchoesophageal grasping forceps again are necessary for this maneuver.

With a curved bistoury introduced either transnasally in the standing horse or transorally in the anesthetized or standing horse[140-142] (Figure 45-24), the entrapping membranes can be incised on the midline from caudad to rostrad. The reported re-entrapment rate ranges from 5% to 15%.[133-136] Serious complications can occur after transnasal axial division using a curved bistoury on standing sedated horses, including partial division of the soft palate and lacerations of the epiglottis or pharynx. Therefore, this technique is not recommended unless a protected bistoury is used, which has been shown in a small number of horses to reduce this complication, but operator experience is necessary.[140]

Inadvertent incision of the soft palate can be avoided by performing the hook technique through the mouth with the horse anesthetized. The re-entrapment rate after transoral hook correction varies between 1%[135,136] and 10%, and up to 10% of horses experienced dorsal displacement of the soft palate.[134-136] Transendoscopic electrosurgical division has the highest complication rate of the axial division techniques (in a series of 5 horses, it resulted in a 40% re-entrapment rate[137]), and it has fallen into disuse.

Special Considerations

An excessively thickened, ulcerated, or fibrotic-appearing entrapping membrane is present in approximately 5% of horses with epiglottic entrapment, and often these horses have severe epiglottic hypoplasia (Figure 45-25). Horses with this endoscopic appearance are generally poor candidates for correction by axial division. Surgical excision of the central one third of these bulky membranes is recommended and can be performed via a laryngotomy, with the horse anesthetized and positioned in dorsal recumbency. Through the laryngotomy, the epiglottis is retroverted into view by placing traction on the aryepiglottic fold tissue on the lateral margin with sponge or Allis tissue forceps. Applying instruments directly onto the epiglottic cartilage for traction should be avoided. The aryepiglottic folds are placed under tension and stabilized in a fan shape with three evenly placed Allis tissue forceps (two lateral and one central) and the triangular shape of the epiglottis is identified by visual inspection and palpation. The outstretched aryepiglottic fold is incised on the midline with Metzenbaum scissors to within 2 to 3 mm of the epiglottic cartilage tip. The aryepiglottic fold is then cut for a length of about 1 to 1.5 cm parallel to each margin of the epiglottis. This cut angles out from each epiglottic edge, beginning adjacent to the cartilage and tapering to the free edge of the outstretched aryepiglottic fold. Two roughly triangular pieces of mucous membrane are excised out of the central portion of the aryepiglottic fold, debulking the membrane and, in most horses, preventing re-entrapment. The laryngotomy is left to heal by second intention, with routine wound cleaning provided twice daily. Exercise can be resumed after 4 weeks if healing progresses normally; however, 8 weeks is the preferred rest period. Re-entrapment can occur and is treated by a second excision.

Alternatively, the membrane can be debulked with the horse standing using transendoscopically guided laser excision via the nares. However, care must be taken to avoid thermal damage to the epiglottic cartilage because adhesions and indurated cicatrix often form between the entrapping membrane and the epiglottis, making their delineation difficult and often inducing complications. Owners should be warned that dorsal displacement of the soft palate may be a sequela to correction of epiglottic entrapment regardless of the technique performed, particularly in horses with severe epiglottic hypoplasia.

Acute Epiglottitis

Acute epiglottitis is manifested endoscopically by mucosal edema, reddening, and thickening of the epiglottis (Figure 45-26). Occasionally, the cartilage tip of the epiglottis is exposed through the membrane. Extensive reddening or purple swelling of the mucous membrane ventral to the epiglottis, elevating the epiglottis dorsally into an abnormal axis, can also be present. The cause is unknown but the problem is seen most commonly in racehorses. The chief complaint is usually respiratory noise and exercise intolerance or coughing.

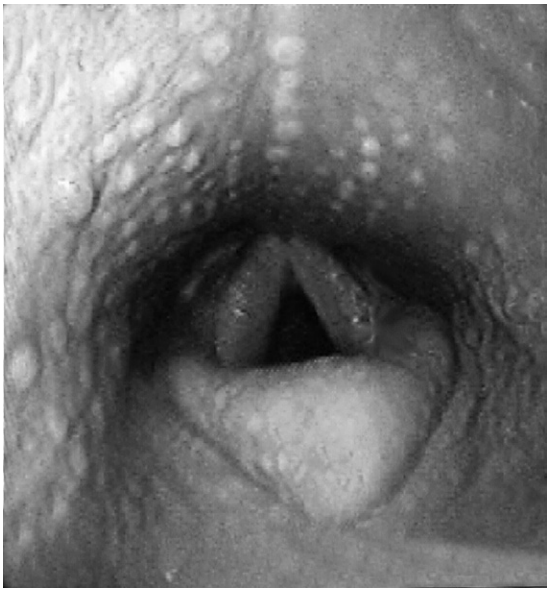


Figure 45-26. Endoscopic appearance of acute epiglottitis shows the typical edema and thickening of the epiglottis with this condition. (Courtesy M. Weishaupt, Zurich.)

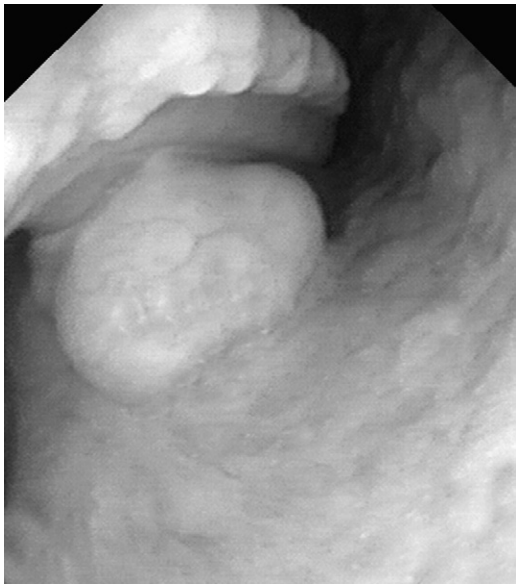


Figure 45-27. Endoscopic appearance of a subepiglottic cyst in a foal; the cyst was resected via laryngotomy.

Exercise should be discontinued. Most horses respond well to systemic NSAIDs and antimicrobial medications and pharyngeal sprays containing anti-inflammatory medication. Initially, systemic corticosteroids may be useful in reducing severe inflammation. Substantial improvement in endoscopic appearance is usually seen in 7 to 14 days. Although many affected horses recover completely and successfully return to work, long-term complications occur about 50% of the time and these include epiglottic deformity, epiglottic entrapment, or intermittent or persistent dorsal displacement of the soft palate.[143]

Subepiglotic Cysts

Subepiglottic cysts are particularly prevalent in young Thoroughbred and Standardbred racehorses, suggesting a possible congenital condition.[144,145] In young foals, subepiglottic cysts may cause coughing, dysphagia, and aspiration pneumonia.[146] Subepiglottic cysts may also be acquired; they are occasionally diagnosed in older horses with no previous history of respiratory tract problems. Older horses are usually presented with a complaint of respiratory noise, coughing, and even dysphagia if the cyst is large.[145]

Diagnosis

A diagnosis of a subepiglottic cyst is made on endoscopic examination. A round or oval, smooth, pink, fluctuant, mucosa-covered mass (15 to 40 mm) can be seen beneath the epiglottis (Figure 45-27). The epiglottis may be asymmetrically elevated slightly above the soft palate by the cyst. Some larger, more fluctuant subepiglottic cysts occasionally at rest or during swallowing slip beneath the soft palate into the oral pharynx, temporarily obscuring the cyst from view and causing the epiglottis to have a normal interface with the soft palate. Concurrent intermittent or persistent epiglottic entrapment by the aryepiglottic folds can also be present. Lateral laryngeal radiographs or contrast pharyngography may help define the location and size of the cyst.

An oral endoscopic and/or digital examination (dependent on hand size) can reveal the exact size and position of the cyst. The horse is anesthetized, positioned in lateral recumbency, and nasotracheally intubated to allow unrestricted access to the oropharynx. With the aid of a mouth speculum and rostrad traction of the tongue, the soft palate is manually displaced above the epiglottis. The cyst can be felt or visualized beneath the epiglottis as a variably sized, smooth, fluctuant mass projecting from the area of the epiglottic base superficial to the hyoepiglotticus muscle. The cyst is submucosal in origin, and hence the cyst base may be poorly defined (sessile) rather than reduced to a distinct narrow, pedunculated stalk. When the oral examination is complete, the horse may be placed in dorsal recumbency for approach via laryngotomy or left in lateral recumbency for an oral approach (see "Treatment," later). Histopathology reveals that subepiglottic cysts are usually lined with a combination of stratified squamous and pseudostratified columnar epithelium. Mucous glands, dilated ducts, and homogeneous eosinophilic mucus are usually present.

Treatment

Removal of the secretory lining is important. If the cyst is only punctured and drained, it usually seals over and refills within days. However removal of excessive oral pharyngeal mucosa overlying the cyst may result in subepiglottic cicatrization, possibly altering the normal range of motion of the epiglottis or disrupting the normal synchrony between the epiglottis and the soft palate. Cicatrization after subepiglottic cyst removal can also result in intermittent or persistent dorsal displacement of the soft palate.[145] Traditionally, subepiglottic cysts have been removed through a laryngotomy with the horse positioned in

dorsal recumbency under general anesthesia.[146] The epiglottis is gently retroverted into view by grasping each aryepiglottic fold with a sponge forceps or Allis tissue forceps. By elevating the epiglottic tip, the cyst can be palpated on the ventral epiglottic surface. The mucosa is sharply incised over the cyst and the cyst is excised submucosally with Metzenbaum scissors. The mucosal incision is usually left to heal by second intention or it may be sutured with 3-0 to 5-0 synthetic absorbable suture. The laryngotomy is normally left to heal by second intention with routine twice-daily cleansing. Exercise is resumed in 3 to 4 weeks if the laryngotomy has healed routinely and follow-up endoscopy is normal.

Two techniques are available for excising subepiglottic cysts via an oral approach.[145] The horse is anesthetized and positioned in lateral recumbency, and a nasotracheal tube is positioned. Using a videoendoscope to visualize the cyst, traction is placed on the mucosa and the underlying cyst with 600-mm bronchoesophageal grasping forceps (Universal bronchoesophageal grasping forceps, 600-mm length, 8280.62, Richard Wolf Medical Instrument Corporation). Using either a Nd:YAG laser or a diode laser with a 600-µm sculpted fiber, a fusiform incision into the mucosa over the cyst is made transendoscopically with 15 to 18 W of power. Laser energy is then used to excise the cyst membrane submucosally.[147,148] This can be difficult to achieve without rupture of the cyst, and operator experience is essential. Occasionally, a small amount of digital dissection is necessary to free the cyst's final attachments. Alternatively, an electrocautery snare, passed via the biopsy channel of the endoscope, may be applied. A cyst snare can be fashioned from a mare urinary catheter and obstetrical wire and used to excise the cyst at its base, taking extreme caution to remove as little overlying mucosa as possible. In either technique, hemorrhage is minimal and the defect created is left to heal by second intention.

A standing transnasal approach using either endoscopic guided laser or electrocautery excision can also be performed with the horse sedated and the head elevated in stocks. This technique is usually reserved for small cysts that can be easily manipulated to a position above the palate. The cyst is anesthetized topically through polyethylene tubing introduced through the biopsy channel of the videoendoscope. Five to 10 mL of local anesthetic solution dripped over the cyst will induce local anesthesia in about 5 minutes. The 600-mm bronchoesophageal forceps are introduced up the opposite nasal passage to the one with the endoscope and the mucosa overlying the cyst is grasped. A fusiform mucosal incision and submucosal dissection of the cyst lining is subsequently performed (as previously described for the oral approach) using either an Nd:YAG or a diode laser and a 600-µm sculpted fiber.[100] A single horse has been successfully treated with two endoscopically guided injections, 14 days apart, of 10% neutral buffered formalin directly into the cyst. The contents of the cyst were aspirated prior to injecting the formalin.[149]

Aftercare

Postoperative inflammation and pain are controlled with a combination of corticosteroids, NSAIDs, and pharyngeal sprays as needed. Exercise is restricted until the pharynx appears endoscopically normal, which is usually within approximately 21 days. In horses with concurrent epiglottic entrapment, removal of the subepiglottic cyst usually can correct the epiglottic entrapment; however, epiglottic entrapment can recur, and it should probably be treated using the tissue-sparing technique described earlier. Foals with concomitant aspiration pneumonia should be treated with broad-spectrum antimicrobial agents based on culture and sensitivity of tracheal aspirates before excision of the cyst.

Dorsal Epiglottic Abscess

Dorsal epiglottic abscess is a rare disease that causes coughing, respiratory noise, and exercise intolerance.[147] Palpation of the laryngeal cartilages may elicit pain. On endoscopic examination, there is a smooth, well-circumscribed, round or oval swelling on the dorsal epiglottic surface. Intermittent dorsal displacement of the soft palate may be observed. The abscess can be incised and drained transendoscopically on an outpatient basis with a contact Nd:YAG or diode laser using a sculpted fiber or chisel probe. After decompressing the abscess, débridement can be achieved with 600-mm-long bronchoesophageal grasping forceps. The abscess cavity can be lavaged transendoscopically, if desired, with 240 polyethylene tubing and a dilute 1 : 10 chlorhexidine solution in physiologic saline. Aftercare includes stall confinement and exercise restricted to handwalking. NSAIDs and systemic antibiotics (procaine penicillin G given intramuscularly twice daily) are given at recommended dosages for 3 days. The abscess usually appears healed by 10 days postoperatively. In a small number of cases, clinical signs resolved and the horses resumed normal exercise without recurrence of the abscess[147]; however, the potential for epiglottic deformity does exist.

Subepiglottic Granulomas

Subepiglottic granulomas originate following acute ulceration of the aryepiglottic fold on the ventral aspect of the epiglottis. In response to infection or chronic irritation, excessive granulation tissue forms. This tissue may be confused with subepiglottic cysts. Clinical signs frequently include coughing, exercise intolerance of varying degrees, and occasionally dysphagia. The horse may have a history of poor performance, and intermittent dorsal displacement of the soft palate may be suspected. It may be necessary to drive the endoscope up under the epiglottis to assess this condition. Large lesions may disrupt the normal anatomic seal between the epiglottis and the soft palate. Because dorsal displacement of the soft palate should be suspected with ulcer formation, a high-speed dynamic endoscopic examination is recommended.

Treatment

Small granulomas and ulcers can respond to antimicrobial and anti-inflammatory medical therapy as long as the horse has received forced rest for approximately 4 to 6 weeks. When ulcers do not heal, enlargement of the granulation tissue frequently occurs when the horse is returned to exercise. In these cases, excision either orally or through a ventral laryngotomy is indicated. Sharp surgical excision alone (performed through a ventral laryngotomy) may not result in complete resolution of the problem. Therefore, sterilization of this area with laser cautery of the surface after resection is indicated. Alternatively, laser photo ablation of the surface of the granulation tissue can be performed with the sculpted fiber using contact technique.

It is imperative that extended periods of rest (up to 60 days) accompany any treatment of this condition.[148]

Epiglottic Hypoplasia, Flacidity, and Deformity

Diagnosis

Epiglottic hypoplasia, flaccidity, and epiglottic deformity have been associated with dorsal displacement of the soft palate and epiglottic entrapment, both of which can cause abnormal respiratory noise and exercise intolerance.[131-136,139,150-153] On endoscopic examination, the hypoplastic epiglottis in the adult horse appears short and narrow, particularly from midbody to the tip, or it may appear very thin (Figure 45-28). The epiglottic contour may appear flaccid, conforming excessively to the shape of the underlying soft palate. In some horses, the epiglottic border is irregular or rounder and much flatter without the normal crenations. Occasionally, the epiglottis has a very shrunken and wrinkled appearance. Easily induced intermittent or persistent dorsal displacement of the soft palate may be observed during the endoscopic examination. A lateral laryngeal radiograph and contrast pharyngography may provide additional information regarding thyroepiglottic length, epiglottic thickness and contour, and the relationship of the epiglottis to other adjacent pharyngeal structures. If the soft palate is persistently displaced dorsad, endoscopy and digital palpation of the epiglottis *per os* with the horse anesthetized and in lateral recumbency may provide useful diagnostic information on the extent of the problem. Epiglottic hypoplasia that is severe enough to result in persistent dorsal displacement of the soft palate is associated with a guarded to poor prognosis for optimal exercise potential.[151,152] Other underlying problems causing dorsal displacement of the soft palate, such as epiglottic entrapment, subepiglottic cicatrix formation, or granulation tissue, can also be fully assessed through a detailed endoscopic and digital oral examination. However, persistent dorsal displacement of the soft palate may occur secondary to severe epiglottic hypoplasia without any other underlying abnormalities or previous surgical intervention.[152]

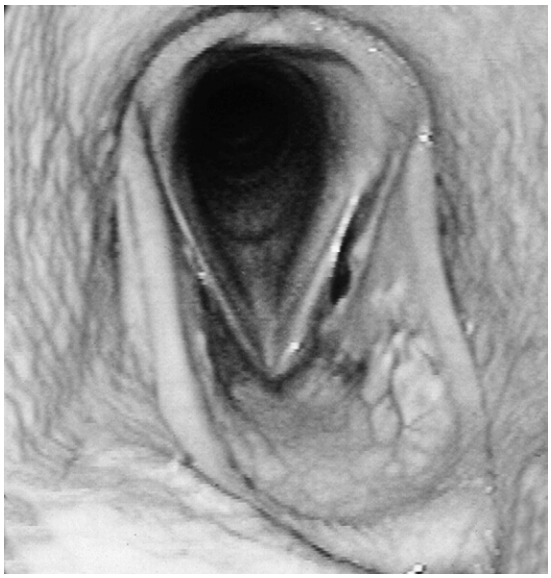


Figure 45-28. Endoscopic appearance of epiglottic hypoplasia/flaccidity, showing how the epiglottis appears very thin. (Courtesy J.T. Robertson, Ohio.)

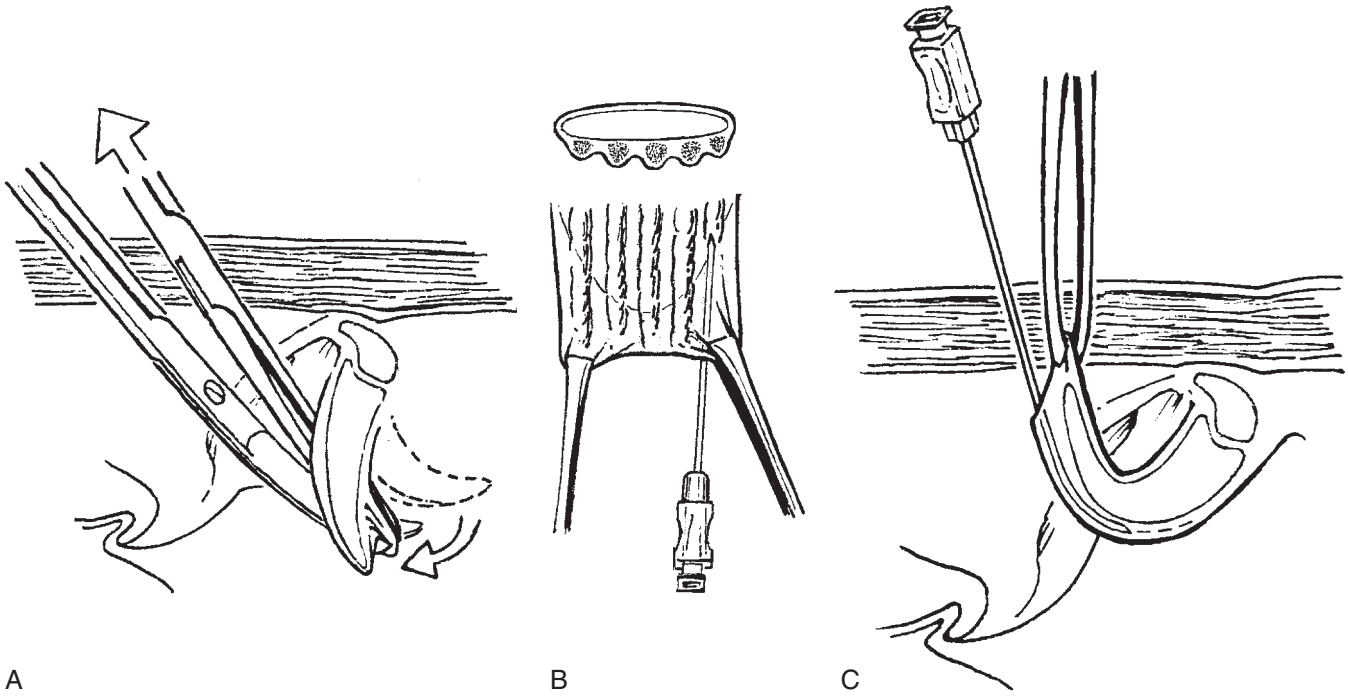


Figure 45-29. Technique used to evert the epiglottis through a laryngotomy incision. **A,** Curved scissors are used to evert the epiglottis until the epiglottic fold can be grasped with Allis tissue forceps. **B,** The endotracheal tube can be replaced and the ventral surface of the epiglottis is injected. **C,** Five linear injections are made equidistant from each other into the aryepligotic fold.

Because epiglottic hypoplasia or flaccidity in adult horses is thought to contribute to epiglottic entrapment and intermittent dorsal displacement of the soft palate, the epiglottis is routinely scrutinized during prepurchase endoscopic examinations in yearling Thoroughbreds. However, no correlation could be found between the appearance of a hypoplastic or flaccid epiglottis in the standing adult horse and dorsal displacement of the soft palate during exercise on a high-speed treadmill.[154] Initially these same visual epiglottic abnormalities in yearlings could not be significantly correlated with racing performance[16]; however, it has now been reported that horses with shorter epiglottises have lower earnings as 2- and 3-year-old race horses.[155] We now recognize that variation exists in the upper airway structure of yearling and adult horses. Compared with adult horses, yearlings have a shorter, narrower, and more flaccid-appearing epiglottis normally, and judgments about epiglottic appearance should be reserved until the horse reaches maturity, except in very severe cases.

Treatment

Epiglottic augmentation is performed only in the adult horse to increase the mechanical rigidity of the epiglottis and to make the hypoplastic or flaccid epiglottis more resistant to dorsal displacement of the soft palate. Several materials including collagen and autogeneic and allogeneic cartilage grafts have been evaluated, but epiglottic augmentation with polytetrafluoroethylene (Polytef Paste; Polytetrafluoroethylene Teflon paste, Mentor polytef paste for injection, Mentor O & O, Inc) has proved to be the most useful.[156,157] Submucosal injection of the ventral epiglottic muosa with polytetrafluoroethylene paste via a laryngotomy (Figure 45-29) using an Arnold-Bruning syringe (Arnold-Bruning intracordal injection syringe, catalog No. 7754) (Figure 45-30) resulted in an increased thickness of approximately 29% to 40% along the margins and midline (Figure 45-31).[157] Epiglottic length was not increased. Polytetrafluoroethylene paste functions by inciting a sterile granulomatous reaction that is incorporated in fibrous connective tissue.

In two studies, an approach to management of intermittent dorsal displacement of the soft palate that most often combined surgical techniques and always included epiglottic augmentation improved racing performance in 66% of treated horses with poor racing performance attributable to epiglottic hypoplasia or flaccidity.[158,159]

Figure 45-30. Photograph of Arnold-Bruning intracordal injection syringe, fully assembled.

Epiglottic Retroversion

Diagnosis

Epiglottic retroversion refers to a condition where the epiglottis assumes a position dorsal to the soft palate and retroverts into the opening of the glottis during inspiration and returns to its normal position with each expiration.[160] Resting endoscopic examination of the pharynx and larynx can appear normal, or on occasion the epiglottis projects slightly above the soft palate. High-speed dynamic or treadmill videoendoscopy is necessary to confirm the diagnosis (Figure 45-32). Retroversion of the epiglottis enables visualization of the ventral (lingual) surface of the epiglottis, but usually the soft palate does not elevate or displace dorsal to the epiglottis. In one horse, the epiglottis rolled up in a tubelike fashion and pointed dorsally, but it did not retroflex into the glottis. During exercise, a respiratory noise may be heard.

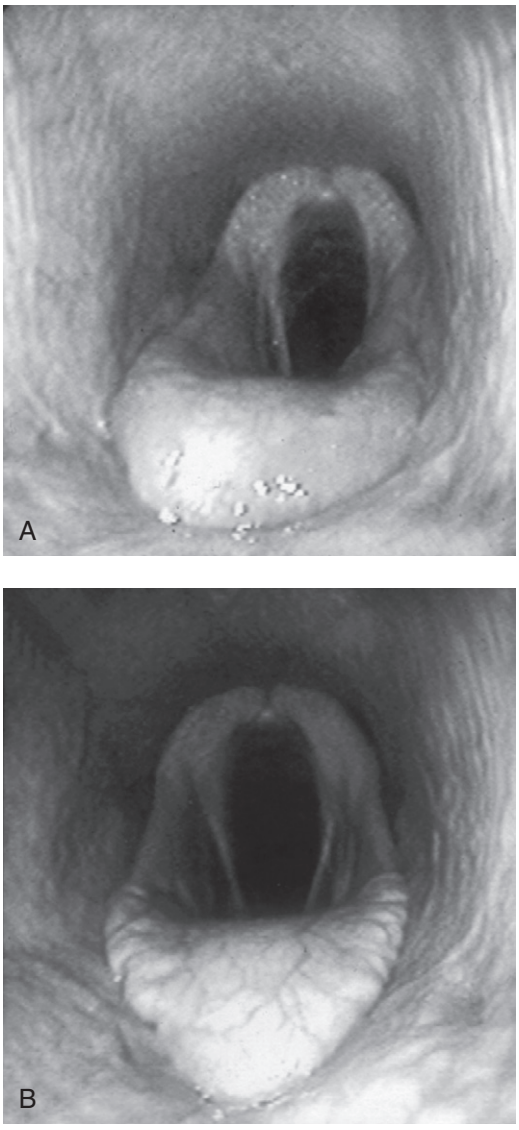


Figure 45-31. A, Twenty-four hours after Teflon injection in the epiglottic fold, the epiglottis becomes edematous; **B,** Thirty days after injection, the epiglottis is clearly thickened and more substantial when compared with its appearance before surgery (see Figure 45-28).

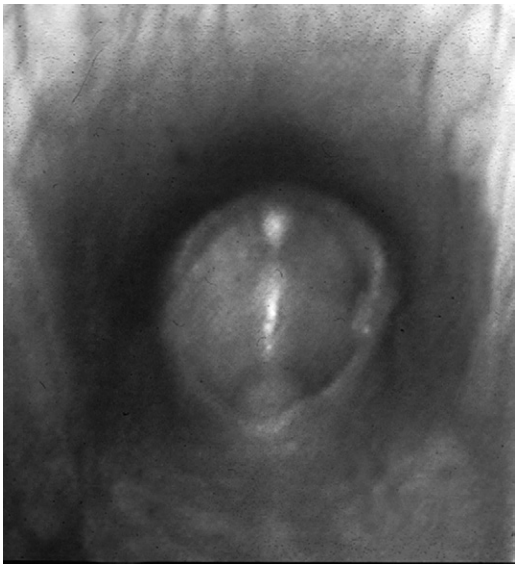


Figure 45-32. Videoendoscopic appearance of epiglottic retroversion as seen during inspiration on a horse exercising on a high-speed treadmill.

Etiology

Experimentally, local anesthesia of the geniohyoid muscle and of the hypoglossal nerves within the guttural pouch have produced epiglottic retroversion during exercise in normal horses.[161] Trauma or inflammation of the normal hyoid musculature or the hyoepiglotticus or geniohyoid muscles may precede epiglottic retroversion.

Treatment

Two horses with this rare problem were treated by epiglottic augmentation with polytetrafluoroethylene paste injected submucosally on the lingual epiglottic surface. One Standardbred was able to return to racing and competed successfully, including winning several races, and one Thoroughbred was retired when it continued to make noise during training after undergoing surgery. In a third horse, a racing Standardbred, a prosthetic suture placed between the epiglottis and the thyroid cartilage was used to stabilize the epiglottis, which allowed it to return to racing.[162]

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CHAPTER 46

Guttural Pouch

David E. Freeman and Joanne Hardy

ANATOMY

Guttural pouches are paired extensions of the eustachian tubes that connect the pharynx to the middle ear.[1] They are found in perissodactyls, such as equids, tapirs, some species of rhinoceros (except for the white rhinoceros), some bats, a South American forest mouse, and hyraxes.[2-4]

The pouches are separated from each other on the midline by the rectus capitis ventralis and the longus capitis muscles and the median septum.[1] Each is in close contact rostrally with the basisphenoid bone; ventrally with the retropharyngeal lymph nodes, pharynx, and esophagus; caudally with the atlantooccipital joint; laterally with the digastricus muscle and the parotid and mandibular salivary glands; and dorsally with the petrous part of the temporal bone, tympanic bulla, and auditory meatus. Each guttural pouch is divided ventrally into a medial and a lateral compartment by the stylohyoid bone, and it communicates with the pharynx through the pharyngeal orifice of the eustachian tube. The pharyngeal orifice is a funnel-shaped opening in the dorsolateral aspect of the pharynx that forms an oblique slit rostral and ventral to the dorsal pharyngeal recess. The small end of the funnel opens into the guttural pouch. The medial lamina of each opening is composed of fibrocartilage directed in a rostroventrad-to-caudodorsad direction. The capacity of guttural pouches in adult horses is 472 ± 12.4 mL and the lateral compartment is approximately one third of the capacity of the medial compartment.[5]

Clinical signs of important guttural pouch diseases are referable to injury of specific nerves and arteries in the guttural pouch and acoustic system. The internal carotid artery (ICA), cranial cervical ganglion, cervical sympathetic trunk, and vagus, glossopharyngeal, hypoglossal, and spinal accessory nerves are all contained in a fold of mucous membrane along the caudal wall of the medial compartment (Figure 46-1). The cranial laryngeal nerve and the pharyngeal branch of the vagus nerve lie beneath the mucosa on the floor of the medial compartment (see Figure 46-1).[1] The external carotid artery (ECA) lies along the wall of the lateral compartment and gives off the caudal auricular artery and superficial temporal artery, and it continues as the maxillary artery (MA) along the roof of the guttural pouch. The facial nerve (CN VII) passes for a short distance over the caudal dorsal aspect of the lateral compartment after it emerges from the stylomastoid foramen. The vestibulocochlear nerve (CN VIII) enters the internal acoustic meatus caudal to the facial nerve and divides into vestibular and cochlear branches that innervate components of the middle ear. This nerve does not enter the guttural pouch but can become involved in guttural pouch diseases that affect the middle ear (e.g., temporohyoid osteoarthropathy). The mandibular nerve, a branch of the trigeminal nerve (CN V), emerges from the foramen lacerum, passes close to the muscular process of the petrous part of the temporal bone, and continues rostrad along the roof of the lateral compartment of the guttural pouch.

The guttural pouch is lined with pseudostratified ciliated epithelium containing goblet cells in both adults and foals.[6] The guttural pouch mucosa has the ability to clear foreign substances, but this ability varies among different regions of the epithelium.[6] In a study on the distribution of various immunoglobulin isotypes and sub-isotypes in the guttural pouch mucosa of healthy horses, immunoglobulin G (IgG) was found in the guttural pouch mucosa, mucosal lymph nodules, and submucosal lymph nodules.[5] IgM was scattered in the mucosal lymph nodules and in the germinal centers of the submucosal lymph nodules. IgGc was recognized only in the submucosal lymph nodules, and IgA was detected in glandular epithelial cells and the surface layer of the mucosal epithelium.[5]

Possible functions of the guttural pouches include pressure equilibration across the tympanic membrane, contribution to air warming, a resonating chamber for vocalization, and a flotation device.[7] A role more recently proposed, on the basis of measurement of lower arterial temperatures in the cerebral side of the ICA compared with the cardiac side, is brain cooling.[8,9] Based on cadaver studies, opening of the pharyngeal orifice of the guttural pouch involves the levator and tensor veli palatini muscles and the pterygopharyngeus and palatopharyngeus muscles. Passive opening of the auditory tube involves a reduced tone in the stylopharyngeus and pterygopharyngeus muscles,

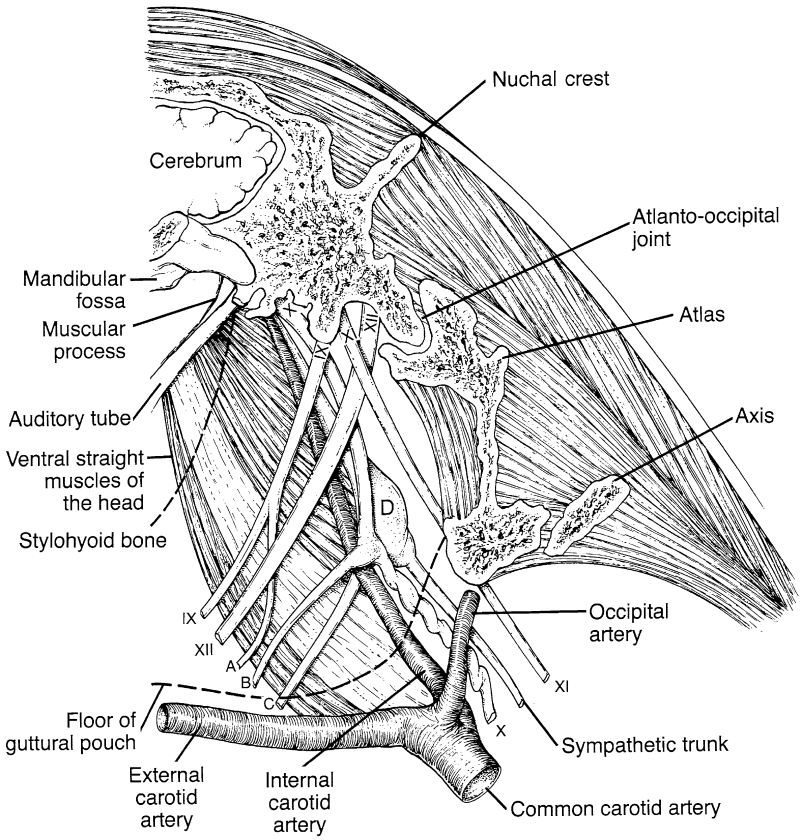

Figure 46-1. Interior of medial compartment of the left guttural pouch, viewed from the lateral aspect in a sagittal section of a horse's head. The section is cut through the styloid process of the petrous temporal bone on a line that divides the guttural pouch into medial and lateral compartments. *IX*, Glossopharyngeal nerve; *X*, vagus nerve; *XI*, accessory nerve; *XII*, hypoglossal nerve; *A,* pharyngeal branch of the glossopharyngeal nerve; *B,* pharyngeal branch of the vagus nerve; *C,* cranial laryngeal nerve; *D,* cranial cervical ganglion. (Redrawn from Freeman DE, Donawick WJ: Occlusion of internal carotid artery in the horse by means of a balloon-tipped catheter: Clinical use of a method to prevent epistaxis caused by guttural pouch mycosis. J Am Vet Med Assoc 176:236, 1980.)

accompanied by increased inspiratory pressure.[10] Although guttural pouch filling was previously reported to occur on expiration, the latter study demonstrated that filling occurs on inspiration.[10]

EXAMINATION

The guttural pouches are examined by external palpation, endoscopy, and various imaging techniques. Enlargement caused by empyema (purulent material in the pouches), but particularly by tympany (air engorgement), can be palpated externally. Guttural pouch endoscopy provides the most information regarding disease. Nonspecific evidence of guttural pouch disease, such as collapse of the pharynx and blood or pus draining from the pharyngeal orifice, can be found on endoscopic examination of the pharynx. However, blood or pus from other respiratory sources may be aspirated into the guttural pouch opening and appear to drain from it, so that direct endoscopic examination of the pouches must be performed (Figure 46-2). With the horse mildly sedated, the biopsy instrument is passed through the biopsy channel of the endoscope and used to guide the endoscope into the guttural pouch. The endoscope is placed so that the biopsy forceps is as close as possible to the lateral wall of the pharynx until successful insertion into the guttural pouch is achieved. Both pouches can be entered in this manner with the endoscope in the same nostril. Alternatively, the pharyngeal opening can be levered open with a Chamber's catheter to allow the endoscope to enter the pouches. The endoscopic appearance of the guttural pouch anatomy is quite consistent between horses (see Figure 46-2), but a double ICA can be seen (Figure 46-3).

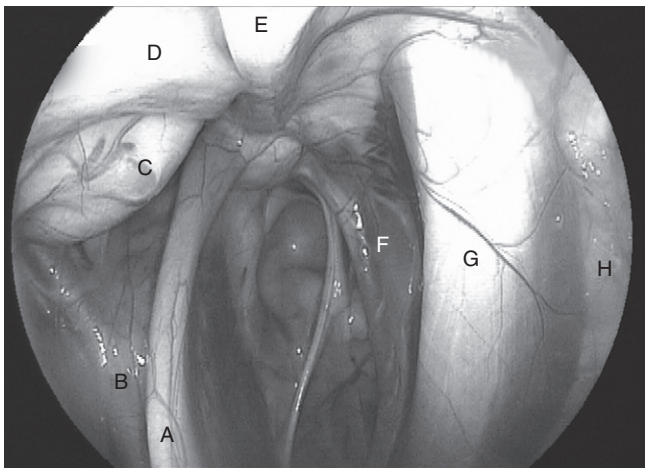

Figure 46-2. Normal endoscopic anatomy of right guttural pouch. The stylohyoid bone *(A)* divides the caudoventral part into lateral and medial compartments. *B,* External carotid artery; *C,* maxillary artery; *D,* tensor veli palatini muscle; *E,* cartilaginous portion of eustachian tube; *F,* internal carotid artery with hypoglossal and glossopharyngeal nerves lateral to it; *G,* ventral straight muscles; *H,* median septum.

Lateral radiographic projections of the guttural pouches can demonstrate fluid lines, fractures and exostoses of the stylohyoid bone, radiopaque foreign bodies, and space-occupying masses.[11] Air distention, as in tympany, can increase dimensions of the affected guttural pouch, sometimes beyond the second cervical vertebra. A dorsoventral or ventrodorsal projection is best used to image the stylohyoid bones and temporohyoid

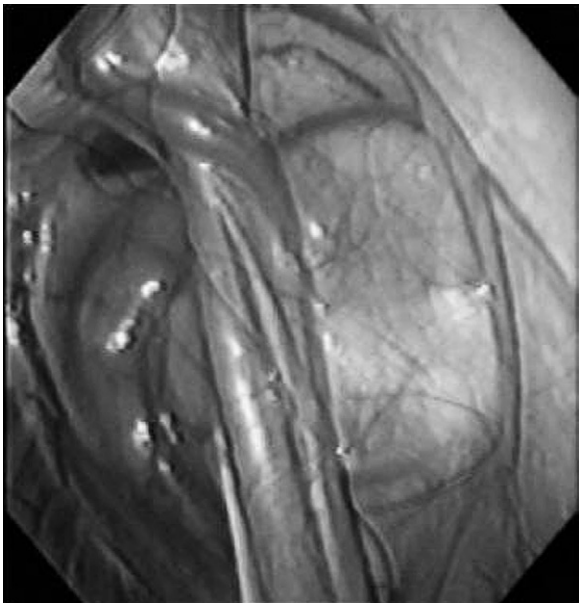

Figure 46-3. Endoscopic photograph of the medial compartment of the right guttural pouch in a normal horse showing a double internal carotid artery.

articulation. Computed tomography can provide an alternative imaging modality, especially for evaluation of the stylohyoid bone, inner ear, and petrous temporal bone in cases of temporohyoid osteoarthropathy.[12-16] Ultrasonography can be used to demonstrate soft tissue lesions in the guttural pouches, such as tumors or muscle damage and associated submucosal hemorrhage.[17,18]

A percutaneous centesis technique through Viborg's triangle has been described for guttural pouch lavage and collection of samples for cytologic and microbiologic examinations.[19] The normal cytologic pattern contains less than 5% neutrophils, a large proportion of ciliated columnar epithelial cells, a few nonciliated cuboidal epithelial cells, and less than 1% monocytes, lymphocytes, and eosinophils.[20,21] The proportion of neutrophils is important, with less than 5% being considered normal and greater than 25% being considered abnormal. There is a high correlation between a high cytologic score and the presence of pathogenic bacteria, such as *Streptococcus equi*.[20,21] The cytologic gradings and neutrophil concentrations of guttural pouch washings are increased in horses whose heads are restrained for more than 12 hours, as would occur during long transportation.[20] Washings from these horses are more likely to contain bacteria and yield potentially pathogenic bacteria.[20]

DISEASES OF THE GUTTURAL POUCH

Tympany

Tympany, as the name implies, refers to the distention of the guttural pouches with air under pressure, sometimes accompanied with some fluid accumulation. This condition is usually unilateral but can be bilateral and is more common in fillies than in colts.[22] In 51 foals with guttural pouch tympany seen at a German clinic between 1994 and 2001, there were approximately three times as many fillies as colts, regardless of breed.[23-26] Significantly more Arabian and Paint horse foals were affected compared to the breed distribution of hospitalized foals at the same clinic.[23] In the 27 Arabian purebred foals affected with guttural pouch tympany, many were from the same stud farm and some were full or half siblings. Complex segregation analyses showed that a polygenic and a mixed monogeneic–polygeneic model best explained the segregation of Arabian foals with this disease.[26] Subsequently, whole genome scan for guttural pouch tympany in Arabian and German Warmblood horses indicated a sex-specific quantitative trait locus, in agreement with the higher prevalence of this disease in fillies.[27]

Possible causes include a mucosal flap (or plica salpingopharyngea) acting as a one-way valve that traps air and fluid in the pouch, inflammation from an upper airway infection, persistent coughing, and muscle dysfunction.[28] In most cases, no gross anatomic abnormality is encountered at the guttural pouch opening.[23]

Clinical Signs

Guttural pouch tympany develops in foals shortly after birth and up to 1 year of age. The affected guttural pouch is distended with air to form a nonpainful, elastic swelling in the parotid region. Although the swelling is most prominent on the affected side, it can extend across the neck and give the impression of bilateral involvement. Severe distention can cause dyspnea, dysphagia, and inhalation pneumonia, and secondary empyema is not uncommon. Because the consequences of the guttural pouch distention are detrimental, it is important to relieve the tympany as quickly as possible.

Diagnosis

Diagnosis is based largely on clinical signs. On endoscopic examination, the pharyngeal openings usually appear normal, but the roof of the pharynx can be collapsed. Guttural pouch enlargement with air and fluid can be seen on radiographs. Distinguishing between unilateral and bilateral tympany can be difficult on radiographs, but usually they can be defined on direct endoscopy of each pouch.[29] The distinction between unilateral and bilateral involvement is important in selecting treatment.

Treatment

Temporary alleviation of guttural pouch tympany can be achieved by needle decompression or by placing an indwelling catheter in the pharyngeal orifice. Chronic catheterization (2 to 3 weeks) using Sovereign catheters has also been anecdotally reported to result in resolution of guttural pouch tympany. However, failure of these methods to eliminate the disease within a brief time can put the foal at risk of developing complications, such as empyema, bronchopneumonia, and permanent deformity and scarring in the cartilage of the pharyngeal ostium.[30] Surgical intervention is aimed at providing a permanent means of evacuating air, either through the unaffected guttural pouch (fenestration of median septum), through the guttural pouch opening (removal of obstructing membrane), or through an artifically created opening into the pharynx (salpingopharyngeal fistula).

For median septum fenestration, the affected guttural pouch is usually entered through Viborg's triangle or through a modified Whitehouse approach (discussed later). The median septum

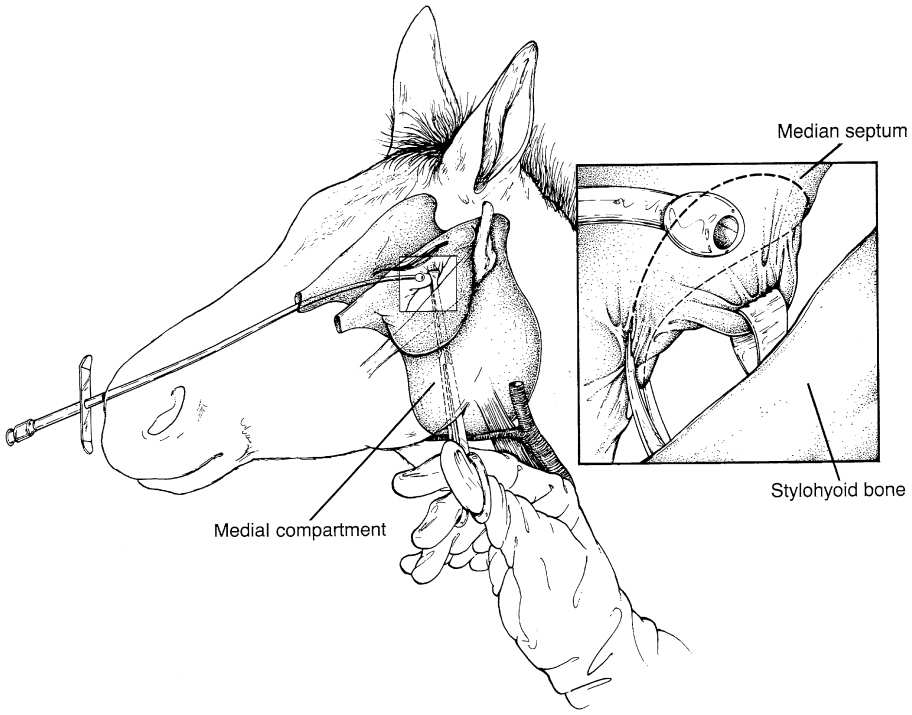

Figure 46-4. Method for creating a fenestration between the abnormal and normal guttural pouches in a foal with unilateral tympany. The affected guttural pouch (on the *left side*) is approached through an incision in Viborg's triangle, and a Chamber mare catheter in the right guttural pouch is used to elevate and expose the median septum. In the *expanded view,* a pair of scissors has been placed to start fenestration *(broken line).* (Redrawn from Milne DW, Fessler JF: Tympanitis of the guttural pouch in a foal. J Am Vet Med Assoc 161:61, 1972; and Deen T: Surgically correcting guttural pouch tympany. Vet Med 83:592, 1988.)

can be fenestrated by removal of a 2 cm^2 segment to allow egress of trapped air from the tympanitic pouch through the normal side (Figure 46-4). A Chambers mare catheter or, preferably, a lighted fiberoptic endoscope can be inserted into the healthy pouch to elevate the septum toward the incision and to facilitate identification (see Figure 46-4). Care must be taken to ensure that the mucosal linings of both sides are removed to complete the opening. Fenestration of the median septum has been completed with laparoscopic instruments, to improve access and reduce the risk of tissue trauma, especially nerve damage.[30] The major disadvantage of the fenestration procedure is that it will only be effective for unilateral cases, because it depends on one functional guttural pouch opening working for two. Fortunately most cases are unilateral, and fenestration can be a simple and effective procedure in such cases.

When bilateral involvement is suspected, the fenestration procedure can be combined with removal of a small segment (1.5 × 2.5 cm) of the medial lamina of the eustachian tube and associated mucosal fold (plica salpingopharyngea) within the guttural pouch orifice; this forms a larger opening into the pharynx.[22,29] The approach incision can be closed or left open to heal by second intention, and systemic antibiotics are given if needed. Alternatively, partial resection of only the plica salpingopharyngea within the gutteral pouch (via a modified Whitehouse approch) as the only procedure has been described with good results.[31]

Uncertainty arises about the true role of the plica salpingopharyngea in the disease process because of inconsistent descriptions of the tissue removed. In one method, the cartilaginous lamina of the eustachian tube and plica salpingopharyngea are removed,[30,32] and in another, only the cartilaginous flap is resected.[33] The plica salpingopharyngea is a mucosal fold on the floor of the tubular entryway into the guttural pouch; it is attached medially to the cartilaginous flap of the eustachian tube and laterally to the lateral wall of the pharynx. It is best viewed during guttural pouch endoscopy as the ventrally located fold that must be negotiated by the biopsy instrument to guide the endoscope under the cartilaginous flap into the pouch.

Transendoscopic electrocautery can be used to create a fenestration in the septum and to make a fistula into the guttural pouch through the pharyngeal recess.[28,34] A high-powered diode or a neodymium:yttrium-aluminum-garnet (Nd:YAG) laser can also be used through a transendoscopic approach to create the fenestration. This can be carried out as a standing procedure or with the horse under general anesthesia.[29,35] An alternative and easier method is to create a salpingopharyngeal fistula using the laser under video-endoscopic control in the wall of the pharynx, caudal to the guttural pouch opening.[35] A Foley catheter is placed through the fistula for 7 to 10 days to act as a stent and to allow lavage of the guttural pouch.[35] The diode laser has been used in contact mode to create bilateral salpingopharyngeal fistulas and thereby successfully treat bilateral guttural pouch tympany in a foal.[36] Location of the salpingopharyngeal fistula is important. If it is placed too far rostrad or ventrad, it could fail to bypass the defective pharyngeal ostium or it could become occluded by inflammation.[30]

Prognosis

The prognosis for complete recovery and a successful racing career is favorable after median septum fenestration by any method,[22,29] although surgery is not always straightforward and repeat surgery is not uncommon. A surgical laser technique was used on 50 foals in one clinic and was successful in 35 foals after one treatment and in 15 foals after two treatments.[23] Long-term follow-up information, available for 44 of the 50 treated horses, revealed no deaths related to the disease or treatment and that all horses up to 2 years of age were healthy.[23]

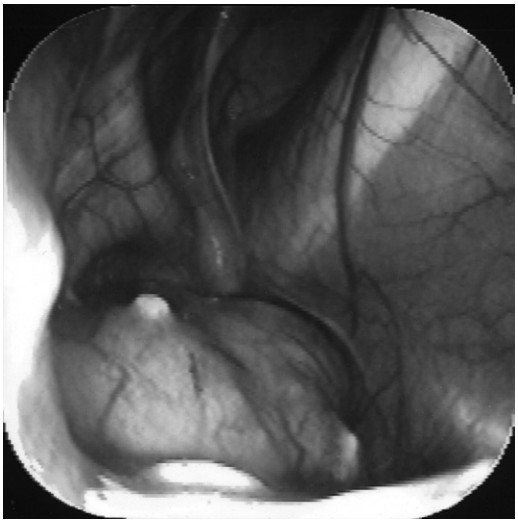

Figure 46-5. Endoscopic view of the interior of the right guttural pouch of a horse with enlargement and drainage from a retropharyngeal lymph node. Note purulent material on floor of the medial compartment.

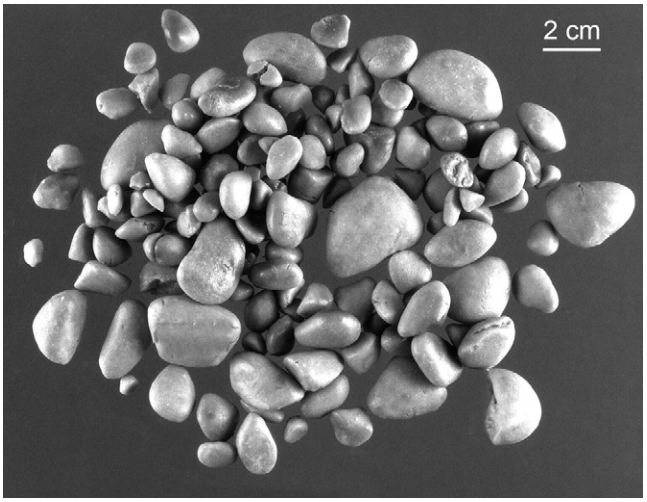

Figure 46-6. Guttural pouch chondroids removed through a modified Whitehouse approach.

Secondary empyema and pneumonia usually resolve spontaneously after successful treatment of the tympany; however, the prognosis is guarded for foals that have aspiration pneumonia and dysphagia secondary to nerve damage induced during surgical correction.[22] The fenestration procedure can fail if the newly created opening seals or the condition is bilateral.[22,29] Resection of the mucosal fold can fail if swelling and inflammation along the cut edges close the pharyngeal orifice.[37] The salpingopharyngeal fistula may seal if the catheter does not stay in place for the period required for the fistula to mature.[35] Horses treated with this method have responded favorably,[35] although effects on upper airway dynamics in the athletic horse need to be determined.[38]

Empyema

Empyema of the guttural pouches is defined as the presence of purulent material (Figure 46-5), and chondroids within one or both guttural pouches. Chondroids consist of inspissated purulent material (usually numerous individual round balls) that forms in some cases (Figure 46-6). Empyema can affect horses of any age but usually occurs in young animals. Upper respiratory tract infections (especially those caused by *Streptococcus* spp); abscessation and rupture of retropharyngeal lymph nodes into the guttural pouch (see Figure 46-5); infusion of irritant drugs; fracture of the stylohyoid bone; congenital or acquired stenosis of the pharyngeal orifice; and pharyngeal perforation by a nasogastric tube may cause empyema.[28,39] Persistence of guttural pouch infection in asymptomatic long-term carriers could be responsible for recurrent outbreaks of strangles.[40] In one study of 91 horses with guttural pouch empyema, 21% had chondroids, and the horses with chondroids were more likely to have retropharyngeal and pharyngeal swelling than those without this complication.[41] The number of chondroids present is variable, ranging from one to many (see Figure 46-6), and both guttural pouches can be affected.[41] Duration of infection does not appear to correlate with development of chondroids.[41]

Clinical Signs

Clinical signs include intermittent nasal discharge in most cases, swelling of adjacent lymph nodes, parotid swelling and pain, extended head carriage, excessive respiratory noise, and difficulties in swallowing and breathing. Many cases with long-standing disease and a heavy load of inspissated pus and chondroids can have a deceptive absence of external swelling. Severe and protracted guttural pouch empyema can cause cranial neuropathy, manifested by dysphagia, recurrent laryngeal neuropathy, and persistent soft palate displacement.[42] Although these signs are rare with this disease, they can persist in some cases after successful resolution of the empyema.[43] The disease can be bilateral.[43]

Diagnosis

On endoscopic examination, a purulent discharge can be seen at the pharyngeal orifice of the affected side, with pharyngeal collapse in some horses. Fluid accompanied by masses seen within the guttural pouch on standing lateral radiographs is suggestive of chondroids.[44] Fluid aspirates or saline washings can be taken from the guttural pouch for culture and sensitivity testing; however, results should be interpreted with caution because microorganisms can be retrieved from the normal guttural pouch and upper respiratory tract. Horses that are carriers of, or are infected with, *S. equi* in the guttural pouches can be identified by culture and polymerase chain reaction tests with repeated swabs.[45]

Treatment

In acute cases, daily irrigation with physiologic saline solution is usually effective. An indwelling catheter, devised from polyethylene 240 tubing with heat-formed coils at one end, can be used for this purpose. Alternatively, a commercially available guttural pouch catheter or one fashioned from a polypropylene dog urinary catheter can be used. Coiled catheters can be straightened to facilitate insertion by inserting a coaxial wire or by passage through a larger curved catheter. The coiled end of the catheter is placed under endoscopic guidance within the

pouch, and the free end is secured by a suture to the alar fold. A Foley catheter can also be used, but it should be advanced until the end is completely in the pouch because distention of the balloon within the pharyngeal opening could cause pressure necrosis. In larger horses, standard Foley catheters are not long enough to reach the guttural pouch. Alternatively, the pouch can be flushed through the biopsy channel of the endoscope, which has the advantage of delivery of the flush solution to areas coated with purulent material. After 7 to 10 days, irrigation should be interrupted briefly to assess the response and allow any inflammation from treatment itself to resolve.

The purpose of lavage with a balanced physiologic solution is to dislodge and remove mucopurulent material from the guttural pouch, which should reduce the bacterial burden and produce a more favorable environment for recovery. Aggressive methods of lavage, such as delivery of large volumes of fluid under pressure through large-bore tubes, are not recommended because they can rupture the guttural pouch lining[43] and spread the infection into tracts from which it cannot be readily removed.[46] In chronic cases with inspissated pus and chondroids, repeated delivery of lavage fluid is usually not effective and could also cause guttural pouch rupture with extension of infection and air into adjacent fascial planes.[46] The cost and potential complications of protracted medical treatment should be weighed against the benefits of surgery if a slow response seems likely.

Hydrogen peroxide or concentrated antiseptic solutions should not be infused because they are irritating and can induce neuritis of the cranial nerves. Topical antibiotic solutions are rarely effective because the contact period is too brief and many are inactivated by the products of inflammation present in the purulent material. However, penicillin gels have gained popularity. Dilute povidone-iodine (1%) is more effective than concentrated solutions because free iodine dissociates more readily from the organic carrier at low concentrations; however, iodine can be neutralized by exudates.[47] Chlorhexidine gluconate at antimicrobial concentrations is not recommended because it can cause severe inflammation of the guttural pouch mucosa.[48] In one horse with empyema that did not respond to saline lavage, infusion of acetylcysteine on four occasions appeared to hasten resolution.[49] However, despite its ability to disrupt disulfide bonds of mucoproteins, acetylcysteine does not alter the viscosity of purulent material in clot tubes compared with the use of saline alone.[49] Additional clinical experiences with acetylcysteine have demonstrated a lack of efficacy for removing inspissated material and chondroids, and there is some concern that it could exacerbate mucosal inflammation.[46] Systemic antibiotics are rarely indicated unless the response to topical treatment is poor or the infection is severe. Drugs of choice are a potentiated sulfonamide, penicillin, or ceftiofur for at least 21 days.[45] Nonsteroidal anti-inflammatory drugs may be used as needed.

Chondroids can be removed by maceration, followed by saline lavage or extraction by endoscopically guided grabbing forceps, a basket snare, or a memory-helical polyp retrieval basket.[45] Another technique involves repeated section of each mass by a diathermic snare or a wire loop, with removal by suction, lavage, or extraction by basket-type endoscopic forceps (Gomco Equipment).[50] In one study, 44% of horses with chondroids were treated successfully by these noninvasive methods, although such methods can be slow and tedious.[41]

If the response to medical treatment is poor or chondroids have formed, surgical drainage of the guttural pouch should be considered (discussed later). Even when accumulated inspissated pus or chondroids have caused little external distention, the amount of material that can be removed surgically can far exceed what one might expect to be the maximum capacity of the guttural pouch. After surgery, the approach incision should be left open to drain and to allow repeated lavage and endoscopy of the pouch. In horses with severe dyspnea caused by guttural pouch distention, a tracheotomy should be performed.

If empyema is the result of occlusion of guttural pouch openings by adhesions, this occlusion may be relieved by blunt division through a surgical approach to the guttural pouch interior.[45] Chronic empyema of the guttural pouches, possibly unresponsive to medical therapy because of poor drainage through the pharyngeal ostia, can be successfully treated by using a laser to establish a permanent pharyngeal fistula into the guttural pouch.[38] In a pony with chondroids and occlusion of the pharyngeal ostium on the affected side by deformity and a fibrotic adhesion, the median septum was fenestrated from the healthy opposite guttural pouch with the Nd:YAG laser as a standing procedure.[51] This allowed immediate drainage of purulent material through the healthy side and repeated access for endoscopic snare or basket removal of all the chondroids. This approach was selected over the creation of a salpingopharyngeal fistula because a catheter could not be inserted into the diseased pouch to guide safe and effective laser penetration from the pharynx.[51]

Prognosis

Response to medical treatment and surgery is usually satisfactory. Neurologic signs usually resolve when the infection is brought under control.

Guttural Pouch Mycosis

Guttural pouch mycosis affects the roof of one guttural pouch, rarely both. There is no apparent age, sex, breed, or geographic predisposition to this disease, and it has been reported in foals.[52,53] The cause of guttural pouch mycosis is unknown, although *Aspergillus* spp can be identified in the lesion, typically as the only fungus or in combination with another.[54] *Aspergillus fumigatus* is the most common isolate, and it is more likely to be found by direct examination of biopsies than by culture.[54] The typical lesion of guttural pouch mycosis is a diphtheritic membrane of variable size, composed of necrotic tissue, cell debris, a variety of bacteria, and fungal mycelia.[28] Aneurysm formation rarely precedes or follows arterial invasion[53] and therefore is not essential to the pathogenesis of arterial rupture.

Clinical Signs

The most common clinical sign is moderate to severe epistaxis, which is caused by fungal erosion of the ICA in most cases (Figure 46-7) and of the ECA and maxillary arteries MA in approximately one third of cases (Figure 46-8, and see Figure 46-3).[52,55-59] However, any branch of the ECA, such as the caudal auricular artery, can be affected. Several bouts of hemorrhage usually precede a fatal episode. Mucus and dark blood continue to drain from the nostril on the affected side for days after acute hemorrhage ceases.

The second most common clinical sign is dysphagia caused by damage to the pharyngeal branches of the vagus and glossopharyngeal nerves (see Figure 46-7).[57] Aspiration pneumonia

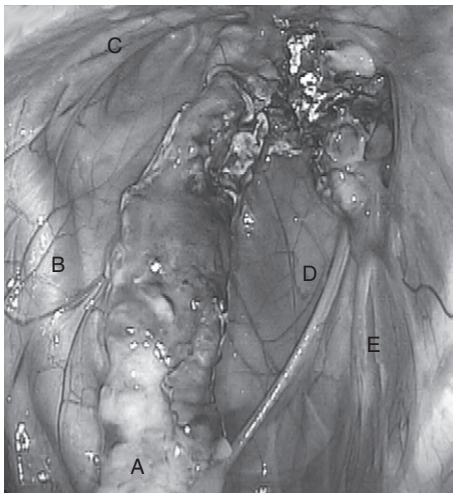

Figure 46-7. Guttural pouch mycosis in the typical location on the roof of the medial compartment (right guttural pouch) involving the internal carotid artery and causing epistaxis. *A,* Stylohyoid bone covered with diphtheritic membrane; *B,* external carotid artery; *C,* maxillary artery; *D,* mucosal fold that contains the glossopharyngeal and hypoglossal nerves; *E,* internal carotid artery. Extension of the plaque along the stylohyoid bone is not unusual and is rarely if ever clinically significant.

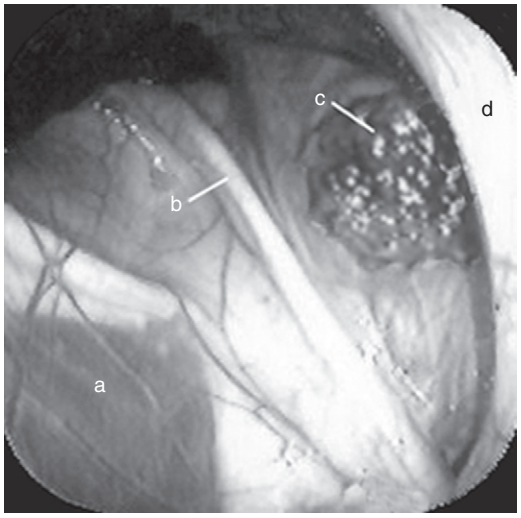

Figure 46-8. Roof of the left guttural pouch as viewed through retroflexion of the endoscope. *a*, Insertion of ventral straight muscles of the head; *b*, cartilaginous flap of the eustachian tube; *c*, mycotic lesion on the left maxillary artery; *d*, dorsal edge of the stylohyoid bone. This approach provides an excellent view of these structures when the rostral end of the guttural pouch is obscured with hemorrhage.

may develop in severe or protracted cases. Abnormal respiratory noise can arise from pharyngeal paresis or laryngeal hemiplegia, the latter as a result of recurrent laryngeal nerve damage.[57] Horner's syndrome may develop from damage to the cranial cervical ganglion and postganglionic sympathetic fibers. The classic signs associated with this denervation are ptosis, miosis, and enophthalmos; patchy cervical sweating; and congestion of the nasal mucosa. The reason for equine sweat glands to increase their activity when denervated is unclear.[60] Equine sweat gland myoepithelium is predominantly under α_2-adrenergic control, with additional α-adrenergic input from receptors.[60] However, sweating after neurectomy may be caused by increased peripheral vasodilation, which increases blood flow and skin temperature.[60] Ptosis is caused by a decreased tone of the superior tarsus muscle, and it is assessed by observing eyelash angles from a frontal view.[60] Pupillary response to decreased sympathetic tone in horses is variable, and the maximal difference in pupil size is usually slight.[60] Enophthalmos, which is the result of decreased smooth muscle retrobulbar tone and unopposed activity of the striated retractor bulbi muscle, is rarely obvious and usually evident as a slight protrusion of the nictitating membrane.[60]

Less common signs of guttural pouch mycosis that can develop singly or in combination with others are parotid pain, mucopurulent nasal discharge, abnormal head posture, head shyness, sweating and shivering, corneal ulcers, colic, blindness, locomotion disturbances, facial nerve paralysis, paralysis of the tongue, and septic arthritis of the atlantooccipital joint.[57-58,61-64]

Diagnosis

Endoscopy is critical for diagnosis, and it should be combined with history and clinical signs. On endoscopic examination of a horse with epistaxis, blood can be seen draining from the pharyngeal orifice. In horses with dysphagia, the roof of the pharynx can be collapsed, the soft palate can be displaced, and the nasopharynx may contain food material. The lesion appears as a white, tan, and black diphtheritic membrane on the roof of the affected guttural pouch (see Figure 46-7), and its size can vary but bears no relationship to the severity of clinical signs. Part of the diphtheritic membrane can coat the stylohyoid bone and the bone can be thickened, but clinical signs usually do not develop from this change. Fistulas may form into the opposite guttural pouch and pharynx[55] and true bilateral involvement has been described in 6 of 31 horses in one report.[53] The presence of serum antibodies to *Aspergillus fumigatus* detected by enzyme-linked immunosorbent assay (ELISA) cannot distinguish between horses with guttural pouch mycosis and healthy horses.[65]

Treatment

NONSURGICAL TREATMENT

The response to topical treatment is generally slow and inconsistent. Daily direct lavage through the endoscope can macerate the diphtheritic membrane, and the biopsy forceps or cytology brush of the endoscope can be used to detach it, provided any eroded artery was occluded beforehand. Topical povidone-iodine or thiabendazole, with or without dimethyl sulfoxide, has been used with mixed results.[55,59,64,66] Nystatin, natamycin, and miconazole have little activity against *Aspergillus* spp, but amphotericin B is effective against this organism, although systemic administration in the horse is limited by its toxicity.[67] Successful delivery of topical medication to the typical site of infection on the roof of the guttural pouch can be accomplished by transendoscopic delivery of a liquid preparation or by insufflation of a powder through a Neilson catheter using an enema syringe.[68]

Successful treatment of dysphagia from guttural pouch mycosis has been reported with a combination of itraconazole (5 mg/kg body weight PO) and topical enilconazole (60 mL of 33.3 mg/mL solution per daily flush) in one horse and with topical enilconazole alone in another.[66,69] Itraconazole at 3 mg/

kg twice a day in the feed can be effective against *Aspergillus* and other fungi in the nasal passage of horses, but treatment may be required for up to 4 or 5 months.[70] Bioavailability of another triazole antifungal agent, fluconazole, can be sufficiently high after oral and intravenous administration in horses to suggest a potential value in treatment of fungal infections.[71]

The response to any treatment method that is measured solely by disappearance of the mycotic lesion should be interpreted with caution because spontaneous regression of the lesion over a variable time course is typical. Horses with blood loss should be treated with polyionic fluids and, if necessary, with blood transfusions, and horses with dysphagia should be fed by nasogastric tube or by esophagostomy and should receive nonsteroidal anti-inflammatory drugs to reduce neuritis.

SURGICAL TREATMENT

The diphtheritic membrane can be detached by gentle swabbing and lavage through a modified Whitehouse approach. This treatment does not eliminate the risk of hemorrhage completely, and it does not retard or reverse progression of neurologic signs, but it does carry the risk of iatrogenic nerve damage and hemorrhage. In horses with epistaxis, the affected artery should be identified by endoscopy and surgically occluded (discussed later). There is some evidence that occlusion of the affected artery hastens spontaneous resolution of the mycotic lesion and thereby renders medical therapy unnecessary.[72] However, this is controversial and must be balanced against evidence that the mycotic plaque can undergo spontaneous regression[57] and within timeframes similar to those described in many horses that underwent arterial occlusion.[53,72] In a report on angiography and transarterial coil embolization in a large series of cases, the fungal lesion regressed completely on endoscopic examination between 30 and 180 days and incompletely between 51 and 269 days.[53] Such timeframes would be difficult to regard as hastened resolution. Also, a single case was reported in which the mycosis progressed and neurologic signs developed after coil embolization of the ICA.[73] Therefore the available evidence suggests that the effects of arterial occlusion on resolution of the mycosis is unknown.

Prognosis

Approximately 50% of horses with hemorrhage die from this complication, but this risk can be abolished or considerably reduced by the occlusion procedures described later.[57] These procedures must be performed as soon as possible after the first bout of hemorrhage to prevent subsequent bouts that could render the horse a poor candidate for anesthesia and surgery.

Although the mycotic lesion disappears with time, this is a very slow process,[53] and neurologic signs can persist after the lesion has disappeared. Recurrent laryngeal neuropathy is one of the more common clnical signs,[53] is typically permanent, but recovery has been reported.[58] Some horses with dysphagia eventually recover, but 6 to 18 months may be required and recovery may be incomplete.[57,58] Most horses recover from Horner's syndrome and facial nerve paralysis.[53]

Rupture of the Ventral Straight Muscles

Rupture of ventral straight muscles of the head (the longus capitis muscle and rectus capitis ventralis muscle) is usually caused by trauma, such as falling over backward, and it can cause severe epistaxis.[17] A nondisplaced impaction fracture at the basisphenoid–basioccipital junction associated with this injury can cause neurologic signs from subdural hemorrhage over the cerebral cortex and brain stem.[74] The same injury may occur without causing epistaxis.[74]

Clinical Signs and Diagnosis

Rupture of the ventral straight muscles causes severe epistaxis, as does guttural pouch mycosis.[18] Some horses show ataxia and other neurologic signs, and hemorrhage from torn muscle into the retropharyngeal tissues can compress the pharynx and even the trachea and cause respiratory obstruction.[17] Rupture of the ventral straight muscles is distinguished from guttural pouch mycosis by a history of trauma with the former, if such history is available, and by endoscopy.[17] In horses with rupture of the ventral straight muscles, the roof of the pharynx is collapsed and both guttural pouches are affected, usually more so on one side than on the other. Unlike in guttural pouch mycosis, the major arteries and the more caudal aspects of the guttural pouches are not involved, and there is no evidence of a diphtheritic membrane (Figure 46-9). Although some dark blood can be seen in the most rostral aspect of the pouch, it usually obscures the view of this area. A more complete view of the source of hemorrhage and swollen muscle bellies can be seen by retroversion of the endoscope (see Figure 46-8).[17] After swelling and inflammation from the original injury have subsided, any displaced bone fragment from a basisphenoid–basioccipital avulsion fracture can be seen on endoscopic examination of the medial compartment of the guttural pouch.[75]

On radiographs, partial obliteration of the guttural pouch by increased soft tissue density, soft tissue impingement on the roof of the pharynx, gas in soft tissues of the head and neck, and even avulsions of the basisphenoid bone may be evident (Figure 46-10).[75] The size and extent of the hematoma and gas in soft tissues can be determined by an ultrasonographic examination.

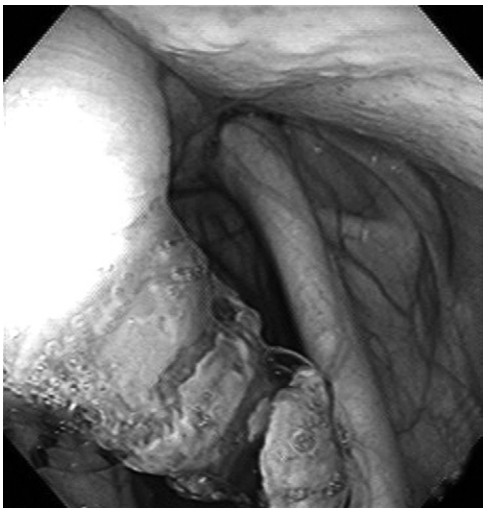

Figure 46-9. Endoscopic view of torn ventral straight muscles of the head in the left guttural pouch. The injury is rostral to the arteries in the caudal part of the guttural pouch. (Courtesy A. Walesby, Louisiana State University.)

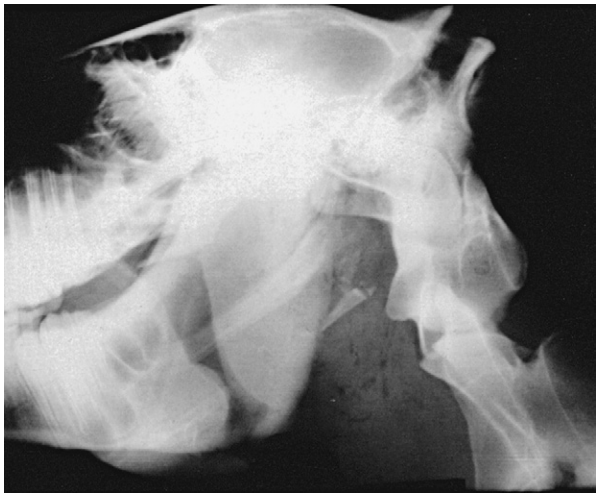

Figure 46-10. Radiograph demonstrating avulsion fracture of the basisphenoid bone, soft-tissue obliteration of the guttural pouch cavity, soft-tissue impingement on the pharynx, and gas in soft tissues in a horse with ruptured ventral straight muscles of the head. (Courtesy J. Foreman, Louisiana State University.)

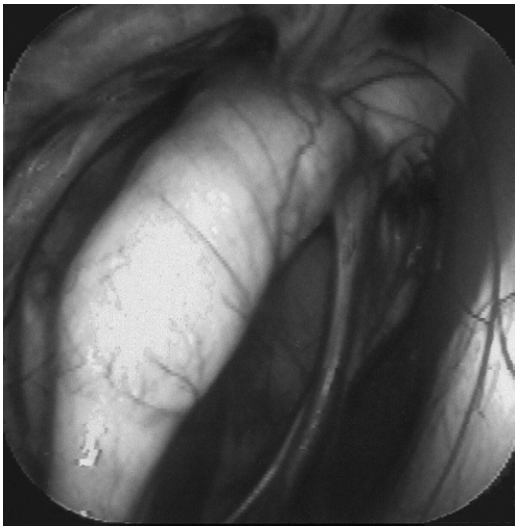

Figure 46-11. Thickened stylohyoid bone with involvement of the temporohyoid articulation in a horse with clinical signs of damage to the vestibulocochlear and facial nerves.

Treatment and Prognosis

Treatment is stall rest for 4 to 6 weeks and limiting head and neck movement that could disturb the damaged muscles and precipitate hemorrhage. Antibiotics are given to prevent secondary infections through the disrupted mucosa. The prognosis is good provided there are no neurologic signs, because these can be permanent and life threatening.[17] Severe neurologic signs, such as inability to rise, depression, and sluggish menace response, may be caused by an associated subdural hemorrhage overlying the cerebral cortex and brain stem and can warrant euthanasia.[74]

Temporohyoid Osteoarthropathy (Middle Ear Disease)

Temporohyoid osteoarthropathy (THO) is a progressive disease of the middle ear and components of the temporohyoid joint, such as the stylohyoid bone, the cartilaginous tympanohyoid, and the squamous portion of the temporal bone.[76] Horses of a wide age range and of any breed or either sex can be affected.[76] The cause is thought to be an inner or a middle ear infection of hematogenous origin that spreads to the aforementioned bones, causing them to thicken and the temporohyoid joint to fuse.[76] Other possible causes range from extension of otitis media or externa infection or guttural pouch infection to a nonseptic osteoarthritis.[77-80] Although guttural pouch mycosis can involve the same bony structures and temporohyoid articulation, clinical signs of temporohyoid osteoarthropathy are rare with this disease.[57] Recent CT and histopathologic evidence of an age-related and bilateral increase in severity of degenerative changes in the normal equine temporohyoid joint, although milder than the changes in horses with THO, suggest that degenerative, rather than infectious processes could underlie development of this disease.[13]

When the temporohyoid joint fuses and the associated bones thicken, forces generated by movement of the tongue and larynx during swallowing, vocalizing, combined head and neck movements, oral or dental examinations, and teeth floating may induce fractures of the petrous part of the temporal bone, resulting in facial nerve (CN VII) and vestibulocochlear nerve (CN VIII) dysfunction.[77-81] Severe new bone production and inflammation can damage the glossopharyngeal and vagus nerves where they leave the medulla caudal to the vestibulocochlear nerve.[1,82] After fracture of the petrous temporal bone, middle or inner ear infection could extend around the brain stem and involve additional cranial nerves and hindbrain structures.

Clinical Signs and Diagnosis

Early signs include head tossing, ear rubbing, refusing to take the bit, refusing to position the head properly when under saddle, resistance to digital pressure around the base of the ears or on the basihyoid bone, and other nonspecific behavioral changes.[76] The disease can cause an acute onset of signs consistently referable to the vestibulotrochlear nerve, including asymmetric ataxia, head tilt with the poll to the affected side, and spontaneous nystagmus with the slow component to the affected side.[76] These signs can be revealed or exacerbated by blindfolding. Signs of facial nerve damage, including paresis or paralysis of the ear on the affected side, deviation of the upper lip away from the affected side, decreased tear production, and inability to close the eyes, are evident in most cases.[81,82] Decreased tear production and inability to close the eyes may cause corneal ulcers, keratoconjunctivitis sicca, and exposure keratitis.[81,82] Dysphagia is rare but can result from damage to the glossopharyngeal and vagus nerves.[76]

Radiographs of the skull may depict proliferation and osteitis of the affected bones; however, endoscopy of the guttural pouch is in most cases a more sensitive method for detecting stylohyoid bone and temporohyoid joint involvement and hence for making the diagnosis (Figure 46-11).[83] Both sides should always be examined, because the disease can be bilateral, with only the more severely affected side showing obvious clinical signs.

Computed tomography (CT) can precisely demonstrate bony and soft tissue changes in the middle and inner ear,[16]

and consistently demonstrates osseous proliferation of the stylohyoid bone and temporohyoid articulation.[12] Also, thickening of the ceratohyoid bone and proliferation of its articulation with the stylohyoid bone can be identified on CT and these changes could have surgical implications.[12] The CT evaluation can also identify subclinical bilateral disease in horses determined to be unilaterally affected based on clinical examination. The stylohyoid bone is significantly wider in horses with neurologic deficits compared with the subclinically affected side.[12]

Treatment and Prognosis

Medical treatment includes broad-spectrum antibiotics for infection, nonsteroidal anti-inflammatory drugs to relieve pain and inflammation, and dimethyl sulfoxide to relieve inflammation.[76,84] Unilateral partial ostectomy of the stylohyoid bone has been used to create a pseudoarthrosis between the cut ends of the bone (Figure 46-12), which decreases the forces on the ankylosed temporohyoid joint and thereby prevents skull fractures.[84] In this procedure, approximately 2 to 3 cm (1 to 1½ inches) of the midbody of the stylohyoid bone is removed (see Figure 46-12). Although this procedure appears to have merit as a prophylactic measure against more-severe bone damage and associated neurologic consequences, it may cause transient dysphagia or injury to the hypoglossal nerve.[84] When performed as a bilateral procedure, it causes permanent problems with prehension.[84]

An additional complication of the partial ostectomy is regrowth of the stylohyoid bone approximately 6 months after surgical resection and recurrence of clinical signs.[82] Because of this complication, a ceratohyoidectomy can be performed as a safer, easier, and more permanent surgical alternative (see Figure 46-12).[82] For this, the horse is placed in dorsal recumbency and the ventral laryngeal region is prepared aseptically. A 10- to 15-cm (4½- to 7-inch) incision is made in the skin medial to the linguofacial vein on the affected side and centered on the basihyoid bone, approximately 2 cm from the midline as it crosses the basihyoid bone.[82] The fibers of the sternohyoid muscle are separated bluntly until the basihyoid bone is exposed. Rostral to the basihyoid bone, the geniohyoid muscle is separated to expose the ceratohyoid bone and, lateral to it, the hypoglossal nerve. The ceratohyoid–basihyoid synovial joint is identified and disarticulated with cartilage scissors. The transected end of the ceratohyoid bone is grasped and the ceratohyoid bone is freed from its attachments to the ceratohyoideus, the hyoideus transversus, and the genioglossus muscles. The hypoglossal nerve and lingual branches of the mandibular and glossopharyngeal nerves can be identified and gently retracted to protect them during these steps. An alternative is to separate the ceratohyoid bone from its soft tissue attachments with a narrow osteotome or periosteal elevator, thereby keeping dissection close to the bone and avoiding the need to expose the adjacent nerves. The cartilaginous articulation between the ceratohyoid bone and the stylohyoid bone is cut by cartilage scissors or continued elevation with the osteotome, taking care to avoid tension on the bone and temporohyoid joint. The separated muscle fibers can be sutured over the defect created by dissection to prevent reattachment of the bone ends, but the benefit of this step is unknown and could be regarded as optional. Subcutaneous tissues and skin are subsequently closed in routine fashion.

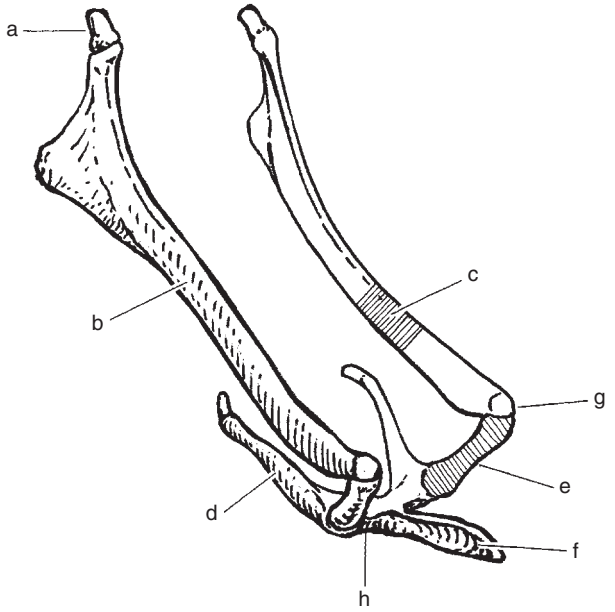

Figure 46-12. The hyoid apparatus, showing sites for ostectomy procedures *(shaded)* for horses with temporohyoid osteoarthropathy. *a*, Tympanohyoid; *b*, stylohyoid bone; *c*, site for partial ostectomy of the stylohyoid bone; *d*, thyrohyoid bone; *e*, ceratohyoid bone; *f*, lingual process; *g*, articulation of ceratohyoid bone with stylohyoid bone and epihyoid; *h*, basihyoid bone.

Although the prognosis is good according to one report, neurologic signs may persist in the majority of horses, especially if treatment is delayed.[77,83] Some degree of facial nerve paresis can persist.[7] In a study in which most horses were treated medically, some residual cranial nerve deficit persisted in the majority of horses, a year or longer was required for maximal improvement, and prognosis for return to some type of athletic function was fair.[80] In another study, substantial improvement was recorded at 1 year after surgery in 89% of horses that underwent a ceratohyoidectomy and in 87% that had a partial stylohyoid ostectomy, with most of the improvement developping within the first 6 months.[85] The corneal ulcers are difficult to treat, because there is an underlying problem with lid closure and tear production. A temporary tarsorrhaphy may help to manage the ocular complications until facial nerve function returns.

Miscellaneous

Although guttural pouch neoplasia is rare, squamous cell carcinoma, round cell sarcoma, fibroma, hemangioma, hemangiosarcoma, and parotid melanoma have been described.[11,86-92] In one case, bilateral squamous cell carcinoma of the guttural pouches and the left middle ear was initially evident as protrusion of a mass in one external acoustic meatus, without other obvious clinical signs.[89]

Parotid melanomatosis can appear as multilobular or multiple individual firm lumps in the parotid region of gray horses with extension into the guttural pouches (Figure 46-13) and may cause dyspnea and dysphagia from compression of the nasopharynx and larynx and facial nerve paralysis from compression of the facial nerve.[86,88] In one case, a malignant amelanotic melanoma caused halitosis, nasal discharge, and cough by

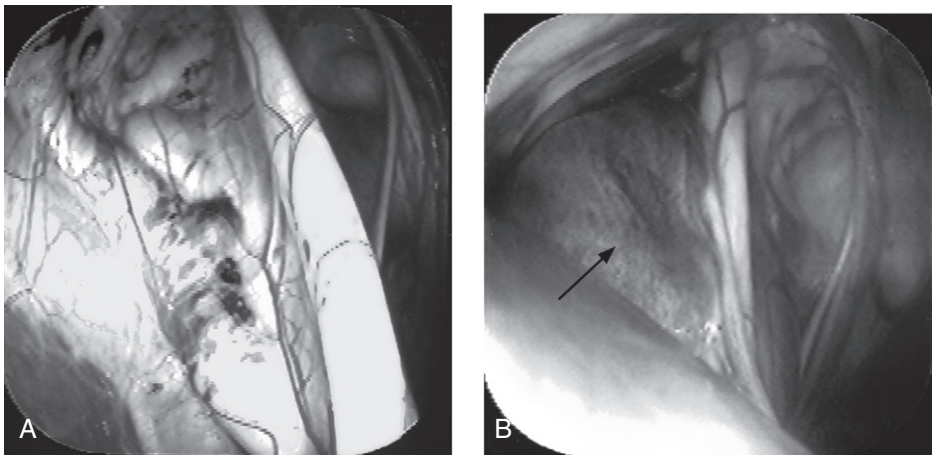

Figure 46-13. Endoscopic views of two horses with melanomas in the lateral compartments of their left guttural pouches that were not causing clinical problems. **A,** Typical melanin staining is scattered along the course of the external carotid artery and branches in an old gray horse without any external evidence of parotid involvement. **B,** A larger lesion *(arrow)* fills the lateral compartment and is evident externally as a firm mass over the parotid gland.

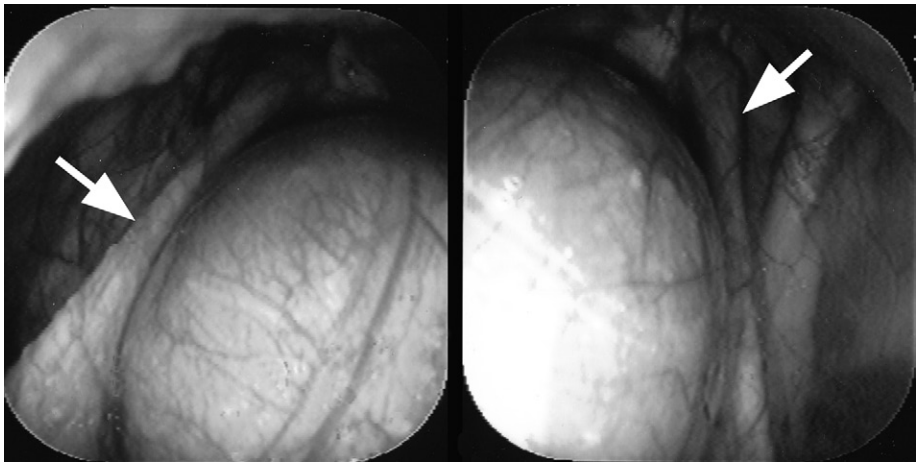

Figure 46-14. Bilateral guttural pouch cysts, each in the medial compartment of a Haflinger foal. *Arrows* point to the stylohyoid bones. This foal had nasal discharge, but the role of the cysts could not be established, there were no other clinical signs referable to them, and they were not treated.

undergoing central necrosis that extended from the guttural pouch into the pharynx.[18] The rate of growth of melanomas is variable and can be rapid after a prolonged initial growth, but even clinically evident lesions can be well tolerated in sedentary horses and allow years of continued health.[86,88] Smaller lesions may not be evident externally, and it is not unusual to find small discrete and scattered submucosal black patches and nodules in the guttural pouch of gray horses (see Figure 46-13). These are unlikely to cause problems. Extensive tumors may infiltrate the parotid gland without apparent lymph node involvement. Surgical resection is rarely indicated and is impossible in most cases because of the extent of infiltration and the close association with vital structures.[86]

Fracture of the stylohyoid bone can be secondary to guttural pouch mycosis or trauma.[11,28,86] Bone necrosis can follow, and clinical signs include dysphagia, pharyngeal swelling, empyema, keratoconjunctivitis sicca, and abscessation.[11,57,81] Reported foreign bodies include segments of catheters and wire.[93] Cystic structures have been described as causes of guttural pouch swelling and dyspnea in young horses (Figure 46-14) and respond favorably to surgical resection and fenestration in the median septum between the two guttural pouches.[94] The cause is unknown, but this lesion could be congenital or acquired.[94]

SURGICAL DRAINAGE OF THE GUTTURAL POUCH

The following approaches can be used to open the guttural pouch for removal of pus, mycotic plaques, and foreign bodies and to establish drainage.

Hyovertebrotomy

A 10-cm–long incision is made 2 cm cranial to and parallel to the wing of the atlas (Figure 46-15). The dense parotid fascia is incised, and the parotid gland and overlying parotidoauricularis muscle are reflected craniad. The guttural pouch lining is exposed beneath a covering of areolar tissue and grasped with rat-toothed or Allis tissue forceps. It is punctured with the closed tips of scissors or a hemostat, and this opening is enlarged by spreading its edges with a hemostat or the fingers. To establish ventral drainage, the pouch is opened ventrally through an incision in Viborg's triangle, guided by a finger within the

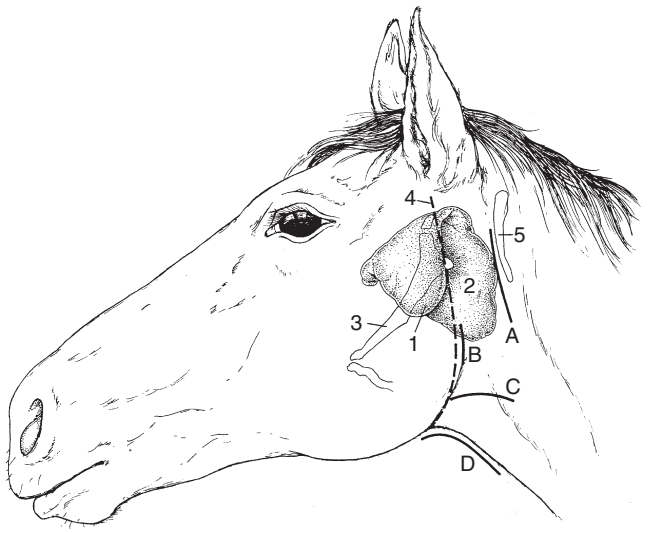

Figure 46-15. Surgical approaches to the guttural pouch. *A,* Hyovertebrotomy; *B,* Viborg's triangle; *C,* modified Whitehouse; *D,* Whitehouse. *1*, Lateral compartment of the guttural pouch, which is partly separated from the medial compartment *(2)* by the stylohyoid bone *(3); 4*, vertical ramus of the mandible; *5*, wing of the atlas. (Redrawn from Freeman DE: Diagnosis and treatment of diseases of the guttural pouch: Part II. Comp Cont Educ Pract Vet 2:S25, 1980.)

pouch. The hyovertebrotomy can be closed or left partly open for infusion of irrigating solutions.

Viborg's Triangle Approach

Viborg's triangle is bordered by the tendon of the sternocephalicus muscle, the linguofacial vein, and the vertical ramus of the mandible (see Figure 46-15). A vertical or horizontal incision is made in this area, taking care to avoid the parotid duct and branches of the vagus nerve along the floor of the guttural pouch. The incision is usually kept open with a soft rubber drain to establish ventral drainage.

Whitehouse Approach

With the horse in dorsal recumbency, a skin incision is made on the ventral midline over the larynx (see Figure 46-15). Dissection is continued between the paired sternohyoideus and omohyoideus muscles and along the larynx to the affected guttural pouch. The guttural pouch is opened medial to the stylohyoid bone, and care is taken to avoid the pharyngeal branch of the vagus nerve and the cranial laryngeal nerve, which are close to the incision.

Modified Whitehouse Approach

In the modified Whitehouse approach, the 12-cm skin incision is made along the ventral edge of the linguofacial artery and vein as if for a laryngoplasty, except 4 to 6 cm further rostrad (see Figure 46-15). The underlying fascia is incised to expose the lateral aspect of the larynx, and blunt dissection is continued until the guttural pouch cavity has been entered. Dissection through a natural fascial plane and avoidance of an incision between the sternohyoideus and omohyoideus muscles represent the the major advantages of this modification.

Advantages of both Whitehouse approaches are direct access to the roof of the guttural pouch, digital access to the lateral compartment, excellent ventral drainage, and simultaneous access through the septum to both compartments. Although both approaches involve deep dissection, they do not appear to have a higher rate of complications than the other approaches. The modified Whitehouse approach has been used in standing horses for management of guttural pouch empyema with inspissated pus.[43]

Modified Garm's Technique

A modified Garm's technique allows access to the lateral compartment of the guttural pouch and can be performed safely as a standing procedure for lavage and drainage in horses with mild empyema.[95] For this approach, a 6-cm skin incision is made 4 cm more rostrally than in Garm's original technique, between the ramus of the mandible and the submandibular lymph node.[95] Blind digital dissection is continued by this approach to the rostroventral aspect of the lateral compartment, where the mucosa can be perforated without risk to important vessels or nerves.[95] Because of the depth of dissection and the tight path created, little can be accomplished through this route except to insert a tube for lavage.

General Comments

Surgery of the guttural pouch through any approach should be a last resort because of risks of iatrogenic nerve damage. Identification of the guttural pouch lining and underlying nerves is difficult, especially in cases in which there is no distention, and can be facilitated by a lighted endoscope inserted into the medial compartment. A fixed structure, such as the stylohyoid bone, should be used as a guide for deep dissection, and entry through the guttural pouch lining is most safely accomplished by blunt penetration with the closed tips of a hemostat. The mucosa should not be incised with a scalpel, scissors, or other sharp instruments, and sharp-pointed retractors should be applied with care or preferably not at all to avoid nerve damage. Because all approaches enter the pouch cavity in the same approximate area, none provides less risk of nerve damage than others.

Open incisions in the guttural pouch are cleaned daily, and the guttural pouch cavity should be flushed daily with a nonirritating solution. Open incisions close spontaneously within 14 days, and the infection should also resolve within this time. Postoperative antibiotics can be given.

PROCEDURES FOR ARTERIAL OCCLUSION

In a horse with hemorrhage caused by guttural pouch mycosis, the involved artery or arteries should be occluded by one of the following procedures, or a combination of them, as soon as the diagnosis is made. The vessel to be occluded is determined by endoscopy. If accurate identification is impossible because landmarks are obscured by blood and/or a diphtheritic membrane, all arteries in the pouch should be occluded, and this can be done safely.[52] Arteriography may also be used to identify the affected vessel and unusual vascular anatomy.[96]

Ligation of the Common Carotid Artery and Branches

In horses with guttural pouch mycosis, fatal or severe hemorrhage has followed ligation of the affected ICA and could be attributed to occlusion of the wrong vessel or to retrograde flow, as from the cerebral arterial circle (circle of Willis).[56,58,97,98] A study was performed on six anesthetized adult horses to determine the effects of selective arterial occlusion on blood flow through the common carotid artery (CCA) and its branches and to thereby establish the most effective ligation for horses with life-threatening hemorrhage from these vessels.[99] Compared with control values, flow through the left ICA was reduced to 38% by simultaneous occlusion of the left and right CCA and to 19% by occlusion of the left ICA.[99] Occlusion of the left mid-cervical CCA induced retrograde flow through the left ICA that was 40% greater than control flow.[99] In the same study, flow through the left ECA was reduced to 30% of control values by occlusion of the left mid-cervical CCA or by simultaneous occlusion of the left and right CCA. Occlusion of the ECA or combined occlusion of the ECA and the major palatine artery reduced flow through the ECA to 1% of control values.[99] However, these data provide no information on blood pressure, which could remain sufficient after some of these ligations to cause substantial hemorrhage. Blood pressure in the segment of ICA in the guttural pouch is not reduced by ligation and is maintained for at least 3 days afterward at the same level as in the contralateral patent artery.[100]

Based on the preceding findings, ligation of the ipsilateral CCA in a horse bleeding from the ICA would increase flow in the affected artery and would be contraindicated; however, the same procedure may provide some immediate benefit in horses bleeding from the ECA and its branches, although any such benefit could be temporary.[56,58,99] Ligation of the affected ICA would decrease flow but not pressure, so bleeding could persist or recur; however, if access to definitive occlusion procedures is not immediate during a severe bleeding crisis, induction of general anesthesia to quiet the horse and ligation of the affected ICA could be attempted.

Success with ligation of the ICA can be attributed to development of a thrombus distal to the ligature over some time after surgery.[56,58] To prevent backflow, an additional ligature can be placed distal to the mycotic infection; however, this is difficult because the artery must be ligated deep within the guttural pouch, where it is likely to be obscured by the diphtheritic membrane.[98] The site for ligation of the ICA is immediately distal to its origin, outside the guttural pouch, using a similar but more ventral approach to a hyovertebrotomy (see Figure 46-15). The ICA is identified on the cardiac side of the occipital artery and deep to that vessel. In some horses, both arteries arise as a single trunk. If necessary, both ICAs can be ligated simultaneously without any apparent risk.[56,97] A transendoscopic clip ligation of the ICA has been evaluated for the standing sedated horse and was used successfully in 4 horses with epistaxis caused by guttural pouch mycosis.[101] However, critical details about the procedure are lacking.

The ECA can be ligated distal to the origin of the linguofacial trunk through an incision similar to that used for the ICA ligation but after extensive rostral dissection.[59] However, this procedure is generally unsuccessful because the ECA and MA have numerous collateral channels that allow retrograde flow to the affected segment.[1,52] Although ligation of the major palatine artery could prevent retrograde flow, a combination of this procedure with ligation of the ECA and ICA can cause ischemic optic neuropathy and permanent blindness.[102]

Balloon Catheter Occlusion of the Internal Carotid Artery

The balloon catheter technique allows immediate intravascular occlusion of the artery and prevents retrograde flow to the site of hemorrhage from the cerebral arterial circle (circle of Willis).[55,103] The ICA is ligated close to its origin, and an arteriotomy is made distal to the ligature (Figure 46-16). A size 6 French venous thrombectomy catheter is inserted through the

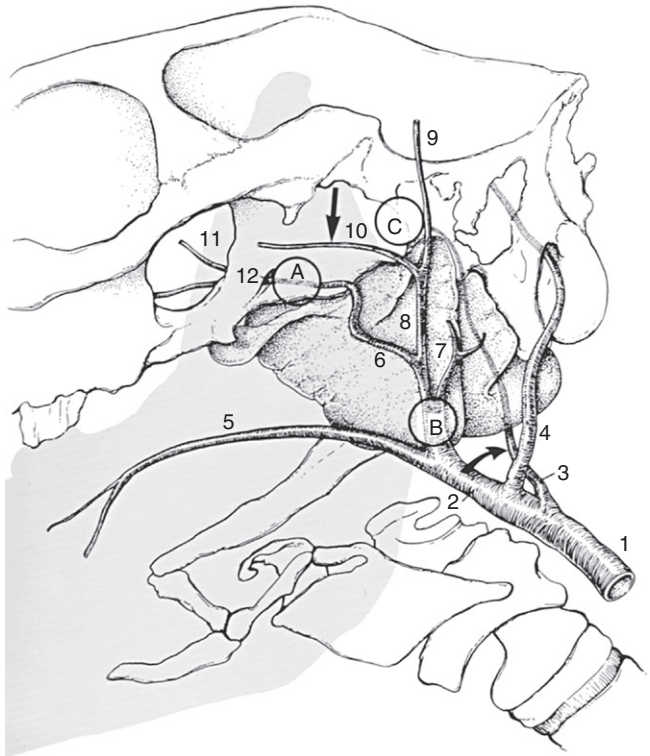

Figure 46-16. Diagram of major arteries close to and underlying the mucosa of the guttural pouch (numbers) and sites of balloon-catheter occlusion (letters). *1*, Common carotid artery; *2*, external carotid artery; *3*, internal carotid artery; *4*, occipital artery; *5*, linguofacial trunk; *6*, maxillary artery; *7*, caudal auricular artery; *8*, superficial temporal artery; *9*, rostral auricular artery; *10*, transverse facial artery; *11*, external ophthalmic artery; *12*, caudal alar foramen. *A,* Balloon inserted in the major palatine artery and guided in retrograde fashion to be inflated immediately caudal to the caudal alar foramen; *B,* balloon of catheter inserted into transverse facial artery *(10)* at *arrow* and directed into the external carotid artery, where it is inflated close to the floor of the guttural pouch; *C,* balloon in internal carotid artery at the sigmoid flexure, dorsal to the roof of the guttural pouch. This catheter is inserted into the internal carotid artery *(3)* at the *arrow*. *A, B, C,* and *arrow* on internal carotid artery are also the sites for obstruction with microcoils delivered through a catheter in the common carotid artery *(1)* in the upper third of the neck. (Redrawn from Caron JP, Fretz PB, Bailey JV, et al: Balloon-tipped catheter arterial occlusion for prevention of hemorrhage caused by guttural pouch mycosis: 13 cases. J Am Vet Assoc 191:345, 1987; and Smith KM, Barber SM: Guttural pouch hemorrhage associated with lesions of the maxillary artery in horses. Can Vet J 25:239, 1984.)

arteriotomy for a distance of approximately 13 cm.[103] At this distance, the balloon tip of the catheter is arrested at the sigmoid flexure of the ICA, within the venous sinuses and distal to the site of infection (see Figure 46-16). Fluoroscopy is not required to confirm placement, but intraoperative endoscopy can be helpful, although this can be difficult if landmarks are obscured by blood or the lesion. The catheter tip should be visible as it passes up the artery, and the balloon can be inflated at intervals to demonstrate its position. However, it does not distend the artery as dramatically as one would like, and this can make it difficult to locate. The balloon is inflated with sterile saline and secured in position by a ligature distal to the arteriotomy, and the redundant portion is buried as the incision is closed.

The horse may resume its usual activity when wound healing is complete and its packed cell volume has returned to normal. Perioperative antibiotics and nonsteroidal anti-inflammatory drugs can be used. It is not necessary to treat the fungal lesion or to remove the catheter after surgery, unless infection tracks along it and invades the surgical incision. For removal of the catheter, the horse is sedated and local anesthetic is infiltrated along and rostral to the original incision. A cut-down procedure is used to locate the coils of the catheter, which are then freed up and elevated. The balloon is deflated and the catheter is removed, usually with considerable traction to get it through the ligature distal to the site of insertion.

Complications

Complications associated with this procedure are rare.[52,55] The catheter rarely penetrates the defect in the artery, and if it does, it can be withdrawn and redirected. Alternatively, the catheter can be left in place to obstruct the hole while another catheter is directed alongside it. This mishap can be detected by intraoperative endoscopy. Infection of the surgical site should be treated by removing the catheter and establishing drainage, and it can be prevented by inflating the balloon to a sufficient diameter (at least 8 mm) to prevent displacement into the infected segment.[55,64] Delayed prolapse of the catheter through the hole in the artery into the guttural pouch is favored by the normal pressure distal to the balloon and the loss of pressure proximal to it (heart side). The use of the more rigid commercially available catheters can also prevent this complication. One of us treated a horse that had undergone balloon catheterization for guttural pouch mycosis 4 years before presenting with a draining tract at the site of catheter placement (Figure 46-17). Although the embolization catheter was removed with some difficulty, the horse developed a fever and severe seizures and was euthanized 2 days after catheter removal. The post-mortem finding of a septic meningitis demonstrates the importance of catheter removal when signs of infection are evident.

Failure to prevent fatal hemorrhage in one case was caused by inadvertent catheterization and occlusion of an aberrant branch from the ICA, which left the affected segment of artery open to retrograde blood flow.[104] To prevent this mishap, approximately 6 cm of the ICA should be exposed to locate any aberrant branch. Such a branch should be ligated so that the catheter can be maintained in the ICA.[104] The most troublesome anatomic aberration is an ICA that leads to the caudal cerebellar artery without following its usual pathway, and that places the inflated balloon in a position that causes neuronal necrosis of the brain stem. If a catheter cannot be inserted to the required distance or passes well beyond the 13-cm mark before it becomes arrested, then it is probably in an aberrant branch.

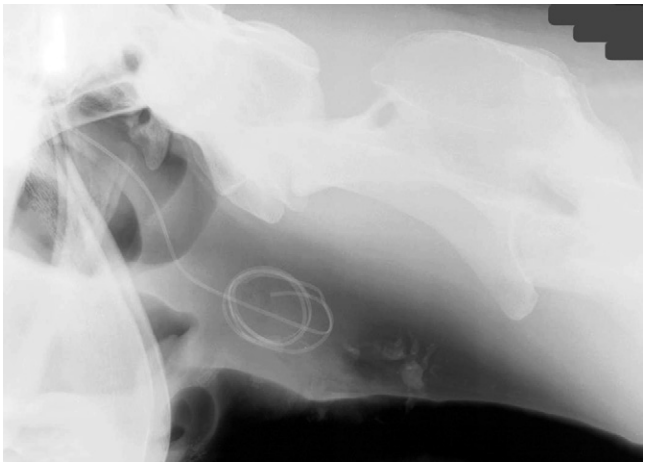

Figure 46-17. Lateral contrast fistulography of the a horse with a draining tract in the throatlatch of approximately 1 year's duration; the draining tract communicated with a previously placed ballooon-tipped catheter placed 4 years previously.

It may be difficult to distinguish between the occipital artery and ICA in some horses, especially those with a thick throatlatch and those in which both arteries arise as a single trunk and bifurcate at a variable distance from the CCA. If both arteries arise in the normal fashion, the recommended method for identification is to dissect them both free for a couple of centimeters and then elevate each one gently with umbilical tape or a Penrose drain. It should be possible to demonstrate that the ICA lies deep to the occipital artery and that it courses more rostrad.

Balloon Catheter Occlusion of the External Carotid Artery and Its Branches

The most likely source of retrograde flow to the ECA and its branches is the major palatine artery, which is a large continuation of the MA.[1,52] This joins the contralateral major palatine artery behind the upper row of incisor teeth to form a large arterial loop around the upper jaw. Attempts to occlude the MA by a single balloon-tipped catheter in the ECA have failed because the catheter tip can readily enter the superficial temporal artery rather than the MA (see Figure 46-16).[52,55] The following procedure was developed to overcome these difficulties.

To prevent normograde flow, the ECA is ligated after the linguofacial trunk branches and, to reduce retrograde flow, a size 6 French Fogarty venous thrombectomy catheter is inserted into the major palatine artery, 3 cm caudal to the corner incisor tooth.[52] This catheter is inserted retrograde for approximately 40 to 42 cm in a 450 kg horse, or the shortest distance from the arteriotomy to the articular tubercle of the temporal bone.[52] The balloon is partly inflated and the catheter is gently retracted until some resistance is encountered, at which point it is assumed that the balloon is at the caudal alar foramen (see Figure 46-16).[52] It is subsequently fully inflated with sterile saline. At this site, the balloon can obstruct retrograde flow to the MA (see Figure 46-16).

As an alternative to ligation, the ECA can be occluded distal to the origin of the linguofacial trunk by a balloon catheter

inserted through the transverse facial artery.[52] This obviates the difficulty of exposing the artery through a hyovertebrotomy and therefore is most useful in a horse that does not require ligation of the ICA. The catheter is inserted 3 cm rostral to the articular tubercle of the temporal bone and advanced in retrograde fashion until its tip enters the ECA (approximately 12 cm from the arteriotomy site in a 450-kg horse) (see Figure 46-16).[52] The balloon is inflated with saline. The redundant ends of catheters in the transverse facial and major palatine arteries are taped to the head or incorporated into a stockinette hood. These catheters are removed after 7 to 10 days without sedation or local anesthesia.

Although the MA has many collateral branches in the segment between the balloons, this procedure has been effective to date and does not cause blindness, even when combined with occlusion of the ICA.[52] This difference from ligation of the major palatine artery can be attributed to prevention of the "steal phenomenon" that occurs with ligation.[100,105] However, the owner should be warned of the risk of blindness in any procedure that involves occlusion of the ECA and its branches, even if the risk is very low.

Occlusion of the Internal Carotid Artery with a Detachable Balloon Catheter System

A detachable, self-sealing, latex balloon was described to successfully occlude the ICA without the need for catheter removal, as required in some cases treated with the nondetachable balloons.[106] Combined with angiography, the detachable system can also be used to occlude aberrant vessels that originate at a distance from the origin of the ICA.[107]

The ICA is approached as described earlier (see Figure 46-16), and a 3-cm segment, 1 to 2 cm distal to the origin of the ICA, is isolated between two Rummel tourniquets of umbilical tape and polyethylene tubing. The balloon delivery system consists of a Tuohy-Borst adapter (rotating Y adapter) that is attached to an 8-French, 95-cm nontapered, thin-walled guiding catheter (GC 8/95). A 2-French, 135-cm balloon-carrier microcatheter (Mini-Tourquer, CTFN 135) is placed through the adapter and guiding catheter, and an 8.5-mm–diameter, detachable, self-sealing latex balloon (GoldValve Balloon, GVB 17) inflated with 0.5 mL of a 1:1 solution of physiologic saline solution and 66.8% iothalamate sodium (Conray 400) is mounted onto the carrier microcatheter. The balloon is subsequently deflated, the carrier microcatheter is withdrawn so that the balloon is recessed 2 mm inside the guiding catheter, and the microcatheter is held in place by tightening the O-ring of the Tuohy-Borst adapter.[106]

A 19-gauge arterial access needle is inserted into the ICA between the ligatures, and a 0.9-mm guide wire is placed through the needle into the artery.[106] The needle is withdrawn over the wire and an 8-French introducer sheath (Pinnacle Introducer Sheath) is advanced over the wire for 4 cm. The dilator and wire are removed, and retrograde blood flow from the cerebral arterial circle is confirmed by bleed-back through the ancillary fluid port of the introducer sheath. Continuous infusion of heparinized saline solution (4 U/mL, approximately 3 mL/min) is then maintained through the introducer sheath. The balloon delivery system is inserted through the flexible diaphragm of the introducer sheath and advanced approximately 13 cm into the ICA or until resistance is met. The carrier microcatheter is advanced 5 to 10 mm within the guiding catheter while the guiding catheter is retracted 1 cm over the microcatheter. The balloon is subsequently inflated with 0.5 mL of the radiopaque solution, and its appropriate positioning and degree of inflation are confirmed by a single lateral intraoperative radiograph. The balloon is detached by gentle traction on the carrier microcatheter, and all proximal ligatures are secured. All catheters are withdrawn from the introducer sheath and immediate occlusion of the distal ICA is confirmed by lack of retrograde blood flow through the ancillary fluid port of the introducer sheath. The introducer sheath is subsequently removed and the proximal Rummel tourniquet is replaced with two ligatures of No. 0 polypropylene. The subcutaneous tissues and skin are closed in layers.

Transarterial Coil Embolization

A transarterial coil embolization technique can selectively occlude the arterial segments involved in a mycotic lesion in horses with guttural pouch mycosis.[53,108] The coil embolization technique combines angiographic studies to image the affected vessels and identify any unusual vessels and sites of bleeding, followed by a selective embolization or occlusion of the affected vessels. In this procedure, a stainless steel coil of appropriate size expands as it emerges from the catheter so that it becomes lodged within the artery, and the Dacron fibers on its surface induce rapid thrombosis in the occluded segment. Compared with the balloon catheter technique, transarterial coil embolization allows visualization of affected vessels throughout the procedure as it is performed under fluoroscopic guidance. This is critical, because aberrant vasculature has been described in horses with guttural pouch mycosis, and failure to identify and occlude such aberrant branches may result in fatal hemorrhage.[104,109] Also, this procedure eliminates the need to protect redundant ends of catheters, which is necessary when nondetachable balloons are used. It is less invasive than the original balloon catheter procedures and requires shorter anesthesia and shorter hospitalization. Transarterial embolization can be performed during active bleeding. The surgical approach for all arteries in the guttural pouch is the CCA exposed through a single incision.

Under general anesthesia, the horse is placed in lateral recumbency with the affected side uppermost, and the head and neck are placed on a radiolucent surface, such as a 30 × 30 cm sheet of Plexiglas, to allow intraoperative fluoroscopy to be performed (Figure 46-18).[110] The fluoroscopy unit must be mobile and covered with a sterile cover if it is to be manipulated intraoperatively by the surgeon. All surgical and anesthesia personnel must wear protective lead aprons during fluoroscopy (see Figure 46-18). The proximal aspect of the jugular groove is clipped and prepared and draped for aseptic surgery. Drapes must cover the front half of the horse, because catheters and guide wires used for the procedure are long and could get contaminated. All catheters, guide wires, and introducers are flushed with heparinized saline (10 IU/mL) and left soaking in a bowl containing heparinized saline until use. A single surgical approach is sufficient to allow access to all affected branches of the CCA, such as the ICA, the ECA, the MA, and aberrant branches (see Figure 46-18). In general, all four sites are occluded when lesions are extensive (see Figure 46-16) or when preoperative hemorrhage precludes identification of the affected vessel. When the lesion is focal, only the affected vessel is occluded.

Figure 46-18. Room organization for coil embolization techniques. The horse's head rests on a thick Plexiglas sheet supported by the surgery table and a surgery cart so that the fluoroscopy unit can be positioned more accurately. The fluoroscope head can be covered with a sterile drape if the surgeon is to manipulate it into position. The surgeon is directed by the image on the monitor to guide catheter advancement. Note the position of the radiation-shielding panels and that the surgeon is wearing radiation protection beneath the gown. The horse's head is exposed for demonstration purposes but should be covered with a sterile drape to prevent contamination of the long catheters.

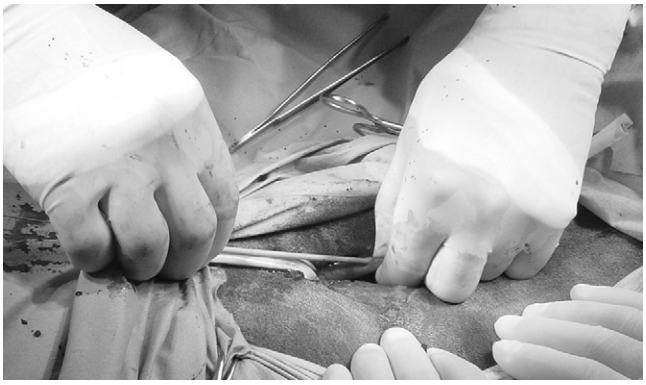

Figure 46-19. The carotid artery is elevated with umbilical tape and is punctured with an angiographic needle. A 6-French introducer system (Cook Inc., Bloomington, IN) is inserted into the artery and guided toward the head.

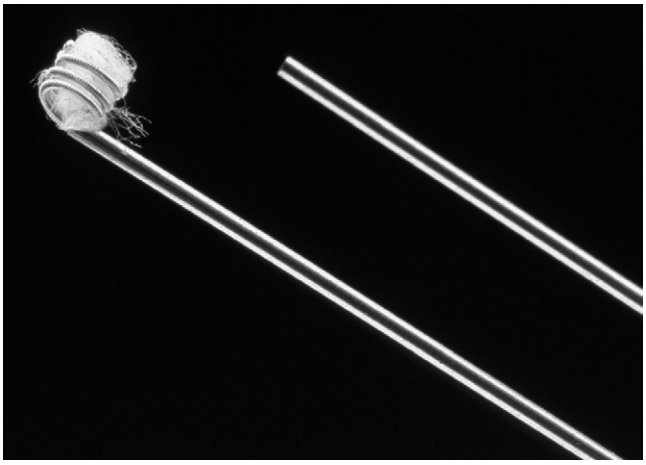

Figure 46-20. Microcoil used for coil embolization. The coils are made of stainless steel coated with Dacron fibers and presented in a stainless steel sheath. Coil formation is achieved by pushing the guide wire into the sheath *(right)* to deliver the coil *(left)* at the desired location.

An 8-cm skin incision is made at the junction of the proximal and middle thirds of the neck, just above the jugular vein.[110] This distal location is important to avoid exposure of the surgeon's hands during fluoroscopy (see Figure 46-18). The brachiocephalicus and omohyoid muscles are bluntly separated, and the carotid sheath is elevated by blunt finger dissection. The vagosympathetic trunk is carefully separated from the CCA and replaced within the incision. Care must be taken to prevent tension on the nerves, which could result in postoperative complications, such as Horner's syndrome or recurrent laryngeal neuropathy. The CCA is elevated with umbilical tape and is punctured with an angiographic needle. A 6-French introducer system is inserted in the artery and guided toward the head (Figure 46-19). The correct position of the introducer is verified by injection of contrast material before proceeding. Misdirection into the cranial thyroid artery precludes advancement of the angiography catheter and requires that the introducer be slightly withdrawn and repositioned into the CCA under fluoroscopic guidance. A 6-French single-end–hole nylon angiographic catheter is advanced rostrad into the CCA to the level of the ICA under fluoroscopic guidance. Coils pass less readily through polyvinylchloride or vinyl catheters, so these should not be used. Angiography is performed during catheter advancement by hand-injection of 3 to 5 mL of a 1 : 2 solution of iohexol in heparinized saline, or iodinated contrast material, to identify the path of each vessel. All injections are made using a double-flush technique through a three-way stopcock. To avoid embolization of thrombi that may have formed within the catheter, heparinized saline is flushed and aspirated before injection of contrast material. Injection of air must be avoided by carefully expelling air from all syringes and by performing injections with the piston of the syringe directed upward.

Preembolization angiography is mandatory for anatomic identification and location of the vessels, exclusion of vascular anomalies, abnormal vascular connections between the ICA and the occipital artery, and correct positioning of the embolization coils.[110] Small or aberrant arterial branches are present in up to one third of horses and require selective embolization. If a connection is present between two embolization sites, continued bleeding could still occur, and therefore such small branches must be occluded separately. The distal (cerebral) side of the lesion in the ICA is embolized first, to protect the cerebral circulation from any intraoperative errors, such as air or clot embolization. The distal ICA is embolized at the level of its superimposition on the basisphenoid bone and caudal to the sigmoid flexure. To select a coil diameter, an estimate of the width of the artery at the embolization site is made by using the known diameter (3 mm) of the angiography catheter as a guide. For the distal ICA coil, diameters of 5 to 8 mm are usually necessary, although 3 mm may be adequate in smaller horses or ponies. A Dacron fiber–covered, stainless steel, occluding spring embolization coil (Embolization Coils) of proper diameter is introduced through the catheter, while its tip is in the desired location, and pushed into the ICA by the stiff end of a guide wire (Figure 46-20). A coil slightly larger than the artery

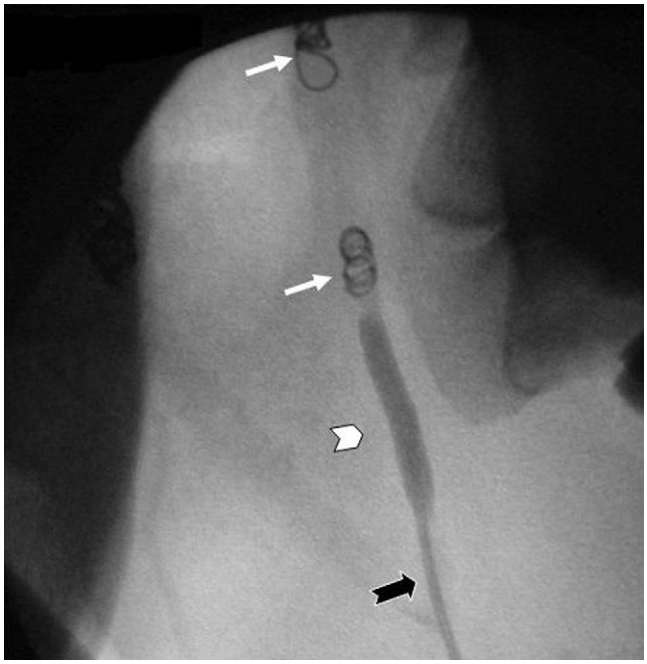

Figure 46-21. Fluoroscopic image obtained during coil embolization showing location of the distal and proximal coils within the internal carotid artery (*white arrows*), the contrast material with complete occlusion of the artery *(arrowhead),* and the angiography catheter within the internal carotid artery *(black arrow).*

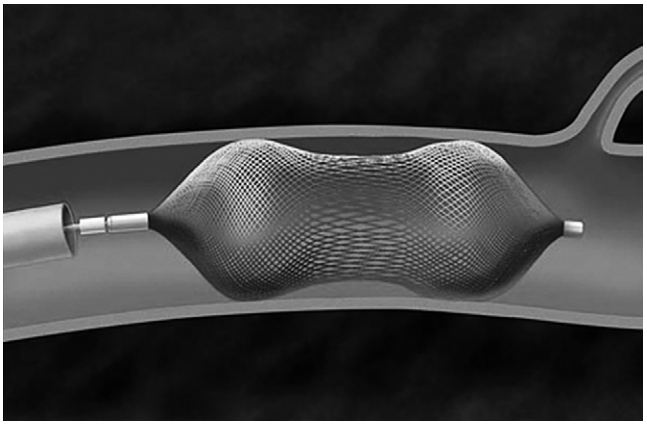

Figure 46-22. Nitinol intravascular plug in an appropriately sized artery demonstrating the dumbbell configuration when impacted at the desired level of obstruction.

is placed first, and additional smaller imbricating embolization coils follow until complete occlusion is obtained. To imbricate the coils, the tip of the catheter is placed within the previous coil.

After occlusion is verified by injection of contrast material, the proximal (cardiac) side of the ICA is embolized next, midway between the first embolization site and its origin from the CCA (Figure 46-21). After occlusion of the ICA, the catheter is withdrawn into the CCA and advanced to repeat the same procedure in the caudal MA and rostral ECA. The MA is embolized distal to the superficial temporal and proximal to the infraorbital, buccal, and mandibular alveolar arteries. Correct identification of the MA is verified by inserting the catheter farther and imaging the ophthalmic artery. The catheter is subsequently withdrawn until it is within the alar foramen, just distal to the curvature of the MA, and cranial to the most cranial aspect of the guttural pouch outline. Coils of 8 to 12 mm in diameter are necessary at this location. The ECA is embolized next, on the caudal (cardiac) side of the origin of the caudal auricular artery. The ECA requires the largest size and greatest number of coils to achieve immediate and complete occlusion, usually 10 to 15 mm diameter.

After coil placement, the catheter and introducer system are removed.[110] The CCA puncture site is closed using 5-0 silk in a cruciate pattern, and the muscle layers and skin are closed in three layers. Phenylbutazone is administered for 3 to 5 days after surgery to minimize inflammation at the incision site. Horses are rested for 30 days and then gradually returned to use.

The disadvantages of this technique are the need for fluoroscopy (and the specialized equipment and expertise involved), positioning of the horse's head for fluoroscopy, and radiation-shielding apparel and equipment. Although these disadvantages would limit this technique to a small number of well-equipped hospitals, coils have been implanted in the ICA using a cut-down procedure similar to that for the balloon catheters.[111,112] Placement can be determined as for balloon catheters and confirmed by lack of backflow through the arteriotomy and assessment of coil placement by radiography.[111] The same approach has been described with an allowance of 10 minutes after coil placement for a clot to form, followed by an injection of contrast material at 200 mm Hg for 3 seconds to determine occlusion.[112] This direct approach to the ICA also obviates the need for a proximal coil occlusion, because a ligature at the site of catheter insertion will prevent normograde flow. The major disadvantages of the cut-down procedure are failure to identify unusual anatomy and aberrant connections and to precisely position the microcoils.

Transarterial Nitinol Vascular Occlusion Plug Embolization

Transarterial occlusion using nitinol plugs was recently described in three horses.[113] A nitinol vascular plug is a self-expanding nickel-titanium wire mesh that deforms from its fully expanded cylindrical shape to a dumbbell configuration when delivered into the vessel to be occluded (Figure 46-22). Both ends are marked with radiopaque platinum marker bands (Figure 46-23) and one end is attached at to a flexible delivery cable from which the plug can be detached as needed by counterclockwise rotation. The procedure is performed under fluoroscopic guidance using an approach similar to that for coil embolization. When the CCA has been isolated, a 7-French vascular access sheath is introduced and angiography is performed. Vascular plugs are placed on the cardiac and cerebral side of the lesion, as with other occlusion techniques; the size needed is based on angiography, with the diameter being 130% to 150% of the angiographic diameter of the artery at the proposed site of occlusion. Available sizes range from 4 to 16 mm.[113] As with other occlusion techniques, if the location of the bleed cannot be identified either preoperatively or intraoperatively, all possible sites should be occluded, because at least one third of the lesions involve the MA. This would require using six vascular

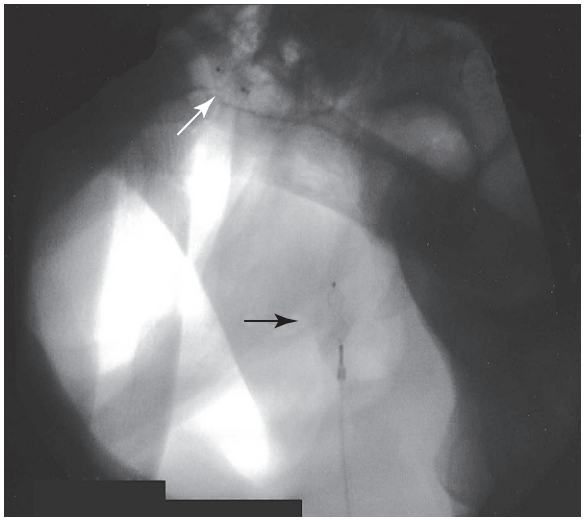

Figure 46-23. Nitinol intravascular plugs placed in the internal carotid artery distal *(white arrow)* and proximal *(black arrow)* to the lesion in a horse with guttural pouch mycosis on this artery. The plugs can be identified by the small radiopaque platinum marker beads at each end. In the case of the proximal plug, the delivery catheter is still attached and the mesh portion of the plug can be seen in its typical dumbbell shape.

plugs. In addition, angiographic identification and possibly occlusion of aberrant vasculature should be performed. In one report, cessation of active bleeding in the MA after vascular plug occlusion took 15 to 20 seconds, but it can take up to 10 minutes when used in large vessels.[113]

Comparisons of Methods for Arterial Occlusion

Since the limitations of ligation methods were recognized, the challenges of occluding the complex vasculature of the terminal branches of the equine carotid arterial system have been addressed through an evolution of methods, such as balloon catheters, detachable balloons, microcoils, and nitinol plugs. The last three transcatheter methods and particularly the last two have emerged as the methods of choice in most university veterinary hospitals, where the necessary equipment is available through an association with small animal cardiologists and interventional radiologists. In fact, the growth of embolization techniques for small animal vascular lesions obviates the need for purchase and storage of expensive equipment in the associated equine hospitals, so that inventory, storage lives, and expertise are no longer limiting factors for use of this methodology in horses in most university hospitals.

The goals of transcatheter techniques should be accurate identification of the involved artery and location of any complicating or affected aberrant branches, accurate and secure placement of the occlusion device, and complete interruption of blood flow to the site of hemorrhage. The transarterial coils and nitinol plugs would seem to come closest to achieving these goals. Atypical anatomy and aberrant branches may be more common than reported with this disease, so preocclusion angiography is critical. The ability to selectively occlude affected vessels with a minimally invasive approach makes transarterial coil or nitinol embolization the preferred treatments.

Coils and plugs are inserted through catheters that can be guided into the relevant arterial branches under fluoroscopic guidance beforehand, and such catheters are designed for accurate placement in multiple vessels remote to a single site of catheter insertion. By comparison, the detachable balloon is delivered without prior selective catheterization of the affected vessel and within the delivery catheter, which is more difficult to manipulate into place if the target segment is a remote branch of the catheterized artery. This can be partly overcome by direct catheterization of the affected artery, such as the ICA, but is a disadvantage if additional arteries need to be occluded. Also, detachable balloons are not currently as readily available as the other devices. We are not aware of a report on use of the detachable balloon system in the ECA and its branches in horses.

One difference between the transarterial coils and the nitinol vascular plug is that a single nitinol plug is delivered at each site whereas at least two transarterial coils are typically used at each site to effectively stop blood flow. The nitinol plug can also be retracted into the delivery cable if unsatisfactory placement is noted, and the plug can also be retrieved after it has been released. Migration or dislodgement from the target vessel is very unlikely because the expanded plug applies radial tension to the arterial wall.[113] There is little difference in cost between the two methods and both suffer from the same disadvantages, such as need for specialized equipment and the expertise of an interventional cardiologist.

Failures following the use of nondetachable balloon catheters and transcatheter embolization methods are rare. In cases of successful arterial occlusion, a small volume of dark blood and serum typically drains from the affected guttural pouch for many days after surgery, but this is from residual blood within the guttural pouch and does not signify ongoing hemorrhage. Hemorrhage from a failed occlusion is typically severe and happens in the recovery stall, leading to death or euthanasia.[53,104] Hemorrhage following occlusion with a nondetachable balloon catheter is typically caused by failure to occlude an aberrant branch that was the site of arterial erosion or that allowed persistent retrograde flow to the site of hemorrhage.[104]

In 27 horses that had transarterial coil embolization for treatment of epistaxis in two hospitals,[53,108] only two had epistaxis after arterial occlusion. In one of these, the disease was bilateral, but arterial occlusion was completed on one side only and the horse subsequently bled to death from the MA on the side that was not occluded.[53] Based on this experience, bilateral arterial occlusion during the same surgery should be considered in such cases.[53] In the second failure, the affected ICA could not be occluded on the rostral (cephalic) side of the lesion and the horse bled in the recovery stall and was euthanatized, despite sucessful occlusion on the cardiac side of the lesion.[53] Failure to place coils in the ICA rostral to the site of hemorrhage has been reported in 3 of 27 horses and attributed to a thrombus in that portion of the vessel or to narrowing of the vessel.[53,110] In another horse, a sharp turn in the MA made it difficult to place a nitinol plug rostral to the lesion.[113] When such difficulties arise, the catheter can dislodge a thrombus and cause intraoperative bleeding.[53,114] Possibly the rigid types of thrombectomy catheter that are commercially available can be advanced beyond any form of distal arterial obstruction or narrowing and can thereby be successfully placed rostral to the site of hemorrhage. Nonetheless, the transcatheter embolization systems (coils and nitinol plugs) remain the methods of choice in those hospitals equipped to use them.

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CHAPTER 47

Trachea

John A. Stick

Although conditions of the equine trachea requiring surgery are rare, those that may require surgical intervention include obstruction of the airway rostral to the trachea, perforation or rupture of the trachea, tracheal collapse, tracheal stenosis, bronchial foreign body, and mucosal granulomas within the trachea lumen. Surgical treatments include tracheotomy, tracheostomy (both temporary and permanent), tracheal resection and anastomosis, and tracheal reconstruction.Successful surgical management requires knowledge of normal anatomy and physiology.

ANATOMY AND PHYSIOLOGY

The equine trachea is a membranous and cartilaginous tube extending from the larynx at the level of the first or second cervical vertebra to the level of the fifth or sixth intercostal space, where it bifurcates into the principal bronchi dorsal to the base of the heart.[1] The trachea is median in position except for its very terminal thoracic part, where it is pushed to the right by the aortic arch. From just caudal to the larynx to its bifurcation, the equine trachea is about 70 to 80 cm (28 to 32 inches) long.

When operating on the trachea, its relationship to other structures is important. The arrangement of the muscles around the trachea should be noted, as well as nerve trunks, vessels, and the esophagus. The sternothyrohyoid muscles are located immediately ventral to the trachea, except at the most cranial extremity of the trachea, where it is related only to the sternohyoid muscle. The sternocephalic, omohyoid, and sternothyroid muscles are found lateral to the trachea near the larynx. An easy spot to perform a tracheotomy is where the sternocephalic muscle bellies diverge and the omohyoid muscle converges. From this area cranial to the larynx, the trachea can be easily palpated. The esophagus is located dorsolaterally to the trachea in the middle of the neck but comes to lie lateral to it in the caudal part of the neck and assumes a position ventral to the trachea at the thoracic inlet. This is another reason the tracheotomy should be performed in the proximal half of the trachea. The common carotid artery, vagosympathetic nerve trunk, and recurrent laryngeal nerve all are enclosed in the carotid sheath and lie alongside the dorsolateral aspects of the trachea. These structures come to lie more laterally near the thoracic inlet. Therefore, when surgery is performed in this area, caution needs to be taken. A tracheotomy cannot be performed blindly. In a young animal, the thymus may extend for a distance into the neck from the thoracic cavity and lie on the ventral and lateral aspects of the trachea. Additionally, in the proximal-most aspect of the trachea, the thyroid gland may have an isthmus that could be encountered if the trachea is surgically approached just caudal to the larynx.

The wall of the trachea can be divided into four layers: mucosa, submucosa, musculocartilaginous layer, and adventitia. The adventitia is a connective tissue layer that blends with the musculocutaneous layer and with the connective tissue surrounding the trachea. It is composed of loose areolar tissue that allows a considerable amount of movement of the trachea along its length. The musculocartilaginous layer consists of the cartilaginous plates, fibroelastic tissue (annular ligaments), and the tracheal muscle. The cartilaginous plates number between 48 and 60 and are composed of hyaline cartilage surrounded by perichondrium.[1] Vascular canals are consistently found within the cartilaginous plates and become obvious when they are traumatized or when incisions are made into the cartilaginous plates. These cartilaginous plates are curved and form incomplete hoops that are open dorsad. Tracheal muscle consisting of smooth muscle fibers extends transversely across the dorsal part of the wall and is attached to the inner surface of the tracheal plates, forming the dorsal tracheal ligament. The tracheal lumen can vary considerably in size as the result of the action of the tracheal muscle, which is facilitated in the cervical region by overlapping of the cartilaginous rings when the muscle contracts. The submucosa contains many elastic fibers and fat cells and many small seromucous tubular glands, which open into the lumen of the trachea.[2]

The mucosa has numerous low longitudinal folds and is lined with pseudostratified columnar ciliated epithelium, which contains numerous goblet cells and rests on a basement membrane. The secretory cells and the mucous glands under the epithelium provide the mucous layer that covers the tracheal epithelium. As the cilia move in a coordinated fashion, they create a wave that carries the mucus and inhaled foreign particles orally, and this is referred to as the *mucosal ciliary escalator.* The rate at which tracheal mucus is transported orally has been determined to be 1.6 ± 0.24 cm/min.[3] This mucosal ciliary escalator is important as a pulmonary defense mechanism. Surgical interventions, as well as foreign bodies and endotracheal tubes, can disrupt this mechanism and may predispose the animal to pulmonary disease.

There is a wide variation in the extrathoracic tracheal cross-sectional shape in horses. In the adult horse, tracheal diameter varies from 5.5 cm cranially, where it is almost round, to up to 7 cm or more distally, where it is flattened dorsoventrally.[1] A recent paper has quantified normal radiographic tracheal height for the Thoroughbred horse.[4] It is recognized that as horses (particularly ponies) age, the trachea may undergo some torsion and additional flattening in the cervical area. This is important when performing permanent tracheostomies, because this surgery needs to be centered in the middle of the tracheal ring, which may become rotated with age.

In a recent cadaveric study of 33 freshly slaughtered horses, neither age nor weight of the horse had any influence on the mechanical properties of the trachea.[5] However, extension decreases the compressibility of the tracheal segments, and the shape of the cross-sectional area has a major influence on mechanical properties. Hyperextension of the neck partly facilitates respiration at high levels of ventilation by elongating the trachea and decreasing its collapsibility. Therefore, muscles that cranially retract the larynx (and hence the trachea) may provide some stability of the tracheal luminal diameter during inhalation. In addition, the tracheal cartilages themselves prevent tracheal collapse during inhalation when intraluminal pressures are lower than atmospheric pressure. The incomplete dorsal structure of the tracheal rings allows tracheal expansion when

large volumes of air are exhaled, especially during periods of exercise. Although the fibroelastic annular ligaments positioned between consecutive tracheal rings allow considerable tracheal flexibility during neck movement, they become more stabilized whenever the trachea is elongated, and this probably occurs during exercise.

EXAMINATION

Preventing and managing complications of tracheal surgery in horses requires a thorough examination of the trachea. Preoperative evaluation should include manual palpation of the trachea. Its ventral aspect can easily be palpated in the cranial half of the cervical trachea. The lateral aspects of the trachea and their rings can be palpated further distally in the neck, but it becomes increasingly more difficult as the musculature thickens in the neck near the thoracic inlet. Cartilaginous rings should be compressible except in older horses; if they are not, they may have become mineralized. However, any change in the amount of compressibility of the tracheal rings may indicate a diseased state.

The evaluation should also include direct flexible tracheoscopy, the goal being to identify airway abnormalities (such as recurrent laryngeal neuropathy) and to determine the extent of the tracheal disorder, thus allowing the surgeon to plan surgical management and achieve the best possible outcome for each patient. A technique for clinical bronchography has been developed in the horse using a barium sulfate powder and an ether vaporizer into which compressed air is blown.[6] Although the value of bronchoscopy as a diagnostic procedure has yet to be proved, the method of powder insufflation provides a safe and accurate means of evaluating bronchial conditions in the horse. This could have some application toward tracheal disease.

CONDITIONS REQUIRING TRACHEAL SURGERY

Rostral Upper Airway Obstruction

Surgery of the trachea is often an emergency procedure to bypass a life-threatening obstruction of the upper respiratory tract. It may also be conducted as a route for endotracheal intubation for general anesthesia when routine nasotracheal or orotracheal intubation limits access to the surgical field. Additionally, surgical interventions are performed to physically rest the upper respiratory tract on a temporary basis or to simply bypass an inoperable upper respiratory tract obstruction (such as bilateral arytenoid chondritis).

Tracheotomy

Temporary tracheotomy and tracheotomy with an indwelling tracheal cannula (tracheostomy) may be performed with the horse standing or under general anesthesia. In the standing horse, the ventral midline of the neck is easier to locate (with regard to all tissue layers) and the dissection of the trachea is easier than in the anesthetized horse in dorsal or lateral recumbency, because the trachea shifts within the neck if the neck is not held in a perfectly vertical position. Tracheotomy is performed at the junction of the upper and middle thirds of the neck. The trachea is located superficially at this level. The horse is sedated in the stocks and the surgical site is prepared for aseptic intervention. Local anesthetic is injected on the ventral midline under the skin and into the paired sternothyrohyoid muscles prior to incision.

If the surgery is to be performed in the recumbent animal, a pad is placed under the head to place the neck in a slightly flexed position after the neck and the trachea have been placed in vertical position and as straight as possible. The objective is to align the skin so that it is in the position it will be in after the horse recovers and is standing normally.

With the horse standing or in recumbency, a 10-cm incision is made through the skin, subcutaneous tissue, and cutaneus colli muscle. The paired sternothyrohyoid muscle bellies are bluntly divided along the ventral midline for a distance of about 8 cm and held in a retracted position with a self-retaining retractor (Figure 47-1). The cartilage of the trachea can then be easily palpated. Although vertical and flap procedures are used in dogs, only the transverse tracheotomy technique is used in the horse to prevent problems with tracheal collapse and granulation tissue formation. Therefore, the annular ligament between two adjacent cartilage rings is sharply incised using a scalpel parallel to the orientation of the rings. The incision between the rings is lengthened to allow placement of a tracheal cannula. The incision should not exceed one half the circumference of the trachea. A large hemostatic forceps or an index finger may be inserted between the rings to guide the tracheal tube in the lumen.

A variety of tracheal tubes are manufactured. Self-retaining tubes are popular because they do not require skin sutures for

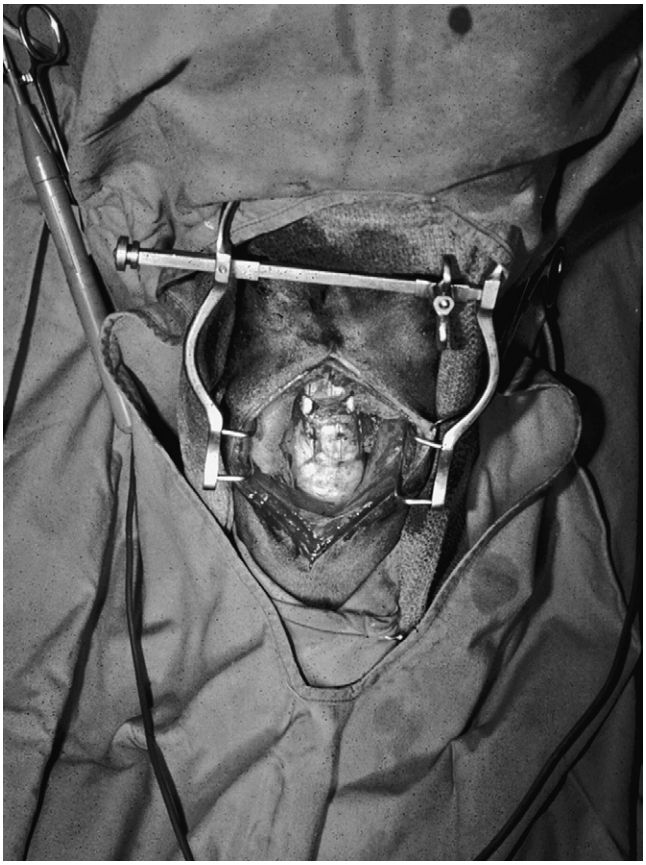

Figure 47-1. After the paired sternothyrohyoid muscles are bluntly divided along the ventral midline, they can be held in place with Weitlaner or baby Balfour retractors as shown. This provides good exposure to multiple tracheal rings.

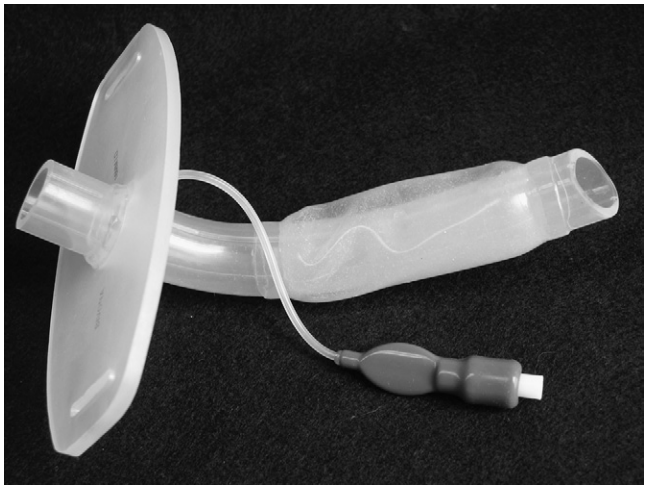

Figure 47-2. A short-cuff tracheal tube made of silicone. The flanges are used to tie the tube in place with gauze passed around the neck, or can be sutured to the skin.

security. Short-cuff silicone tubes are a good choice (Figure 47-2) because their compliance makes them more comfortable than rigid metal tubes. However, the cuff should not be inflated if the tubes are used long term; they will produce mucosal erosions.[7] Therefore, they are usually tied in place using the flanges on either side of the tubes.

In emergency situations, a segment of stomach tube, garden hose, or large plastic syringe casing with the tip removed may provide an airway until a better option is available. In an emergency situation, tracheotomy is difficult when performed on a horse that is struggling because of airway obstruction. It may be necessary to allow the horse to collapse; however, from that point forward, speed is imperative because the horse can die shortly after this stage is reached. The tracheotomy site should be cleaned and the indwelling cannula should be removed twice daily to remove accumulated tracheal secretions that could cause airway obstruction.

When direct tracheal intubation through a tracheotomy is carried out to avoid orotracheal or nasotracheal intubation, the procedure can be accomplished in the standing sedated or recumbent anesthetized horse. Precautions are taken to ensure that the skin incision is made on the ventral midline in the recumbent horse, because inappropriate positioning may create excessive skin tension or stoma obstruction at the tracheotomy site on recovery. The endotracheal tube should not extend past the carina because endobronchial intubation will impair ventilation.

COMPLICATIONS

Early complications can be reduced by using aseptic technique and by performing minimal soft tissue dissection to limit the amount of dead space. Complications after aseptic tube placement are uncommon. Occasionally, the wound becomes infected or the horse develops subcutaneous emphysema. More commonly, obstruction of the tracheal cannula with mucous secretions occurs and needs clearing. This problem should be closely monitored and the tube replaced at least every 24 hours, preferrably every 12 hours. Occasionally, the tracheal cannula is inadvertently placed peritracheally because of the loose areolar tissue around the trachea. This can be recognized immediately by the lack of air movement through the tube while the horse is breathing.

Long-term complications, including damage to tracheal cartilage or intraluminal granulation tissue and mucosal stricture, are extremely rare. Attention to the soft tissue dissection and aseptic technique whenever possible reduces these complications. Removal of the endotracheal cannula as soon as possible also reduces long-term complications. After the cannula is removed, the wound is left to heal by second intention with daily cleansing.

Tracheostomy

Permanent tracheostomy is used for correction of permanent impairment of the laryngotracheal apparatus, such as bilateral laryngeal hemiplegia or chondritis. Permanent tracheostomy has been used to provide a large, stable, permanent tracheal stoma with minimal complications.[8] A retrospective study that followed 42 horses for an average of 4.8 years found that stomas remained functional and 91% of owners would have it done again.[9] Good success also has been had with long-term function of tracheal stomas created in a number of adult horses.

Permanent tracheostomy can be performed with the horse sedated and standing or anesthetized and positioned in dorsal recumbency. The procedure should be performed under general anesthesia until the surgeon is comfortable with it before it is attempted in the standing horse.

Surgical Technique with General Anesthesia

The horse is anesthetized and positioned in dorsal recumbency with the ventral cervical area prepared for surgery. A 10-cm ventral midline incision is made through the skin, subcutaneous tissues, and cutaneus colli muscle in the cranial third of the cervical region just caudal to the larynx (Figure 47-3). The paired sternothyrohyoideus muscles are separated and retracted laterally to expose four tracheal rings. (The most cosmetic appearance is achieved if tracheal rings two to five are removed.) A 3-cm wide band of each of these muscles is bluntly separated and transected on either side of the midline so that the skin will lie easily on the trachea without tension. The area is packed with gauze sponges to stem the bleeding as the rest of the surgery progresses. A ventral midline incision and two paramedian incisions 15 mm on either side of the midline are made through the tracheal cartilages without disrupting the tracheal mucosa underneath. The rectangular cartilage pieces are carefully dissected from the tracheal submucosa and removed. The tracheal mucosa and annular tracheal ligaments are incised in a double-Y pattern and stay sutures used to maintain mucosal traction. The intersection of the legs of each Y in the mucosa is elevated and sutured to the skin with 2-0 polypropylene in a simple-interrupted pattern. The main tracheal mucosa is apposed to the skin lateral to the stoma in a similar manner.

The stoma is cleaned twice daily until the sutures are removed and then once daily afterward. The sutures can be removed 10 to 14 days after surgery; however, by that time many of the sutures have sloughed, and with time all of them will be extruded if they cannot be easily removed. This technique provides a cosmetically acceptable, structurally sound stoma needing minimal aftercare (Figure 47-4). The stoma is unlikely to be obstructed by accumulated secretions, even if daily cleaning is not done.

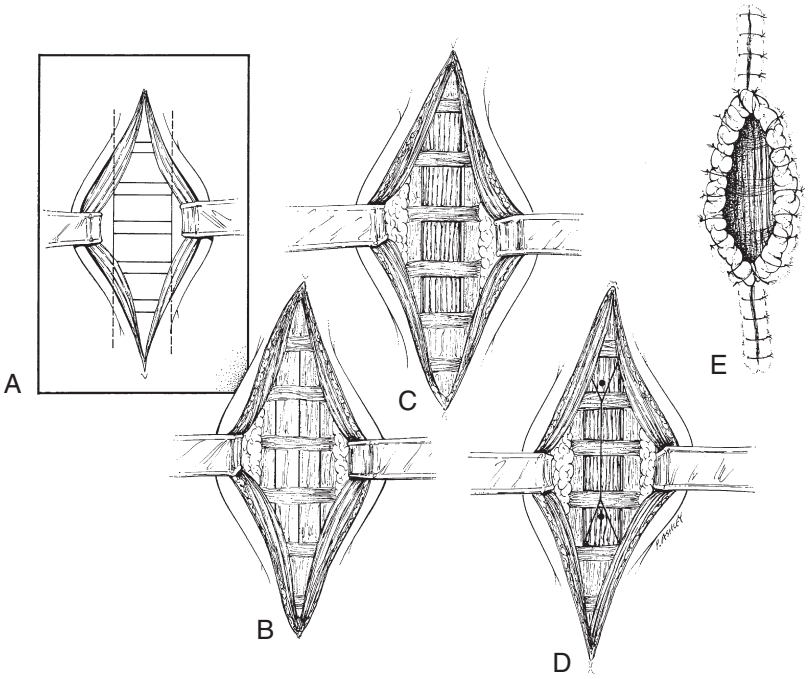

Figure 47-3. Surgical technique for permanent tracheotomy. **A,** Exposure of tracheal cartilage rings after separation of the sternothyrohyoideus muscles. **B,** Incision of tracheal cartilages. **C,** Removal of eight tracheal cartilage rectangles. **D,** Line of incision of tracheal mucosa and placement points for stay sutures *(circles).* **E,** Mucosal epidermal apposition.

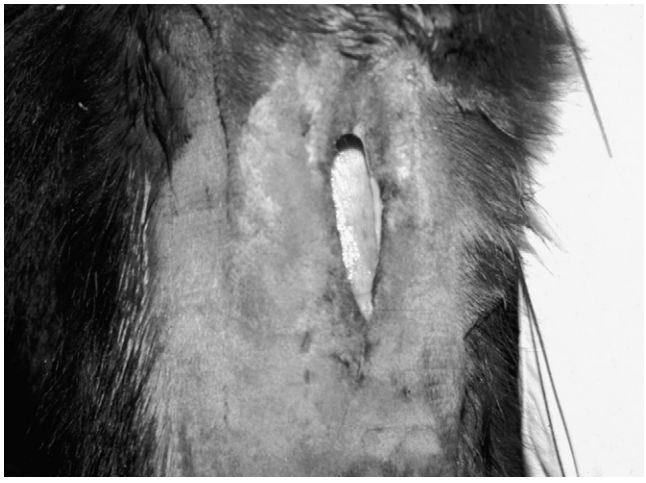

Figure 47-4. Permanent tracheostomy provides a cosmetically acceptable, structurally sound stoma requiring minimal aftercare.

The stoma can be located at any site in the proximal third of the neck. However, for best cosmetic results, this stoma can be located beginning at the second tracheal ring. This often provides a permanent stoma that is not easily recognizable to the untrained observer. It is not recommended that the first tracheal ring be removed.

SURGICAL TECHNIQUE IN THE STANDING HORSE

If permanent tracheostomy is performed with the horse sedated and standing, the horse is restrained in stocks and cross-tied with the head extended. The horse is sedated and a local anesthetic infiltrated subcutaneously in an inverted-U pattern dorsal and lateral to the second through the fifth tracheal rings. The 6-cm–long incision is made through the skin centered over the ventral midline beginning 3 cm distal to the cricoid cartilage. The paired sternothyrohyoid muscles are isolated and removed as previously described. The tracheal mucosa is then desensitized by injecting 30 mL of 2% lidocaine with a 1-inch, 23-gauge needle into the lumen of the trachea proximal to the surgery site. The ventral third of the second through the fifth tracheal rings is resected as previously described, leaving the tracheal mucosa intact. The longitudinal double-Y incision is subsequently made through the tracheal mucosa and annular tracheal ligaments, and the stoma is completed by suturing the tracheal mucosa and the submucosa to the skin with simple-interrupted sutures of 2-0 polypropylene in the simple-interrupted pattern.

COMPLICATIONS

Although the permanent stoma affects pulmonary defense mechanisms by reduced airway temperature control and altered humidification of inspired gases, the mucosal ciliary escalator continues to work, and mucus is continuously extruded through the stoma. Horses with existing pulmonary disease may have exacerbation of chronic pulmonary disease, or signs of respiratory disease may be induced, but this has not been my clinical experience.

Tracheal collapse will occur postoperatively if care is not taken to ensure that segments of the cartilage are removed from the ventral third of the cartilage rings only and not close to the trachealis muscle. In older animals, the tracheal ring may become rotated, and if that is the case removing the center third of the cartilage ring is important even if it is off-center. The amount of tension that is experienced from the skin can be adjusted by removing more of the sternothyrohyoid muscle if necessary. Also, to avoid tracheal collapse, no more than one third of the entire circumference of the ring should be removed.

Tracheal Perforation or Rupture

Disruption of the integrity of the tracheal lumen is usually caused by trauma.[10,11] Clinical signs of tracheal trauma depend on the site of the tracheal injury, but trauma can cause tracheal

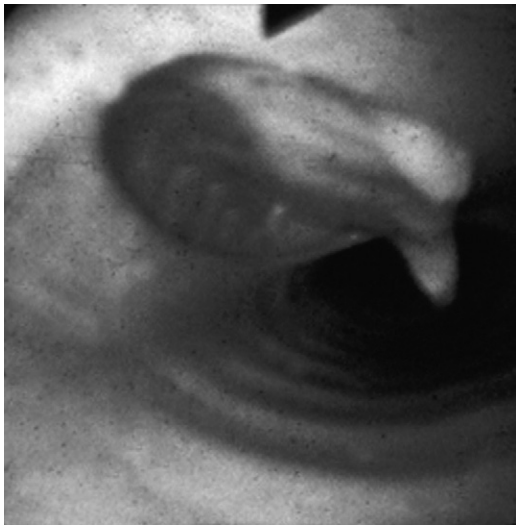

Figure 47-5. Endoscopic view of a tracheal rupture through the annular ligament. This horse had subcutaneous emphysema extending over the thorax.

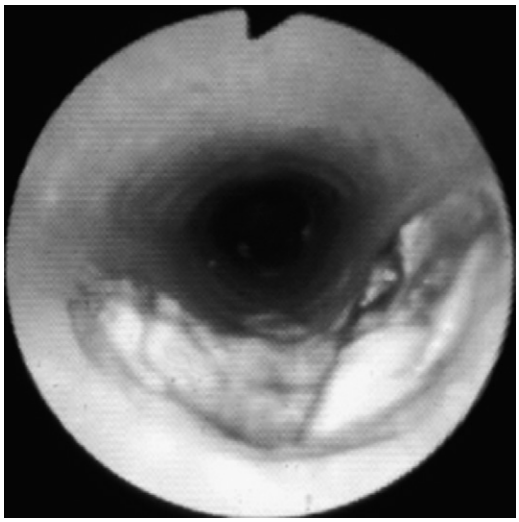

Figure 47-6. Endoscopic view of a tracheal perforation. Although this tear formed a fibrin seal, the presenting signs were subcutaneous emphysema.

perforation without concurrent cartilage damage or obvious skin damage or even rupture of the annular ligament (Figure 47-5). Clinical signs include subcutaneous emphysema and harsh tracheal sounds. Often, the perforation or disruption is not recognized until regional subcutaneous emphysema develops. When these signs occur in mares separated from their foals by wire fences at weaning, tracheal trauma should be suspected. The subcutaneous swelling is usually nonpainful, soft, easily indented, mobile, and crepitant. When tracheal trauma is suspected, thoracic radiographs should be taken to check for pneumomediastinum. Continued leakage into the mediastinum may lead to pneumothorax. Small tissue perforations in the absence of cartilage damage may not be apparent on radiographs, but both large and small lesions are usually apparent with tracheoscopy (Figure 47-6). Although the small tears rapidly form a fibrin seal, large ruptures should be treated promptly to avoid infection, obstruction from peritracheal tissue, progression of subcutaneous emphysema to the mediastinum, and development of pneumothorax. Small defects may respond to a pressure bandage over the affected region, which reduces the subcutaneous emphysema, or alternatively may respond to temporary tracheotomy.[11]

Although small tears can be managed conservatively and generally resolve within 24 to 48 hours, large tears should be managed surgically as soon as the horse's condition allows such an intervention. If there is a wound, the tears in the cervical trachea can be approached and directly sutured after débridement of devitalized tissue.[12] Wound margins are apposed in a simple-interrupted pattern of No. 0 absorbable suture material. Injuries that involve the trachealis muscle or the adventitia of the trachea, or that are rotational in nature, can be repaired by rotating the trachea to expose the dorsal surface. Closed suction drains are recommended in the postoperative period and should be removed between 48 and 72 hours, depending on the appearance of the wound and the amount of drainage. When the tracheal defect is closed, the subcutaneous emphysema usually resolves in 7 to 10 days.

Resection and Anastomosis

When tracheal trauma is extensive or when complete rupture occurs between tracheal rings, tracheal resection and anastomosis is indicated.[13-15] Before attempting a tracheal resection and anastomosis, the horse must be trained to wear a Martingale-type harness, which restricts dorsal movement of the head and reduces tension at the anastomotic site. This is particularly important if sections or rings need to be removed. If sections or rings do not need to be removed and the harness is quickly tolerated, the surgical preparation can occur immediately. Broad-spectrum antibiotics are administered prophylactically and the horse is anesthetized and positioned in dorsal recumbency.

The ventral cervical midline is prepared for aseptic surgery. A ventral midline cervical incision is made through the skin, subcutaneous tissue, and subcutaneous colli muscles over a distance of about 40 cm centered over the affected area. The sternothyrohyoid muscles are divided to expose the trachea. The trachea is separated from adjacent tissues along the entire cervical region within the incision site. If cartilage rings are to be removed, stay sutures are placed in tracheal cartilages adjacent to the segment to be removed. Up to five cartilages can be removed; however, tension on the anastomotic site will be excessive, and as few cartilages as possible should be removed (Figure 47-7). Tracheal mucosa and annular ligament are incised to divide the trachea into proximal and distal segments. The endotracheal tube is removed and replaced in the distal segment. The tracheal mucosa is turned back over the open ends of the proximal tracheal segment and sutured to the adventitia. The same procedure is repeated on the distal segment, working around the endotracheal tube. The head is subsequently flexed at a right angle with the neck. The endotracheal tube is replaced through the mouth and advanced into the distal segment of the trachea. If this is not possible because of the distal location of the injury, the endotracheal tube needs to be placed through another tracheotomy incision proximal to the anastomotic site. Tracheal ends are apposed with towel clamps and anastomosed with 25-gauge stainless steel wire in a simple-interrupted pattern without mucosal penetration. After the anastomosis is

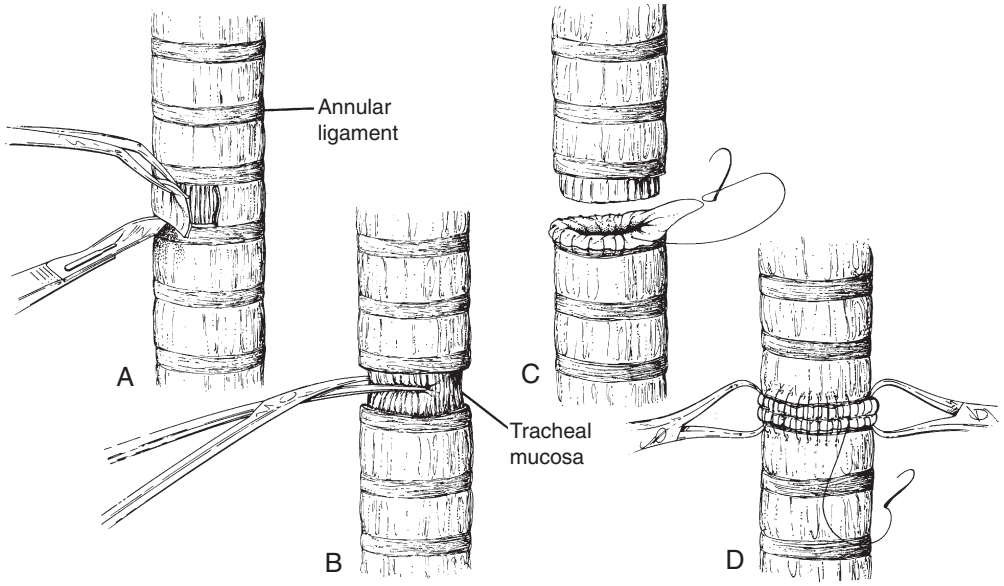

Figure 47-7. Surgical technique for tracheal anastomosis. **A,** Removal of a tracheal cartilage without penetration of mucosa. **B,** A 360-degree incision of mucosa between remaining cartilage rings. **C,** Eversion of mucosa, followed by suturing of mucosa to adventitia. **D,** Anastomosis of tracheal segments with stainless steel sutures.

complete, it is checked for air leaks prior to completely withdrawing the endotracheal tube. A continuous suction drain is placed in the soft tissue, and the remaining tissue layers are closed. The harness is applied before recovery and left in place for 3 weeks. Antibiotics are usually continued for 5 days. Complications include breakdown of the anastomosis and infection. Fistulation around the steel sutures persists if infection is present, and development of intraluminal abscesses in the anastomotic site is possible. Therefore, these sutures may need to be removed if abscesses develop after the trachea is healed.

Tracheal Collapse

Tracheal collapse can be a sequela to peritracheal abscesses, tumors, any injury causing cartilage trauma, malformation of the cartilages, or chronic pneumonia.[16-18] An idiopathic primary collapse syndrome has also been reported in the horse.[19] Primary tracheal collapse is recognized by widespread or segmental dorsoventral flattening of tracheal cartilage rings. Neurologic, congenital, or nutritional abnormalities may cause collapse in other species. Tracheal collapse uncomplicated by trauma is reported only in mature horses. It also occurs after emergency tracheotomy when vertical incisions were made through the rings. When tracheal collapse occurs from dorsoventral flattening of the trachea in aged horses and particularly ponies and American Minature Horses, the tracheal rings form shallow arcs and the dorsal tracheal membrane is stretched, creating an elliptical rather than a circular cross-section.[20]

Clinical signs vary with the degree of tracheal obstruction. Most animals experience varying degrees of respiratory distress and exercise intolerance and usually exhibit some stridor, although some animals may be asymptomatic until they become stressed. Diagnosis can be confirmed by auscultation, palpation, endoscopy, fluoroscopy, and radiography. Turbulent airflow may be auscultated within the cervical trachea and may thus allow localization of the site. Palpation may reveal the lateral edges of flattened tracheal rings in the jugular groove. Endoscopy will show the abnormal shape of the tracheal lumen, fluoroscopy will demonstrate the dynamic collapse of the the trachea, and lateral radiographs will depict the narrowed tracheal outline.

Treatment of tracheal collapse depends on the etiologic factors, length of the trachea involved, and accessibility of the affected area. Tracheal reconstruction has included complete replacement of tracheal segments with prostheses, external prostheses, imbrication of collapsed tracheal rings, plication of the trachealis muscle to reduce laxity of that structure, and resection of damaged cartilages with anastomosis of unaffected tracheal regions. The primary goal of the surgery is to restore the tracheal diameter without affecting the mucosal ciliary apparatus. This is best accomplished with an extraluminal prosthetic device that restores tracheal diameter by limiting redundant dorsal tracheal membrane and providing rigid support for the tracheal wall. Polyethylene syringe cases have commonly been used in ponies, but any nonreactive material of similar shape could be used. Partial chondrotomy is usually required on the ventral aspect of each collapsed tracheal ring to correct some deformities that occur when this dorsoventral flattening has occurred over time. Failure of the prosthesis to work will result in resection and anastomosis as a second choice; however, no more than five rings can be removed, and frequently dorsoventral collapse of a chronic nature involves more rings than this.

Tracheal Stenosis

Tracheal stenosis refers to a narrowing or stricture of the tracheal lumen that can occur during healing of a tracheotomy made by transverse incision through more than 50% of the circumference of the annular tracheal ligament (Figure 47-8). This produces instability and abnormal scarring. Other causes, particularly in foals, include *Streptococcus equi* abscesses in the peritracheal mediastinal lymph nodes and trauma. Tumors occur in this region, but tracheal collapse and stenosis secondary to tumors are unusual. Prolonged endotracheal intubation

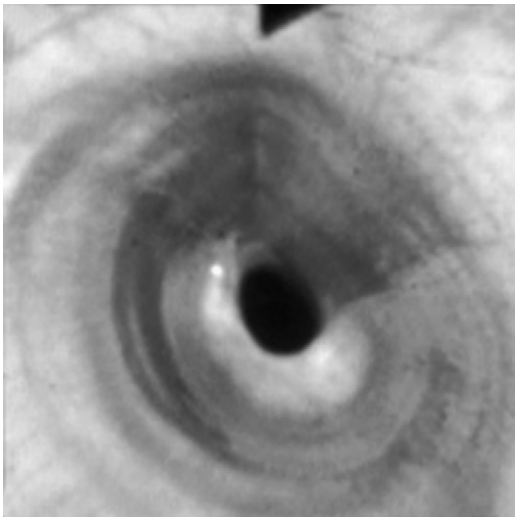

Figure 47-8. Endoscopic appearance of tracheal stenosis after transection of more than 50% of the annular ligament during temporary tracheostomy.

is associated with mucosal hemorrhage, necrosis, and stricture in people, and these lesions are frequently treated with surgery for removal of the circumferential fibrous tracheal stenosis.[21] Additionally, penetrating wounds that disrupt the tracheal mucosa, with or without infection, may lead to tracheal stenosis.

Surgical procedures used in the treatment of tracheal stenosis include tracheal resection and anastomosis, extraluminal polypropylene prostheses, surgical drainage of peritracheal abscesses, and multiple tracheal ring chondrotomies with placement of retention sutures anchored to the skin.

Foreign Body

Tracheal foreign bodies are uncommon in mature horses because of the size of the lumen of the trachea; however, endobronchial foreign bodies, although rare as well, are reported in equine patients.[22-25] They usually contain plant material, which may be inhaled accidentally, particularly during food prehension. The removal of foreign bodies from the respiratory tract is usually accomplished under general anesthesia and by way of tracheotomy. Frequently, removal involves endoscopic retrieval or endoscopically assisted surgical techniques. Foreign bodies in the tracheobronchial tree usually have thorns that cause them to lodge in the bronchi. The clinical signs usually include coughing and epistaxis, and a bloody nasal discharge is not uncommon. Usually, despite the severity of bronchial lesions, improvement in the bronchial mucosa is observed very soon after removal of the plant material, and few long-term effects are reported.

Granulomas

Granulomatous nodules are occasionally observed in the trachea during endoscopic examination. Often these are in the proximal trachea near the larynx, but occasionally they are associated with healing tracheotomy incisions, foreign body irritation, or inappropriate passage of a stomach tube that damages the tracheal mucosa. Although they are usually an incidental finding and not associated with clinical signs, if they are of sufficient size to create airflow obstruction they can be removed using an endoscopically guided laser technique.

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CHAPTER
48 Thoracic Disorders

Joel Lugo and Elizabeth A. Carr

Thoracic disorders in horses amenable to surgical intervention are primarily caused by thoracic trauma but also include pleuropneumonia, pleural effusion, and infectious or neoplastic masses. This chapter's emphasis is on thoracic trauma, its associated disorders, and the management of pleuropneumonia.

THORACIC TRAUMA

Thoracic injuries in horses occur as a result of blunt or penetrating trauma. Blunt trauma is commonly seen in neonatal foals and is believed to result from excessive compression of the pelvic canal of the dam during parturition.[1] In adult horses, penetrating trauma caused by collision with an object is most common.[2] The many reported sequelae of thoracic injuries include pneumothorax, hemothorax, rib fractures, flail chest, pulmonary contusion/laceration, and diaphragmatic hernias. These can occur alone or in combination.

The pathophysiology of respiratory distress in horses with thoracic injury is complex. Pain associated with thoracic trauma restricts full expansion of the thorax during inspiration. This can induce hypoventilation, eventually resulting in hypoxia and hypercarbia, which in turn can lead to myocardial dysfunction, lactic acidosis, and ultimately death if not corrected in time.[3] Pneumothorax, hemothorax, and contusion of pulmonary parenchyma can develop secondary to injury and can contribute to respiratory insufficiency.

Evaluation of a horse presented for thoracic trauma must be thorough but rapid. If necessary, potentially life-threatening conditions must be identified and addressed immediately before undertaking further diagnostic measures. Emergency management of thoracic trauma patients is directed at providing respiratory support to restore adequate ventilation and oxygenation, providing analgesia, and managing hypovolemic shock.

Clinical Presentation

Horses with thoracic injuries may be presented with a range of clinical signs all depending on the extent of trauma to external and/or internal thoracic structures.[4] Horses with extenal injuries (e.g., lacerations, rib fractures) may be presented with stiffness at the walk, anxiety resulting from thoracic pain, or severe signs of hypovolemic shock from hemorrhage. Otherwise, clinical signs of internal injuries usually result in respiratory distress including excessive respiratory effort, nostril flaring, tachypnea, dyspnea, accentuated thoracic excursion, and cyanotic mucous membranes.[5,6] Clinical signs may be subtle or dramatic, specific or nonspecific, and immediate or delayed in onset. This is especially true in horses with axillary wounds, where severe subcutaneous emphysema leading to pneumomediastimun can occur days after the injury.[7] Horses with concurrent abdominal involvement may develop colic symptoms because of injury to the viscera as occurr in cases of diaphragmatic hernias. Initial evaluation of the respiratory system should include good observation of the thoracic excursions, auscultation and palpation of the trachea and thorax, thoracic radiographs, thoracic ultrasonography, and arterial blood gas analysis.

Diagnosis

Physical Examination and Triage

The physical examination of a horse with thoracic trauma should be thorough and systematic. Observation of the rate, depth, and effort of respiration may help the clinician identify specific conditions. Rapidly developing respiratory distress is generally the result of pneumothorax or severe pulmonary contusion. Horses with hemothorax and pneumothorax develop a rapid and shallow respiratory pattern because of the restriction of lung expansion caused by the presence of fluid or air in the pleural cavity. A similar pattern of respiration can occur in horses with thoracic wounds or rib fractures, and this is most likely the result of restriction of thoracic wall movements due to pain. Paradoxical movement of the thoracic wall can be observed and is caused by collapse of a portion of the rib cage on inspiration when multiple rib fractures create an unstable flail chest wall. In this situation, the flail segment will move inward during inspiration and outward during expiration. This is sometimes better appreciated by palpation than inspection.

The thoracic wall should be palpated to evaluate the potential presence of subcutaneous emphysema (crepitus), fractured ribs, hematomas, and unstable (flail) segments. Localized crepitus with or without a hematoma suggests the presence of fractured ribs, whereas more diffuse subcutaneous emphysema may indicate the presence of pneumothorax, pneumomediastinum, or axillary wounds.[7,8] Thoracic auscultation and percussion help determine the presence of pneumothorax and hemothorax. The absence of respiratory sounds and increased resonance on percussion strongly suggest pneumothorax. However, auscultation of the thorax should not be relied upon as the sole means of diagnosing pneumothorax in horses. In a retrospective study, only 55% of the horses with diagnosed pneumothorax had decreased lung sounds dorsally.[7] When fluid accumulates in the pleural cavity, as with hemothorax, lung sounds are diminished ventrally, heart sounds resonate over a larger area, and a fluid line can be detected on percussion of the ventral thorax.[5] If crackling lung sounds are heard, pulmonary contusions should be suspected.

Several additional parameters should be monitored during the initial examination. The hematocrit and plasma total solids should be assessed at presentation and periodically afterward to determine if there is ongoing hemorrhage. Moreover, arterial blood gas analyses can help determine ventilatory function and the ability to oxygenate the tissues. If arterial blood cannot be obtained, a venous sample can allow assessment of ventilation. Hypoventilation is present if the PCO_2 is greater than 50 mm Hg, and hypoxemia is identified by an arterial PO_2 of less than 80 mm Hg. Frequent blood gas analyses can help assess if a horse's respiratory system is deteriorating or responding favorably to treatment.

Horses with thoracic trauma may suffer concurrent abdominal injury because of the close proximity of the abdomen to the thorax. Evaluation of the abdominal cavity is especially indicated when lacerations are present caudal to the sixth rib, or when a deep penetration or diaphragmatic trauma is suspected.[7] Abdominocentesis, diagnostic imaging, laparoscopy, thoracoscopy, or exploratory celiotomy can be used to rule out abdominal visceral involvement or diaphragmatic rupture. Musculoskeletal injuries can also accompany thoracic trauma, but they are typically of secondary importance to systemic stabilization.

Radiography and Ultrasonography

Radiography and ultrasonography are valuable diagnostic tools for evaluating of the equine thorax. Animals in severe respiratory distress must be stabilized with thoracocentesis and oxygen therapy before diagnostic imaging. Pneumothorax is evident radiographically by the separation of the lung margins from the diaphragm caudally and the vertebral bodies dorsally, thus allowing the pleural surface of the collapsed lung to be observed (Figure 48-1). Atelectatic lung lobes are radiopaque in contrast to the radiolucent, air-filled pleural space created when the lung collapses and retracts from the parietal pleura. Pneumomediastinum is diagnosed by the presence of air that outlines the mediastinal contents, and it can coexist with pneumothorax, usually secondary to trauma. Radiographs can also reveal other lesions, such as rib fractures, foreign objects, and diaphragmatic hernias. Intrathoracic and pulmonary masses may be seen as areas of increased soft tissue opacity that summate with or displace structures in the mediastinum, lung tissue, or thorax.

Thoracic ultrasonography is more valuable than radiography for confirming the presence of pleural fluid, for identifying rib fractures, and for determining the best site for thoracentesis or placement of indwelling chest tubes. In neonates, ultrasonography is more sensitive than radiography to detect rib fractures and concurrent internal thoracic trauma.[9] Ultrasonographic examination may permit the identification of foreign material, masses in the pulmonary parenchyma and cranial mediastinum, rib fractures, and diaphragmatic hernias.[10-14] However, pneumothorax may be difficult to diagnose because free air in the dorsal thorax and aerated ventral lung lobes appear ultrasonographically similar.

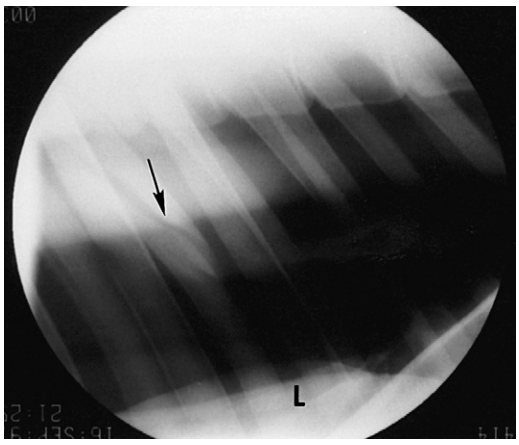

Figure 48-1. Thoracic radiograph of a horse with a rib fracture *(arrow)* that resulted in pneumothorax, as evidenced by air within the chest *(appears black).* The collapsed lung *(L)* lies ventral to the accumulated air.

Thoracocentesis

Thoracocentesis can be performed to obtain a sample of pleural fluid for analysis or to drain air or fluid from the pleural space. Aspirated fluid should be submitted for cytologic analysis, aerobic and anaerobic cultures, and antibiotic sensitivity testing. Sensitivity testing is important when hemothorax is caused by penetrating wounds because the risk of contamination and subsequent infection is great. The site for thoracocentesis is best guided with ultrasonography. Thoracocentesis carries the risk of pulmonary parenchyma injury, which results in iatrogenic pneumothorax or hemothorax; however, proper technique can minimize complications.

Thoracoscopy

Equine thoracoscopy can be used to provide a more accurate diagnosis and prognosis of thoracic diseases when less-invasive diagnostic tools fail to yield an accurate assessment. Common uses of thoracoscopy include the following[15-17]:

- Exploration of the thorax of horses with suspected neoplasia, diaphragmatic hernias, or pleural effusion of unknown origin
- Drain placement for relief of pleural effusion and abscesses
- Transection of pleural adhesions
- Guided pulmonary biopsy
- Window pericardectomy
- Exploration of the thoracic cavity to identify best site for thoracotomy

In addition, thoracoscopic surgery has been used to diagnose and assist in the repair of diaphragmatic hernias (see Chapter 39), to evaluate and repair thoracic esophageal obstruction, and to explore of the thorax in horses with open penetrating wounds.[18-20]

Thoracoscopy can be performed with the horse standing or under general anesthesia. Thoracoscopy in the standing horse has proved to be safe, well tolerated, and associated with minimal detrimental effects to the cardiovascular and pulmonary function of healthy horses and horses with chronic lung disease.[21,22] The presence of a pneumothorax during thoracoscopy can cause a transient hypoxemia attributed to ventilation/perfusion mismatch and a slight decrease in cardiac output secondary to decreased venous return.[21,22] Analysis of arterial blood gases is considered essential to evaluate ventilation and oxygen exchange, especially in horses with compromised lung function.

For thoracoscopy in standing horses, the animal is restrained in stocks and is sedated with xylazine (Rompum) at 0.5 to 1.1 mg/kg IV, romifidine (Sedivet) at 0.04 to 0.10 mg/kg or detomidine (Dormosedan) at 8 to 10 mg/kg or by use of a continuous IV drip infusion of detomidine (an 8 mg/kg loading dose followed by an administration rate of 0.3 to 0.5 mg/kg per minute until the desired effect is observed). During surgery, analgesia is provided by a single bolus of butorphanol tartrate (Torbugesic) (0.04 mg/kg IV) and local anesthesia of the skin, subcutaneous tissue, intercostal musculature, and costal pleura.

(Additional information on sedation for standing surgery can be found in Chapter 22). The thoracic chest wall is clipped and prepared for aseptic surgery. The sites for the telescope and instrument portals depend on the surgical intervention. The thoracic cavity can be better evaluated when the telescope is introduced into the thorax just cranial to the tenth to twelfth intercostal spaces, avoiding the vessels and nerves located caudal to the ribs.[23] The mobility of the ribs at this location allows manipulation of the telescope for extensive exploration of the thorax. Selection of a thoracoscopic portal caudal to the fifteenth intercostal space can result in inadvertent injury of the diaphragm during insertion of the trocar–cannula system.[22] On the other hand, placing portals too far craniad at the eighth intercostal space causes some pain and discomfort during manipulation of the instruments or telescope as a result of distraction of the ribs.[23] A 1-cm stab incision should be made through skin and intercostal muscles just ventral to the epaxial muscles. To prevent damage to the lung by the trocar–cannula system through which the thoracoscope is inserted, a pneumothorax is induced by inserting a teat cannula into the thoracic cavity through the incision and the intercostal muscles into the pleural cavity. This allows atmospheric air to enter the thorax, resulting in an iatrogenic pneumothorax. After the pneumothorax is induced, the teat cannula is replaced by the trochar–cannula system. The trocar is subsequently removed and replaced by the telescope. After the telescope is inserted, a systemic exploration is performed and the desired surgical intervention is performed using the triangulation technique (see Chapter 13). Multiple instrument portals are placed at least 10 to 15 cm apart (two to three intercostal spaces) to avoid obstruction of vision or restriction of instrument manipulations. Thoracoscopic viewing of the surgical field prevents injuries to thoracic structures during insertion of instrument and trocars. At the conclusion of the procedure, the thoracic cavity is inspected one final time and thoracic negative pressure is reestablished by removing all the air from the pleural cavity through a suction system connected to the thoracoscope cannula. Lung reinflation is observed through the telescope, which is retracted to the distal end of the endoscopic cannula, as inflation occurs.[19] The telescope is removed and the skin over the portals is closed with nonabsorbable suture material using simple interruped sutures.

Thoracoscopy under general anesthesia is performed with the horse in sternal, lateral, or dorsal recumbency.[17,19] To perform the procedure under general anesthesia, positive-pressure ventilation is an absolute necessity, because bilateral pneumothorax can occur more easily in the anesthetized than in the standing horse. Single-lung ventilation has been widely used during thoracoscopy in humans and dogs because it provides more surgical space in the operated hemithorax. Although this technique has been clinically used in some horses, the pulmonary effects with this ventilation technique require further investigation. After surgical preparation of the ventral aspect of the thorax, a pneumothorax is induced as previously described, and the telescope and instrument portals are made as dictated by the surgical intervention. Thoracoscopy under general anesthesia improves the viewing of the ventral lung surfaces, ventral thoracic cavities, diaphragmatic surfaces, and lateral surface of the heart.[19]

On some occasions, CO_2 insufflation of the pleural cavity is used to facilitate surgical exposure. A recent investigation demonstrated that CO_2 insufflation for 20 minutes during standing thoracoscopy was not detrimental to the horse's cardiopulmonary parameters.[24] However, the ocurrence of bilateral pneumothorax was more common than without CO_2 insufflation. CO_2 insufflation during general anesthesia is also used, but careful monitoring is essential because intrapleural pressure greater than 3 to 4 mm Hg in dogs resulted in cardiovascular and pulmonary deterioration.[25]

Thoracoscopically guided lung biopsy has been described as a safe and a successful technique to facilitate the harvest of tissue samples for histologic and microbiologic examination.[22] The technique can be performed in normal horses and in horses with pulmonary function impairment (heave-affected horses during clinical exacerbation) without harmful effects.[22] Pulmonary tissue samples are collected from the caudodorsal aspect of the lung, because, when lung disease is diffuse, this site appears to be representative of the remainder of the horse's lung.[26] Direct observation of the surgical biopsy and excellent hemostasis enable surgeons to avoid complications reported by others when performing percutaneous lung biopsies.[27] Furthermore, multiple resections can be performed or larger wedge resections can be obtained when necessary. The main disadvantage of this technique is that the tissue access is limited to the periphery of the lung and therefore samples are useful only for peripheral lesions or diffuse interstitial diseases. If pulmonary lesions are seen during thoracoscopy on the broad surface of the lung, a biopsy can be performed with fine-needle aspirates, endoscopic biopsy forceps, or other biopsy instruments.

Thoracoscopically guided lung biopsy is performed with the horse standing under chemical restraint. The telescope portal is placed at the twelfth or thirteenth intercostal space. One instrument portal is placed one or two intercostal spaces cranial and 15 cm ventral to the telescope, and the other instrument portal is located at the fifteenth intercostal space and approximately 10 cm ventral to the telescope portal. The caudal aspect of the caudal lung lobe can be grasped with atraumatic forceps (Babcock), and the wedge resection sample is obtained by the use of endoscopic staples (Figure 48-2). When necessary, the atraumatic forceps and staple device are interchanged between portals to facilitate the approach to the tissue specimen. Because the tissue sample is usually too large to be withdrawn through the trocar–cannula system, it is withdrawn through the instrument portal with the cannula. The thoracic cavity and biopsy site are inspected for evidence of hemorrhage. Negative pressure in the thoracic cavity is reestablished by removing all the air from the chest, and the skin is closed routinely.

Another reported technique to obtain the lung sample is using a pretied ligating ligature loop and endoscopic scissors instead of the endoscopic staple device. Limitations of this technique include a relatively high rate of complications because of slippage of the ligating loop from the biopsy site (31% of the time) and the inability to predetermine the biopsy size.[28]

Thoracoscopy is not without risk, and complications can occur. The most common complications include (1) injury to lung parenchyma or diaphragm during trochar penetration, especially in horses with lung adhesions to the parietal pleura; (2) injury to intercostal vessels; (3) pain during manipulation of telescope and/or instruments; (4) residual pneumothorax; (5) difficulty exposing the area of interest or manipulating the instruments inside the chest cavity; and (6) mild respiratory distress during induction of pneumothorax or surgery.[20-23]

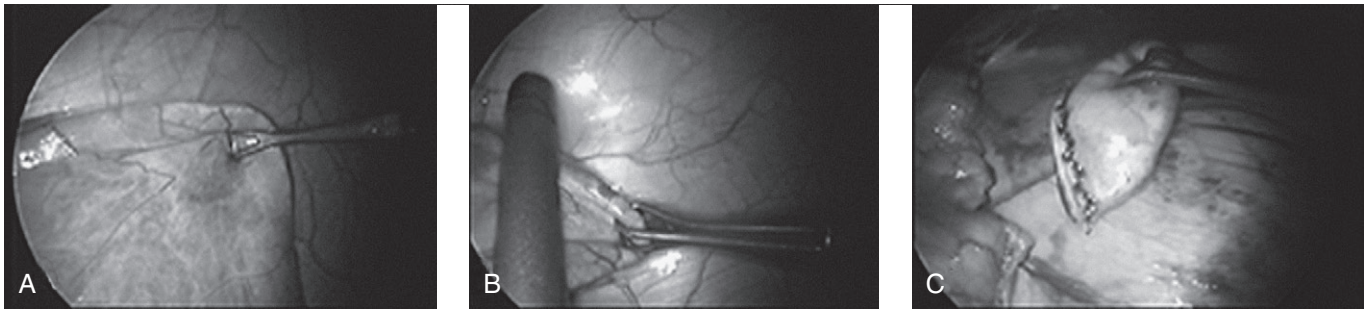

Figure 48-2. Thoracoscopically guided lung biopsy in a horse. The caudodorsal aspect of the lung lobe is grasped with Babcock forceps **(A),** and, using endoscopic staples **(B),** the pulmonary wedge resection is performed **(C).**

PNEUMOTHORAX AND HEMOTHORAX

Clinical Signs

Pneumothorax refers to the accumulation of air in the pleural cavity, which occurs as a result of pulmonary or chest wall injury.[29] When a communication between the atmosphere and the pleural space is created, a pressure gradient between the negative pressure in the pleural space and the alveolar or atmospheric pressure develops. Air enters the pleural cavity until the pressure gradient is eliminated or the communication is sealed. Consequently, the lung collapses to its minimal volume and this compromises pulmonary function. Meanwhile, the increase in pleural pressure causes a shift of the mediastinum to the contralateral side, enlarges the ipsilateral hemithorax, and depresses the diaphragm.[30] Pneumothorax in horses has been reported secondary to thoracic trauma, secondary to pleuropneumonia, and as a complication of upper airway surgery and thoracoscopy.[7,22] Traumatic pneumothorax can be classified by its physiologic nature as open or closed or as a tension pneumothorax. Injury to the chest wall (e.g., from penetrating objects, wounds, gunshots) that creates a communication between the pleural cavity and the external environment produces an open pneumothorax. A closed pneumothorax results from leakage of air from the pulmonary parenchyma or an airway tear and commonly takes place after blunt trauma causing rib fractures that injure the lung parenchyma. Tension pneumothorax results when air leakage, whether from the thoracic wall or lung parenchyma, acts like a one-way valve, allowing entry of air into the pleural cavity but not permitting its escape. Air continues to accumulate inside the pleural cavity during subsequent respiratory cycles, thus progressively increasing intrathoracic pressure to a point that exceeds atmospheric pressure (positive pressure). Increasing intrathoracic pressure causes severe cardiovascular and pulmonary compromise; if not treated promptly and aggressively, this condition can be fatal.[30] Severe pleuropneumonia is another cause of pneumothorax in horses.[7] Slow leaks of air from necrotic pulmonary tissue or the formation of bronchopleural fistulas may be the source of pleural air in these horses.

The main physiologic consequences of pneumothorax are a decrease in the vital capacity of the lung and a decrease in the partial pressure of arterial O_2 (PaO_2).[30] Patients with pneumothorax have a reduced PaO_2 and an increased alveolar–arterial oxygen difference (A-a gradient). The hypoxia in turn may result in myocardial dysfunction and lactic acidosis. These physiologic changes appear to be caused by ventilation/perfusion mismatching (low $\dot{V}/\dot{Q}$ ratio), intrapulmonary shunts, and alveolar hypoventilation resulting in hypercarbia and respiratory acidosis, pulmonary hypertension, and increased work for the right side of the heart.[30,31] Therefore, if pneumothorax is not treated in time can ensue shock, cardiopulmary deterioration, and ultimately death. Unless the mediastinum is sealed by another underlying inflammatory condition (e.g., pleuropneumonia, hemothorax), pneumothorax is frequently bilateral because an incomplete mediastinum exists in the horse.[32]

Clinical signs associated with pneumothorax include restlessness, cyanosis, tachypnea, dyspnea, unilateral expansion of the thorax, and accentuated respiratory excursions. In the absence of lower airway disease, the most probable cause of pneumothorax is trauma, and further examination may reveal evidence of a penetrating wound. Acute clinical signs are usually observed in horses with acute onset of pneumothorax or tension pneumothorax, whereas a more insidious onset of clinical signs is typical in horses with pleuropneumonia.[7] Decreased lung compliance and increased intrathoracic pressure increase the work of breathing.[29] To minimize the work of breathing, respiratory frequency increases and the tidal volume decreases, hence producing the characteristic shallow, rapid breathing pattern observed in horses with pneumothorax.[5]

Hemothorax can develop secondary to any trauma that causes laceration of the intercostal blood vessels, myocardium, pulmonary parenchyma, or thoracic wall musculature. A horse with massive hemorrhage from laceration of the heart or one of the great vessels usually does not survive. Horses with hemothorax commonly present with signs of hypovolemic shock, anemia, and pain. Signs of respiratory distress develop when a large accumulation of blood in the pleural cavity restricts the full expansion of the lungs or when hemothorax is combined with pneumothorax.

Management

The emergency management of patients affected by pneumothorax is aimed at providing adequate ventilation, oxygenation, and systemic perfusion. Immediate therapy includes establishing a patent airway, reestablishing adequate alveolar ventilation by removing the free air, and preventing recurrence. Supplemental oxygen therapy must be initiated for the treatment of hypoxemia (PaO_2 less than 80 mm Hg). Nasal insufflation of oxygen at a flow rate of 15 L/min is recommended in adult horses to increase the inspired oxygen concentration.[5] In addition, patients with evidence of shock, such as tachycardia, weak peripheral pulses, peripheral vasoconstriction, and prolonged

capillary refill time, require aggressive fluid volume replacement therapy.

Treatment of uncomplicated closed pneumothorax (e.g., following thoracoscopy) can be as simple as restricting exercise and close monitoring. An open penetrating wound should be sealed immediately with a sterile, airtight bandage to convert the condition to a closed pneumothorax. After the wound is sealed, thoracocentesis and reestablishment of negative pleural pressure can proceed. A large-gauge needle, teat cannula, or thoracostomy tube is inserted between the twelfth and fifteenth intercostal spaces just below the epaxial muscles.[6] Air should be removed slowly from the pleural cavity, using a 60-mL syringe and a three-way stopcock or a suction device. If negative pressure cannot be reached or repeated air aspiration is required to alleviate respiratory distress (more than three or four aspirations per 24 hours), tube thoracostomy should be considered for intermittent or continuous evacuation. Thoracostomy tubes can be attached to a Heimlich valve (see Figure 17-7) for one-way flow or to a pressure-regulated suction system such as the commercially available Pleuro-Evac. Resolution of pneumothorax results in dramatic improvement in hemodynamic parameters, although abnormalities in gas exchange persist for 60 to 90 minutes after recovery and are associated with a decrease in pulmonary compliance.[31] After reinflation of the lung, some areas of $\dot{V}/\dot{Q}$ mismatch and intrapulmonary shunts persist for several hours.

Unilateral pulmonary edema, also known as reexpansion pulmonary edema, can occur after rapid expansion of the lung following a period of collapse as the result of pneumothorax or pleural effusion.[33] Pulmonary edema acutely develops after reinflation and causes hypoxemia, decreased cardiac output, transient hypotension, and may lead to death.[34,35] The pathophysiology is poorly understood; it appears that multiple factors contribute to increased vascular permeability in the alveolar capillary bed. The inflammatory response occurring after reexpansion of the lung is a strong predisposing factor for the development of pulmonary edema. The increase in capillary permeability is believed to be secondary to (1) mechanical vascular injuries to the alveolar capillary membrane, (2) reperfusion injury as blood flow returns to the now fully expanded lung, and (3) abnormalities in surfactant production and function.[36,37] Typically, the chance for development of reexpansion pulmonary edema is increased if the pneumothorax has been present for several days or when extremely negative pressure is used to evacuate the pleural cavity. A pressure of less than 20 mm Hg should be applied to the pleural cavity, and slow evacuation is recommended to minimize the chances of developing this complication.[38] During surgical reconstruction of diaphragmatic hernias in small animals, current recommendations to prevent reexpansion pulmonary edema call for chronically atelectatic lungs to be reinflated by gradual reexpansion over several hours (8 to 12 hours) after surgery rather than complete reinflation at the end of surgery.[39]

The rate of pleural air absorption can be accelerated by tracheal administration of 100% supplemental O_2.[40] When 100% O_2 is administered, the partial pressure of nitrogen in the pulmonary vessels is decreased to near zero, whereas the partial pressure of O_2, CO_2, and H_2O remain unchanged. The absence of nitrogen in the dissolved gases in the pulmonary vessels establishes a diffusion gradient for nitrogen between the air in the pleural cavity and the gases in the pulmonary vessels. After the nitrogen is absorbed, O_2 is 100% of the gas in the pleural cavity, which results in a gradient for uptake of O_2. This gradient can increase the speed of absorption of free air to approximately 10 times the absorption achieved while breathing room air.[41] This therapy has been proved to be clinically effective in humans, and it would be logical to assume that it would work in horses as well.

Treatment of hemothorax involves restoring the blood volume by use of intravenous fluids, blood transfusion, and pleural drainage. Removing the free blood from the thorax and reexpanding the lung improves ventilation. In horses with open thoracic wounds, free blood should be removed to reduce the risk of septic pleuritis and pleural adhesions. Treatment of the simple hemothorax, a complication of lung biopsy, thoracoscopy, or blunt trauma may be conservative unless the horse displays signs of respiratory distress, but careful monitoring is mandatory.[22,42] In these horses, absorption of blood occurs within days and pleural adhesions are unlikely.

Thoracocentesis is commonly used to reestablish adequate alveolar ventilation in patients with pneumothorax and pleural effusion. Evacuation of air or fluid from the chest improves $\dot{V}/\dot{Q}$ matching and reduces intrapulmonary shunting.[43] The site for thoracocentesis should be based on auscultation or, ideally, on radiography and/or ultrasonography. In the case of hemothorax and pneumothorax, blood and air can be evacuated from the same site. A large area over the selected site, usually the seventh and eighth intercostal space, should be clipped and prepared for an aseptic intervention. Local anesthetic is infiltrated into the skin, subcutaneous tissue, intercostal musculature, and costal pleura. A large-gauge needle, teat cannula, Chamber's catheter, or chest tube can be used for the procedure. The site for thoracocentesis is selected cranial to the rib to avoid the intercostal vessels located caudal to the rib. A small stab incision is made through the skin, and the selected instrument is advanced through the intercostal muscles until resistance is decreased as the pleural space is penetrated. At this moment, fluid or air can be drained and the catheter can be redirected if neither fluid nor air is aspirated. Thoracostomy tubes can be left in place after securing the tubes to the body wall with purse-string sutures. A Heimlich valve may be attached to the tube to provide one-way flow.

THORACIC WOUNDS

Etiology

Injury to the thorax in horses is uncommon but not rare, possibly because automobile accidents involving horses are infrequent. Penetrating wounds to the thorax generally occur after an impact with sharp objects (e.g., running into trailers, gates, trees, fences). Penetrating wounds are usually located in the lateral aspect of the thoracic wall or in the region of the thoracic inlet (axillary region). The defect may look like a simple perforation requiring no specific treatment, or it may be extremely large. Open pneumothorax and subcutaneous emphysema are the most common clinical findings that develop subsequent to penetrating thoracic wounds.[2] The prognosis for horses with penetrating thoracic wounds is favorable if no other abdominal or musculoskeletal injury is present.[2] However, septic pleuritis can alter the prognosis in these horses.[2,44,45] Although a low incidence of septic pleuritis has been reported following thoracic trauma, approximately 50% of horses that develop pleuritis die or are euthanatized.[45]

Axillary wounds create interesting clinical signs for affected horses. The injury might not involve the thorax initially; however, marked subcutaneous emphysema may develop because the wounded tissue becomes a one-way valve for air as the horse moves. When extensive, this subcutaneous emphysema can migrate to the mediastinum (pneumomediastinum) and extend into the thorax, leading to life-threatening pneumothorax.[8] Therefore, horses with axillary wounds should be kept in strict confinement and monitored closely for signs of respiratory distress.

Management

Initial management of horses with thoracic wounds includes (1) immediately covering the wound with a sterile dressing to prevent any further ingress of air to the thoracic cavity and (2) assessing the patient for intrathoracic injuries. Pneumothorax and hemothorax are likely to require treatment. If the wound is properly sealed, a one-time thoracocentesis or needle aspirate can alleviate the pneumothorax and associated ventilatory consequences. Any residual air in the pleural cavity will be reabsorbed within a few days. In horses with tension pneumothorax, emergency thoracocentesis is necessary to equilibrate pleural pressure with atmospheric pressure and relieve the respiratory insufficiency.

An important goal in the management of thoracic trauma is pain relief. All horses with thoracic wounds can suffer from severe pain that limits chest wall excursions, and this often results in ventilatory impairment. Appropriate analgesia allows patients to maintain effective ventilation and oxygenation and to preserve the cough mechanism necessary to eliminate pulmonary secretions. Pain can be reduced by use of systemic analgesics and intercostal perineural anesthesia. Analgesics must be used carefully because of the potential for respiratory depression. Intercostal nerve blocks are performed with long-acting local anesthetic agents, such as 5% bupivacaine. The block is performed dorsal and one to two intercostal spaces cranial and caudal to the injury to provide optimal local analgesia. Deeper infusion of local anesthetics is necessary to desensitize nonmyelinated C fibers located within the parietal pleura, which are a source of pain when stimulated by the penetrating object, fractured ribs, or wounds.[22]

After life-threatening conditions are controlled, thoracic wound care consists of exploration, débridement, and closure. These manipulations can be accomplished with the horse standing under adequate analgesia or recumbent under general anesthesia. Treatment with the horse standing is preferred to avoid potential respiratory and cardiovascular complications that may develop under general anesthesia. General anesthesia is indicated for extensive wounds, for wounds that may involve underlying structures (e.g., the diaphragm), or for fractious animals. The horse must be stabilized by treating shock and respiratory impairments before induction of anesthesia and, if necessary, surgery should be delayed for 2 to 3 days. The wound must be thoroughly explored for the presence of foreign material and bone fragments, which can become the source of septic pleuritis or of recurrent purulent discharge. Thoracoscopy can be used to remove foreign objects while allowing a thorough inspection of thoracic structures. The telescope is initially inserted into the thoracic cavity via a portal away from the wound, and a detailed thoracic exploration is performed. Meanwhile, pleural lavage with sterile saline solution can be performed. The wound itself can also be explored with a rigid or flexible telescope.

Immediate or delayed closure techniques may be used depending on the hemodynamic status of the patient and the degree of contamination and tissue damage. The wound is irrigated with sterile saline solution and subjected to débridement. To facilitate assessment of damaged ribs, diaphragm, and lung lobes, the wound may need to be enlarged. Any foreign body is removed, and, ideally, primary closure of the wound is attempted. However, large thoracic defects may require reconstruction with primary muscle flaps, diaphragmatic advancement flaps, and/or prosthetic mesh. Creation of muscle pedicles from the longissimus dorsi and external abdominal oblique muscles to close a thoracic wall defect in a horse has been described.[46] If the wound defect cannot be closed, the wound is packed and sealed with moist, sterile laparotomy sponges to prevent the entry of air and leave to heal by second intention. A stent and thoracic bandage around the thorax holds the packing in place. The wound is allowed to heal by secondary closure with fresh bandage changes daily until the thoracic cavity seals. Horses should be confined to a stall and in cases with axillary wounds cross-tied to minimize limb motion and limit further air penetration into the tisuues. Horses are also given broad-spectrum systemic antibiotics, nonsteroidal anti-inflammatory drugs, analgesic medications, and tetanus prophylaxis.

FRACTURED RIBS

Rib fractures are common after thoracic trauma. Complications of rib fractures include underlying pulmonary contusion and thoracic wall pain. The most substantial physiologic impairment after rib fracture is a decrease in dynamic compliance, resulting in ventilatory impairment.[47] In addition, underlying pulmonary contusion renders a portion of the pulmonary parenchyma unavailable for adequate ventilation and oxygenation, resulting in hypercarbia and hypoxemia.[48] Chest radiography can be used to assess the presence of pneumothorax or hemothorax, although thorough ultrasonography examination of the rib cage is a better method to identify displaced or nondisplaced fractures as well as concurrent internal thoracic trauma.

Most isolated fractures heal spontaneously and surgical fixation is not necessary. Treatment in adult horses usually consists of managing pneumothorax, hemothorax, pleuritis, pulmonary contussion, wound care, and analgesia. The main goal for management of rib fractures is pain relief. Analgesia can be provided by administering nonsteroidal anti-inflammatory drugs and by using local intercostal nerve blocks. In the case of penetrating wounds, bone fragments are removed and rib edges are smoothed with rongeurs to prevent trauma to the pulmonary parenchyma.[4] The horse should be strictly confined and monitored for signs of respiratory distress.

Flail chest is uncommon in adult horses but when it ocurrs it causes severe ventilatory impairment. Flail chest occurs when multiple ribs are fractured in two places (dorsal and ventral), resulting in the loss of stability of a segment of the thoracic wall, which is known as the flail segment, and consequently exhibits paradoxical respiratory movement.[47] Respiratory efforts are hindered by the failure of the affected flail segment to expand and participate in the normal process of respiration. However, the underlying pulmonary contusion, and not the

mechanical disruption of the thoracic wall, causes the respiratory insufficiency.[47] Fracture stabilization of the flail segment and pain management are necessary to restore adequate ventilation. Surgical stabilization of a flail chest was achieved in a horse using an external splint secured to the ribs with orthopedic wires.[2] However, septic pleuritis developed in this horse as a consequence of ascending infection through the orthopedic wires that surrounded the ribs inside the pleural cavity. The use of internal fixation methods similar to the ones described for foals should be considered.

Rib Fractures in the Neonate

A study revealed that approximately 21% of the foals up to 3 days of age have thoracic trauma, which can include rib fractures and costochondral dislocation.[1] In addition, a study reported that a high number (up to 65%) of neonatal foals presented to equine referral centers experienced fractured ribs.[9] In most foals, no clinical problems develop and the outcome is favorable. However, thoracic trauma and fractured ribs can have harmful effects and are a substantial contributor to morbidity and mortality in affected neonates.[12,49] Hemothorax, pneumothorax, diaphragmatic hernia, and even sudden death can result after laceration of intrathoracic vessels, lung parenchyma, and cardiac tissue by displaced fracture ribs. Underlying pulmonary contusion can also result in pulmonary insufficiency and predispose the foal to the development of pneumonia. Thoracic trauma in neonates usually involves three or more ribs, frequently occurs in one hemithorax, and is consistently located in the cranioventral aspect of the thorax.[50] The most common site for injury is the costochondral junction and the area immediately proximal to it. Dystocia and foaling in primiparous mares are two factors associated with thoracic trauma in foals.[12,50] These clinical findings and the occurrence of this condition during the first week of life suggest that injury occurs during parturition. The cause is believed to be pressure on the thorax during passage of the foal through the pelvic canal, but the thoracic circumference or the foal's weight does not appear to be a factor in the development of thoracic trauma.

Clinical signs that may create suspicion of fractured ribs in foals include asymmetry of the thoracic cavity, groaning or grunting with respiration, plaques of subcutaneous emphysema overlying the ribs or ventral thorax, and crepitation or pain on palpation of the affected ribs. In a field study, 90% of affected foals were detected by palpation with the foal standing, but the thoracic cage asymmetry was better appreciated with the foal in dorsal recumbency.[1] As mentioned before, ultrasonography is better than radiography to determine the number of ribs fractured and the severity of fracture. In addition, ultrasonography permits characterization of associated thoracic abnormalities. The presence of unilateral or bilateral pleural fluid, pulmonary contusion, or pericardial effusion, the number of fractured ribs, and the degree of fragment displacement are better evaluated with ultrasonography than with radiography.[49] Because more than three ribs are usually involved, affected foals can present with flail chest. The flail segment can also develop when ribs fracture in a single dorsal plane and the flexible costochondral junction becomes the second movement plane.[51]

Management of foals with simple rib fractures consists of strict confinement for 1 to 4 weeks and supportive care with analgesics, anti-inflammatory drugs, antibiotics, and antiulcer medications. Foals with flail chest and concomitant pulmonary trauma are kept recumbent with the affected side down, and intranasal oxygen insufflation is administered. Lateral recumbency with the affected lung down minimizes ventilatory impairment of the better-ventilated lung (on top).[12] Foals are kept in recumbency and sedated as necessary until the respiratory difficulties improve. However, lateral recumbency may lead to intrathoracic damage if axial displacement of the fracture is present. In these cases, foals can be placed in sternal recumbency. Surgical intervention is usually not necessary, but it has been pursued in foals when there is a potential for intrathoracic injury, which can occur with axial displacement of fracture fragments. There are reports of the use of both external coaptation and internal fixation of the affected ribs.[49,52] External coaptation was achieved by placing stainless steel suture material around the ribs and fixing the wires to cast material molded to the rib cage.

Internal fixation seems to be a better option to successfully manage foals with complicated rib fractures, or when the risk of intrathoracic injury is high. A report evaluated the repair of rib fractures using internal fixation techniques in 14 foals.[52] Surgery was performed because the foals were considered at great risk for developing life-threatening injuries. A surgical procedure was performed on an average of two ribs (one to three ribs). After fracture reduction was achieved, a 2.7-mm reconstruction plate was contoured to the rib and applied with 2.7-mm–diameter cortex screws engaging both cortices (Figure 48-3). Four to six cortices of fixation on each fracture fragment were recommended. In addition, cerclage wire (18 to 22 gauge) was placed encircling the rib and plate at two sites above and two sites below the fracture. Application of the cerclage wire prevented implant failure from pullout of the cortex screws. Stall confinement was recommended for 4 to 6 weeks or until healing, which was evaluated with the use of ultrasonography. Of 14 foals treated using internal fixation, 12 were discharged from the hospital. Follow-up information on seven foals revealed that all were in good health and performing as expected, with the plates left *in situ*. Complications occurred in six foals and included implant failure, seroma formation, and subcutaneous hematoma.[52]

Another internal fixation technique described to repair multiple rib fractures in foals consisted of using 80-pound nylon strand (SCCLRS).[53] After the fracture fragments are exposed and adequately reduced, the nylon strand is passed in a figure-of-eight fashion through holes drilled in the cranial cortex of the proximal and distal fracture fragment ends and then secured with SCCLRS crimp clamps (Figure 48-4). The technique is repeated for each fractured rib. A similar technique was described by Down and Rodgerson but using commercially available, gas-sterilized nylon cable ties placed through drilled holes in the fracture fragment.[54] The surgical method was successful in the treatment of seven foals, and no complications directly related to the surgical technique were reported. These two similar techniques for rib fracture repair in foals offer a safe, relatively easy, and economically advantageous procedure to use when surgical intervention is deemed necessary.

The use of locking internal fixation implants, such as LCP technology, seems a logical, but more expensive, option (see Chapter 76).

The risk of operating so close to the thoracic cavity is present with any of these methods of fixation. Care must be taken not to enter the thoracic cavity when dissecting the tissue, reducing the fracture, and drilling the implant holes to avoid creating a pneumothorax and injuring internal thoracic organs. In all

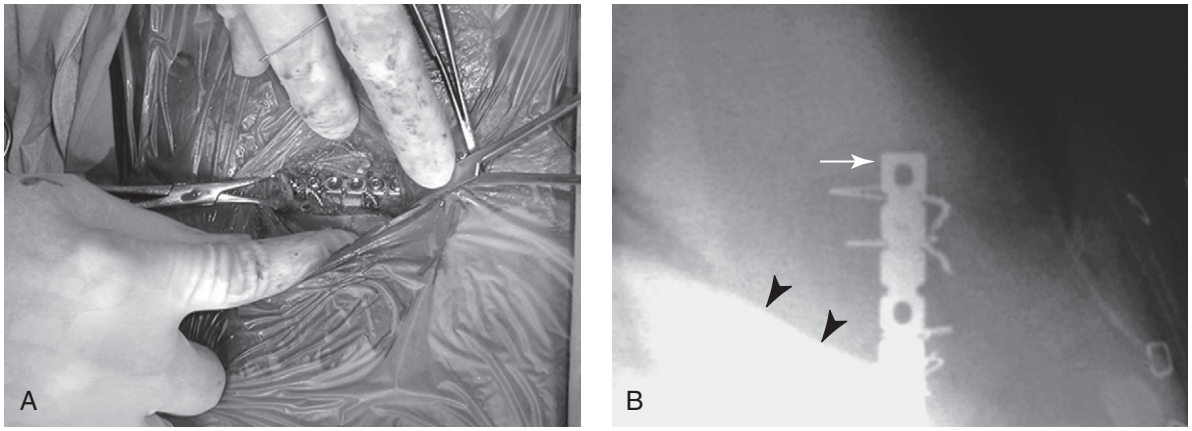

Figure 48-3. A, Intraoperative view during repair of a displaced rib fracture in a foal. Notice the reconstructive plate, cortical screws, and cerclage wires. Towel calmps were used to reposition and align the fractures fragments; **B,** Postoperative radiograph of a fractured rib in a foal, repaired with the same tchnique of reconstructive plate *(white arrow),* cortical screws, and cerclage wires. Note the caudal aspect of the distal humerus *(black arrowheads).* (Courtesy Dr. F. Bellezzo and Dr. D. Rodgerson.)

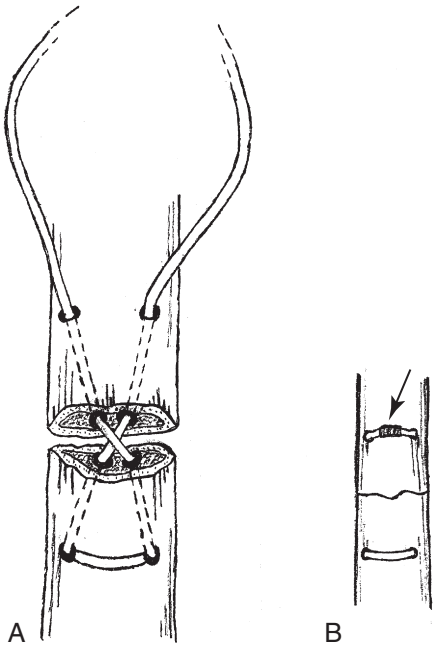

Figure 48-4. A, Schematic of a fractured rib (cranial is to the right and ventral is at the bottom) showing suture placement using an 80-pound nylon strand (SCCLRS, Securos Veterinary Orthopedics, Charlton, MA); **B,** Complete reduction of the fracture after application of the tension device and closure of the SCCLRS crimp clamp *(arrow).*[53] (Courtesy Dr. Beth Krauss.)

cases, broad-spectrum systemic antibiotics, nonsteroidal anti-inflammatory drugs, and analgesic medications are recommended.

PLEUROPNEUMONIA

Pleuropneumonia is a serious disease in the horse, and it has a significant economic impact on the equine industry. Treatment of horses with bacterial pleuropneumonia is frequently expensive, and it is time consuming. Resolution of the infection and response to therapy can be prolonged, even with early detection and aggressive treatment. Survival rates vary from 43% to 75% depending on the microbial pathogens involved, the rapidity of diagnosis, the institution of appropriate treatment, and the development of secondary complications.[55-57] Many survivors are unable to return to their previous level of performance.[55]

Risk Factors

Bacterial pleuropneumonia most commonly occurs in horses subjected to physiologic stress, including long-distance transport, high-speed exercise, general anesthesia, and recent viral infection.[55,56,58,59] Horses subjected to long-distance transport have higher numbers of circulating neutrophils, an increased neutrophil-to-leukocyte ratio, elevated plasma cortisol levels, and decreased phagocytic function of peripheral blood neutrophils.[60,61] In addition, the practice of shipping horses with their heads secured in an elevated position results in increased bacterial and pollutant contamination of the lower airway compared with individuals allowed to lower their heads during transport.[62,63] Many horses refuse to drink appropriately during transport, and subsequent dehydration further compromises mucociliary clearance mechanisms.[64] During extended travel, periodic rest stops allowing the horse to lower its head and drink are necessary to resolve this contamination problem and to facilitate the correction of dehydration. However, rest stops may be infrequent with commercial hauling operations; consequently, the shipped horse is at increased risk of infection of the lower airway.

High-intensity exercise results in transient decreased peripheral blood neutrophil function and oxidative burst activity of pulmonary alveolar macrophages.[65,66] In the United States, Thoroughbred horses in race training are 4.3 times more likely to develop pleuropneumonia than nonracing Thoroughbreds, suggesting that career, not breed, is the risk factor.[59] In contrast, in Great Britain, show jumpers have the highest risk of developing pleuropneumonia.[67] The reason for this difference may be partly because most races are run on turf in the United Kingdom but on dirt in the United States, which would contaminate the airways during races. Additionally, European show jumpers frequently travel long distances for competitions, which may explain the increased risk in horses in this discipline.[67]

Endotracheal intubation, dorsal recumbency, and anesthetic-induced depression of the respiratory defense mechanisms may all increase the risk of lower airway contamination with general anesthesia. Respiratory viral infections result in damage to airway epithelium and breakdown of clearance mechanisms, further increasing the risk of bacterial colonization of the lower airway.[59] Penetrating thoracic injuries and pulmonary aspiration secondary to esophageal obstruction or upper airway dysfunction have also been associated with an increased risk of pleuropneumonia.[56,59,67]

Clinical Signs

Clinical signs vary depending on the chronicity and severity of disease and on the volume of pleural effusion. In the peracute stage, fever, inappetence, lethargy, depression, and exercise intolerance are common. Pleurodynia (pleural pain) secondary to pleural inflammation may be manifested by anxiety, pawing, reluctance to move, abduction of the elbows, and grunting when the thorax is percussed. A soft cough, dyspnea, tachypnea, and tachycardia may or may not be present during the peracute phase. Lung sounds vary depending on the severity of pulmonary and pleural changes, and a rebreathing examination is recommended to increase the depth of respiration and to accentuate audible lung sounds. Although lung sounds may be relatively normal during the peracute stages, the patient often becomes distressed by the rebreathing examination (because of increased pleural pain) and may begin to cough violently.

With progression of the disease and accumulation of pleural effusion (acute to chronic stages of the disease), auscultation reveals ventral dullness with pleural friction rubs and adventitial sounds (crackles and wheezes) in the dorsal lung fields. Areas of dullness can also correlate with pulmonary or pleural abscessation. Cardiac sounds frequently radiate over a larger than normal area as a result of the pleural effusion and improved sound conduction. Nasal discharge can vary between absent and copious and can range in consistency from serous to purulent. A chronic brown, bloody nasal discharge indicate pulmonary hemorrhage and infarction.[68] A foul, fetid odor to the breath is frequently associated with anaerobic infections.[69,70]

Accumulation of pleural effusion results in an increased respiratory effort with a slow prolonged pattern of inspiration and expiration. This pattern is the result of pleural fluid accumulation and its effects on lung volume and expansion of the chest wall.[71] Edema can frequently be palpated between the pectoral muscles and along the ventral midline, and it is a nonspecific finding with pleural effusion. Dramatic weight loss over a relatively short time results from the catabolic nature of the disease coupled with a decreased (or absent) appetite.

Diagnosis

The diagnosis of bacterial pleuropneumonia is based on clinical findings (fever, pleural pain, abnormal lung sounds), abnormalities detected in a complete blood count, ultrasonographic and radiographic findings, and positive bacterial culture of a tracheal wash and pleural fluid sample. Thoracic percussion can be used to detect and outline the extent of pleural effusion or pulmonary consolidation. The line of demarcation between resonant percussive sounds and dull sounds is usually the dorsal margin of pleural fluid accumulation. Fluid results in a horizontal line of percussion abnormality, whereas pulmonary consolidation without effusion generally results in a more irregular outline of dullness.

Thoracic ultrasonongraphy has largely replaced the need for percussion and is a simple and accurate way to assess the volume and type of effusion, if one is present (Figure 48-5). Pleurocentesis is indicated if fluid is detected, and ultrasonography is used to determine the ideal site for the centesis. An ultrasonographic examination of the thorax in the peracute stage may reveal pleural irregularities (Figure 48-6) without significant effusion or evidence of pulmonary consolidation. As the disease process progresses, pleural effusion generally increases and pulmonary consolidation becomes increasingly evident (Figure 48-7). Because of the large reflection of ultrasonic waves at an air–tissue interface, ultrasound is less useful in determining deep lung abscessation or consolidation if the superficial lung is aerated.[72] In this situation, thoracic

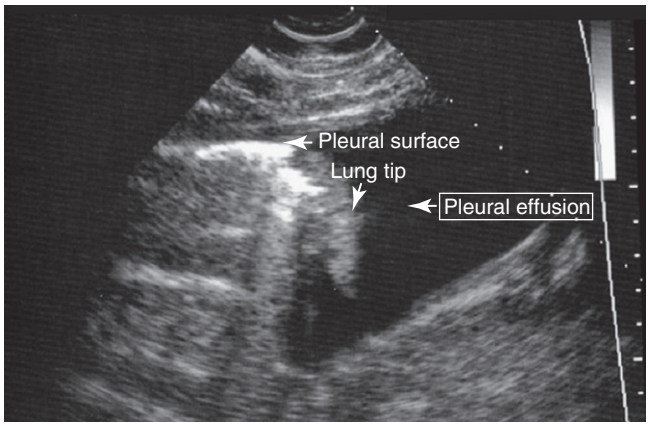

Figure 48-5. Ultrasonographic image of the left thoracic cavity of a horse with pleural effusion. A moderate volume of pleural effusion has resulted in collapse (atelectasis) of the ventral tip of the lung.

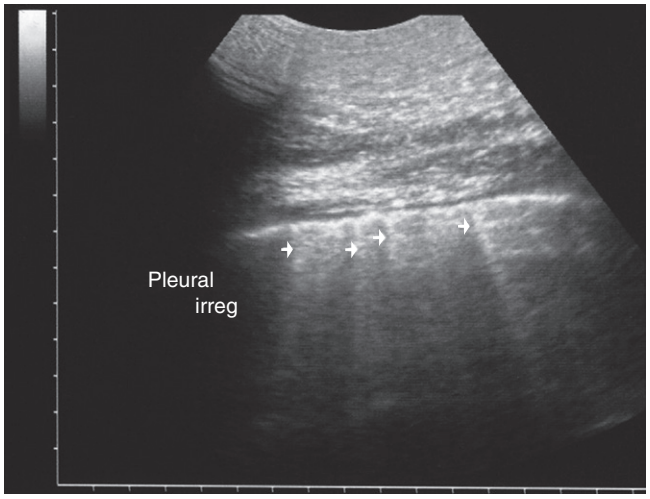

Figure 48-6. Ultrasonographic image of the left visceral pleura of a horse with peracute pleuropneumonia. The pleural surface casts irregular acoustic shadows or "comet tails" *(arrows)*. These irregular acoustic shadows are the result of pleural surface roughening. Subsequent reevaluation revealed progressive accumulation of pleural fluid and pulmonary consolidation.

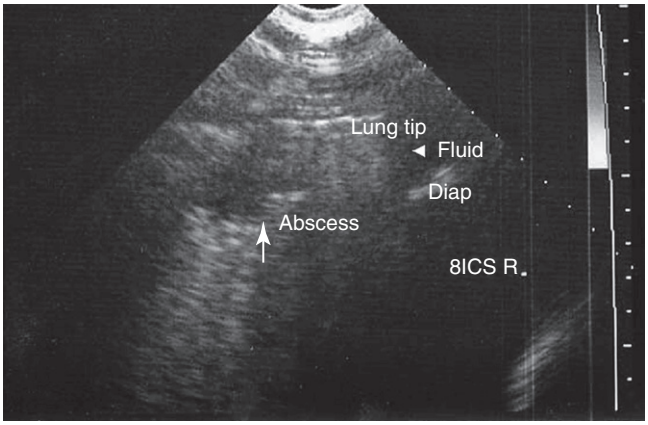

Figure 48-7. Ultrasonographic image of the right thorax of a horse with chronic pleuropneumonia. Minimal pleural effusion is evident *(arrowhead).* Consolidated or abscessed lung is evident by the break in the pleural surface and the ability to image the deeper parenchyma *(arrow).*

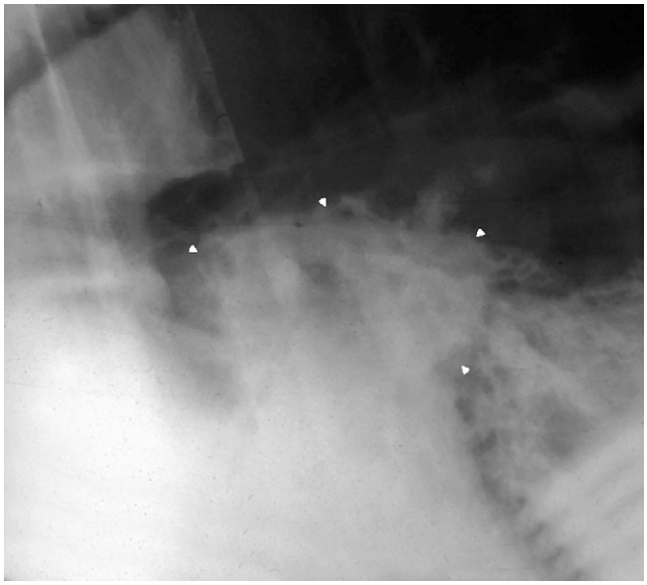

Figure 48-8. Thoracic radiographs of a horse with chronic pleuropneumonia and a large pulmonary abscess *(arrowheads).* Pleural effusion was removed via pleurocentesis prior to radiographic evaluation.

radiography is a more effective tool for evaluating deeper pulmonary parenchymal lesions (Figure 48-8). Drainage of pleural effusion prior to thoracic radiography facilitates accurate evaluation because pleural fluid obscures the lung parenchyma and makes it difficult to evaluate the extent or presence of consolidation or abscessation. Radiographs are useful to determine the extent of pulmonary consolidation and to evaluate progression over time; however, radiographic resolution of disease lags behind clinical resolution and, in our experience, radiographic reevaluation is more useful in evaluating long-term progression than short-term changes.

Complete blood count and serum chemistry abnormalities are relatively nonspecific and reflect the chronicity and severity of the inflammatory processes. Hyperfibrinogenemia and a neutropenia with a toxic left shift are frequently seen in the acute stage of pleuropneumonia. Other common abnormalities include hyperbilirubinemia (or anorexia) and azotemia (prerenal or renal). Neutrophilia is more commonly seen in the chronic stage of pleuropneumonia; an elevated serum globulin and anemia of chronic disease are also common. Although the abnormalities seen are nonspecific, they are useful in evaluating the response to treatment. A gradual decrease in serum fibrinogen and globulin levels, an improvement in the anemia, and a decrease in unconjugated bilirubin concentration (improved appetite) would be considered positive responses to therapy.

Aerobic and anaerobic microbial culture of tracheal wash and pleural fluid samples should be performed prior to instituting antimicrobial therapy. Alternatively, antimicrobials can be withdrawn for 24 hours prior to sample collection. Tracheal wash samples can be collected percutaneously using a nested trocar or via endoscopic techniques. Both methods are effective in experienced hands. Complications of the percutaneous method include localized cellulitis and chondritis; therefore, the site should be monitored carefully for 48 hours after sample collection. If multiple samples cannot be obtained from the chest or if financial constraints exist, the tracheal wash is the preferred sampling technique, yielding positive results with a greater frequency than pleural fluid samples.[51] However, in some cases, only the pleural fluid yields bacterial growth. Consequently, when possible, it is best to submit both sample types for culture. The most common organisms isolated include *Streptococcus, Escherichia coli, Actinobacillus, Klebsiella, Pseudomonas,* and *Staphylococcus* spp.[55,57,67-69,73] Common anaerobes include *Bacteroides* and *Clostridium* spp. Anaerobic bacteria were isolated in 25% to 68% of cases.[56,57,70,73]

In addition to culture, cytologic analysis of a sample of transtracheal aspirate can be useful to assess the type of infectious process present and to aid in determining the appropriate treatment protocol while awaiting culture results. For example, the presence of large, gram-positive coccobacilli from the transtracheal wash of a weanling foal may indicate of a *Rhodococcus equi* infection. Alternatively, the presence of fungal elements, particularly if a horse has been on long-term antimicrobial treatment, may indicate infection with fungal agents.[74]

Pleural fluid cytology alone is of limited value in determining sepsis and should be coupled with the determination of pleural fluid glucose, pH, and lactate.[75] In general, septic effusions are expected to have a low pH and high lactate and low glucose levels when compared with plasma. The finding of sepsis is a clear indication for drainage. Serial pleural fluid protein measurements and cell counts are useful to determine progress and resolution of infection. A trend toward normalization of pleural fluid values is expected with appropriate treatment and control of the infectious agents.

Medical Management

The cornerstone of medical treatment of bacterial pleuropneumonia is early institution of appropriate antimicrobial therapy. Treatment should be initiated with a broad-spectrum antimicrobial combination targeted against the most common bacterial pathogens isolated from equine pleuropneumonia cases, until specific culture results are available. A combination of a penicillin (or cephalosporin) and an aminoglycoside is a typical selection. Although many anaerobes found in equine pleuropneumonia are sensitive to penicillins, the addition of metronidazole is recommended if there is a lack of response to initial

treatment or a resistant anaerobe (such as *Bacteroides fragilis*) is cultured. Depending on the chronicity of the disease, the type of sample cultured, and prior treatment, a culture may not always identify the pathogens involved; consequently, it is critical to monitor the patient's response to treatment. Repeat cultures or alternative antimicrobial therapy is indicated if the response to therapy is inadequate. In our experience, a poor response to treatment typically occurs for one of three reasons: inappropriate antimicrobial treatment, inadequate pleural drainage, or the presence of abscessation or necrotic lung tissue. Therefore, daily assessment including ultrasonography is critically important in the successful treatment of pleuropneumonia. When subcutaneous air prevents an accurate ultrasonographic evaluation, thoracoscopy is a useful and relatively safe tool to assess the development of pleural abscesses or necrotic lung tissue.

The second important part of medical management is drainage of the pleural effusion. In mild cases, with minimal effusion, antimicrobial therapy alone may be effective in the resolution of the infection and effusion. The need for drainage should be determined on an individual basis. Indications include large volumes of effusion with or without evidence of respiratory distress, purulent or septic effusions, the presence of gas echoes consistent with an anaerobic infection, and pleural abscessation or loculation. In the acute stages of pleuropneumonia, drainage of one hemithorax frequently results in bilateral resolution of the effusion. Over time, the accumulation of fibrin and debris results in closure of the mediastinal fenestrations, requiring bilateral thoracocentesis to remove bilateral effusion.

Drainage can be facilitated by ultrasound-guided placement of small cannulas or catheters that are temporarily placed into the pleural space and removed after completion of drainage, or via placement of a semipermanent indwelling tube. Indwelling tubes have the advantage of allowing frequently repeated drainage; however, in our experience, over time these tubes become obstructed with fibrin or debris, necessitating their removal and replacement. Attachment of a one-way valve allows continuous drainage, but this can be risky if the horse rolls or otherwise breaks the valve. Alternatively, the drain can be sealed and opened multiple times during the day to allow more control of the drainage. Localized cellulitis is a frequent complication of indwelling cannulas. Although this is generally not a life-threatening complication, it results in increased thoracic pain and is frequently associated with a poorer appetite and general demeanor. Depending on the severity of the underlying pulmonary parenchymal disease and the type of bacteria involved, drainage can become increasingly more difficult as fibrin is produced and abscessation and loculation develop. Prior to the development of thorascopic techniques, we successfully treated cases with multiple, mature, pleural abscesses by repeated ultrasound-guided pleurocenteses and drainage. Although this was time consuming, it can be a useful technique to manage cases with multiple abscesses or loculations.

The judicious use of anti-inflammatory and analgesic drugs is recommended to control pleural pain and fever, minimize malaise, and improve attitude and appetite. Nonsteroidal anti-inflammatory drugs (NSAIDs) are potent analgesics and block production of inflammatory mediators that produce many of the systemic signs seen with pleuropneumonia. The value of NSAIDs in the treatment of pleuropneumonia needs to be weighed against the risks (nephrotoxicity and gastrointestinal ulceration), particularly in patients that are not eating or drinking adequately. The short-term use of small intramuscular doses or continuous-rate infusions (CRIs) of the α_2-agonist detomidine, a potent analgesic, has been effective in controlling severe, nonresponsive pleural pain.[76] α_2-Agonists should be avoided in febrile or hypovolemic patients until these conditions are corrected. Butorphanol, a potent opiate analgesic, can be administered intramuscularly or via CRI as well, and it may have the added benefit of suppressing the cough response (see Chapter 23).[77] Lidocaine infusions have also been reported to have an analgesic effect and may be an additional choice for pain control.[78] The use of combination therapies offers several advantages over single-drug treatment protocols, including more effective pain control and the ability to minimize potential toxic side effects.

Supportive Care

Intravenous fluid therapy may be necessary in the initial treatment of the hypovolemic or dehydrated patient, but it is rarely required for chronic cases of pleuropneumonia. In addition to fluid therapy, nutritional support should be addressed. Many of these patients have a decreased (or absent) appetite, which, coupled with the catabolic nature of the disease, results in significant, often dramatic, weight loss if not addressed quickly. Feeding of palatable feeds, access to grass, and gradually increasing concentrates in the diet are important methods to increase caloric intake. The addition of corn oil or rice bran to the diet may also be of benefit. In some individuals, enteral supplementation via nasogastric intubation or parenteral nutrition may be required to adequately supply the patient's metabolic needs. Continued anorexia may indicate inadequate pain control, uncontrolled infection, or severe underlying lung pathology (pulmonary necrotic debris, abscessation, or neoplasia). Patients that continue to be anorexic despite aggressive therapy have a poor long-term prognosis.

Surgical Treatment

Surgical intervention is typically used to assist in the medical treatment of equine pleuropneumonia. Long-term antimicrobial therapy is rarely effective in penetrating and resolving large abscesses or loculations when substantial fibrosis or septic fluid is present.[79] Open and closed thoracotomy, with or without rib resection, has been successfully used for drainage of thoracic abscesses and resection of adhesions.[80-83] Surgical manipulations can be used to reduce excessive fibrin, which can result in loculations and adhesions, drain pulmonary or extrapulmonary abscesses, resect necrotic lung tissue, or perform pericardectomy in cases of septic pericarditis.[17,80,84,85]

Vachon and Fischer reported the use of thoracoscopy as a useful therapeutic tool in 16 cases of pleuropneumonia.[17] Thoracoscopy facilitated the placement of thoracic drains in abscesses and loculated pleural effusion and the transection of pleural adhesions to disrupt loculations, and it allowed direct evaluation of the lungs and pleural cavity (Figure 48-9). Thoracoscopy provides a superior exploration of the pleural cavity compared with incisional thoracotomy.[23] Furthermore, the minimally invasive nature of thoracoscopy results in less morbidity and aftercare.

After establishing adequate pleural drainage, pleural lavage can be performed with isotonic balanced fluid solutions through

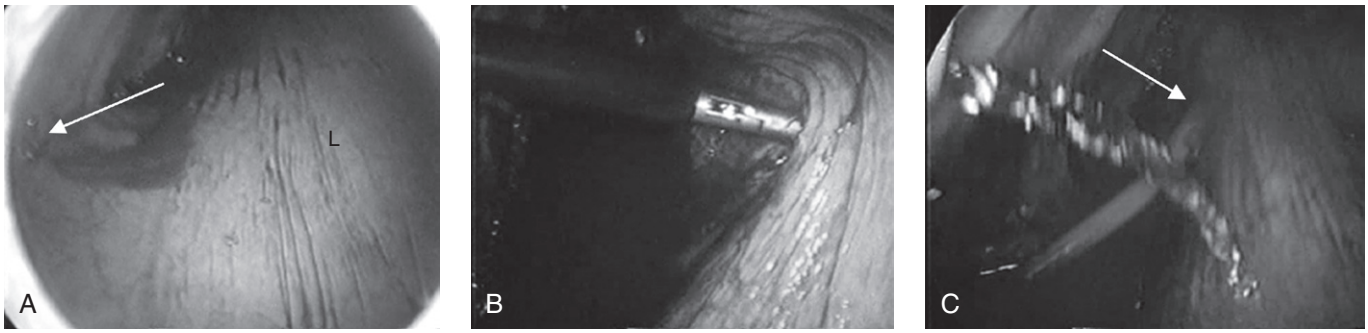

Figure 48-9. Thoracoscopic view of the left pleural cavity in a horse with pleuropneumonia. **A,** The collapsed lung *(L)* is adhered to the parietal pleura by fibrinous adhesions *(arrow).* **B,** With the use of endoscopic instruments, the lung is separated from the adhered parietal pleura. **C,** After adequate drainage was established, a large chest tube *(arrow)* was introduced into the thorax and the pleural cavity was lavaged with isotonic saline.

the cannula system. Adhesions are more readily disrupted the first week after formation when the tissue is fibrinous rather than fibrous. Transection of mature, fibrous adhesions can be difficult and tedious, and it can cause profound hemorrhage. Therefore, conservative transection of mature adhesions is recommended. Careful insertion of instruments or portals into the thorax is mandatory in horses with severe pleuropneumonia and with extensive pleural adhesions or anaerobic infections. Adhesions between the lungs and the thoracic wall may not allow complete collapse of the lung, making the manipulation of the instruments more difficult and limiting the view of the pleural cavity. This can increase the risk of injury to vital organs (e.g., lungs, diaphragm).[17] Before introducing the trocar–cannula system in horses with possible adhesions, digital palpation of the lungs and pleura through the endoscope portal is recommended to avoid iatrogenic injury to these structures. In addition, the use of CO_2 insufflation (at less than 5 mm Hg) can facilitate and maintain collapse of the lung, improving surgical exposure.

Open drainage via thoracotomy should be considered for a chronic pleural effusion that has responded poorly to medical therapy, for localized or unilateral disease processes, for walled-off abscesses, or when tube drainage is inadequate.[79,85] Before performing open thoracic drainage, the horse's tolerance for pneumothorax must be assessed. This can be done with thoracoscopy or by inserting a large chest tube into the targeted cavity and leaving it open prior to thoracotomy.[17,85] If no respiratory distress is observed, the diseased pleural hemithorax is likely to be isolated from the rest of the thoracic cavity and is amenable to open drainage. Because these horses have compromised pulmonary function and are poor candidates for general anesthesia, the procedure is typically performed in a standing position. However, general anesthesia with the horse in lateral recumbency is recommended to drain abscesses in the cranial mediastinum or when an abscess is not isolated from the rest of the pleural cavity.[86] Positive-pressure ventilation with a mechanical ventilator is mandatory. The use of isoflurane or sevoflurane is also recommended for gas anesthesia, because these drugs do not depress the equine myocardium as profoundly as halothane. Therefore, the combination of assisted ventilation, oxygen, and isoflurane or sevoflurane should be used to support horses undergoing a thoracotomy procedure when general anesthesia is required.

The preferred surgical approach to drain intrathoracic abscesses is determined by careful localization of the abscess by radiography and ultrasonography.[87] For the standing approach, local anesthesia is infiltrated into the skin, subcutaneous tissue, intercostal musculature, and costal pleura at the proposed incision site, and a local nerve block of the intercostal nerves is performed two intercostal spaces cranial and caudal to the site. A 15- to 25-cm (6- to 10-inch) incision is made through the skin, intercostal musculature, and pleura over the selected intercostal space into the thorax. Manual exploration and débridement of the purulent material is followed by lavage with isotonic balanced saline solution. The incision is left open and the diseased cavity is irrigated twice daily until the surgical wound heals by second intention.

If thoracotomy with rib resection is elected, a 30-cm (12-inch) incision is made over the selected rib, through the skin to rib periosteum. In the cranial aspect of the thoracic wall, the cutaneus trunci muscle is incised at a right angle to its fibers. The *M latissimus dorsi* and *M serratus ventralis* are subsequently incised separately. The external thoracic vein is identified near the dorsal edge of the pectoral musculature, isolated, and retracted. Careful dissection of thoracic wall musculature avoids damage to the intercostal vessels and nerves, which are located at the caudal aspect of each rib. To expose the entire length of the rib, the fascia from the attachment of the external intercostal muscle is bluntly dissected. The periosteum is incised along the superficial surface of the rib for a distance of 25 cm (10 inches) and reflected circumferentially with a periosteal elevator (Figure 48-10). The periosteum is separated from the rib, and an obstetric wire is passed around the most proximal aspect of the rib and transected. The rib is subsequently disarticulated at the costochondral junction and removed. A small stab incision should be made through the periosteum and pleura, and the opening is enlarged with scissors. A Finochietto rib retractor can be used to separate the ribs for better surgical exposure, but this can cause discomfort in the standing horse. Thoracic exploration and débridement is subsequently performed. Fluid and necrotic tissue are manually removed, taking care not to disrupt any mature adhesions.[87] If a localized abscess is encountered, a Foley catheter can be introduced from a separate stab incision through the thoracic wall into the abscess. The abscess can then be drained and lavaged, and the catheter can be removed or left *in situ* and secured to the chest wall for future irrigation. The thoracic incision is usually left open to heal by second intention.

If the thoracotomy is closed, three layers of sutures are recommended. A rib contraction device (Bailey-Gibbon rib

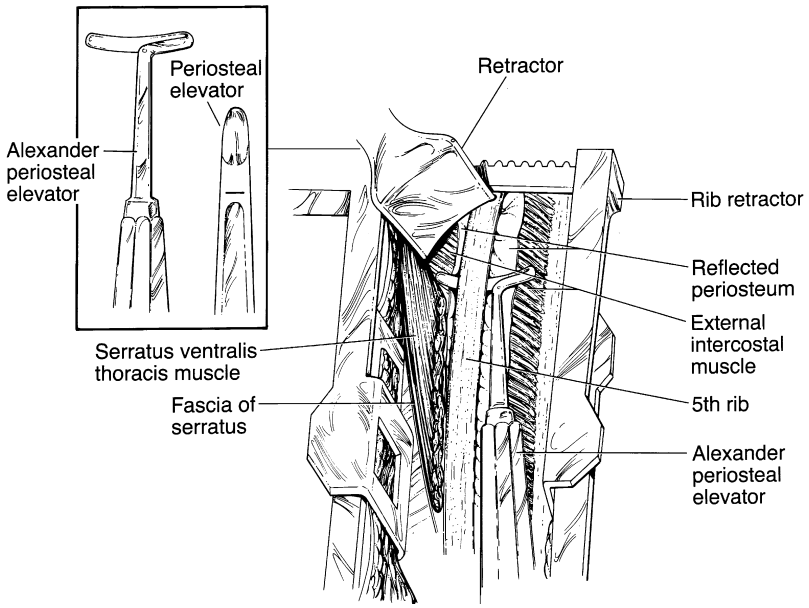

Figure 48-10. Surgical removal of the fifth rib with appropriate rib retractors and periosteal elevators. (Redrawn from the original by Gheorge M. Contantinescu, DVM, PhD, Dr hc.)

contractor) may facilitate the apposition of the thoracic wall. First, the periosteum is carefully apposed with size 0 or 1 absorbable suture material with a simple-interrupted pattern. The final suture in the first layer of the thoracic wall closure should be tightened while the lungs are fully inflated to reestablish the normal negative pressure of the pleural cavity. The intercostal musculature is subsequently closed in routine fashion with size 1 or 2 absorbable sutures. Finally, routine apposition of the skin edges with nonabsorbable suture material is recommended. Thoracic tubes are placed as necessary to alleviate a potentially remaining pneumothorax or to drain pleural fluid.

Complications after thoracotomy include cardiac arrhythmias, thoracic wall abscesses, chronic draining tracts, and severe bilateral pneumothorax.[82,83] In contrast to thoracotomy, only mild discomfort during manipulation of instruments was reported during thoracoscopy in horses affected by pleuropneumonia.[17] However, thoracoscopy is not without risk; complications including injury to vital organs (such as the lung and diaphragm) have been reported.[21,22] Surgical treatment does not replace medical management of equine pleuropneumonia but can, in selected cases, improve the prognosis of these horses.

Prognosis

In retrospective reports, the survival rate for horses with bacterial pleuropneumonia varied from 43% to 75%.[55-57] The presence of anaerobic bacteria has been associated with a poorer survival rate in some studies but not others. Delay between the onset of clinical signs and the diagnosis and start of treatment is also associated with a poorer outcome.[63] The majority of horses that do not survive bacterial pleuropneumonia are euthanized for reasons including poor response to treatment, development of complications, expense involved in treatment, or a poor prognosis for return to performance. In a study of racehorses that survived pleuropneumonia, 61% returned to racing, with 56% of those winning at least one race. However, racing class was not examined in this report.[58] Complications associated with pleuropneumonia include bronchopleural fistulas, spontaneous pneumothorax, cranial thoracic abscessation, jugular vein thrombosis, pulmonary infarction, laminitis, and, rarely, pericarditis and peritonitis.[55-59,67-69,73,86] A positive response to treatment is indicated by resolution of fever, return of appetite, and gradual reduction in the volume of effusion. Negative responses to treatment include recurrent fevers, continued inappetence and weight loss, continued effusion (particularly with progression of the effusion to an exudate), and development of the complications mentioned previously. These findings, coupled with a continued elevation in serum fibrinogen and globulins and an abnormal WBC count, should stimulate careful reevaluation of the individual. Even with appropriate and early treatment, the response is often slow, with treatment durations ranging from 3 weeks to several months. After resolution of the underlying infection at least 3 to 6 months of recovery time should be allowed prior to return to training and performance.

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CHAPTER
49

Anatomy and Physiology of the Nervous System

Robert J. MacKay

DEVELOPMENT AND ORGANIZATION OF THE NERVOUS SYSTEM

The nomenclature relating to embryologic, medical, and common description of the parts of the brain is inconsistently applied and can be confusing. Table 49-1 shows the principal naming systems used for the parts of the brain, Table 49-2 gives numeric and medical names for the cranial nerves, and Figure 49-1 depicts the locations of the cranial nerves on the ventral aspect of the brain.

The nervous system derives from a specialized strip of ectoderm, the neural plate, which runs along the dorsal midline of the developing embryo.[1] Ventral to the neural crest is the primordial vertebral organ, the notochord, which develops in concert with the central nervous system (CNS). The sides of the neural crest fold upward and medially to meet in the midline and form the neural tube.

Beginning at the brain–spinal cord junction, the neural tube closes progressively both rostrad and caudad to encircle the developing brain and spinal cord.[2] The rostral part of the neural tube buds large vesicles laterally on either side, which develop caudad and ventrad to form the hemispheres of the cerebrum (telencephalon), leaving the rest of the rostral tube to form the thalamus and hypothalamus (diencephalon) (see Table 49-1). The lumen of the neural tube (the neural canal) forms the interconnected ventricular system of the diencephalon (third ventricle) and cerebrum (lateral ventricles). Together, these structures become the *forebrain* (prosencephalon). Cranial nerves I and II arise from the forebrain (see Table 49-2). Successively more caudal dilatations of the neural tube develop into the *midbrain* (mesencephalon) and *hindbrain* (rhombencephalon), respectively. The brain structures directly derived from the neural tube proper—namely, the diencephalon, midbrain, and hindbrain—comprise the *brain stem*. Development of the *neuropil*, the dense feltwork of interwoven cytoplasmic processes of dendrites, neurons, and glial cells, of the midbrain encroaches on the neural canal until it is a narrow tube, the mesencephalic aqueduct. The rostral part of the hindbrain (metencephalon) develops into the pons and cerebellum, while the caudal part, which is contiguous with the spinal cord, forms the medulla oblongata (myelencephalon). Within the pons and medulla, the neural canal forms the fourth ventricle, connecting the mesencephalic aqueduct rostrally with the spinal (central) canal caudally. Cranial nerves III and IV (midbrain), V (pons), and VI to XII (medulla) develop in the ventrolateral brain stem.

Within the neural tube of the developing spinal cord, primitive neurons become organized into mantle and marginal layers that become white and gray matter, respectively.[2] Ventral mantle neurons grow through the surrounding marginal layer and course outside the neural tube to form ventral spinal nerve roots. Dorsal ganglia and roots form from the growth of neural crest cells centrad into the neural tube and distad to sensory structures. The developing gray matter becomes divided morphologically and functionally into dorsal, intermediate, and ventral columns. Ventral and dorsal roots and gray matter divide spinal cord white matter into three regions called funiculi. These are dorsal, lateral, and ventral on each side of the spinal cord (Figure 49-2).

TABLE 49-1. Naming Systems for Principal Parts of the Brain

Primary Segments	Secondary Segments	Principal Divisions	Common Usage	Cavities
Prosencephalon (forebrain)	Telencephalon	Cerebrum	Brain	Lateral ventricle
	Diencephalon	Thalamus	Brain stem	3rd ventricle
		Hypothalamus		
Mesencephalon (midbrain)	Mesencepahlon	Midbrain	Brain stem	Mesencephalic aqueduct
Rhombencephalon (hindbrain)	Metencephlon	Cerebellum	Brain stem*	4th ventricle
	Myelencephalon	Pons	Brain stem	
		Medulla oblongata	Brain stem	

*Some authors exclude the cerebellum from definitions of the brain stem

TABLE 49-2. Names of Cranial Nerves

Number	Name
I	Olfactory
II	Optic
III	Oculomotor
IV	Trochlear
V	Trigeminal
VI	Abducens
VII	Facial
VIII	Vestibulocochlear
IX	Glossopharyngeal
X	Vagus
XI	Accessory
XII	Hypoglossal

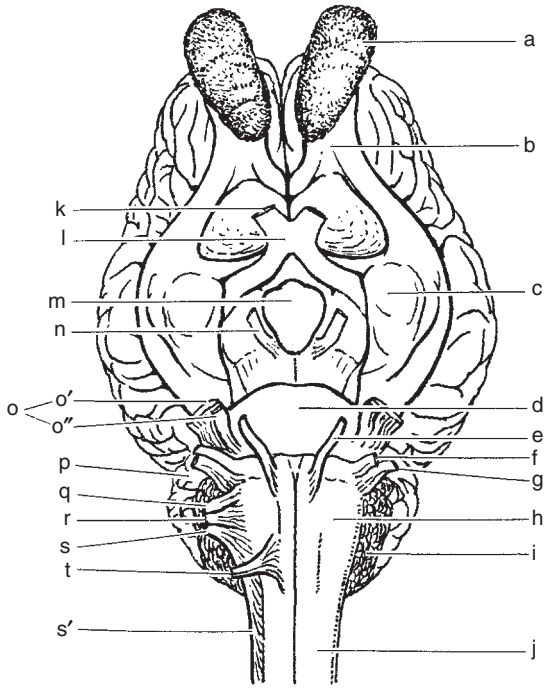

Figure 49-1. Base of the brain showing cranial nerve roots. *a,* Olfactory bulb; *b,* olfactory tract; *c,* piriform bulb; *d,* pons; *e,* abducens nerve; *f,* facial nerve; *g,* vestibulocochlear nerve; *h,* medulla oblongata; *i,* choroid plexus of the fourth ventricle; *j,* spinal cord; *k,* optic nerve; *l,* optic chiasm; *m,* pituitary gland, *n,* oculomotor nerve; *o,* trigeminal nerve (*o′,* motor root, *o″,* sensory root); *p,* cerebellum; *q,* glossopharyngeal nerve; *r,* vagus nerve; *s,* spinal accessory (medullary root); *s′,* spinal accessory (spinal root); *t,* hypoglossal nerve.

The Cranium

The cranium is made up of the occipital, sphenoid, ethmoid, interparietal, parietal, frontal, and temporal bones.[3,4] These bones enclose a cranial cavity with an approximate 650-mL volume in an adult horse. The roof of the cranium features large rostral and caudal concavities, which accommodate the hemispheres of the cerebrum and cerebellum, respectively (Figure 49-3). The internal occipital protuberance separates these cavities dorsally and provides attachment for the tentorium cerebelli, the tent-shaped extension of the dura mater that forms a partition between the cerebrum and cerebellum. The occipital

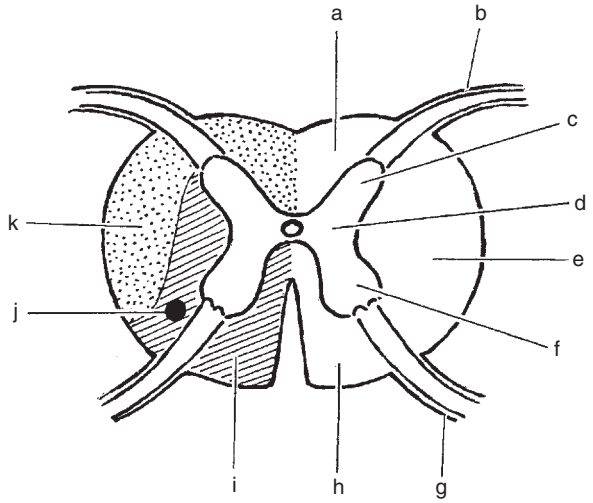

Figure 49-2. Simplified diagram of the functional *(left)* and anatomic *(right)* organization of the spinal cord at the level of the seventh cervical segment. *a,* Dorsal funiculus of the white matter; *b,* dorsal root; *c,* dorsal column of the gray matter; *d,* intermediate column of the gray matter; *e,* lateral funiculus of the white matter; *f,* ventral column of the gray matter; *g,* ventral root; *h,* ventral funiculus of the white matter; *i,* upper motor neuron *(descending)* tracts; *j,* sensory/pain *(ascending)* tracts; *k,* proprioceptive *(ascending)* tracts.

and petrous temporal bones encase the cerebellum dorsally and laterally. The cerebrum is covered dorsally largely by the parietal bones, with smaller contributions from the interparietal bone caudally and the frontal bones rostrally. The rostral or nasal wall of the cranial cavity is formed by the cribriform plate of the ethmoid, which separates the cranium from the nasal cavity. It is perforated by numerous foramina through which olfactory nerve bundles pass. On the dorsal midline is the internal parietal crest, which furnishes attachment to the falx cerebri, the sickle-shaped fold of dura, which separates the cerebral hemispheres. The forebrain may be injured by impact to the frontal or parietal area of the skull.[5] As a consequence of swelling, cerebral lobes may herniate under the falx or tentorium and compress other parts of the brain.

The floor of the cranial cavity is organized into three fossae.[4] The rostral fossa supports the frontal and olfactory parts of the cerebrum. It is formed chiefly by the sphenoid bone. Caudally in this fossa is a bony shelf, which covers the entrance to the optic canals and supports the optic chiasm. The optic nerves are firmly attached within the optic canals and may be injured within the canals by the to and fro motion of the brain after skull impact.[6] From this point caudad to the junction between the basilar portions of the occipital and sphenoid bones is the middle fossa. This is the largest and widest part of the cranial cavity. In its middle is the hypophyseal fossa and the caudal aspect supports the midbrain. On either side are two grooves, the medial one provides passage for cranial nerves III, IV, V (ophthalmic branch), and VI to the optic, trochlear, and orbital foramina; the lateral one leads to the foramen rotundum and contains the maxillary nerve (V). The posterior fossa is formed by the basilar part of the occipital bone. This bone supports the pons and medulla oblongata and the posterior fossa continues dorsad to enclose the cerebellum. On either side of the basilar part of the occipital bone is the foramen lacerum, through which course cranial nerves V (mandibular branch), IX, X, and

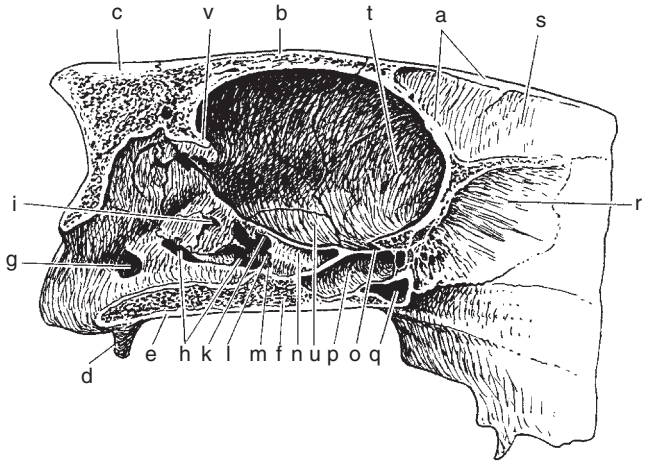

Figure 49-3. Sagittal section of cranium. *a,* Frontal bone; *b,* parietal bone; *c,* occipital bone (squamous part); *d,* paramastoid process; *e,* basioccipital bone; *f,* basisphenoid bone; *g,* hypoglossal foramen; *h,* foramen lacerum; *i,* internal acoustic meatus; *k*, spinous notch; *l,* oval notch; *m,* carotid notch; *n,* groove for maxillary nerve; *o,* optic foramen; *p,* sphenoidal sinus; *q,* central compartment sphenoidal sinus; *r,* perpendicular plate of ethmoid bone; *s,* septum between frontal sinuses; *t,* orbital wing of sphenoid bone; *u,* temporal wing of sphenoid bone; *v,* internal occipital protuberance.

XI. Caudolaterally, at the level of the occipital condyles, is the hypoglossal foramen through which passes cranial nerve XII. The ventral aspect of the basilar bones display tubercles at the points of attachment of the large paired rectus capitis ventralis major (longus capitis) and minor muscles. After poll impact, which typically results from a horse flipping over backwards, the wrenching action of these muscles may fracture or separate the basilar parts of the occipital or sphenoid bones and damage nerves in the adjacent foramen lacerum.[7]

The temporal bone forms most of the lateral wall of the cranium.[4] It is related to the occipital caudally, the parietal dorsally, the frontal rostrally, and the sphenoid bone ventrally. There are two distinct parts, the squamous and petrous bones. The squamous bone is plate-like in shape and overlaps laterally much of the petrous bone. The petrous bone is a dense pyramid-shaped bone that forms the lateral margin of the foramen lacerum. The external acoustic meatus opens on the lateral side. Housed within the petrous temporal bone are the cavities and specialized bones of the auditory and vestibular systems, including the large ventrally-projecting osseous bulla. Medially, the petrous temporal bone is related to the cerebellar hemisphere and transmits cranial nerves VII and VIII through the internal acoustic meatus. The temporal bone is vulnerable to fracture after poll impact.[5] A short rodlike projection, the hyoid process, projects from the base of the petrous slightly in front of the stylomastoid foramen, through which passes cranial nerve VII. The temporohyoid joint is formed by attachment of the hyoid process to the tympanohyoid cartilage and thus to the stylohyoid proper. A condition involving remodeling and fusion of this joint (temporohyoid osteoarthropathy) subjects the petrous bone to unusual stresses, which may result in fracture and signs of cranial nerve VII and VIII injury.[8]

The caudal fossa is continuous with the vertebral canal via the foramen magnum. As a result of increased pressure in the caudal fossa, the caudal part of the cerebellum may be squeezed through the foramen magnum (tonsillar herniation).[9]

Joints between the plates of the cranium are immobile synarthroses[4]; some, such as the joint between the basilar parts of the occipital and sphenoid bones, are bound together by cartilage (synchondroses) and others, such as the temporoparietal joint, are united by fibrous tissue (sutures). These joints usually ossify and become completely fused. The spheno-occipital synchondrosis fuses at about 5 years of age. The suture between the parietal bones ossifies at 4 years, the parieto-occipital suture at 5 years, and the parietotemporal suture at 12 to 15 years.

The Vertebral Column

The vertebral column extends from the skull to the end of the tail. There are five regions: cervical, thoracic, lumbar, sacral, and coccygeal (caudal). The typical formula for the horse is C7, T18, L6, S5, Cy15-21.[3] Although regions of the spine vary as to details, the vertebral structure conforms to a common plan. Each vertebra comprises a body, an arch, and various processes. The body is a cylindrical mass oriented along the median plane and connected to other vertebrae by fibrocartilaginous discs. Together with the dorsal surface of the body, the arch completes the vertebral foramen or ring. The arch consists of two lateral pedicles and a dorsal lamina. The intervertebral foramina, formed by the spaces between the arches of adjacent vertebrae, provide passage for spinal nerves and vessels. Successive vertebral rings are strung together by dorsal and ventral longitudinal ligaments to form the vertebral canal, a protective cylinder for the spinal cord, nerve roots, and cauda equina. The arch gives rise to articular processes at either end, a dorsal spinous process, and laterally projecting transverse processes. In some vertebrae, there are additionally mamillary or accessory processes, or costal facets. There are at least five centers of ossification in each vertebra: one body, two arches, and epiphyseal plates at either end of the body.

The spine can be modeled biomechanically as three "columns." The ventral column is composed of the ventral longitudinal ligament and the ventral half of the vertebral bodies and discs. The middle column is formed by the dorsal longitudinal ligament and dorsal half of the bodies and discs, and the dorsal column consists of the arches, intervertebral joints, and associated ligaments.[10] The degree of instability after spinal injury increases as more columns are impacted; the prognosis for recovery decreases correspondingly.

In its neutral position, the spine presents a series of curves in the median plane. The cranial part of the cervical spine is concave ventrally, the cervicothoracic junction is markedly concave dorsally, the thoracolumbar spine exhibits gentle ventral concavity, and the lumbosacral junction has more marked ventral concavity.[4] The vertebral canal varies in diameter as it progresses along the spine; within the atlas it is wide so as to house the dens and the spinal cord, and there are cervicothoracic and lumbosacral dilations to accommodate the corresponding intumescences of the spinal cord. The sagittal diameters of the cervical vertebral foramina in horses of various sizes have been determined radiographically.[11] Mean sagittal diameters for horses greater than 350 kg varied from 21.3 mm for C4 to 27.4 mm for C7.

The cervical vertebrae are relatively large and long. The arch is thick and strong and forms large articular processes. The cranial articular processes are directed dorsomedially; the posterior, ventrolaterally. Disordered vertebral development, possibly related to osteochondrosis, commonly causes malformation

and arthropathy of the cervical vertebrae in young adult horses (see Chapter 51). These changes may cause cervical vertebral stenotic myelopathy with compression of the spinal cord and ataxia of the limbs and trunk. Horses with these clinical signs are commonly referred to as "wobblers."

The first two cervical vertebrae are modified to accommodate the movements and weight of the head.[4] The atlas is composed of dorsal and ventral arches, lateral masses, and wings. The ventral arch has on its upper surface, the fovea dentis, on which the dens rests. The dens projects forward from the body of the axis and is attached by strong transverse, alar, and apical ligaments to the atlas and occiput. The upper cervical area of performance and racing horses is prone to severe hyperflexion, hyperextension, and/or axial compressive forces caused by collisions at speed.[12] Such stresses may fracture any of the bony components of the upper (cranial) part of the neck or separate the dens from the atlas.

Thoracic vertebrae are relatively constrained in their ranges of intervertebral movement. Added stability is provided to this region by the bracing action of the ribs and sternum. Perhaps because of the comparatively unconstrained movement of the cranial lumbar spine, fractures or dislocations of the vertebrae around the thoracolumbar joint occur quite commonly.

The sacrum is formed by the fusion of five sacral vertebrae.[4] It is wedged between and attached to the ilia. Branches of the first four sacral nerves course through the dorsal and ventral sacral foramina. Fusion proceeds from the front of the sacrum caudad and may not be complete even in adult horses. The sacrum houses the roots of the cauda equina and a small terminal part of the spinal cord that supplies the tail. These nerve roots are vulnerable to damage as a result of sacral fractures. Such injury may manifest as cauda equina syndrome (S2 to S5 roots)[9] and/or gait abnormalities in the pelvic limbs (S1 to S2).

Cerebrospinal Fluid

Cerebrospinal fluid (CSF) envelops, bathes, and protects the CNS.[2,9] It is produced at high rate by the choroid plexuses of the lateral, third, and fourth ventricles and more generally by the meninges and ependyma. CSF passes from sites of production in the ventricles, out through the lateral apertures of the fourth ventricles, and subsequently flows forward over the cerebrum and caudad over the spinal cord (Figure 49-4). The rate of production is independent of CSF pressure. Absorption takes place in convoluted arachnoid structures, which project, mushroom-like, into cerebral veins or venous sinuses. Flow at these arachnoid villi is pressure dependent and unidirectional from CSF to blood.

The CSF plays an extremely important role in CNS homeostasis. Changes in CSF composition, as occur in subarachnoid hemorrhage[13] or bacterial meningitis, cause clinical changes out of proportion to the minor structural changes found in the underlying neuropil.

Cerebrospinal fluid is a modified transudate with lower potassium and calcium and higher chloride, sodium, and magnesium concentrations than are found in plasma. Protein (60 mg/dL or more) and cell (6 cells/μL or more) concentrations are lower than in most other body cavities. In disease, changes in CSF composition are a useful diagnostic tool. CSF collection and analysis and myelography are described in Chapter 50.

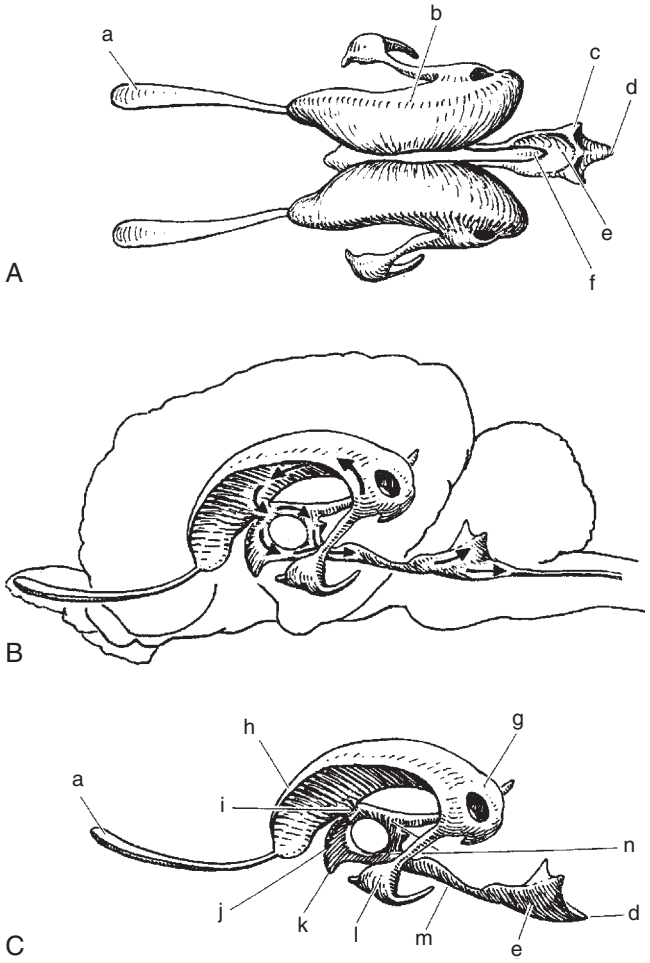

Figure 49-4. Ventricular system of the horse. **A,** Dorsal view; **B,** Lateral view showing direction of flow of cerebrospinal fluid; **C,** Lateral view. *a,* Cavity of olfactory bulb; *b,* lateral ventricle; *c,* lateral recess of the fourth ventricle; *d,* central canal; *e,* fourth ventricle; *f,* suprapineal recess, third ventricle; *g,* body of the lateral ventricle; *h,* rostral horn of the lateral ventricle; *i,* interventricular foramen; *j,* optic recess; *k,* infundibular recess; *l,* caudal horn of the fourth ventricle; *m,* mesencephalic aqueduct; *n,* third ventricle.

The CNS is physically separated from circulating blood by an extensive blood-brain barrier and a relatively minor blood-CSF barrier.[14] The blood-brain barrier is formed by endothelial cells, support cells such as astrocytes of the neuroglia and pericytes of the vascular system, and basal lamina. Endothelial cells of CNS capillaries are cemented together by continuous tight junctions. Foot processes of astrocytes (end feet) and long cytoplasmic processes of pericytes are closely adhered to the basal (i.e., nonlumenal) surfaces of CNS endothelial cells. These cells affect and support the barrier function of the endothelium. The basal lamina surrounds the endothelial cells and pericytes. A biochemical barrier against potentially injurious endogenous substances is provided by various endothelial enzymes including monoamine oxidases (e.g., epinephrine degradation) and aminopeptidases (e.g., enkephalins). Carrier and transport systems control traffic of some molecules across the blood-brain barrier. There is a Na/K-ATPase pump to regulate Na^+ and K^+, glucose transporters (including GLUT-1), amino acid

transporters, and transcellular endocytosis systems for receptor-mediated transfer of insulin, immunoglobulin G, and transferrin into the CNS. Generally, diffusion of molecules across the blood-brain barrier depends on physicochemical properties such as lipid solubility, polarity, and molecular weight. There is a close association between lipid solubility of compounds and their ability to cross the blood-brain barrier. The free, non–protein-bound, nonionized form of most drugs can passively diffuse into the CNS.

In healthy animals, the blood-CNS barrier limits the passage of immunoglobulins, complement, and potentially injurious unconjugated bilirubin. Penicillin and aminoglycosides are excluded from the normal CNS; however, the blood-brain barrier reportedly allows passage of therapeutic amounts of sulfonamide-trimethoprim, chloramphenicol, enrofloxacin, and ceftriaxone into the CNS of horses.

PHYSIOLOGY OF NEUROTRANSMISSION

The transmission and processing of information in the nervous system depends on the presence and distribution of different ion channels in neurons, the localization of synaptic circuits, and the synaptic effects of neurotransmitters.[14]

The nervous system functions through propagation of electrical signals via changes in cell membrane potential. The resting membrane potential is determined by flow of K^+ through channels open at rest and by the activity of the Na-K/ATPase pump. In response to stimuli, neurons generate local potentials that can summate. When these potentials reach the threshold for opening voltage-gated Na^+ channels, an action potential is triggered. The opening of Na^+ or Ca^{2+} channels produces neuronal depolarization, whereas opening of K^+ and Cl^- channels decreases neuronal excitability.

Neurochemical pathways in the CNS can be divided into two main groups: relay systems and diffuse projection systems.[2] *Relay systems* include the long sensory and motor pathways that connect neurons of peripheral nerves to centers in the brain. Spinothalamic, visual, vestibulospinal, and reticulospinal pathways are examples of relay systems. *Diffuse projection systems* modulate brain function by distributing signals from individual nuclei to multiple other (usually higher) areas of the brain. They consist of cholinergic and monoaminergic neurons in nuclei of the brain stem and subcortical areas.

The excitatory neurotransmitter for *relay systems* is L-glutamate and, in some areas, aspartate. Local inhibitory circuits along these relay systems use γ-aminobutyric acid (GABA) or glycine. Numerous studies in experimental animals have implicated glutamatergic neurotransmission in toxicity associated with trauma, ischemia-reperfusion, and epilepsy. Experimental inhibitors of the glutamatergic system are being investigated as therapy for brain injury.[15] GABA is synthesized from glutamate by the action of glutamic acid decarboxylase (GAD). Impaired GABAergic transmission is involved in some forms of epilepsy, and autoantibodies against GAD are thought to be the cause of the spastic gait seen in horses with stiff-horse syndrome.[16]

Diffuse projection systems include cholinergic, dopaminergic, noradrenergic, serotonergic, and histaminergic neurons. These systems are targets for numerous drugs used for psychiatric and movement disorders in humans. Dysfunction of cholinergic neurotransmission is responsible for the signs of fluphenazine (an antipsychotic sedative drug) and metaclopramide (a prokinetic agent) reactions in horses.[17]

CLINICAL NEUROANATOMY

The following section is designed to group neurologic functions according to major areas of the CNS. This section complements the information provided in Chapter 50.

Whole Brain

Level of Consciousness

One of the critical functions of the reticular formation of the brain stem is activation of the cerebral cortex for the awake state. This component of the formation, known as the ascending reticular activating system (ARAS), is an ill-defined meshwork of cells that richly invests the rostral half of the brain stem but also extends into the medulla oblongata. The ARAS receives afferent input from all parts of the CNS and projects excitatory stimuli cortically.

Focal to extensive lesions anywhere in the brain stem may reduce the level of consciousness, whereas cortical injury must be diffuse to cause noticeable obtundation. Injury to the rostral sections of the brain stem is more likely to cause severe obtundation than is injury to the medulla. The ARAS also is involved in the initiation and maintenance of sleep. Abnormalities of production or action of hypothalamic arousal peptides (hypocretins/orexins)[18] or imbalances of brain stem neurotransmitters may result in narcolepsy/cataplexy sleep disorders.

Upper Motor Neuron System: Voluntary Movement

The upper motor neuron (UMN) system is responsible for the initiation of voluntary movement and regulation of posture through support against gravity.[2] The latter is effected in part by modulation of the anti-gravity myotatic reflexes of the limbs. The component of the UMN system originating in the hypothalamus is responsible for the control of muscular activity associated with visceral functions (respiratory, cardiovascular, urinary).

Direct cortical influence over motor activity in the horse via the *pyramidal* system is largely limited to fine control of the muzzle and lips.[9] The much more important *extrapyramidal* system is a multisynaptic pathway from the brain to the lower motor neurons (LMNs) of the spinal cord and brain stem. The frontal and parietal lobes of the cerebrum, basal nuclei, diencephalon, midbrain, and hindbrain all contribute to the UMN system. Upper motor neurons, particularly those originating in the midbrain, generally are inhibitory to myotatic reflexes. Damage to the neurons or axons of these tracts in the caudal midbrain, hindbrain, or spinal cord, increases extensor tonus and may result in limb spasticity ("stiffness"). With UMN injury there also is limb weakness, hyperactive extensor reflexes, and crossed extensor reflexes, all in the ipsilateral limbs.

Movement disorders are characteristic of UMN disease in the rostral brainstem. Abnormal involuntary movements include dystonia (writhing movements of the muscles of the head and spine), ballism (violent flailing of a limb), chorea (repetitive jerky movements of different muscle groups), and myoclonus (repetitive movements of a single muscle group).[14] Nigropallidal encephalomalacia is a disease of this type in horses that is caused by the chronic consumption of the neurotoxin repin in yellowstar thistle or Russian knapweed plants.[19] There is dystonia and rigidity of the muscles of the head resulting in lip retraction, tongue protrusion, and inability to prehend food.

More generalized extrapyramidal syndromes in horses reportedly are associated with reactions to fluphenazine (an antipsychotic sedative drug) and metaclopramide (a prokinetic agent).

Forebrain

Behavior (Limbic System, Temporal Lobes)

Normal behavior requires integration of signals from the entire CNS but principally involves the forebrain. Most important in controlling intrinsic behavior is the *limbic system:* a connected series of structures in the cerebrum and diencephalon. A minor component is also found in the midbrain. Included are the amygdala, hippocampus, fornix, cingulate gyrus, and septal area. A closely associated region, which is important in primate behavior, is the temporal lobe of the cerebrum.[2] It is thought that behavior based on conditioning and experience (i.e., learning) is controlled by the temporal lobes. Structural, metabolic, or psychological disturbances affecting these areas may result in behavioral abnormalities (i.e., dementia). Dementia can be defined as changes in normal habits, personality, attitude, reaction to the environment, or loss of learned skills. Some of the signs that may be seen include disorientation in a familiar environment; failure to recognize familiar humans, animals, or objects; loss of the ability to be led; frequent yawning; head-pressing; irritability; unprovoked kicking or biting; compulsive walking or circling; and dramatic changes in eating or drinking habits.

Almost any disturbance of the forebrain potentially can cause dementia. Encephalitis, head trauma, space-occupying lesion, malformation, infarct, and metabolic disorders all are likely to cause changes in behavior. Important examples in horses include rabies, eastern equine encephalomyelitis (EEE), leukoencephalomalacia, neonatal encephalopathy (neonatal maladjustment syndrome), cerebral abscess, cholesterol granuloma, frontal/parietal trauma, post-seizure encephalopathy, hepatic encephalopathy, hyperammonemia, and hydrocephalus. It is likely that structural or metabolic forebrain disease is the cause of dementia if other neurologic abnormalities are found by neurologic examination or imaging studies. In the absence of such supportive findings, abnormal behavior (such as self-mutilation[20]) may have a psychological basis.

Seizures

Seizures are sudden, transient attacks of abnormal motor and/or behavioral activity attributable to paroxysmal depolarization of part to all of the brain.[9] Depolarization either occurs simultaneously throughout the brain or originates from a hyperirritable focus somewhere in the forebrain. Seizures originating from a focus likely will initially have asymmetric clinical signs and there may be additional signs of forebrain disease between seizures that are revealed by neurologic examination.

Seizures frequently originate in the frontal (motor) cortex and involve muscle fasciculations and tremors around the head or abnormal movements of the jaws and tongue ("chewing gum fits"). Convulsions characteristic of neonatal encephalopathy often are of this type. In their severest (generalized) form, seizures manifest as sudden recumbency, with a brief phase of extensor tonus, followed by clonic ("galloping") movements of the legs, loss of consciousness, and a variety of signs of autonomic discharge (e.g., sweating, urination, defecation, pupillary dilation). Mild motor seizures are often accompanied by behavioral signs such as obtundation, compulsive walking, vocalization, hyperresponsiveness to stimuli, or other signs of dementia. Seizure foci in the forebrain may occur at sites of previous or current trauma or inflammation. Examples include skull trauma, equine protozoal myeloencephalitis (EPM), EEE, intracranial neoplasia, and aberrant parasite migration. Causes external to the brain include hyperammonemia, hyponatremia, hyperthermia, and hepatic or renal failure. Juvenile epilepsy of Arabian foals occurs because of transiently increased susceptibility to seizures and manifests as repeated generalized seizures without other evidence of brain disease in foals 1 to 9 months old.[9]

Perception of Pain (Parietal Cortex, Cranial Nerve V)

Pain sensation is transmitted from the body to the brain in multisynaptic spinothalamic tracts.[2] Signals initiated by stimulation of pain receptors on one side of the body pass through spinothalamic tracts on both sides of the spinal cord. Axons in these tracts course rostrally to terminate in the thalamus. From there, cell bodies project axons to the sensory (somesthetic) cortex for conscious perception of pain or other sensory modalities. It is thought that the somesthetic cortex is located principally in the parietal lobe of the cerebrum.

The pathways for pain perception in the head pass through the maxillary, ophthalmic, and mandibular branches of the trigeminal nerve. The central component of this pathway is predominantly contralateral. Unilateral lesions of the sensory parts of the forebrain (thalamus, internal capsule, sensory cortex) thus cause contralateral facial hypalgesia. Because the central components of vision and facial pain perception are close anatomically, it is common to find unilateral facial hypalgesia and blindness (on the same side) in the same horse.

Diseases associated with abnormalities of facial pain perception are the same as those described later for disorders of vision perception.

Smell (Olfactory Bulbs, Cranial Nerve I)

Olfactory nerves pass through the cribriform plate and into the olfactory bulbs of the cerebrum. Information on smell is relayed through the thalamus to centers in the unconscious (limbic system) and conscious cerebrum.

Vision (Thalamus, Occipital Cortex, Cranial Nerve II)

In horses, 80% to 90% of optic nerve fibers from one eye cross at the optic chiasm and 80% of fibers in the optic tracts synapse at the lateral geniculate nucleus in the thalamus.[2] The remainder course to the midbrain to function in the pupillary light reflex (see later). Neurons in the lateral geniculate nucleus project via the internal capsule to the visual cortex in the occipital lobe of the cerebrum. This area is caudal in the cerebrum, immediately rostral to the tentorium cerebelli and caudal to the parietal cortex. The pathway from the eye to the contralateral visual cortex via the optic nerve, lateral geniculate nucleus, and internal capsule must be intact for normal vision. Although the pathways for vision and menace responses are assumed, for clinical purposes, to completely cross over at the optic chiasm, a thin medial slice of the nasal visual field does project to the ipsilateral cortex.

Lesions in the pathway caudal to the optic chiasm result in blindness in the opposite eye and lesions in the optic

nerve rostral to the chiasm affect vision in the ipsilateral eye. Damage to the cortex should not affect the pupillary reflex pathway.

Visual perception is evaluated by obstacle tests (with and without blindfolding of one eye) and by the menace response. The menace response requires the central visual pathway just described plus normal facial nerve function. Integrity of the cerebellar cortex is also needed, although it is not known if the pathway that mediates this response actually passes through the cerebellum. *It is important to note that the menace response does not develop in foals until they are 1 to 2 weeks old.*

Any disease that causes diffuse, severe cerebral dysfunction is likely to cause blindness. Examples include EEE, post-seizure encephalopathy, hyperammonemia, leukoencephalomalacia, and frontal/parietal trauma. Asymmetric frontal/parietal skull trauma, EPM, cerebral abscess, and cholesterol granuloma are diseases that may cause unilateral visual deficits by affecting the forebrain on the opposite side. Arabian foals with cerebellar abiotrophy have normal vision, but menace responses are absent.[21]

Midbrain

Pupillary Light Reflex, Pupil Size (Midbrain, Cranial Nerves II, III)

In the normal horse, pupil size reflects the balance of sympathetic (dilator) and parasympathetic (constrictor) influences on the smooth muscle of the iris. Preganglionic neurons for sympathetic supply to the head arise in the gray matter of the first four thoracic segments of the spinal cord and subsequently course rostrally in the cervical sympathetic trunk. After synapse in the cranial cervical ganglion adjacent to the guttural pouch, the postganglionic sympathetic neurons continue to the smooth muscle of the orbit and act to cause pupillary dilation. Emotional and other influences on sympathetic pupillary tone are governed by hypothalamic centers that act through UMN tracts descending from the midbrain.

Interruption of pre- or postganglionic sympathetic nerves to the eye causes Horner's syndrome, with miosis of the pupil, enophthalmos, ptosis, and spontaneous sweating and vasodilatation over the side of the face. Injury to the sympathetic UMN tracts in the brain or spinal cord not only may cause Horner's syndrome but also may result in increased sweating over the entire side of the body. Preganglionic neurons may be injured in the proximal thorax or along the neck in the vagosympathetic trunk. The cranial cervical ganglion and postganglionic neurons may be destroyed by fungal invasion in horses with *guttural pouch mycosis.*[22] Rarely, signs referable to interruption of sympathetic UMN tracts are found in horses with brain stem or spinal cord lesions (e.g., EPM, spinal cord trauma).

Parasympathetic preganglionic neurons arise in the midbrain and exit the skull in the oculomotor nerve (CN III). These neurons synapse behind the eye in the ciliary ganglion. Postganglionic neurons pass along the optic nerve to innervate the ciliary muscle and constrictor of the pupil. This system is responsive to the amount of light received by each eye. The afferent part of the pupillary light reflex passes via the optic nerves and optic tracts, past the thalamus, to terminate in the midbrain. There is extensive decussation of these tracts both in the chiasm and midbrain, so light directed into one eye causes reflex pupillary constriction in both eyes.

Injury to an optic nerve renders the ipsilateral pupil unresponsive to direct light, but it remains responsive to light directed into the contralateral eye. In contrast, injury to the midbrain or oculomotor nerve causes mydriasis in the ipsilateral eye that is unresponsive to light directed into either eye. Focal diseases (such as EPM, aberrant parasite migration, or midbrain hemorrhage) potentially could involve the oculomotor nuclei, although this is rare. More commonly, the oculomotor nerves are compressed by swelling and subtentorial herniation of the cerebral hemispheres. Early in the process of cerebral injury, pupils often are miotic; progression to bilateral mydriasis is a grave prognostic sign.[2]

Midbrain/Hindbrain

Eye Position (Midbrain; Pons; Cranial Nerves III, IV, VI)

From nuclei in the midbrain and pons, the oculomotor, trochlear, and abducens nerves exit the cranial cavity through the orbital fissure and ramify in the periorbital tissues to innervate the rectus and oblique muscles of the eye. The oculomotor nerve also supplies the levator palpebrae and pupillary constrictor muscles and the abducens nerve innervates the retractor bulbi muscle. Lesions in these nerves (or nuclei) cause abnormal eye position (strabismus). The direction of eye deviations resulting from specific nerve lesions is not known for the horse and cannot necessarily be inferred from data for other species.[9] Unlike vestibular strabismus, which can be corrected by rotating the head (usually in the direction of the lesion), strabismus caused by lesions in extraocular nerves persist in any head position. The finding of mydriasis (CN III) or defective eyeball retraction (CN VI) may provide additional information on lesion location in horses with strabismus.

Hindbrain

Mastication (Pons, Cranial Nerve V)

The LMNs of the trigeminal nerve arise in the pons and pass through the petrous temporal bone and the foramen ovale adjacent to sensory trigeminal neurons and are distributed to the muscles of mastication: masseters, pterygoids, temporals, and rostral digastricus.

With unilateral damage to the trigeminal nucleus (or nerve), there is deviation of the lower jaw toward the normal side. By 2 weeks after injury, there is obvious muscular atrophy. Bilateral severe involvement of the trigeminal nuclei (or nerves) causes a dropped jaw, weak jaw tone, slight tongue protrusion, and inability to prehend or chew feed. Damage to the nucleus of CN V, either unilaterally or bilaterally, is found in some horses with EPM.[23]

Facial Expression and Movement (Medulla, Cranial Nerve VII)

Normal facial tone, expression, and movements are dependent upon the integrity of the facial nerves. These nerves arise from nuclei in the rostral medulla (hindbrain) and exit the calvarium with cranial nerve VIII via the internal acoustic meatus. The nerve courses through the facial canal in the petrous temporal bone adjacent to the middle ear and emerges through the stylomastoid foramen. The facial nerve is distributed to the muscles of facial expression including those of the ear, eyelid, nose, and lips, and the caudal belly of the digastric muscle.

The effects of interruption of this motor pathway depend upon the site of damage. With involvement of the nucleus or proximal nerve, there is drooping of the ear and lip, ptosis, and collapse of the nostril, and the muzzle is pulled toward the normal side. Saliva often drools from the affected side of the mouth, and the horse has difficulty prehending food, especially grain. There may be exposure keratitis secondary to eyelid paralysis.

The facial nerve also contributes parasympathetic neurons to lacrimal glands (and some salivary glands). Damage to the facial nerve proximal to the middle ear likely will affect tear production by this mechanism. Tear production can be measured and compared between sides using the Schirmer tear test. The sensory component of the facial nerve contains fibers from the tongue (taste) and middle ear.

Facial nerve motor function is evaluated by testing "flick" reflexes of the lip, eyelid, and ear. These reflexes involve afferent sensory input via trigeminal neurons that terminate in the medulla on the LMN of the facial nerves. Note that these reflexes do not involve the forebrain and thus can occur without conscious perception. Because they are close together in the hindbrain and skull, the facial and vestibulocochlear nerves are often damaged together. Examples of diseases that often affect both nerves are West Nile virus encephalomyelitis, EEE, EPM, migrating parasite (central), polyneuritis equi, lightning strike, petrous temporal bone fracture, temporohyoid osteoarthropathy, or extramedullary neoplasm (peripheral). To distinguish clinically between central and peripheral causes, it is important to look for other signs of brain disease. For example, a horse with a lesion in the medulla affecting the nuclei of cranial nerves VII and VIII, may also be obtunded with weakness and ataxia of ipsilateral limbs.

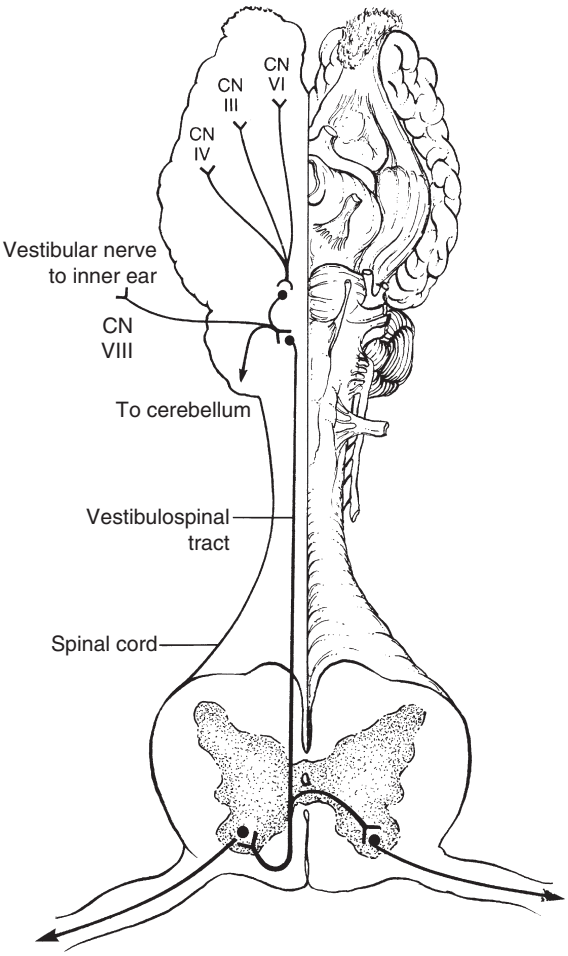

Figure 49-5. The vestibular system showing afferent input from the inner via cranial nerve VIII *(CN VIII)* to the vestibular nuclei in the hindbrain. There is complex integrated output to the cranial nerves controlling extraocular muscles *(CN III, CN IV,* and *CN VI),* the cerebellum, and the trunk and limbs *(vestibulospinal tracts).*

Balance and Equilibrium, Hearing (Medulla, Cranial Nerve VIII)

The vestibular system is responsible for orientation of the horse relative to gravity (Figure 49-5). The receptor is in the bony labyrinth of the inner ear. The membranous labyrinth includes three semicircular ducts containing endolymph that connect to vestibular nerve endings at the cristae ampullares. Vestibular neurons pass centrally through the internal acoustic meatus to penetrate the rostral medulla and terminate in four vestibular nuclei. The first pair or two of cervical nerves also contributes to vestibular function via sensory fibers, which pass to the vestibular nuclei. These nuclei have numerous projections to the cerebellum and the spinal cord and to nuclei controlling extraocular muscles.

The vestibular system controls the conjugate movements of the eyes during movement of the head through extensive connections with the nuclei of cranial nerves III, IV, and VI. Vestibular-cerebellar pathways pass through the caudal cerebellar peduncle. These pathways function to smoothly coordinate the movements of the eyeballs, trunk, and limbs with those of the head. Vestibulospinal tracts descend ipsilaterally to synapse on LMN and facilitate extensor muscles of the limbs while inhibiting flexor muscles. Some vestibulospinal tracts cross and reduce extensor tonus in contralateral limbs.

Unilateral disease involving the peripheral part of the vestibular system causes asymmetric ataxia *with preservation of strength*. The poll rotates towards the side of the lesion and the head and neck may be turned toward the lesion. The body leans, falls, or rolls toward the side of the lesion and the horse may stagger in tight circles. This type of circling should be distinguished from the compulsive walking in circles (without head tilt) that is characteristic of horses with asymmetric frontal cerebral lobe lesions. Because there is some visual compensation for vestibular ataxia, blindfolding exacerbates the signs and is a useful clinical test in mild cases of suspected vestibular disease.

In horses with central vestibular disease, there usually are additional signs of hindbrain disease (e.g., obtundation and *limb weakness*) and head tilt may be either toward or away from the side of the lesion. The latter presentation is known as paradoxical central vestibular disease and usually follows involvement of vestibular connections within the cerebellum. Unilateral vestibular disease often causes spontaneous or positional nystagmus and physiological (vestibular) nystagmus may be absent or abnormal. In *peripheral disease*, the nystagmus is always horizontal, rotatory, or arc-shaped, with the fast phase away from the lesion. With *central involvement* of the vestibular system, nystagmus also may be vertical. Typically, the eye on the affected side rotates ventrally in the orbit while the eye on the normal

side rotates dorsally (especially when the head is extended). This abnormal eye position is termed *vestibular strabismus.* Bilateral vestibular disease is characterized by severe symmetric ataxia and wide, sweeping movements of the head from side to side. In addition to the diseases described earlier that also affect the facial nerve, an idiopathic vestibular syndrome is reported to occur in the horse, possibly as a result of viral neuritis or labyrinthitis.[9]

Neurons of the cochlear division of cranial nerve VIII pass from receptors in the middle/inner ear to auditory centers in the midbrain and thalamus. A variety of local reflexes are initiated by stimulation of the cochlear nerve. In addition, there is projection of conscious pathways for hearing from the thalamus to the cortex (temporal lobe?). Deafness is rarely detected in the horse.

Taste (Cranial Nerves VII, IX, X)

Taste buds are found on the surface of the tongue and also in the soft palate, pharynx, lips, and cheeks. Sensory gustatory innervation is provided by the facial nerve (rostral two thirds of the tongue), glossopharyngeal nerve (caudal one third of the tongue), and vagus (pharynx and palate). General sensory innervation provided by the trigeminal nerve probably also contributes gustatory information. Perception of taste involves the forebrain including the limbic system. Deficiencies in the sense of taste are very difficult to detect by clinical testing.

Movement of Pharynx and Larynx (Cranial Nerves IX, X, XI)

Motor innervation of the larynx and pharynx originates in neurons in the nucleus ambiguus, a fusiform structure extending the length of the medulla. This nucleus provides axons for the glossopharyngeal, vagus, and spinal accessory (internal branch) nerve roots. These roots form nerves that innervate the soft palate, pharynx, larynx, and cranial esophagus via the pharyngeal plexus and cranial and recurrent laryngeal nerves. The nucleus ambiguus is continued in the spinal cord as the nucleus of the external branch of the spinal accessory nerve (innervation of trapezius and parts of brachiocephalicus and sternocephalicus). The facial and hypoglossal nerves innervate several of the muscles that control movements of the hyoid apparatus; therefore, impaired movement of the hyoid apparatus caused by paralysis of these nerves could affect movements of the larynx and pharynx. Clinically, such effects are minor in horses at rest, but they can be revealed by intense exercise.

Unilateral (hemiplegia) or bilateral pharyngeal paralysis interferes with swallowing and manifests as signs of dysphagia: coughing and gagging during eating with return of saliva, feed, and water through the nostrils and mouth. With complete unilateral laryngeal hemiplegia (i.e., paralysis of adductors and abductors), there is exercise-induced respiratory stridor and aspiration of feed into the trachea. In horses with bilateral laryngeal paralysis, inspiratory stridor occurs at rest and there is aspiration pneumonia. Some or all of these pathways can be affected peripherally in horses with botulism, guttural pouch mycosis, extramedullary neoplasia, or basilar skull fracture. Examples of diseases affecting the hindbrain that may cause laryngeal or pharyngeal dysfunction are EEE, EPM, leukoencephalomalacia, bacterial meningitis, and West Nile viral encephalomyelitis (WNVE).

Tongue Movement (Cranial Nerve XII)

The muscles affecting movement of the tongue are supplied by the hypoglossal nerve. Neurons of the hypoglossal nerve originate in the hypoglossal nucleus in the caudal aspect of the medulla and emerge from the medulla as a horizontal row of rootlets, which combine to form the nerve as it enters the hypoglossal foramen. After emerging from this foramen, the hypoglossal nerve runs forward and ventrad in association with the guttural pouch and stylohyoid bone to innervate the geniohyoideus and muscles of the tongue.

Interruption of the hypoglossal pathways causes hemiparesis of the tongue, evident as deviation of the apex of the tongue toward the affected side. Within 1 to 2 weeks, atrophy of the tongue will become noticeable. The peripheral nerve may be damaged by extramedullary neoplasia, upper cervical or caudal skull trauma, or guttural pouch mycosis. Tongue hemiplegia and atrophy has been seen in horses with EPM although any cause of caudal hindbrain disease can damage the hypoglossal nuclei.

Cerebellum

The cerebellum sits in the caudal fossa of the skull and is separated from the cerebral hemispheres by the tentorium cerebelli. It is divided into the flocculonodular lobe, also known as the vestibular cerebellum, and the much larger body of the cerebellum. The cerebellar body consists of a median region, the vermis, and two lateral cerebellar hemispheres. Connections with the rest of the CNS are via three peduncles: efferent connections pass through the rostral peduncle and afferent pathways enter the cerebellum via the middle and caudal peduncles.

The cerebellum regulates and smoothes motor activity initiated by the UMN system. It also acts to maintain equilibrium and appropriate body posture during rest and motion. Proprioceptive information is gathered via afferent connections from the spinal cord (spinocerebellar tracts) and vestibular system and is notified of UMN activity via extensive connections with brain stem UMN nuclei (including the olivary nucleus). Efferent cerebellar neurons project to vestibular nuclei and other parts of the brain stem including the thalamus. There is virtually no projection of cerebellar efferents into the spinal cord.

Cerebellar disease is usually diffuse and manifests as symmetric ataxia without weakness. There is defective regulation of the rate, range, and force of movement. Limbs may appear spastic with excessive (hypermetric) or inadequate (hypometric) flexion during protraction. Signs are most obvious when there is a change in the force or direction of voluntary movement. At rest, the body may sway, laterally or backward and forward, and there may be coarse head bobbing or tremor that is exacerbated by voluntary movement, such as reaching the head out for food. Extensor muscle tone is increased and limb reflexes may be hyperactive. With diffuse cerebellar cortical disease, the menace response is absent, although vision is normal (see earlier).

Signs of diffuse cerebellar disease are seen in Arabian horses[24] or Götland pony foals with cerebellar abiotrophy. This inherited disease first manifests at any time from birth to 3 months of age and may be stable or progressive for weeks to months. Rarely, horses with poll trauma have a head bob indicating cerebellar injury. The rhabditid nematode *Halicephalobus gingivalis* has a propensity to invade the caudal fossa and cause cerebellar destruction.[25] Tremorgenic mycotoxins produced by

parasitized perennial ryegrass or *Paspalum* species cause signs suggestive of cerebellar dysfunction, although other parts of the CNS are also affected.

Spinal Cord

Upper Motor Neuron: Muscle Tone and Voluntary Movement

Axons of the UMN extrapyramidal and vestibular systems travel from cell bodies throughout the brain and pass predominantly in reticulospinal and vestibulospinal tracts to LMN in the ventral and intermediate columns of the gray matter of the spinal cord. As described in the section on UMN of the brain, this system provides tonic support of the body against gravity and recruits spinal reflexes for the initiation of voluntary movement. UMNs act by influencing α and γ motor neurons in the spinal cord. These LMNs, in combination with afferent nerves and stretch receptors in the neuromuscular spindles and tendons, control muscle tone and movement by myotatic and antimyotatic reflexes. Most descending UMN tracts are inhibitory to extensor motor neurons. Removal of these influences by transection through the midbrain causes extension of head, neck, trunk, and limbs, signs that are characteristic of decerebrate rigidity.[2] Basic locomotor activity involves recruitment and control of these reflexes by distinct postural and voluntary UMN systems.

Interruption of UMN tracts in the spinal cord causes signs of ipsilateral weakness of the trunk and limbs. Signs of paresis range in severity from slight toe-dragging and delayed protraction to recumbency and inability to rise. Because myotatic reflexes are released from inhibitory UMN influences, there may also be spasticity (stiffness) of limb movement. This is most obvious in the thoracic limbs, which may appear to "float" during walking. The stiff appearance reflects decreased flexion of the limb during the protraction phase of the stride. Interference with UMNs also may manifest as delayed initiation of voluntary movement or alterations in gait cadence. For example, some horses with spinal cord disease have a lateral "pacing" gait at walking speed. If LMNs are not affected, spinal reflexes (e.g., patellar) are either normal or exaggerated on the side of the spinal cord lesion and crossed extensor reflexes may be seen. *It should be noted that crossed extensor reflexes are normal in newborn foals.*[9]

Spastic paresis is especially common with focal compression of the cervical spinal cord in horses with cervical stenotic myelopathy (CSM) or spinal cord trauma. Other significant causes of UMN disease (in the spinal cord) include EPM, equine degenerative myeloencephalopathy (EDM), spinal cord abscess or tumor, and aberrant parasite migration. In keeping with the basic principles of neuroanatomic localization, UMN lesions from C1 to T2 (inclusive) may cause neurologic signs in all four limbs, lesions from T3 to S2 can only affect the pelvic limbs, and lesions caudal to S2 do not directly affect gait. With external compression of the cervical spinal cord (as in CSM), signs typically are worse in the pelvic limbs than in the thoracic limbs. The principles of neuroanatomic localization of spinal cord lesions are discussed more fully in Chapter 50.

Lower Motor Neuron and Spinal Cord Reflexes

The function of the CNS is exerted entirely through the actions of LMN on striated (skeletal) and smooth muscle. LMNs to skeletal muscles are found in the ventral columns of the gray matter, and those of the autonomic nervous system are located in the intermediate columns. In the horse, there are 8 cervical, 18 thoracic, 6 lumbar, 5 sacral, and a variable number of coccygeal (caudal) spinal cord segments. LMNs originating in the gray matter of a segment form a ventral root, which then exits the vertebral canal through the intervertebral foramen of the vertebra of the same name. The ventral root joins with the dorsal sensory root to form the segmental spinal nerve. In the cervical vertebrae, this foramen is at the cranial end of each vertebra. For the remaining roots, the foramina are at the caudal end. The more caudal spinal cord segments have long nerve roots because the spinal cord segments are shifted cranially with respect to the vertebrae. The first three sacral segments are in the sixth lumbar vertebra and the spinal cord ends within S2.[2]

The LMNs form the efferent part of spinal reflexes, which are central to the neurologic examination and the process of neuroanatomic localization. The neurons of the afferent (sensory) component of reflexes course from receptors in the skin, muscle, or tendon, through the spinal nerve and dorsal root, into the dorsal horn of the gray matter where they terminate on interneurons. The interneurons then complete the pathway by passing to the LMN. An exception to this is the patellar reflex, wherein the sensory neuron terminates directly on the LMNs in the ventral horn. Long (i.e., multisegment) spinal cord reflexes, including slap tests, cervicofacial and cutaneous trunci reflexes, and caudal reflexes, including anal and tail-clamp, are routinely evaluated during neurologic examination. Limb reflexes, including withdrawal, patella, and triceps, are always evaluated in neonatal foals and in older horses that are recumbent. The specifics of individual spinal cord reflexes are covered in Chapter 50.

Abnormalities of LMN (in gray matter, ventral root, peripheral nerve, or neuromuscular junction) manifest as flaccid muscle weakness (paresis, paralysis) with hypotonia and hyporeflexia. Within 1 to 2 weeks, muscle atrophy is noticeable, and this neurogenic muscle atrophy progresses rapidly.[9] *Note that neurogenic atrophy is a consequence only of LMN disease; UMN involvement results in weakness without atrophy.* Ventral nerve roots contribute to multiple peripheral nerves, and peripheral nerves are derived from multiple roots, so injury to gray matter of an individual segment or to a ventral nerve root produces less-severe neurologic signs than does loss of function in a peripheral nerve.[2] Examples of diseases that affect the LMN in horses are botulism (neuromuscular junction), trauma, ischemia associated with recumbency, neuritis of the cauda equina/polyneuritis equi, EPM, postanesthetic myelomalacia, and equine motor neuron disease (EMND).

Proprioception

Through a system of receptors in muscles, tendons, and joints, the general proprioceptive system is able to monitor the position of the body or limbs in space. Proprioceptive information is passed centrally in sensory nerves that terminate in the dorsal gray column on cell bodies of neurons in the spinocerebellar tracts. These tracts pass cranially and provide information for the cerebellum to use in its role of regulating posture, muscle tone, locomotion, and equilibrium. Other proprioceptive pathways pass from the spinal cord to the somesthetic sensory cortex via relay nuclei in the midbrain and thalamus.

Interruption of spinal cord proprioceptive pathways interferes with recognition by the brain of the positions in space of

the body and limbs. This is manifest as ataxia (incoordination). Signs of proprioceptive deficit in the horse include base-wide or base-narrow limb placement, swaying of the trunk and torso during walking (but not usually at rest), and overstriding, especially in the pelvic limbs. During circling, limb ataxia is evident as circumduction in the pelvic limbs and interference in the thoracic limbs.

Urination

Parasympathetic LMNs to the bladder's smooth muscle (detrusor) originate in the intermediate column of the gray matter of sacral segments 2 to 4. These neurons exit in ventral roots and contribute to the pelvic plexus, a network that supplies autonomic innervation to the smooth muscle of the bladder and rectum. Sympathetic LMNs to the bladder begin in the gray matter of L1-4, exit the vertebral canal, and course to the pelvic plexus. Postganglionic sympathetic neurons terminate on smooth muscle in the body and neck of the bladder and proximal urethra. These autonomic LMNs function in local reflexes. Afferent neurons pass from stretch receptors in the bladder wall and enter the spinal cord in dorsal sacral nerve roots to exert inhibitory influences on parasympathetic and sympathetic LMN. Striated muscle of the urethra is innervated by somatic LMN in the pudendal nerve. Urination occurs when there is stimulation of parasympathetic nerves to the detrusor muscle, inhibition of sympathetic nerves to the detrusor muscle, and inhibition of sympathetic and somatic nerves to the urethra. The net effect of this activity is contraction of the smooth muscle of the body of the bladder and relaxation of the proximal urethra. Centers in the midbrain and hindbrain receive sensory information from the bladder and modulate reflex activity via UMNs passing caudad in the spinal cord. Forebrain influence on these centers is responsible for initiation of voluntary voiding.

In horses with severe spinal cord disease cranial to S2, there may be loss of voluntary control of urination. Within 2 weeks, "spinal-reflex bladder" function develops, which results in intermittent voiding with retention of small amounts of urine. Horses with severe CSM, EPM, vertebral trauma, or epidural abscess may present in this way. When there is a spinal cord lesion between L4 and S2, reflex pathways for inhibition of sympathetic activity may be interrupted, resulting in increased urethral tone and functional obstruction of urinary outflow. This is the case in some horses with equine herpesvirus type 1 (EHV-1) myeloencephalopathy, although others have signs of LMN bladder (see later).[26]

With injury to the sacral spinal cord segments or nerves, the bladder and urethra are atonic and distended and there is overflow incontinence. Although a small amount of intrinsic reflex bladder contraction ("automatic bladder") may occur, it is ineffectual, and large volumes of urine and sediment remain. Trauma, epidural abscess or tumor, EHV-1 myeloencephalopathy, sorghum-Sudan grass toxicity, and neuritis of the cauda equina are diseases that can cause these signs. Idiopathic atonic bladder (also known as sabulous cystitis) is found in middle-aged geldings and some stallions and resembles the presentation of horses with cauda equina syndrome; however, the idiopathic syndrome does not appear to be caused by denervation of bladder muscle and there are no other signs of cauda equina syndrome. In cases of LMN urinary incontinence, additional nonurinary signs of sacral nerve injury are expected. These are the signs of cauda equina syndrome and include fecal incontinence, paralysis of the anus, tail, and penis, and perineal analgesia.

Defecation

The smooth muscle of the rectum and anus is innervated by postganglionic parasympathetic neurons in a way that parallels that described for the bladder. Innervation of striated muscle of the anus is provided by the pudendal nerve. Spinal cord lesions cranial to S2 are unlikely to affect defecation; however, involvement of the sacral segments results in rectal obstipation and may cause colic. Diseases causing rectal paralysis are the same as those described under the section on urination.

Sensation

Sensory information from pain, thermal, and touch receptors is conveyed to the spinal cord by peripheral nerves, spinal nerves, and dorsal roots. Branches pass several segments both craniad and caudad from the site of spinal cord penetration and terminate on spinothalamic neurons or interneurons involved in spinal reflexes. Spinothalamic pathways servicing a single dorsal nerve root course craniad on both sides of the spinal cord. Pain perception requires interpretation of afferent information by the forebrain and was described earlier. The flexor (withdrawal) reflexes require only sensory nerves, contiguous spinal cord segments, and LMNs. In the thoracic limbs, spinal cord segments C6 to T2 are required and L6 to S2 are involved in the pelvic limb reflex. Evaluation of sensation over the trunk and limbs requires knowledge of the common autonomous zones for sensory nerves. This is described in more detail in Chapter 54.

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CHAPTER
50 Diagnostic Procedures

Robert J. MacKay

NEUROLOGIC EXAMINATION

A competent neurologic examination is not unlike an evaluation for lameness, where the initial evaluation is by systematic, "passive" examination and this is followed by an assessment of the gait. This chapter contains a description of an ordered approach to neurologic examination (Box 50-1 is a summary). There is considerable emphasis on evaluation of symmetry during each element of the examination. To accurately assess symmetry, it is essential that a mirror-image technique be used when comparing sides—that is, if the right hand of the examiner is used to test one side, the left hand should be used for testing the opposite side. The only equipment needed is a transilluminator, a hemostat, and a patella hammer (for neonatal examinations). The findings are used to localize a lesion or lesions. This is the basis for formulation of an initial rule-out list and for planning additional diagnostic tests.

In this discussion, all abnormal findings are interpreted in the context of a neurologic explanation. It is clear that many of these signs could be caused by lesions outside the nervous system. Full general physical and lameness examinations should also be performed to assess possible involvement of other body systems.

BOX 50-1. Items for Neurologic Examination

Mentation
Orientation and coordination of the head
Muscles of mastication
Facial tone
Facial sensation
Menace response
Size of pupils and pupillary light reflex
Position and movement of the eyeballs
Swallowing
Tongue
General examination of the neck, trunk, and limbs
Slap test
Cervicofacial reflex
Cutaneus trunci reflex (panniculus)
Back reflexes
Tests for limb strength
Gait
Testing of recumbent horses
Tail and anus

Mentation

Alertness and behavior are not evaluated by specific testing but are assessed from the history and general observation throughout the examination.

Alertness

Assess level of consciousness or alertness on a continuous scale from normal (i.e., bright and alert) to comatose. Progressive levels of obtundation are termed *lethargy, stupor, semicoma,* and *coma*. With lethargy, there is a somewhat blank facial expression with slight drooping of the ears and eyelids, sluggish responsiveness to stimuli, and reduced voluntary activity. It should be noted that some conditions cause horses to lose facial expression while maintaining normal alertness. Botulism is a disease of this type. A stuporous horse stands in one place with the head held low and responds only to strong stimuli. Stuporous horses that are recumbent are defined as semicomatose, and recumbent horses that do not respond to any stimulus are comatose. Some horses with reduced alertness appear irritable and anxious, walk compulsively, or otherwise interact abnormally with handlers. An obtunded horse showing such behavior may be termed *delirious.*

It is very unlikely that a horse with hypersomnia (excessive sleepiness) or narcolepsy/cataplexy will have a sleep attack while being examined; thus, assessment of possible narcolepsy usually rests on description by the owner or video recordings of an attack. Occasionally, neonatal foals collapse during examination and briefly appear comatose. These are typically benign

episodes of cataplexy and are probably an exaggerated version of the collapse response by which a normal foal can be cast into recumbency.

Behavior

Abnormalities of behavior are termed *dementia.* Behavior is assessed from history and general observation in the course of the examination. Typical abnormal behaviors resulting from central nervous system (CNS) disease include self-mutilation, head-pressing, compulsive walking (often in a circle), yawning, aggression (including unprovoked biting or kicking), hyperresponsiveness to normal stimuli, timidity, loss of affinity of a foal for its dam, and loss of learned behaviors and skills. Inability or refusal of a trained horse to follow when being led is a form of dementia that may be obvious during neurologic examination.

Lesion Location

Obtundation reflects damage anywhere in the brain stem or diffusely in the cerebrum. Injury to the rostral half of the brain stem often causes severe obtundation. Dementia is a sign of forebrain damage.

Associated Clinical Signs

Limb ataxia or weakness and signs of cranial nerve dysfunction are common in horses with obtundation secondary to brain stem disease. Compulsive turning of the head and neck in one direction, blindness, and seizures are other signs of forebrain dysfunction that may be seen in demented horses.

Orientation and Coordination of the Head

Examination

Evaluate the orientation of the head from directly in front. Any head tilt is described from the patient's perspective; thus, if the poll is rotated to the horse's left (i.e., clockwise from the examiner's point of view), the abnormality is described as a left head tilt. Repeatedly straighten and release the horse's neck and head and observe whether or not the head returns to one side. Asymmetric disease of the vestibular system causes the head to tilt and turn, whereas asymmetric cerebral disease may cause the head and neck to turn without tilting. Carefully blindfold the horse and observe the effect on head position (Figure 50-1). Blindfolding removes visual input to head position and exacerbates abnormalities caused by either vestibular or cerebral disease. Observe the head and neck from the side. Persistent horizontal or low position of the head may indicate neurologic or muscular weakness of the neck, whereas extended head position may be found in horses with upper cervical vertebral problems or guttural pouch disease. Offer feed or a treat to the horse and observe the way the horse moves its head in response. Horses with cerebellar disease often make jerky or bobbing movements of the head as they move toward the offered feed.

Lesion Location

Head tilt suggests involvement of the vestibular labyrinth, nerve, or root peripherally, or the vestibular nuclei and connections in the medulla oblongata or cerebellum centrally. It has also been suggested that lesions in the first one or two pairs of cervical

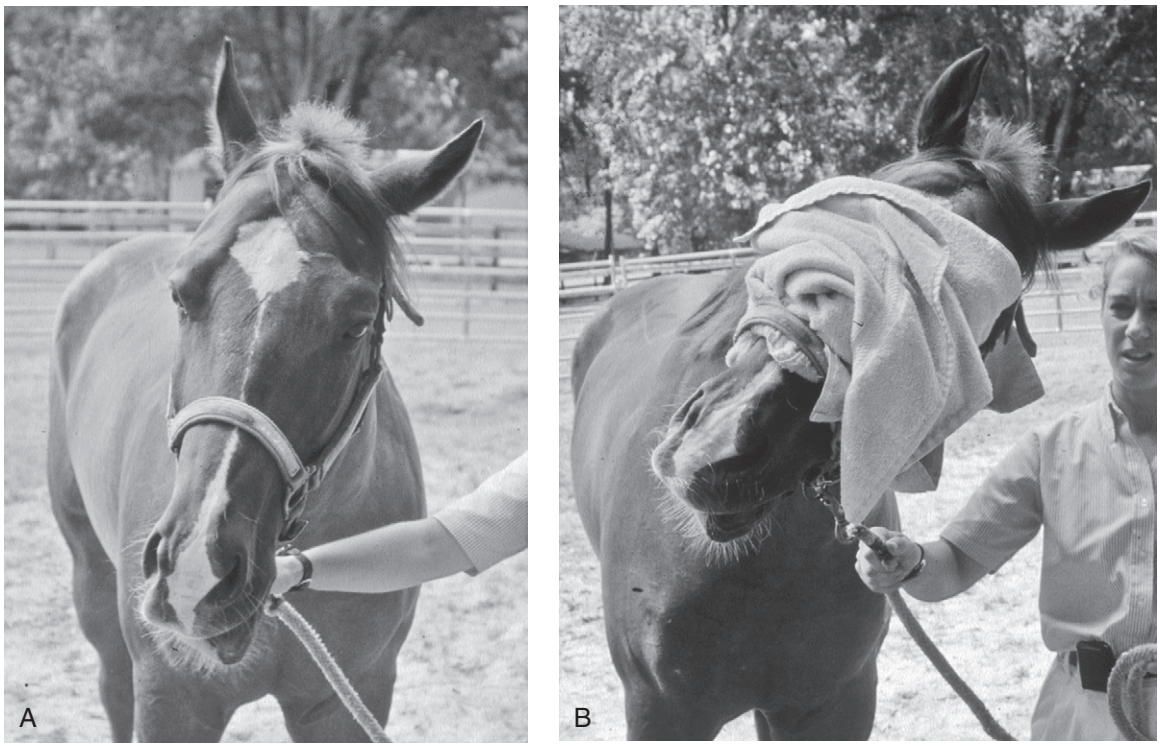

Figure 50-1. Head tilt associated with temporohyoid osteoarthropathy (THO). Six days earlier, this 7-year-old Quarter Horse gelding was presented with signs of acute-onset left-sided vestibular disease and facial paralysis. **A,** With anti-inflammatory therapy, the head tilt improved gradually to the time of presentation. **B,** Blindfolding removed visual input and exacerbated the head tilt.

sensory nerves may cause signs suggestive of vestibular disease.[1] If the head is turned without tilting, involvement of the forebrain is likely. Coarse or fine head bobbing, especially intentional, indicates diffuse cerebellar dysfunction.

Associated Clinical Signs

Because of the close association of cranial nerves (CNs) VII and VIII, facial paralysis is often seen in horses with vestibular disease. With peripheral vestibular disease, there often is spontaneous horizontal or arc-shaped nystagmus with the fast phase directed away from the side of the lesion. In horses with involvement of the central components of the vestibular system, there may be horizontal or vertical nystagmus. Damage to adjacent structures in the hindbrain may additionally cause obtundation, other signs of cranial nerve dysfunction (such as dysphagia and masseter atrophy), and weakness or ataxia of the limbs on the side of the lesion. Horses with cerebellar cortical disease may lack menace responses despite having normal vision.

Muscles of Mastication

Examination

If the mouth hangs open and the tongue protrudes, there is probably bilateral paresis of the muscles that close the jaw (temporalis, masseter, and pterygoid). Grasp the upper and lower jaws at the level of the interdental space and attempt to pull the lower jaw downward. The jaws pull apart easily in horses with bilateral paresis of the masticatory muscles. Tuck the forelock behind one of the ears and compare the temporalis muscles from in front of the horse. Asymmetry may indicate neurogenic atrophy of the smaller side (Figure 50-2). Turn the head from side to side and observe and palpate the masseter muscles. The pterygoid muscles and cranial belly of the digastricus muscle (the only muscle innervated by CN V that is devoted to opening the jaws) are located on the medial side of the mandible, so they are not readily palpable; however, atrophy of the pterygoids on one side is often evident as relative deepness of the supraorbital fossa on the same side. Peel back the upper and lower lips and examine the alignment of the symphyses of the upper and lower jaws. With acute unilateral paresis of the masticatory muscles, even before atrophy is apparent, the lower jaw may be deviated toward the normal side (see Figure 50-2).

Lesion Location

Weakness or atrophy of the muscles of mastication indicates involvement of the motor division of the trigeminal nerve, its roots, or the trigeminal motor nucleus in the pons.

Associated Clinical Signs

Injury to the proximal part of the trigeminal nerve may also affect the sensory component, resulting in reduced facial sensation (hypalgesia) over one side of the face. With trigeminal damage at the level of the pons, there may be additional signs of hindbrain disease, such as obtundation, other signs of cranial nerve involvement (e.g., dysphagia, head tilt, or facial paralysis), and weakness and ataxia of the limbs on the same side.

Facial Tone

Examination

Examine the head carefully for symmetry of facial expression, particularly with respect to the ears, eyes, and muzzle. With complete unilateral facial paralysis, there is drooping of the ear, upper eyelid (ptosis), and lower lip and immobility, narrowing, and lengthening of the affected external naris (Figure 50-3). The muzzle is deviated away from the affected side, and saliva may drool from the mouth. Any or all of these components can be affected separately. Next, evaluate facial nerve function by testing "flick" reflexes on each side of the face. Each of these reflexes requires intact trigeminal sensory branches, central

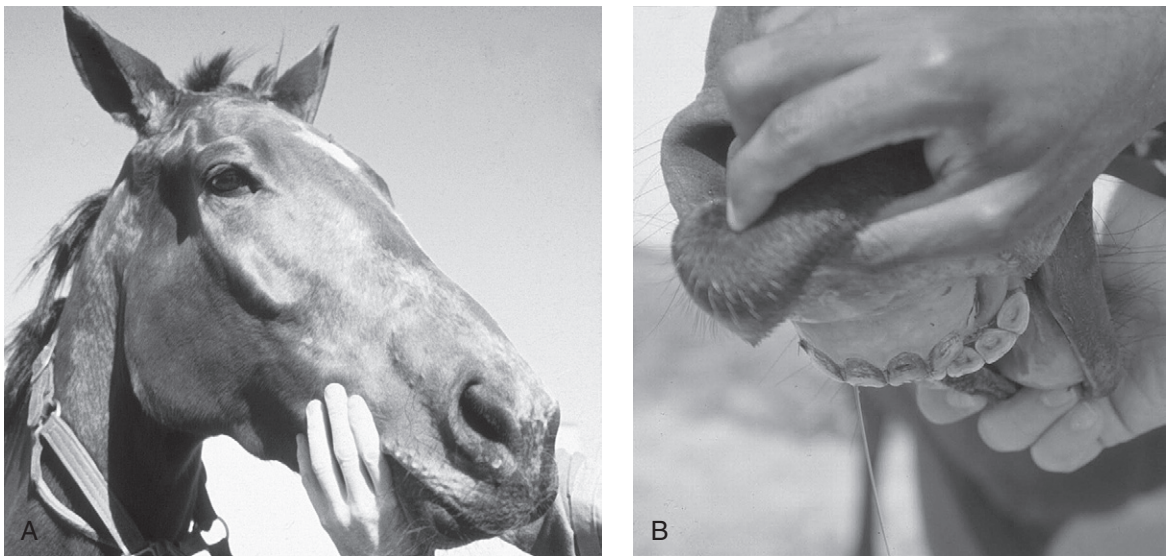

Figure 50-2. Atrophy of the muscles of mastication in a horse with equine protozoal myeloencephalitis. Beginning about a month after foaling, this 11-year-old Thoroughbred mare had an insidious onset of ataxia and limb weakness. **A,** Six weeks later, ataxia and weakness were worse, especially in the left limbs, and there was obvious atrophy of the masseter and temporalis muscles on the right side. **B,** The lower jaw was deviated away from the affected side.

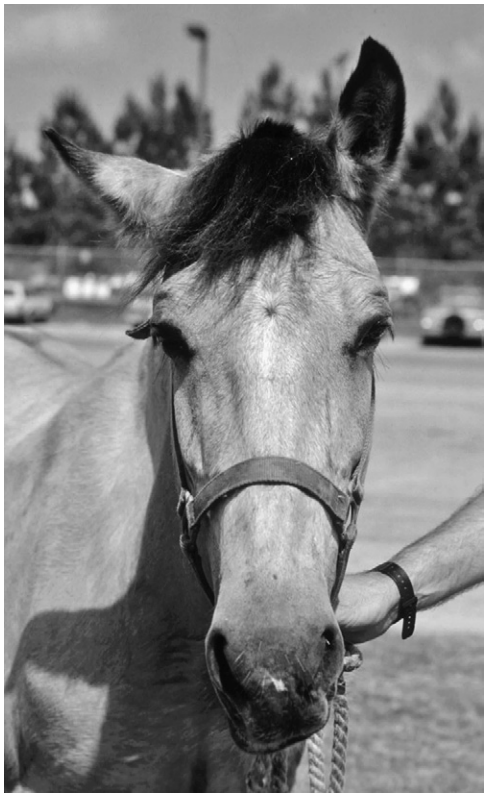

Figure 50-3. Facial paralysis in a horse with polyneuritis equi (neuritis of the cauda equina). Over the previous 2 weeks, this 13-year-old Quarter Horse mare had several exacerbations and remissions of right-sided facial paralysis. Beginning 3 days ago, there were also signs of cauda equina syndrome, including a weak tail and paralysis of the anus.

connections in the hindbrain, and functioning facial nerves. To test these reflexes, touch in turn the commissure of the lips, the medial and lateral canthi of the eye and the supraorbital fossa, and the ear. Appropriate responses are retraction of the commissure of the lip, blinking of the eye, and flick of the ear, respectively. It is helpful to have the handler cover the ipsilateral eye during testing of the lip and ear.

Test facial innervation of lacrimal glands by performing Schirmer tear tests. If tear production is reduced to less than 50% of normal on the side of the face that is paralyzed, there is likely to be a lesion affecting secretomotor fibers proximal (central) to the middle ear. Further test the symmetry of facial tone by using the thumbs or fingers to assess resistance simultaneously on both sides to retraction of lip commissures, elevation of upper eyelids, and forward extension of the ears. Examine the throat latch area. If there has been facial paralysis for more than 2 weeks, there usually is an obvious subcutaneous depression running ventrad from the base of the ear along the back of the mandible. This reflects atrophy of the parotidoauricularis muscle. Finally, test the cervicofacial reflex (see later) as part of the examination of long spinal reflexes. This reflex is not used as a primary test of facial nerve function but, clearly, its interpretation is affected by facial nerve paralysis.

Lesion Location

Abnormalities of facial tone reflect damage to the facial nucleus in the medulla oblongata, facial nerve root, or facial nerve or its motor branches, including dorsal and ventral buccal nerves, posterior and internal auricular and auriculopalpebral nerves, and the digastric nerve. If the Schirmer tear test gives a normal result on the affected side, the lesion is most likely at or peripheral to the middle ear; if tear production is reduced more than 50%, the lesion is central to the middle ear.

Associated Clinical Signs

Commonly there is also head tilt and spontaneous nystagmus because of involvement of the vestibular system. In horses with facial paralysis caused by brain disease, there may be other signs of hindbrain disorder (such as obtundation), signs of abnormal cranial nerve function (such as dysphagia or masseter atrophy), or limb ataxia and weakness on the same side as the lesion. Nonneurologic signs include exposure keratis or keratoconjunctivitis sicca and inspiratory stridor during exercise.

Facial Sensation

Examination

The sensory branches of the trigeminal nerve have already been tested as flick reflexes of the lip, eye, and ear. While standing in front of the horse, place the palm of the right hand on the muzzle, work the thumb into the right nostril, and touch the nasal septum on that side. In mirror-image fashion, use the left thumb to stimulate the left side of the septum. The normal response to this noxious stimulus is vigorous movement of the head away from the side of the stimulus. Compare the intensities of the response on each side. If there is obvious asymmetry of response, test cutaneous sensation at multiple locations over the face by pinching the skin with hemostats.

Lesion Location

The pathway for perception of this noxious stimulus is input through the sensory division of the trigeminal nerve to the pons and, from there, to the somesthetic area of the contralateral cortex. If flick reflexes are normal, a reduced or absent response to a noxious stimulus suggests involvement of the contralateral forebrain. In contrast, hypalgesia and hyporeflexia of flick reflexes of part to all of one side of the head indicates involvement of the trigeminal nerve or branches.

Associated Clinical Signs

Blindness, obtundation, and abnormal behaviors may be seen when hemifacial hypalgesia is a result of forebrain disease. Peripheral lesions of the trigeminal nerve may involve motor fibers and result in weakness and atrophy of masticatory muscles.

Menace Response

Examination

Test menace responses while standing in front of the horse. Use the palm of the hand to make a threatening gesture toward the eye. Test from both temporal and nasal directions on each side. Stimulate the horse just before each menace gesture by tapping the skin below the eye. For safety reasons, always hold the noseband of the halter with one hand while using the other for testing. A normal menace response is blinking of the eye,

sometimes accompanied by evasive movement of the head and neck. Compare carefully the intensity of the menace responses elicited from each side. In this regard, menace should be considered a quantitative response. Unlike simple reflexes, which are assessed as present or absent, a menace response can be considered abnormal if it is less vigorous than that elicited from the opposite (normal) side.

Lesion Location

The menace response can be interrupted anywhere in its pathway from the eye via the optic nerve to the contralateral optic tract, diencephalon, internal capsule, and visual cortex. From the visual cortex, the menace response pathway continues to the facial nucleus and nerve on the side being tested and receives essential input from the cerebellum. If there is no other sign of cerebellar involvement, and if facial nerve function and pupillary light reflexes are intact, the lesion is contralateral, central to the optic chiasm, and most likely in the forebrain. In normal neonates and in older horses with diffuse cerebellar cortical disease, there is no menace response, but the horse can see. In these settings, vigorous threatening gestures toward the eye may cause evasive movements of the head without blinking of the eye.

Associated Clinical Signs

There may be other signs of asymmetric forebrain disease, such as compulsive walking in circles, dementia, head and neck turn, obtundation, seizures, and facial hypalgesia on the side opposite the lesion (i.e., the same side as the defective menace). If the site of the lesion causing a menace deficit is the optic nerve or fundus of the eye, the pupillary light reflex on the same side should be abnormal. Head-bobbing, dysmetria, and ataxia are expected in horses with defective menace responses resulting from cerebellar dysfunction. If eyelid paralysis is preventing the menace response, the palpebral (eyelid flick) reflex should also be abnormal and there may be additional signs of facial paralysis.

Size of Pupils and Pupillary Light Reflex

Examination

If possible, examine the eyes in subdued or dim light so that the pupils are large enough to easily allow appreciation of reflex constriction. Stand in front of the horse while holding the noseband of the halter and swing the light back and forth from one side to the other so as to obliquely and briefly illuminate each eye without causing constriction of the pupils. Unequal pupillary size is termed *anisocoria*, a constricted pupil is *miotic*, and a dilated pupil is *mydriatic*. From this examination, determine whether or not the pupils are of equal size and if the diameter of each pupil is appropriate for the conditions. In this way, refine the diagnosis of anisocoria to miosis or mydriasis affecting a single eye.

Next, move closer to the horse and aim the light at the skin below one eye. Redirect the beam directly into the eye. This strong light should elicit both a dazzle reflex in the ipsilateral eye and pupillary light reflexes (PLRs) in both eyes. The dazzle reflex refers to eyelid closure in response to bright light. The full dazzle response also includes retraction of the eyeball and movement of the head away from the light. A normal PLR is immediate constriction of the pupils of both eyes in response to light directed into one eye. Next, test consensual (indirect) reflexes. To perform the consensual reflex, watch the pupil in one eye while an assistant shines the light into the opposite eye.

Lesion Location

The dazzle reflex is a subcortical reflex that is mediated via afferent input through the optic nerve to regions of the midbrain distinct from those required for the PLR and by efferent output along the facial nerve. It is possible for a horse to be cortically blind but still have dazzle reflexes, or to have PLRs but not dazzle reflexes. For a PLR, the stimulus pathway is via the optic nerves and optic tracts to the midbrain and thence to the efferent parasympathetic fibers of the oculomotor nerve. Fibers in the afferent limb of the reflex (i.e., optic nerve, optic tracts) can either pass on the same side or cross over at the optic chiasm and in the midbrain. Results of testing of menace responses along with direct and consensual PLRs usually allow anatomic localization of a lesion in the PLR pathways. In brief, without consideration of effects on consensual PLR, a lesion in an optic nerve affects the direct PLR and the menace on the same side; a lesion in an optic tract (rostral to the thalamus) affects the contralateral menace but does not affect direct PLR from either side, and a lesion in the midbrain or oculomotor nerve affects the ipsilateral PLR but not the menace responses.

Mydriasis is caused either by increased sympathetic dilator tone, by interference with the afferent arm of the PLR, or by damage to the oculomotor nucleus or nerve. Mydriasis caused by sympathetic overstimulation is usually bilateral, and PLRs are normal. With a complete optic nerve lesion on one side, slight ipsilateral mydriasis is seen. Light directed into the opposite eye constricts the mydriatic pupil. With complete oculomotor nerve lesions, the ipsilateral pupil is fixed and dilated and unresponsive to light directed into either pupil; however, light directed into the fixed pupil causes reflex constriction of the pupil on the opposite side.

Miosis occurs because of removal of sympathetic dilator influence to the pupil or possibly because of excessive stimulation of pupillary constrictors. The sympathetic pathway can be affected anywhere along the three-neuron pathway from the midbrain to the eye. With damage anywhere along the preganglionic (second) or postganglionic (third) neurons of the sympathetic supply to the head, there are signs of Horner's syndrome in addition to ipsilateral miosis. If the lesion affects the first (upper motor) neuron, there most likely will be widespread areas of spontaneous sweating over one half of the head, neck, and torso, with additional signs of brain stem or spinal cord disease (such as obtundation and ataxia and weakness of limbs). Early in the course of cerebral swelling, there often is miosis bilaterally. This is thought to be the result of either removal of sympathetic influences to the eyes or facilitation by upper motor neurons of the constrictor effects of oculomotor nerves.

Associated Clinical Signs

Horses with abnormal PLR because of cerebral swelling have other signs of forebrain disease, such as obtundation, dementia, and cortical blindness. Within 4 weeks of optic nerve injury, abnormal pigmentation of the fundus develops and there is

atrophy of the vasculature of the optic disc. In addition to miosis and ptosis, interruption of the sympathetic supply to the head causes other signs of Horner's syndrome, including spontaneous facial sweating and hyperemia of mucous membranes.

Position and Movement of the Eyeballs

Examination

While continuing to stand in front of the horse, observe the position and size of the pupils while the head is held level (i.e., a line through the center of each eyeball is parallel to the ground). While keeping the head level, lift the chin slowly. The eyeballs should remain stationary while the chin moves upward; thus, the eyes rotate ventrad relative to the long axis of the head. In horses with vestibular disease, abnormal eye positions are exaggerated by this maneuver.

If the pupils are in abnormal positions, try to position the head in such a way (usually by rotation) that the pupils are normally oriented relative to the transverse axis of the head. For example, a horse with vestibular disease often has ventral deviation of the eyeball on the side of the lesion and dorsal deviation on the opposite side (Figure 50-4). Eye position can be normalized relative to the axis of the head simply by rotating the head in the direction of the ventrally deviated eye. The abnormal position of the eyes in horses with vestibular disease is termed *vestibular strabismus.* True strabismus is eye deviation that cannot be corrected by repositioning the head and usually reflects dysfunction of nerves to the extraocular muscles (i.e., oculomotor, trochlear, or abducens nerves). In horses with strabismus, the eyeball may be rotated medially or laterally, clockwise or counterclockwise. Newborn foals normally have slight dorsomedial rotation of the pupils compared with older horses.

Further assess abducens nerve function by performing a modified corneal reflex. Hold the eyelids closed and, through the eyelid, push the eyeball medially. The normal response to this maneuver is retraction (adduction) of the eyeball. This reflex requires intact sensory branches of the trigeminal nerve, central connections in the hindbrain, and motor fibers of the abducens nerve.

Move the horse's head in a horizontal arc from side to side and observe the movements of the eyeballs. Signs of physiologic nystagmus should normally be elicited—namely, a series of horizontal movements of the eyeball consisting of a rapid phase in the direction of head movement followed by a slow phase in the opposite direction. Each fast phase is accompanied by an eye blink. Physiologic nystagmus is normal and should be distinguished from eye movements characteristic of vestibular disease: spontaneous nystagmus, which occurs when the head is stationary and in a neutral position, and positional nystagmus, which occurs only when the head is moved to certain positions. In horses with asymmetric vestibular disease, physiologic nystagmus is often abnormal or absent when the head is moved toward the side of the lesion.

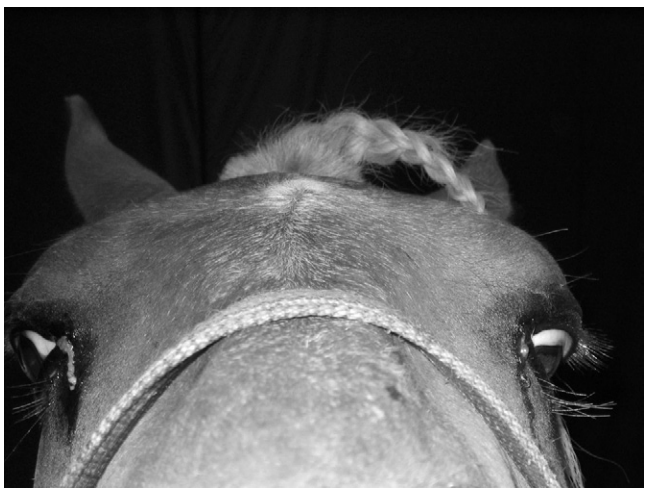

Figure 50-4. Vestibular strabismus in a horse with eastern equine encephalomyelitis (EEE). This yearling Quarter Horse presented initially with high fever and a left head tilt. When the head was straightened out and the chin lifted, the left eye deviated downward and the right eye deviated upward.

Lesion Location

For horses with vestibular strabismus, the causative lesion could be anywhere in the peripheral or central vestibular system (including connections with the cerebellum and nerves to the extraocular muscles), usually on the same side as the ventrally deviated eye (described more fully under "Orientation and Coordination of the Head," earlier). A horse exhibiting true strabismus most likely has a lesion in CNs III, IV, or VI (or its roots or nuclei). These nerves exit the midbrain or hindbrain and pass a short distance to the oblique and rectus muscles of the eye.

Associated Clinical Signs

The roots of the nerves to the extraocular muscles are subject to dorsal pressure exerted by the cerebrum as it swells and herniates under the tentorium cerebelli. Other signs associated with cerebral swelling are obtundation, blindness, seizures, and fluctuant to dilated, fixed pupils. There may also be jerky head movements as a result of pressure on the cerebellum. As compression of the midbrain and rostral hindbrain becomes more severe, obtundation progresses to coma with decerebrate posturing. With more focal lesions of the nerves or their nuclei, mydriasis (III) or defective modified corneal reflex (VI) is likely.

Swallowing

Examination

It is difficult to assess competence for swallowing during a physical examination. On the basis of history and observation, note whether feed, water, or saliva return through the nose, especially when the horse eats or drinks. Pass a nasogastric tube into the pharynx and assess effectiveness of swallowing movements as the horse attempts to move the tube into the esophagus.

Lesion Location

Involvement of the nucleus ambiguus in the hindbrain, or the roots or peripheral parts of one or both glossopharyngeal, vagus, or spinal accessory nerves, can cause dysphagia. These nerves are particularly vulnerable to damage as they pass over the guttural pouches.

Associated Clinical Signs

There is frequently coughing, especially during eating, and signs of pneumonia secondary to aspiration of feed. With bilateral laryngeal paresis because of involvement of the vagus nerves or branches, there may be inspiratory stridor at rest. At exercise, minor or unilateral laryngeal paresis (involvement of CN X or branches) may be evident as a "roaring" sound during inspiration; involvement of CNs IX, X, XI, and possibly XII may lead to displacement of the soft palate and expiratory stridor. The slap test, which is described later, is negative on the paralyzed side.

Tongue

Examination

Pull the jaws slightly apart and observe the movements of the unrestrained tongue. With unilateral weakness, the tongue curls toward the normal side. Grasp the tongue from one side after inserting the hand through the interdental space. Note resistance of the tongue to being stretched and look for atrophy (Figure 50-5) and muscular fasciculations. Gently pinch the side of the tongue with a hemostat and look for reflex retraction and behavioral response. Pull the tongue out one side of the mouth, release it, and look for retraction of the tongue back into the mouth. In normal horses, one or two chewing movements occur as the tongue is quickly retracted.

Lesion Location

Tongue deviation, muscle atrophy, and/or fasciculations reflect involvement of a hypoglossal nucleus, root, or nerve. Delayed or absent retraction of the tongue back into the mouth can occur with any brain disease, probably because of involvement of upper motor neurons that regulate the actions of hypoglossal nerves.

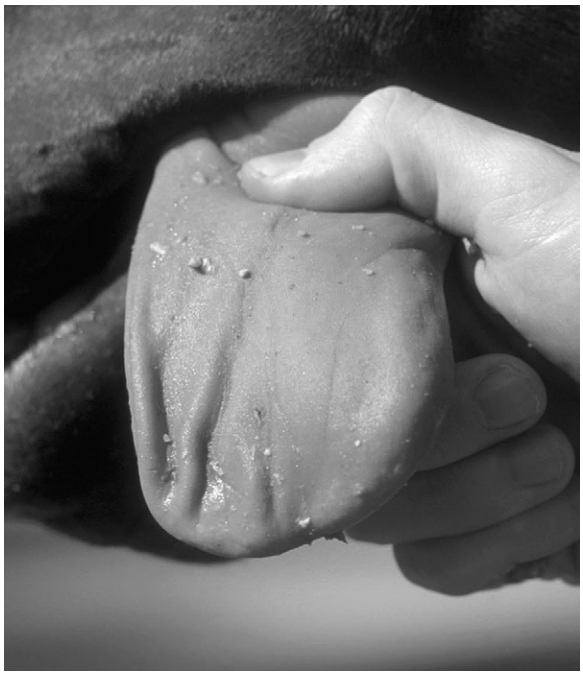

Figure 50-5. Unilateral tongue atrophy in a horse with possible equine protozoal myeloencephalitis. Note the characteristic longitudinal folds on the atrophied (right) side. The tongue intermittently deviated toward the left side when the mouth was held open.

Associated Clinical Signs

With delayed retraction caused by extensive brain disease, there is usually profound obtundation, often with ataxia and weakness of the trunk and limbs. Because the hypoglossal nerve is close to CNs IX, X, and XI as they pass over the guttural pouch, signs of dysphagia and respiratory paralysis may accompany tongue atrophy in horses with guttural pouch disease.

General Examination of the Neck, Trunk, and Limbs

Examination

With the horse standing squarely, assess muscle mass, paying particular attention to asymmetries. Note any circumscribed or asymmetric areas of sweating. Firmly press the cranial edge of each of the cervical transverse processes from C3 to C6 on each side to test for a pain response. Put pressure on the C6-C7 intervertebral joints by pushing medial to the deep pectoral muscle in front of the shoulder on each side. Test lateral neck flexion by enticing the horse to move its head toward feed held at the point of the elbow.

Lesion Location

Neck pain revealed by palpation or reluctance to turn laterally may follow any neck trauma but usually reflects arthritis of intervertebral joints.

Severe or rapidly developing muscle atrophy indicates denervation and is a localizing sign. Neurogenic muscle atrophy is caused by damage to the lower motor neuron in the ventral column of the gray matter, ventral nerve roots, or peripheral nerves supplying that muscle. Neurogenic atrophy of thoracic limb musculature results from lesions of the C6 to T2 spinal cord segments or roots, brachial plexus, or peripheral nerves, whereas atrophy of pelvic limb muscles reflects involvement of L3 to S2. Anesthesia of a strip of skin is caused by loss of the segmental sensory nerve, dorsal nerve root, or connections in the spinal cord. Because sympathetic fibers are distributed with spinal nerves, spontaneous sweating may occur over denervated skin.

Associated Clinical Signs

Arthritic neck pain may be detected in wobblers, especially those older than 2 years. Neurogenic atrophy usually causes limb weakness and gait abnormality and may be associated with other signs of spinal cord disease. For example, there may be ataxia and weakness in a pelvic limb because of upper motor neuron damage at C6, and there may be atrophy of the triceps and weakness of the thoracic limb on the same side because of a gray matter lesion in the same segment.

Slap Test

Examination

While standing on the left side, reach under the horse's neck and hook the index and middle fingers of the left hand over the highest palpable point of the right side of the larynx—the

muscular process of the arytenoid. Have the handler move the head slightly to the left of midline. Gently strike the horse behind the withers with the palm of the right hand. The expected response is slight palpable movement (adduction) of the arytenoid in response to each slap. The adductor response is most reliably obtained if the withers region is slapped when the horse is breathing out. Repeat the procedure from the right side. More information on this test is found in Chapters 42 and 44.

Lesion Location

Sensory input to this reflex is the sensory nerves and roots under the area that is slapped (approximately T7 to T11). Central pathways are thought to cross to the other side at this level and then pass rostrad to the nucleus ambiguus. Next, efferent fibers pass out in the vagus nerve via the recurrent laryngeal nerve to innervate the contralateral laryngeal adductor muscles. Severe cervical spinal cord disease often affects this test bilaterally, and the vagus and recurrent laryngeal nerves may be affected at the guttural pouch or within the jugular groove. Variations in neck anatomy make this reflex easy to palpate in some horses but difficult or impossible in others.

Associated Clinical Signs

Other signs of cervical spinal cord disease include ataxia and weakness of the trunk and limbs. Dysphagia and aspiration pneumonia are further signs of guttural pouch disease, and Horner's syndrome may also be seen when there is involvement of the vagosympathetic trunk deep to the jugular groove.

Cervicofacial Reflex

Examination

Place the left index and middle fingers at the left commissure of the lips, then strike the skin over the brachiocephalicus muscle with the closed tip of the hemostat. Begin at the cranial end of the neck and continue back to the shoulder. The expected responses are facial contraction (detected as retraction of the commissure of the lip) and contraction of the brachiocephalicus and cutaneus colli (observed as shrugging of the shoulder, lateral jerking of the head, and twitching of the skin of the neck).

Lesion Location

Interruption of reflex components in the facial nerve, cervical nerves or roots (sensory or motor), or local cervical spinal cord segments can affect the cervicofacial reflex. This reflex is typically reduced at the level of a cervical spinal cord lesion but is normal cranial and caudal to the lesion.

Associated Clinical Signs

There may be other signs of facial paralysis or cervical spinal cord disease (e.g., limb ataxia and weakness). With involvement of cervical sensory nerves or roots, there may be a strip of cutaneous anesthesia that corresponds to the area of diminished cervicofacial reflex. Asymmetric damage to the most cranial cervical nerves may lead to signs suggestive of peripheral vestibular disease.

Cutaneus Trunci Reflex (Panniculus)

Examination

When examining the reflex on the left side of the horse, use the extended right thumb to firmly prod the skin of the sensory field, beginning cranially just behind the shoulder and extending caudad to the last intercostal space. Check every intercostal space both ventrally and dorsally. For safety, firmly grasp the back of the mane with the left hand and face backward when testing the reflex, because horses that resent this test will try to kick the examiner. A normal response is twitching of the skin, with or without indication of conscious perception of the stimulus. Irrespective of the site of stimulation, the twitch response is the same.

Lesion Location

The reflex pathway is input from sensory thoracic nerves to the ipsilateral spinal cord, where it courses rostrad via interneurons to end in the C8 and T1 segments, and then via the brachial plexus to the lateral thoracic nerve and the cutaneus trunci muscle. Interruption of this pathway in the spinal cord white matter results in loss of the reflex from approximately the point of the lesion caudad. A lesion of the sensory nerve will affect only the reflex within the same dermatome (skin strip), whereas loss of lateral thoracic nerve function ablates the entire ipsilateral reflex.

Associated Clinical Signs

A strip of cutaneous anesthesia may overlay the cranial edge of the hyporeflexic zone when the spinal cord lesion affects interneurons coursing cranially and sensory connections from the segmental spinal nerve. Ataxia and weakness of the ipsilateral pelvic limb is usually found in such cases.

Back Reflexes

Examination

After making sure that the pelvic limbs are positioned equally and squarely, stroke the closed tip of the hemostat caudad along the skin over the longissimus dorsi muscle, from mid thorax caudad to the level of the tuber coxae. For safety, hold the back part of the mane with the other hand. The expected response is brisk extension of the back and pelvis followed quickly by return to normal posture. Next, stroke the hemostat caudad along the skin over the gluteal muscles. This should elicit flexion, followed by relaxation of the lumbar spine and pelvis. Common abnormal reactions to these tests include (1) partial collapse in the pelvic limbs, (2) wobbling of the pelvis from side to side, and (3) no response.

Lesion Location

Abnormal results of the back tests correlate with truncal and pelvic limb weakness and ataxia caused by spinal cord disease.

Associated Clinical Signs

Expect other signs of pelvic limb weakness and ataxia, such as toe-dragging and circumduction.

Tests for Limb Strength

Examination

During the passive part of the examination, it is difficult to detect mild thoracic limb weakness. Only very weak horses buckle a thoracic limb in response to downward pressure exerted over the withers. The hopping test is more sensitive. From the left side, hold the halter with the left hand. Pick up the left leg, then push the head toward the right side while leaning against the left shoulder (Figure 50-6). Normal horses use the right limb to hop briskly around a circle centered on the pelvic limbs. With limb weakness, the response may be delayed so that the horse leans markedly before hopping, or the limb may buckle after landing.

Strength of the pelvic limbs is evaluated by resistance to pressure over the pelvis and to a sideways pull on the tail. Additional insight is provided by the back reflexes described earlier. To test resistance to dorsal pressure, stand on one side of the horse and hook the fingers over the opposite tuber sacrale. Use maximal effort to try to collapse the near limb. Only weak horses buckle in response to such pressure. The tail-pull test is done both at rest and while the horse is walking in a straight line. With the horse standing squarely, take the tail and pull sideways with gradually increasing force. After initial slight movement in the direction of pull, normal horses usually cannot be moved sideways, even with strong pressure. Next, pull sideways on the tail while the horse is walking in a straight line. Always work on the same side as the handler. Exert moderate lateral pressure during all phases of the stride, and then rhythmically increase pressure as the horse supports weight on the near leg. Normal horses of 450-kg body weight can be moved only slightly to the side. Response to this test varies according to the size and strength of the examiner and the bodyweight and temperament of the horse. Perform the test in mirror-image fashion from the right side (i.e., with the horse being led from the right side). Interpretation of this test requires practice and experience on the part of the examiner.

Figure 50-6. Force the horse to hop on each front limb to evaluate limb strength.

Lesion Location

If a full-size adult horse can easily be pulled sideways at rest, there is probably interruption of the antigravity myotatic reflex pathway. Practically, such a lesion most likely is located either in the ventral gray matter, in the roots (L3 to L5) that form the femoral nerve, or in the femoral nerve itself. If cutaneous sensation over the saphenous vein is absent or reduced, the lesion is central to the ventral aspect of the shaft of the ilium (the site at which the sensory saphenous nerve joins the femoral nerve). The much more common finding—namely, lack of resistance to tail-pull only during walking—is consistent with an ipsilateral spinal cord lesion affecting descending upper motor neurons anywhere from C1 to S2, inclusive.

Associated Clinical Signs

Depending on the location of the lesion, there may be interference with long spinal reflexes, hypalgesia of skin strips, spontaneous sweating of skin strips, or atrophy of musculature, in addition to alterations in gait.

Gait

Examination

Have the handler walk the horse in straight lines, leading alternately from the left and the right side. The handler should be instructed to keep the horse's head and neck as straight as possible during walking. When the horse turns its head to one side, there is reciprocal and potentially confusing movement of the pelvis toward the same side. Follow directly behind the horse. From this vantage point, evaluate leg position and stride symmetry. Also, watch for excessive (1) side-to-side (wobbling) movement of the pelvis, (2) up-and-down movement of the tuber coxae (pelvic roll), and (3) side-to-side swiveling of an imaginary line from the tailhead to a point between the tuber sacrale (pelvic yaw). Next, watch the gait from the side while walking in stride with the pelvic and then thoracic limbs. Note any toe-dragging, knuckling, stride-length asymmetries, and abnormal protractive movements, such as hyperflexion, stiffness (hypometria), or excessive range of movement (hypermetria). Often, these signs are most obvious as the horse transitions from standing still to walking. Repeat this part of the examination with the horse's chin lifted and with the horse walking up and down a modest slope. These maneuvers exacerbate most gait abnormalities, especially stiffness of the thoracic limbs.

Take the horse in hand for the next part of the examination. Hold the lead rope with the left hand and, by walking backward, lead the horse in counterclockwise circles (Figure 50-7). It is very important that the horse be always walking forward

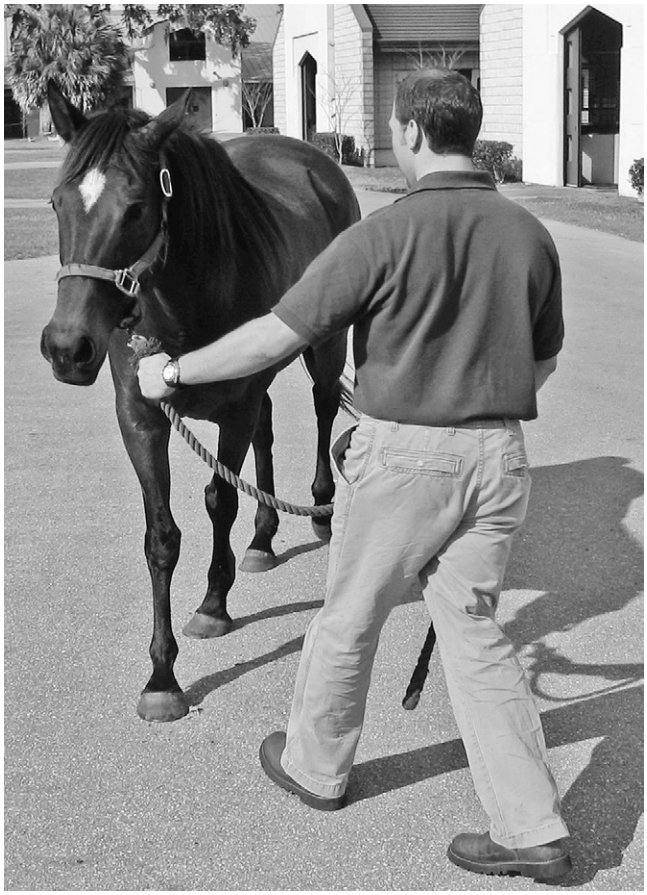

Figure 50-7. Testing for strength: walk backward and pull the horse in counterclockwise circles, then repeat in mirror-image fashion in clockwise circles.

Figure 50-8. Gait analysis: pull the horse sideways and backward in tight circles so that it pivots around a point midway between the thoracic and pelvic limbs. Repeat on the right side.

in these circles. Vary the diameter, making the circles alternately small and large. Observe carefully the motion of the right (outside) pelvic limb by looking under the horse's torso. This limb will often arc out widely on the outside of the circle (i.e., circumduct) in horses with spinal cord disease. In mirror-image fashion, lead the horse from the right side in clockwise circles.

Next, pull the horse sideways in tight circles in either direction. This is done from a position slightly behind the shoulder by pulling the lead rope at an angle sideways and caudad (Figure 50-8). If done correctly in normal horses, the opposite thoracic limb should cross in front of the supporting limb and the pelvic limbs should move reciprocally, causing the horse to pivot around a point midway between the thoracic and pelvic limbs. Horses that are weak and ataxic tend to sag backward in the hindquarters before they start to move, and then pivot the front part of the body around one or both pelvic limbs. There is often also interference between or otherwise inappropriate and inconsistent placement of thoracic limbs.

Use the lead rope to push the horse straight backward. A normal horse should move backward in two-beat fashion, with simultaneous movement of diagonally opposite pairs of limbs (e.g., left thoracic and right pelvic limbs). A horse with spinal cord disease may sag backward before moving and slide its hooves along the ground rather than picking them up and placing them.

Lesion Location

Signs of limb weakness and ataxia suggest spinal cord (or, rarely, peripheral nerve) damage at or cranial to the affected limb. If there is obvious ataxia and weakness in thoracic and pelvic limbs, there is likely to be at least one lesion in the spinal cord somewhere between or within the C1 and T2 spinal cord segments. When the signs are caused by external compression of the cervical spinal cord (e.g., in cervical vertebral stenotic myelopathy), signs in the pelvic limbs are usually worse than those in the thoracic limbs. When, in such cases, the pelvic limb signs are mild, thoracic limbs may appear normal. In contrast, when thoracic limbs are normal but there is moderate or severe ataxia and weakness in the pelvic limbs, there is likely to be at least one lesion caudal to T2 and cranial to S3. If one or both thoracic limbs are abnormal in a horse that has normal pelvic-limb gait, the gray matter of the C6 to T2 spinal cord segments (without white matter involvement), the roots or nerves of the brachial plexus, or the peripheral nerves to the thoracic limbs are probably affected. Asymmetric lesions in the spinal cord cause signs that are more severe on the side of the lesion. Occasionally, there are signs of weakness without ataxia (e.g., botulism, equine motor neuron disease) or ataxia without weakness (e.g., cerebellar abiotrophy, peripheral vestibular disease). Additional signs, such as defective reflexes (cervicofacial, slap, or cutaneus trunci), neurogenic muscle atrophy, or cutaneus anesthesia, often help to localize the spinal cord lesion.

Associated Clinical Signs

Abnormal responses to tests for limb strength, reduced or absent long spinal reflexes, and strips of cutaneous anesthesia or spontaneous sweating may accompany lesions of the spinal cord or peripheral nerves.

Testing of Recumbent Horses

Examination

Note whether or not the horse moves its limbs voluntarily without stimulation. If possible, assist and stimulate the horse in such a way as to assess which of the following best describes the horse's maximal voluntary motor function: (1) lifts head off the ground; (2) rolls shoulders and chest into a sternal position; or (3) straightens thoracic limbs and assumes a "dog-sitting" position.

Check long spinal reflexes (cervicofacial, cutaneus trunci, slap) and test cutaneous sensation systematically over the limbs and torso. Test sensation at each site by grasping a fold of skin between the jaws of the hemostat and firmly squeezing the skin and watching for evidence of a conscious response by the horse. This is a behavioral reaction and must be distinguished from a reflex response. Sensory fields for some peripheral nerves of horses have been described.[2]

Test pelvic limb reflexes and function. First, assess extensor tone in the limb by testing resistance to passive flexion. Next, perform the flexor test by pinching skin on the distal limb with a hemostat. If there is no response, try pinching skin elsewhere on the leg. A normal response is flexion of the limb, usually with some evidence that the horse can feel the skin pinch. When abnormal, the flexor response may be reduced or absent, and it may be accompanied by reflex extension of the contralateral digit (crossed extensor reflex). To test the patella reflex, hold the pelvic limb in a moderately flexed position, and strike the skin over the middle patella ligament (Figure 50-9). A twitch handle works well for this purpose in full-sized horses; a patella hammer or reversed hemostat can be used in foals. The expected response is brisk extension of the stifle. If the reflex is absent, move the leg into different positions and retest. Classify the response as absent, normal, or increased. Other extension reflexes in the pelvic limb cannot be elicited reliably but should be tested for comparison with the opposite leg. These include (1) the tibial reflex—tap just behind the greater trochanter; (2) the gastrocnemius reflex—strike the Achilles tendon close to its insertion; and (3) the long digital extensor reflex—strike the middle of the long digital extensor muscle.

Test thoracic limb reflexes and function. Assess extensor tone and evaluate the flexor reflex as described for the pelvic limb. No other reflex can be obtained consistently in the thoracic limbs, but, for comparison with the opposite limb, test the (1) triceps reflex—with the limb in flexed position, strike the triceps muscle and watch for elbow extension; (2) test the biceps reflex—strike the front of the elbow and watch for extension of the shoulder and flexion of the elbow joint; and (3) strike the middle of the extensor carpi radialis muscle and look for extension of the carpus.

Lesion Location

If a horse can dog-sit, the principal spinal cord lesion is probably behind T2. Inability of a horse to roll from lateral into sternal recumbency is associated with severe lesions (usually involving gray matter) of the caudal cervical or rostral thoracic segments, whereas serious injury to the spinal cord in the rostral part of the neck may also prevent a horse from raising its head off the ground.

With involvement of lower motor neurons to limb muscles, there is reduced or absent extensor tone. Limb reflexes are reduced or abolished if the sensory nerves, motor nerves, or central components of the reflexes are affected. The afferent, central, and efferent parts of important reflexes are listed in Table 50-1. In contrast, extensor tone and limb reflexes may be exaggerated beginning several days after injury to descending upper motor neurons. For example, after trauma to the spinal cord at the T13 segment, patella reflexes and pelvic extensor muscle tone may be exaggerated.

If there is no response to strong pinching of the skin over caudal regions of the body, there is probably catastrophic

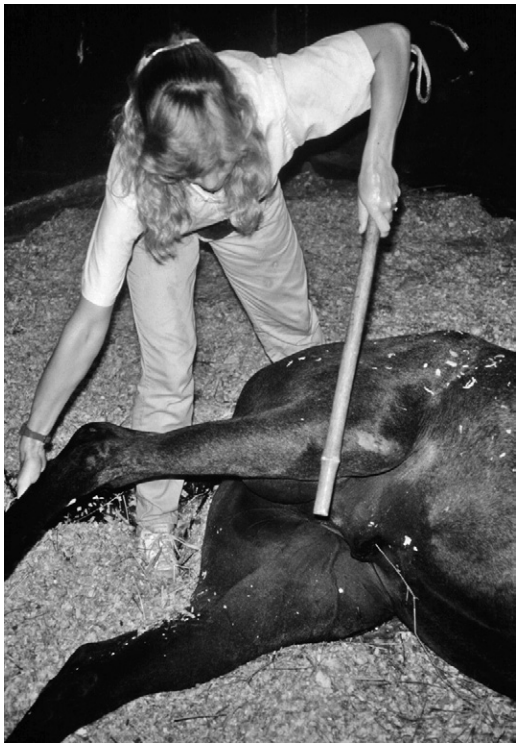

Figure 50-9. Testing the patella reflex in a recumbent horse. The limb is held in a flexed and slightly abducted position, and the skin over the middle patella ligament is struck with a twitch handle.

TABLE 50-1. Limb and Tail Reflexes

Reflex	Spinal Cord Segments	Peripheral Nerves
Flexor (thoracic)	C6-T2	Median, ulnar, axillary, musculocutaneous
Triceps	C7-T1	Radial
Biceps	C7-C8	Musculocutaneous
Patella	L3-L5	Femoral
Flexor (pelvic)	L5-S2	Sciatic
Anal	S3-S4	Pudendal
Tail clamp	S3-S4	S1 to coccygeal segments

damage to the spinal cord cranial to the anesthetic area. If there is no response to deep pain for more than 24 hours, there is at least functional transection of the spinal cord.

Associated Clinical Signs

Large horses that remain recumbent for more than a few hours often have reduced skin sensation in the distal limbs secondary to pressure-induced injury of superficial sensory nerves. This complicates interpretation of tests for flexor reflexes and for presence of deep pain.

Tail and Anus

Examination

Assess tail strength by lifting (extending) the tail. Prod or pinch the skin adjacent to the anus and observe the anal contraction and tail-clamp reflexes. If these reflexes are abnormal or if the history suggests possible cauda equina syndrome, perform a rectal examination to assess rectal tone and bladder size and tone. Assess muscular symmetry of the tail and test cutaneous sensation over the tail and caudal structures.

Lesion Location

Anesthesia and areflexia of the tail, penis, and perineum and paralysis of the anus, rectum, bladder, and penis are signs of cauda equina syndrome. Lesions of the spinal cord or nerve roots caudal to the S2 spinal cord segment cause some or all of the signs of cauda equina syndrome. The bundled roots forming the cauda equina are vulnerable as they pass through the sacrum and proximal coccygeal vertebrae.

Associated Clinical Signs

With involvement of S2 and more rostral nerve roots, expect to see signs of pelvic limb weakness in addition to cauda equina syndrome. Colic caused by obstipation may be the presenting sign of rectal paralysis, and urinary overflow incontinence is a sign of bladder paralysis.

CEREBROSPINAL FLUID COLLECTION

Atlantooccipital (AO) cerebrospinal fluid (CSF) collection under general anesthesia is simple, and iatrogenic blood contamination is seldom a problem. Lumbosacral (LS) collection has the advantage of not requiring anesthesia, but the procedure is technically more challenging than AO collection and significant blood contamination may occur. Because CSF flows caudad over the spinal cord from sites of production in the brain, LS samples can theoretically reflect changes anywhere in the CNS, whereas AO samples may not reflect caudal spinal cord disease. In general, in cases where the horse's physical condition does not dictate the procedure used, select the collection site that is closer to the likely site of the lesion.

Atlantooccipital Cerebrospinal Fluid Collection

Anesthetize the horse (e.g., 1 mg/kg of xylazine and 2 mg/kg of ketamine, both given IV) and position it in lateral recumbency. This procedure should not be performed in standing horses. Clip and surgically prepare a rectangle of skin approximately 10 cm wide and 15 cm long centered on the dorsal midline and beginning just behind the forelock. Extend the prepared area on the side facing up so that a 2.5 cm strip is clipped over the cranial border of the wing of the atlas. In horses heavier than 100 kg bodyweight, use an 8.23 cm 18- or 20-gauge stiletted spinal needle.[3] In foals, use a regular 3.75 cm 20-gauge needle. Flex the atlantooccipital joint in the median plane to an angle of 90 degrees or less. This is conveniently done if the collector kneels in such a way as to flex the head with the inside of the knee while leaning over the neck to place the needle. The needle is inserted at the intersection of lines running along the front of the atlas and along the dorsal midline (Figure 50-10). Insert the needle through the skin and then pass it in the median plane by aiming at the middle of the lower jaw. The needle is passed through the funicular part of the ligamentum nuchae and advanced until there is a popping sensation as the needle penetrates the atlantooccipital membrane, the cervical dura, and arachnoid mater. In practice, the stilette is withdrawn from the needle each time there is a popping sensation. CSF should flow freely from the hub when the subarachnoid space is entered. This can happen anywhere from 2.5 to 8.3 cm beneath the skin surface, depending on the size, sex, breed, and condition of the horse. Collect two or three aliquots of 1 to 5 mL of CSF, depending on need, either by aspirating drops as they fall from the hub of the needle or by attaching a syringe to the hub and aspirating directly. The last aliquot is least likely to be contaminated with blood and should be retained for analysis. Replace the stilette and withdraw the needle.

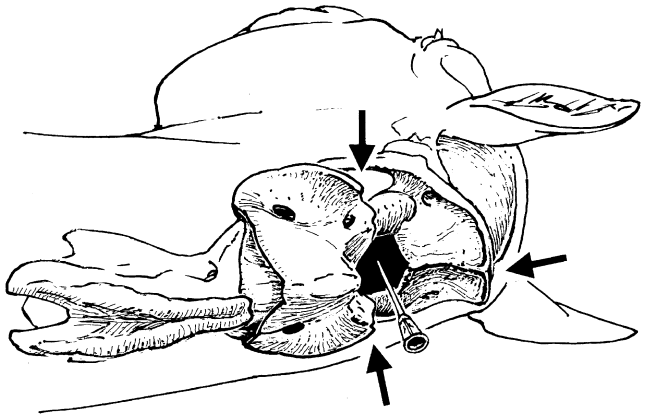

Figure 50-10. Atlantooccipital cerebrospinal fluid collection. The needle is inserted through the midline using the cranial border of the wings of the atlas and the nuchal crest of the skull *(arrows)* as landmarks.

There is very little morbidity associated with this procedure; however, occasionally a horse will hold its head stiffly in an extended position for a few days afterward. This is not associated with fever (in which case infection would be likely) and may be analogous to the headache that occurs in some humans after CSF aspiration. The condition responds well to the use of a nonsteroidal anti-inflammatory drug (NSAID).

Lumbosacral Cerebrospinal Fluid Collection

This procedure is conveniently performed in stocks so that the collector can stand on a raised platform alongside the horse; however, it also can be done in a stall with the horse pushed

against a wall. In either case, it is preferable that only a nose twitch be used. If a sedative must be given, use the lowest dose that controls the horse without causing the hindquarters to sway and wobble unduly.

The key landmarks for this procedure are the tuber sacrale and the dorsal spinous process of the second sacral vertebra. The paired tubera sacrale are located near the midline and can be found between the caudal margins of the tuber coxae at approximately the highest point of the rump. The dorsal (superficial) edge of each sacral tuberosity can be palpated through the skin as a slender ridge beginning adjacent to the sacral spine and progressing craniad and slightly laterad for several centimeters. When viewed from above, the paired tubera sacrale thus form a V with the vertex caudal. The correct tap site is located on the midline within or slightly in front of the V. To find the site, palpate the dorsal spinous processes of the caudal sacrum with the thumb, then slide the thumb forward until a 2- to 3-cm–long depression is located on the midline (between the spinous processes of the second sacral and sixth lumbar vertebrae). Clip and surgically prepare a rectangular section of skin centered on this site (Figure 50-11).

Infiltrate 0.5 to 1 mL of local anesthetic into the skin of the tap site, and make a sagittally oriented stab through the skin with a No. 15 scalpel blade. Rest both forearms on the horse's back while holding the hub of the needle with both hands. The needle is inserted along the intersection of median and transverse planes. It is helpful to have an assistant watch from behind the horse to guide side-to-side orientation of the needle. Advance the needle until (1) a slight "pop" is felt as the needle traverses the ligamentum flavum, (2) there is an involuntary response from the horse (usually brisk extension of the tail and pelvis), or (3) the point of the needle strikes the bottom of the vertebral canal. Remove the stilette, attach a syringe, and apply gentle aspiration pressure. To obtain flow of CSF, it may be necessary to rotate the needle or slightly retract it (or both) during attempted aspiration. Advance the needle only if the stilette is in place. CSF may flow more easily if both jugular veins are occluded (thus increasing intracranial pressure). Collect an initial sample of 2 to 5 mL (or more if the sample is visibly contaminated with blood) to "clear" iatrogenic blood contamination; then collect another sample for analysis. Reinsert the stilette and remove the needle.

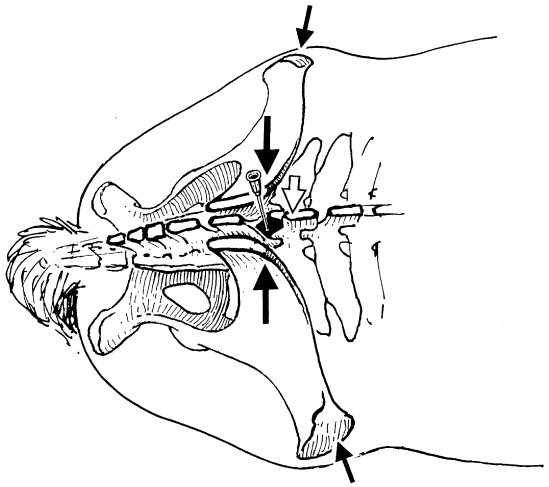

Figure 50-11. Lumbosacral cerebrospinal fluid collection. The caudal border of the tuber coxae *(small dark arrows),* the cranial edge of the tuber sacrale *(large dark arrows),* and the spine of L6 *(open arrow)* are used as landmarks.

Rarely, horses are sensitive to touch near the tap site for several days after the procedure. Occasional cases of cauda equina syndrome have occurred because epidural abscessation develops at the tap site.

CSF Analysis

At least 1 mL of CSF anticoagulated with ethylenediaminetetraacetic acid (EDTA) should be submitted for cytologic examination. A slide for this purpose should be prepared within 2 hours of collection by low-speed centrifugation of the sample. Alternatively, cell morphology can be preserved by mixing the sample with an equal volume of 50% ethyl alcohol. Standard chemistry analyses and immunoassays can also be performed on these samples. Normal values are shown in Table 50-2.[4,5] It is important to minimize blood contamination of the sample so that results do not reflect admixture of plasma and CSF. For most purposes, a red blood cell count of less than 50 µL is acceptable.

DIAGNOSTIC IMAGING EXAMINATION

Survey Radiographs

The primary reference for "Survey Radiographs" is reference 6.

Head

Lateral, oblique, and dorsoventral (in the standing animal) or ventrodorsal (in the anesthetized animal) views of the cranium and associated soft tissues can be obtained with portable film-screen or digital systems. On lateral radiographs, the ovoid shell of the cranial vault is seen. In foals younger than 3 months, the dorsal profile of the cranial cavity is interrupted by the fontanel. Other relevant structures on lateral radiographs are the petrous temporal bone, basilar bones, occipital condyles, ethmoid turbinates and ethmoid plate, frontal sinus, hyoid apparatus, guttural pouches, soft palate, and epiglottis. Oblique radiographs are useful to separate the paired stylohyoid bones and petrous temporal bones. Ventrodorsal or dorsoventral views compare these structures more directly and also show the tympanic bullae.

The parietal-occipital suture and the synchondrosis between the basisphenoid and basioccipital bones disappear by 5 years of age.

Radiographs of the head are used to diagnose frontal and parietal fractures, fractures or separation of the basilar bones, fractures of the petrous temporal bone, hemorrhage into and around the guttural pouches, soft palate displacement, stylohyoid fractures, and orbital fractures.

Cervical Spine

Radiographic examination of the cervical spine is indicated when there is deformity or palpable abnormality of the neck, ataxia or weakness of the limbs, or neck trauma, stiffness, or pain.

Acceptable views of C1 to C6 are readily obtained in standing horses with portable equipment, especially with fast rare-earth screens, grids, and appropriate film, imaging plates used for

TABLE 50-2. Reference Values for Equine Cerebrospinal Fluid

Parameter	Adult (AO)*	Adult (LS)*	Neonate (AO)†
Specific gravity	1.004-1.008	1.004-1.008	—
WBC count (cells/mL)	0-7	0-7	—
RBC count (cells/mL)	<600	<600	—
Total protein (mg/dL)	10-120	10-120	109 ± 9.7
Albumin (mg/dL)	24-51	24-56	—
IgG (mg/dL)	3-8	3-10.5	—
Glucose (mg/dL)	30-80	40-75	98.8 ± 12
Creatine kinase (U/L)	0-8	0-8	—
LDH (U/L)	0-8	0-8	—
AST (U/L)	15-50	15-50	—
Lactic acid (mg/dL)	1.92 ± 0.12	2.3 ± 0.21	—
(mmol/L)	0.21 ± 0.01	0.26 ± 0.01	—
Sodium (mEq/L)	140-150	140-150	148 ± 7.2
Potassium (mEq/L)	2.5-3.5	2.5-3.5	3.01 ± 0.17
Chloride (mEq/L)	95-123	95-123	—
Calcium (mg/dL)	2.5-6	2.5-6	—
Phosphorus (mg/dL)	0.5-1.5	0.5-1.5	—
Urea nitrogen (mg/dL)	5-20	5-20	—

*From Green EM, Kroll RA, Constantinescu GM: Equine cerebrospinal fluid: Analysis. Comp Cont Educ Pract Vet 15:288, 1993.
†From Furr MO, Bender H: Cerebrospinal fluid variables in clinically normal foals from birth to 42 days of age. Am J Vet Res 55:781, 1994.
AO, From atlantooccipital cerebrospinal fluid; *AST,* aspartate transaminase; *LDH,* lactate dehydrogenase; *LS,* from lumbosacral cerebrospinal fluid; *RBC,* red blood cell; *WBC,* white blood cell.

computed radiography, or direct digital systems. For normal 450-kg horses, more powerful equipment (75 to 100 kV, 75 to 100 mAs) is required for views of C7 to T1. Complete radiographic assessment of the cervical spine of a full-size horse requires four or five overlapping views: occiput and C1, C1 to C3, C3 to C5, C5 to C7, and C7 to T1 (Figure 50-12). It is very important to obtain true lateral projections, because a slight obliquity complicates interpretation of the image. In anesthetized horses, additional views including lateral-flexed, lateral-extended, and ventrodorsal (C1 to C5 or C6), can be obtained.

The horse has seven cervical vertebrae. The cervical spine of the standing horse adopts a reverse-S shape when viewed from the left, with gradual flexion at the cranial end and extension caudad. Within each vertebra is a rectangular-appearing vertebral foramen; the continuous cylinder formed by these foramina is the vertebral canal. Absolute values for minimal sagittal diameter, sagittal ratio, and corrected minimal sagittal diameter of the cervical vertebral canal within each vertebra have been reported. Preliminary normal ranges for minimal intervertebral sagittal diameters and sagittal ratios have been published. Measurement of sagittal diameter is used to detect stenosis of the vertebral canal and is described in detail in Chapter 51.[7-10]

The atlas (C1) has no body or articular processes. The axis (C2) has a large dorsal spinous process and a cranially projecting dens (odontoid process). There are separate ossification centers for the dens, head, body, and caudal epiphysis of C2. The body of each of the C3 to C7 vertebrae has cranial and caudal epiphyses, and there is an extra center of ossification in the ventral processes of C6. The transverse processes of C6 differ from those of C3 to C5 by presenting an additional ventral process. This ventral process contributes to the ventral profile of C6 in lateral projections. The caudal aspect of each of the bodies of C3 to C7 is smoothly concave and accommodates the convex cranial aspect of the next vertebra caudally. Intervertebral discs are radiolucent and of uniform width dorsal to ventral. The cranial and caudal articular processes of C3 to C7 form pairs of true articulations (facet joints) that are visible as smooth ovoid radiodense structures at the dorsal aspect of each intervertebral foramen. C7 is a relatively short vertebra with a small dorsal spinous process. In most horses, the dens fuses with the head of C2 at about 7 months of age, and the cranial and caudal physes of C3 to C7 close by 2 and 5 years, respectively.

Vertebral abnormalities including occipitoatlantoaxial malformation, cervical vertebral stenotic myelopathy, degenerative arthritis of the facet joints, osteomyelitis, fracture, discospondylitis, and multiple myeloma are examples of cervical spinal diseases that can be diagnosed in plain radiographs.

Thoracolumbar Spine

Thoracolumbar radiography may be performed on horses that have a history of trauma to the back, deformity of the back, acute or chronic back pain, or signs of limb weakness and ataxia that localize to this area.

Radiography of this area of the spine is difficult and requires specialized and powerful (75 to 120 kV, 100 to 250 mAs) equipment and fast rare-earth screens and grids or computed radiograph cassettes. With this equipment, it is technically possible to obtain lateral views from T1 to L3. Caudal to L3, superimposition of the ilial wings over the lumbar vertebrae precludes useful views. Ventrodorsal images from about T9 to L6 are possible in anesthetized horses. For all areas of the thoracolumbar spine, two lateral exposures are needed for film-screen systems: one optimized for the vertebral bodies and one for the dorsal spinous process. In some cases, separate processing algorithms can be used to obtain optimized images of bodies and spinous processes from a single exposure when a computed radiography system is used.

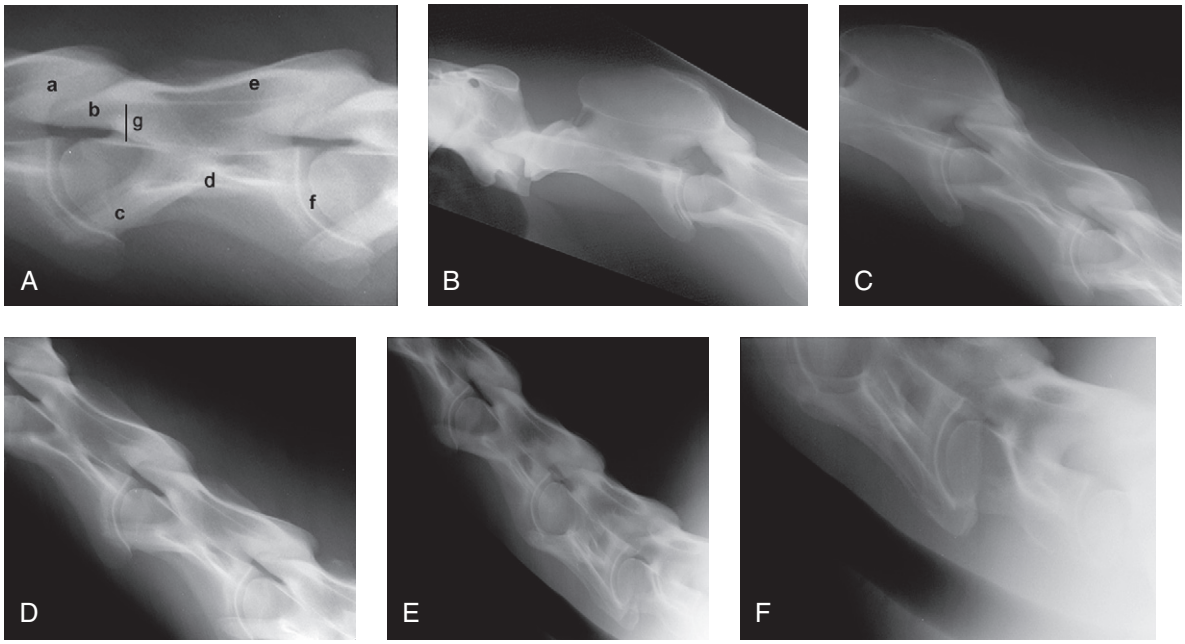

Figure 50-12. Survey radiographs of the neck of a standing normal horse. **A,** Fifth cervical vertebra. *a,* Caudal articular process of C4; *b,* cranial articular process; *c,* transverse process (cranial branch); *d,* vertebral body; *e,* dorsal lamina; *f,* intervertebral disc space; *g,* vertebral body. The *line* shows the sagittal diameter of the vertebral foramen. **B** to **F,** Five overlapping views of a complete cervical series. (Courtesy Dr. Greg Roberts, University of Florida.)

There are 18 thoracic vertebrae and usually six lumbar vertebrae. Some horses and all donkeys have only five lumbar vertebrae. Vertebral bodies are rectangular and the vertebral canal is of uniform sagittal diameter throughout. Disc spaces are curved and of uniform thickness, whereas facet joints vary over the length of the thoracolumbar spine. Thoracic and lumbar facet joints are difficult to image on lateral radiographs. In thoracic vertebrae, ribs articulate dorsal to the disc spaces. The dorsal spinous processes of the thoracic vertebrae increase in length to T6 or T7 and become shorter in the more caudally located vertebrae. Separate ossification centers are present in the dorsal spinous processes of T2 to T8. Thoracic dorsal spinous processes slope caudally, T15 is vertical (anticlinal), and the dorsal spinous processes of lumbar vertebrae slope cranially. The transverse processes of lumbar vertebrae are larger than those of the thoracic vertebrae and can be identified on lateral projections.

The cranial physes close by 12 months and the caudal physes by 4 years of age.

Degenerative joint disease, ossifying spondylosis, osteomyelitis, discospondylitis, and fractures and dislocations can be seen on plain films of the thoracolumbar vertebrae (see Chapter 53).

Sacrococcygeal Spine

Reasons for sacrococcygeal radiography include signs of cauda equina syndrome and pain or deformity over the sacrum or tail. Lateral views of the sacrum may be obtained with techniques similar to those used for the lumbar spine. The sacrum is usually poorly resolved because of overlying soft tissues, but the ventral profile can usually be seen as a straight horizontal line. The last segment of the sacrum (usually S5) is variably separated from the main body of the sacrum and may be confused with the first coccygeal vertebra. Ventrodorsal images can be obtained under anesthesia by using the pelvic technique described in Chapter 53. The sacroiliac joints can be seen on this projection.

Scout films of this area are most often useful in horses with signs of cauda equina syndrome caused by sacral or coccygeal fractures or dislocations.

Myelography

Myelography involves introducing contrast medium into the subarachnoid space followed by radiography of the brain and spinal cord to outline the spinal cord and adjacent structures. In the horse, cervical myelography is commonly used and is indicated when cervical survey films or clinical examinations have suggested the presence of a cervical spinal cord lesion.

Myelography should be performed under general inhalation anesthesia. An NSAID (preferably flunixin meglumine or ketoprofen) should be given prior to the procedure. All of the following views should be completed within 90 minutes, and preferably within 60 minutes. Begin in lateral recumbency and take plain lateral survey films. For these views, the head and neck need to be supported in horizontal position by radiolucent cushions. Perform atlantooccipital cisternal puncture with an 18-gauge 8.3-cm needle as described earlier. Connect a short extension tube to the needle and slowly withdraw 10 mL CSF per 100 kg bodyweight (e.g., 50 mL in a 500-kg horse). Retain a sample of CSF for analysis. Next, inject the same volume of contrast material (iopamidol or iohexol) into the subarachnoid space at constant rate over 5 minutes. Withdraw the needle, then elevate the head and neck at approximately 30 degrees from the horizontal and allow another 5 minutes for caudad flow of contrast material to occur. Bring the head and neck back to the horizontal position and repeat the radiographs of the cervical spine. Next, tie the horse's head in position to exert flexion pressure focused on the middle of the neck. Radiograph C1 to C5. Retie the head so that the caudal part of the neck is

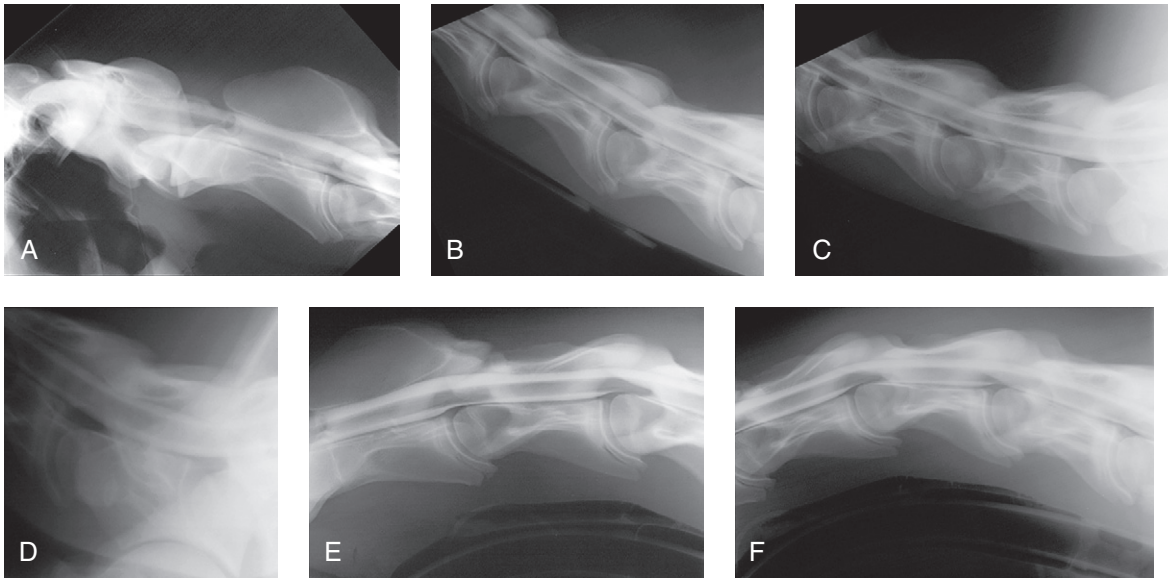

Figure 50-13. Cervical myelographic series in a normal horse. **A** to **C,** The neck is in a neutral position. Note that the transverse processes of C6 in this horse do not have the typical ventral extension. **D,** Now centered caudally, the neck is in extended position. **E** to **F,** Cranial and middle views of the neck in flexed position. (Courtesy Dr. Greg Roberts, University of Florida.)

hyperextended, and radiograph C5 to C7 (Figure 50-13). If indicated, position the horse in dorsal recumbency and take ventrodorsal views. Such views are possible from C1 to the cranial half of C6.

Neutral, flexed, and extended views are examined for evidence of dynamic or static vertebral canal stenosis or extradural compression by soft tissue. Typically, flexion reveals dynamic compression in the middle and cranial part of the neck, whereas extension views are best for dynamic compression in the caudal part of the neck. Criteria for interpretation of myelograms in cases of suspected cervical vertebral stenotic myelopathy have been reported and are described in Chapter 51.

NSAID therapy should be continued for 3 to 5 days after the procedure, and the horse should be closely observed and monitored during the first week. Fever and inappetence are common during this period if NSAIDs are not given, and they occasionally occur after NSAIDs are stopped. Seizures occur occasionally, usually within 24 hours of myelography. These are usually controlled with diazepam (0.05 to 0.1 mg/kg IV) and the horse is put on preventive antiseizure medication (e.g., phenobarbital, 5 mg/kg PO twice a day) for at least 7 days.

Computed Tomography

Computed tomography (CT) is a cross-sectional imaging method that provides additional diagnostic information in situations where survey radiography is unsatisfactory.[11] This is particularly true of the skull, where multiple soft-tissue and bony structures are superimposed on each other in two-dimensional radiographic images. Even in the cranial neck, where radiography is very useful, contrast-enhanced CT had better specificity for detection of spinal cord compression than did cervical myelography.[12] Compared to radiography, CT can discriminate much smaller differences in density (0.5% versus 10%). The gray matter of the brain can be distinguished from white matter, and acellular fluids can be distinguished from blood and pus.

The principles of CT have been reviewed (see Chapter 75).[11] In brief, CT images are produced in slices by an x-ray beam that rotates in a circular gantry around the object. The gantry and table carrying the patient move in precise relation to each other in a series of increments or slices. Slice thickness is typically 0.4 to 1 cm. As in radiographs, images are rendered in gray scale. Iodinated contrast medium can be administered to enhance vascular mass lesions and to better detect diseases that disrupt the blood–brain barrier. Intravenous doses for equine CT contrast enhancement have been reported.[13] Data can be reconfigured to produce images in alternative planes or in three dimensions.

CT-based studies of the anatomy of the head in adult horses and foals have been reported.[14,15] At present, the technique is usually performed under general anesthesia, although techniques and equipment for standing CT of the head are being developed. The skull and first four or five cervical vertebrae can be imaged in adult horses, whereas the entire body of the neonatal foal can be passed through the gantry (50 to 60 cm in diameter). The technique has been used to detect or evaluate temporohyoid osteoarthropathy, brain abscess, cholesterinic granuloma of the choroid plexus, fracture of the basisphenoid and calvaria, and guttural pouch disease.[16]

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) of the skull and spine is a cross-sectional imaging technique that is less widely available than CT (see Chapter 76).[11,16] The strong magnets (0.5 to 1.5 tesla) required for this type of imaging preclude the use of "standing" units designed for imaging distal limbs. Horses must be anesthetized and all metallic objects removed.

MRI provides a gray-scale map of the behavior of tissue protons in a strong magnetic field, and it yields cross-sectional slices in any plane. Data can be used to reconstruct images in three dimensions. T1-weighted images reveal fine anatomic

detail but lack tissue contrast. T2-weighted images are grainier than T1-weighted images, but they are more useful for detecting fluid and pathologic change. Additional contrast during T1 pulse sequence can be provided by injecting gadolinium.

Reference studies for the head have been published, and cases of intracranial neoplasia, abscesses, temporohyoid osteoarthropathy, hydrocephalus, and cholesterinic granuloma have been diagnosed.[16] MRI is generally regarded as superior to CT for soft tissue images, whereas CT is better for bone images.

Ultrasonography

The last 10 years has seen rapid development of techniques for ultrasonographic examination of the equine spine and its component intervertebral disks, dorsal intervertebral joints, sacroiliac joints, interspinous and supraspinous ligaments, segmental spinal nerves and blood vessels, and paraspinal muscles.[17-20] Most examinations are performed transcutaneously; caudal components of the spine are also imaged *per rectum*. The spinal cord, CSF, and meninges can be imaged ultrasonographically in cross-section at the atlanto-occipital and lumbosacral spaces.[21,22] Although the principal use of spinal ultrasonongraphy has been for strictly musculoskeletal abnormalities (see Chapter 68), several applications are suitable for neurologic examination and treatment and many more will be developed over the coming decade. The principal such application already in use is ultrasonongraphic guidance of needles for injection of corticosteroids into the arthritic cervical dorsal intervertebral joints of horses with limb ataxia or stiff necks.[23] Ultrasonography also is used to detect fractures or osteomyelitis of the vertebrae and skull, to facilitate CSF collection at AO and LS sites, and to guide injection of contrast for myelography at the AO site.[21,22]

Endoscopy of the Vertebral Canal

Direct examination of the epidural and, to a lesser extent, intrathecal spaces by endoscopy is an important tool for diagnosis and therapeutic intervention in human neurology.[24] Techniques for cervical epidural and intrathecal endoscopy of horses[25] and epiduroscopy of the sacrum in cattle[26] have recently been published and likely will have clinical application in equine specialty practices.

ELECTRODIAGNOSTIC TECHNIQUES

Electromyography

Needle electromyography (EMG) is the diagnostic recording of the electrical activity of muscle.[27] The EMG records spontaneous activity and response to needle insertion, voluntary or reflex muscle activity, and nerve stimulation. Abnormal results are consistent with muscle damage or motor denervation of at least 2 weeks' duration.[1] Use of the EMG allows objective mapping of the size and margins of involved muscle. This is discussed more fully in Chapter 87.

Electromyography requires needle electrodes, amplifier, cathode-ray oscilloscope, and speaker. Concentric and monopolar electrodes combine reference, ground, and exploring electrodes in different combinations. The examination can be performed standing, with or without sedation, or under general anesthesia. After the needle is pushed into muscle, insertional activity, motor unit action potentials (MUAPs) during muscle contraction, and abnormal spontaneous activity are measured. Normal and abnormal responses have been described for horses. Abnormal responses include fibrillations, fasciculation potentials, positive sharp waves, complex repetitive discharges, and myotonic discharges. Each of these has characteristic electrical profiles and sounds. Reference values for amplitude and duration of normal MUAPs in conscious horses have been published.[28,29] Values for horses with myopathies differed markedly from these normal values, and it is expected that values in denervated muscle would also be abnormal.

Nerve conduction studies combine nerve stimulation with EMG recording to evaluate conduction speeds in large motor nerves. These studies are performed under general anesthesia. Techniques have been described for the radial and median nerves in horses, and tibial nerve testing has been performed in horses with stringhalt.

Electroencephalography

Electrical activity arising from the cerebral cortex is recorded by subcutaneous electrodes inserted over the frontal/parietal area and displayed graphically on paper or captured for computerized analysis. Electroencephalography (EEG) is a useful tool for determining the presence, location, and extent of cerebral disease. Because of the expanded use of CT and MRI for evaluation of brain disease, the EEG is now used less frequently than it was 20 years ago.

Evoked Response Testing

The integrity of various parts of the central and peripheral nervous systems can be tested by providing a stimulus at one site and recording the electrical activity at another site. Techniques that have been used in horses include magnetic motor-evoked potentials, brain-stem auditory-evoked responses, and cortical somatosensory-evoked potentials.[30-32]

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CHAPTER 51

Developmental Vertebral Anomalies

Bonnie R. Rush

Cervical stenotic myelopathy (CSM) and occipitoatlantoaxial malformation (OAAM) are the most notable developmental abnormalities of the vertebral column of the horse. Other congenital vertebral anomalies, such as hemivertebrae, block vertebrae, and spina bifida, occur rarely in horses, with individual reports involving a small number of foals. The pathogenesis of vertebral malformations is largely unknown, but it is suspected to be multifactorial, including heredity, dietary imbalance, and *in utero* insult (trauma and toxin). After a brief description of normal vertebral development, the clinical presentation and pathogenesis of vertebral malformations and anomalies are described.

NORMAL VERTEBRAL DEVELOPMENT

The critical phase of vertebral development is during gastrulation (the formation of the three primary germ layers: ectoderm, mesoderm, and endoderm) and neurulation (the formation of the notochord, neural crest, and the precursors of the nervous system).[1] The spine has its embryologic origins in the cell that is induced to migrate out of the somite, toward the notochord and the neural tube. A mass of sclerotomal cells collect segmentally at the embryonic midline, surrounding the neural tube and the notochord, and these cells begin to separate into a cranial portion and a caudal portion. Failure of segmentation produces a block vertebra. The cranial portion of each sclerotome recombines with the caudal portion of the directly superior sclerotome in a resegmentation process known as *metameric shift*. Failure of recombination produces hemivertebrae.[1] After the metameric shift, spinal nerves, which originally left the neural tube to go to the center of the sclerotome, are able to pass between the precartilaginous vertebral bodies to innervate the segmentation myotomes.

The atlas and axis form by a mechanism that is different from that of the other vertebral bodies. Part of the first cervical sclerotome plus the cranial portion of the second cervical sclerotome contribute cells to form the odontoid process and the arch of the atlas.[2] In the cervical region, eight cervical somites generate only seven cervical vertebrae because the cranial portion of the first cervical sclerotome contributes to the formation of the occiput, and the caudal portion of the eighth cervical sclerotome contributes to T1. This is the process by which eight cervical spinal nerves become associated with seven cervical vertebrae.[1] Because the occiput, atlas, and axis are formed from shared sclerotomes, developmental abnormalities of these structures occur concomitantly, as in OAAM of Arabian foals. The first cervical spinal nerve passes between the base of the skull and the first cervical vertebra. The eighth cervical nerve exits below the seventh cervical vertebra and above the first thoracic vertebra. The remainder of the nerve roots exit below their corresponding vertebral bodies.

As the sclerotomes undergo shift to form the vertebral bodies, the adjacent visceral organs are differentiating, so that a noxious influence affecting development of a particular

vertebral segment may affect adjacent organs at the same time. Hence, cardiac anomalies may occur with congenital malformation of the thoracic spine, and renal anomalies may be associated with congenital malformation of the lumbar spine. During axial embryogenesis, interdependence exists between the spinal cord and the axial skeleton. If this balance is distorted by genetic, traumatic, toxic, or nutritional factors, a malformation may occur. The type of malformation depends on the stage of development of the nervous system at the time of insult.[3] Vertebral anomalies can be divided into three categories from the standpoint of basic developmental pathogenesis: malformation, disruption, and deformation.[4]

Malformation results from a failure of embryologic differentiation of a specific anatomic structure, causing it to be absent or improperly formed during the embryonic stage of gestation. Hemivertebrae, butterfly vertebrae, and block vertebrae are examples of malformation abnormalities. Once the malformed vertebra is anatomically established, the defect may continue to adversely affect spinal development throughout the subsequent fetal and postnatal periods. The eventual type of malformation and its severity depend on the stage of the developmental or maturation cycle that is specifically affected.

Disruption is a structural defect resulting from destruction of an anatomic part during the fetal stage of gestation that formed normally during the embryonic period. This mechanism involves distal structures (e.g., flexural limb deformity or wry nose) more frequently than the axial skeleton.

Deformation is defined as alteration in the shape or structure of an individual vertebra or vertebral segment that had initially differentiated normally, during the fetal and/or postnatal periods (e.g., cervical stenotic myelopathy). Deformation abnormalities are not considered true congenital abnormalities.

CERVICAL STENOTIC MYELOPATHY

Etiology

Cervical stenotic myelopathy (CSM) (cervical vertebral malformation, wobbler syndrome) is a common cause of spinal ataxia in young horses. This developmental disorder is characterized by postnatal deformation of the cervical vertebrae, resulting in stenosis of the vertebral canal and spinal cord compression.[5-7] The etiology of CSM appears to be multifactorial, and genetic predisposition and nutrition appear to play the most significant roles in the development of the disease.[7-9] Cervical stenotic myelopathy has been reported in most light and draft horse breeds; however, Thoroughbreds, Tennessee Walking Horses, and Warmblood breeds appear particularly predisposed, and male horses are more frequently affected than females.[10,11] The age of onset is typically 6 months to 3 years, although mature horses (4 to 20 years) are identified with acute onset of spinal cord compression due to cervical stenosis and arthropathy.[11] Affected horses often demonstrate rapid growth and are more likely to have developmental orthopedic disease of the appendicular skeleton than peers.[8]

Vertebral deformation associated with spinal cord compression includes flaring of the caudal epiphyses of the vertebral bodies, abnormal ossification of the articular facets, malalignment between adjacent vertebrae, osteoarthritis of the articular facets, and extension of the dorsal laminae.[12] In addition to these bony deformations, the vertebral canal is narrowed from C3 through C6, regardless of the specific site of spinal cord compression.[13] Spinal cord compression nearly always occurs in a dorsoventral plane, although lateral compression of the spinal cord has been demonstrated via computed tomography. Caudal epiphyseal flare occurs secondary to abnormal biomechanical forces on the vertebral growth plate because of subluxation and malalignment between adjacent vertebrae. Vertebral subluxation produces dynamic compression of the spinal cord during ventroflexion and is most commonly observed at C3-4, C4-5, and C5-6. Extension of the dorsal laminae contributes to spinal cord compression, because it ventrally displaces the roof of the vertebrae to produce a funnel shape, or "coning," of the vertebral canal.[12] Abnormal ossification of the articular facets often results from osteochondrosis of the joint surface, which quickly progresses to osteoarthritis.[12] Degenerative arthropathy of the articular processes occurs most frequently from C5 through C7 and is associated with spinal cord compression with the neck in the neutral (static) or hyperextended position.

Histopathologic evaluation of the spinal cord reveals necrosis of the white matter, with focal loss of neurons. Neuronal fiber (wallerian) degeneration is observed in ascending tracts cranial to the site of compression and descending tracts caudal to the site of compression.[6]

Clinical Signs

The clinical signs of CSM result from focal compression of the cervical spinal cord between C1 and C7. Compression may occur at a single vertebral site or at more than one site in the same horse. Horses with CSM demonstrate upper motor neuron deficits to all four limbs, characterized by symmetric weakness, ataxia, and spasticity.[3] At rest, affected horses may have a base-wide stance and demonstrate delayed responses to proprioceptive positioning. At a walk, weakness is manifested by stumbling and toe dragging; horses with prolonged clinical signs of CSM may have hooves or shoes that are chipped, worn, or squared at the toe. Ataxia (proprioceptive loss) appears as truncal sway at a walk and is manifested by circumduction and posting (pivoting on the inside limb) of the hind limbs during circling. Moderate to severely affected horses have lacerations on the heel bulbs ("wobbler heels") and medial aspect of their forelimbs from overreaching and interference. Spasticity, characterized by a stiff-legged gait and decreased joint flexion, is often observed in moderately affected horses. Clinical signs can be exaggerated during the neurologic examination by manipulation such as tight circling, walking on a slope, or walking with the animal's head elevated. When prompted to back, horses may stand base-wide, lean backward, and drag their forelimbs. In most instances, the rear limbs are more severely affected than the forelimbs; neuronal tracts to the rear limbs are more superficial and thus susceptible to external compression. Occasionally, forelimb ataxia may be more severe in horses with stenosis of the caudal cervical vertebrae (C6 through C7) because of compression of the cervical intumescence.

Asymmetric ataxia may be observed in horses with dorsolateral compression of the spinal cord by proliferative, degenerative articular processes; periarticular soft tissue proliferation; or a synovial cyst.[11] Rarely, clinical signs of nerve root compression are seen, such as cervical pain, atrophy of the cervical musculature, cutaneous hypalgesia, and hyporeflexia of cervical reflexes adjacent to the site of spinal cord compression.[14] These signs are more commonly observed in horses that are older than 4 years and that have moderate to severe arthropathy of the caudal

cervical vertebrae (C5 through C7), and they result from peripheral nerve compression by proliferative articular processes as the nerve root exits the vertebral canal through the intervertebral foramen.[5,11] In some instances, arthropathy of the caudal cervical vertebrae may produce cervical pain and forelimb lameness as a result of peripheral nerve compression, without producing clinical signs of spinal cord compression.[15] Affected horses typically travel with a short cranial phase of the stride and a low foot arc of one or both forelimbs, and they may stand or travel with their head and neck extended.

Diagnosis

The diagnosis of CSM requires collection of an accurate history, thorough physical and neurologic examinations, and radiographic evaluation of the cervical vertebrae.[7] The clinical signs of spinal cord compression often progress for a brief period and then stabilize.[5-7] Owners often report a traumatic incident with the onset of clinical signs of CSM. The traumatic incident may be the result of mild neurologic deficits, with the injury exacerbating the clinical signs of spinal cord compression. The following neurologic disorders should be considered in the differential diagnosis for cervical stenotic myelopathy: equine protozoal myeloencephalitis (EPM), equine degenerative myeloencephalopathy (EDM), equine herpesvirus myelitis, OAAM, spinal cord trauma, vertebral fracture, and vertebral abscess. Cerebrospinal fluid analysis may be performed during the diagnostic process, and it is usually unremarkable in horses with CSM. In CSM-affected horses with acute compression, mild xanthochromia or increases in protein concentration may be observed.

Assessment of plain film radiographs of the cervical vertebrae can determine the likelihood of CSM in horses with spinal ataxia.[13] Cervical radiographs should be evaluated for subjective assessment of vertebral deformation and objective determination of vertebral canal diameter. The five characteristic deformations of the cervical vertebrae in horses with CSM include flare of the caudal epiphysis of the vertebral body, abnormal ossification of the articular processes, subluxation between adjacent vertebrae, extension of the dorsal laminae, and osteoarthritis of the articular processes (Figures 51-1 and 51-2).[15,16] Osteoarthritis of the caudal cervical vertebrae is the most frequent and severe deformation observed in CSM-affected horses. Nonetheless, degenerative arthropathy occurs in 10% to 50% of nonataxic horses, and it is the most frequent and severe vertebral deformation in horses without CSM.[17] Therefore, subjective evaluation of osteoarthritis of the articular processes leads to a false-positive diagnosis of CSM. Although the presence of characteristic deformations supports the diagnosis of cervical stenotic myelopathy, subjective evaluation of cervical radiographs does not reliably discriminate between CSM-affected and unaffected horses.[18] Objective assessment of the vertebral canal diameter is more accurate than subjective evaluation of vertebral deformation for identifying CSM-affected horses.

The intravertebral sagittal ratio measurement was developed to objectively determine the vertebral canal diameter in horses.[12] The sensitivity and specificity of the sagittal ratio for identifying CSM-affected horses is approximately 89% for vertebral sites C4 through C7.[7] Generalized stenosis of the vertebral canal may be the most important factor in the development of CSM. The sagittal ratio is calculated by dividing the minimal sagittal diameter of the vertebral canal by the height of the vertebral body. The minimal sagittal diameter of the vertebral canal is obtained by determining the narrowest diameter measured from the dorsal aspect of the vertebral body to the ventral border of the dorsal laminae (see Figure 51-2). The vertebral body width is measured perpendicular to the vertebral canal at the widest point of the cranial aspect of the vertebral body. Because the vertebral body is located within the same anatomic plane as the vertebral canal, determination of this proportion negates the effects of magnification that result from variability in object-to-film distance. The sagittal ratio should exceed 52% from C4 through C6, and 56% at C7, in horses heavier than 320 kg.

Intervertebral measurements may provide additional information to identify horses with CSM based on plain film radiography.[19] The minimum intervertebral sagittal diameter ratio

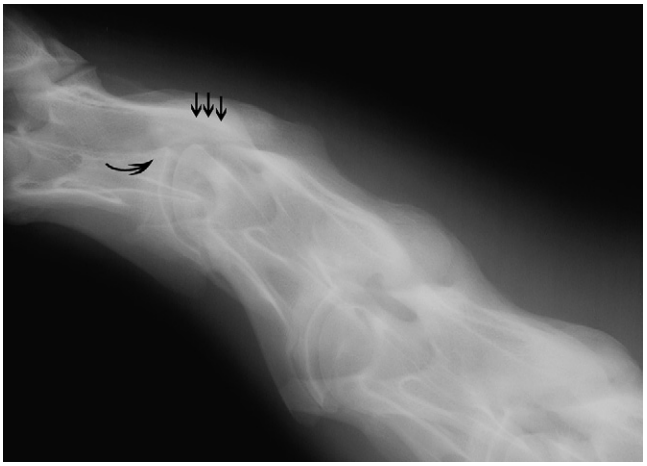

Figure 51-1. Lateromedial cervical radiograph of a 2-year-old Quarter Horse filly with spinal ataxia as a result of cervical stenotic myelopathy (CSM). Bony deformations consistent with CSM include flare of the caudal epiphysis *(curved arrow),* caudal extension of the C3 dorsal lamina *(arrows),* and malalignment of the C3-to-C4 articulation.

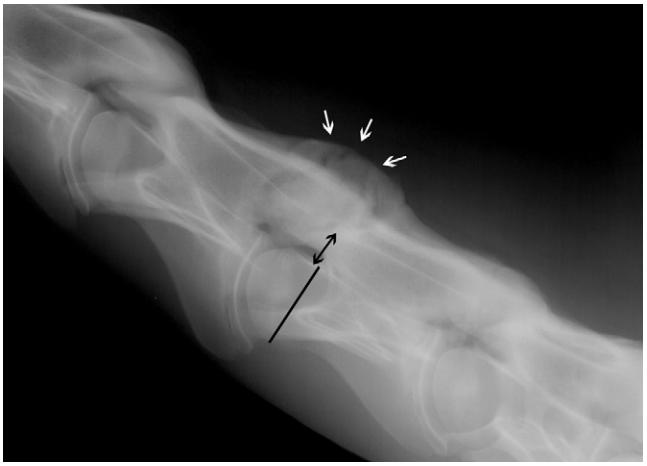

Figure 51-2. Lateromedial cervical radiograph of a 13-month-old Thoroughbred filly with spinal ataxia as a result of static spinal cord compression at C4-5. Severe osteoarthritis of the articular processes *(arrows)* is present at C4-5, and moderate osteoarthritis of the articular processes is seen at C5-6. The sagittal ratio is determined by dividing the intravertebral minimal sagittal diameter *(double arrow)* by the width of the vertebral body *(line),* as demonstrated using C5 in this radiograph.

is measured from the caudal aspect of the dorsal lamina of the vertebral arch of the more cranial vertebra to the dorsocranial aspect of the body of the more caudal vertebra, or from the caudal vertebral body of the more cranial vertebra to the cranial dorsal lamina of the vertebral arch of the more caudal vertebra, whichever is smaller. For vertebrae C2-3 to C5-6, the minimum diameter is typically based on the former measurement. Similar to the intravertebral sagittal ratio, the intravertebral measurement is divided by the width of the vertebral body. Horses with an intervertebral sagittal ratio of 0.485 or less should be classified as having CSM at that site. The diagnostic cutoff for intervertebral sagittal ratio parameter was based on evaluation of eight CSM-affected horses.

The clinician's goal for interpretation of radiographic projections of the cervical vertebrae is to classify the patient into one of the following categories:

- Low sagittal ratio (less than 48% at C4 through C6), moderate to severe bony deformation: perform myelographic examination to identify sites of spinal cord compression and classify lesions as static or dynamic.
- Marginal sagittal ratio (48% through 56%), mild to moderate bony deformation: perform myelographic examination to confirm or exclude CSM.
- High sagittal ratio (greater than 56%), minimal bony deformation: pursue alternative diagnoses.

Accurate assessment of cervical radiographs requires a precise lateral radiographic projection of the cervical vertebrae. Obliquity of the cervical vertebrae results in indistinct margins of the ventral aspect of the vertebral canal, producing erroneous values for minimum sagittal diameter (MSD) and vertebral body width. It is difficult to obtain precise lateral radiographs of the cervical vertebrae in recumbent horses. Plain film radiographs should be obtained in the standing sedated horse whenever possible.

Although plain film radiography can determine the likelihood of CSM, myelographic examination is indicated to confirm the diagnosis, identify lesion location and number of affected sites, and classify spinal cord compressive lesions. Spinal cord compression has been defined myelographically in three ways: (1) as a 50% or greater decrease in the sagittal diameter of the dorsal and ventral contrast columns, (2) as a 20% reduction in the dural diameter (DD), or (3) as a dorsal contrast column of less than 2 mm.[16,18,20] Methods that use a relative decrease in contrast column as diagnostic criteria (i.e., 1 and 2) compare the suspected site to the midvertebral site, cranial or caudal to the compressed site. The ventral column, however, is often obliterated at the intervertebral space in normal studies, particularly in the flexed position. A 20% reduction in DD is an effective diagnostic criterion for identifying CSM at C6-7.[20] Identification of spinal cord compression in the midcervical region is the most challenging. In the midcervical area, with the cervical spine in the neutral position, a 20% DD reduction has high specificity, but it does not detect all cases (low sensitivity). During hyperflexion, midcervical compression is easier to detect; however, false positive diagnosis is more common (low specificity). Most clinicians agree that the criterion of a less-than-2-mm dorsal contrast column is no longer recommended, because it is associated with unacceptable specificity.[20]

In addition to providing the definitive diagnosis of CSM, myelographic examination can differentiate between dynamic (type I) and static (type II) spinal cord compression.[18] Horses with dynamic spinal cord compression (type I) demonstrate narrowing of the dorsal and ventral contrast columns during ventroflexion of the neck, whereas spinal cord compression is not apparent with the neck in the neutral position (Figure 51-3). Dynamic spinal cord compression usually occurs in younger horses (less than 2 years old) and is associated with instability of the cervical vertebrae, particularly from C3 through C6. Dorsal laminar extension, caudal epiphyseal flare, and abnormal ossification patterns often occur in horses with dynamic spinal cord compression. Static vertebral canal stenosis (type II) is characterized by constant spinal cord compression, regardless of cervical position (Figure 51-4). Static compression usually occurs in slightly older horses (2 to 4 years) and results from osteoarthritis of the articular processes and proliferation of periarticular soft tissue structures. In some cases of caudal cervical arthropathy, flexion of the neck stretches the ligamentum

Figure 51-3. Myelographic examination of C2 through C4 of a 2-year-old Quarter Horse filly with dynamic spinal cord compression between the C3 and C5 articulations. The dorsal and ventral contrast columns are obliterated with the neck in the ventroflexed position. Spinal cord compression was not apparent with the neck in the neutral position (not shown).

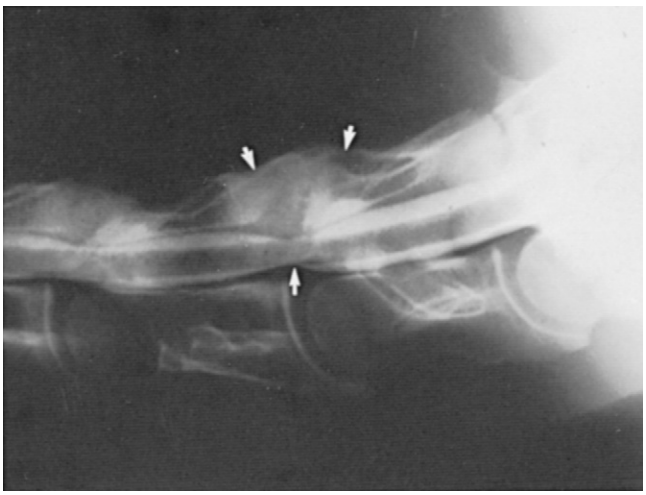

Figure 51-4. Osteoarthritis of the articular processes of C6-7 *(upper arrows)* producing static spinal cord compression *(lower arrow).*

flavum and relieves spinal cord compression, whereas extension exacerbates compression.

A complete myelographic study should include neutral and stressed (flexed and extended) views of the cervical vertebrae.[18] In horses with obvious sites of spinal cord compression on neutral myelographic views, excessive flexion and extension of the neck should be avoided while obtaining dynamic views to prevent exacerbation of spinal cord injury. Horses should be monitored for 24 hours after the myelographic procedure for depression, fever, seizure, and worsening in neurologic status. Worsening of the neurologic status after myelography may result from spinal cord trauma during hyperflexion, iatrogenic puncture of the spinal cord, or chemical meningitis. Administration of phenylbutazone (4.4 mg/kg PO every 24 hours) 1 day before and through 1 day after the myelographic examination will attenuate fever and depression associated with chemical meningitis.

Treatment

Nutritional Adjustments

Successful conservative management of CSM has been achieved using the "paced diet" program in foals younger than 1 year.[15] The goal of this dietary program is to retard bone growth, enhance bone metabolism, and allow the vertebral canal diameter to enlarge to relieve spinal cord compression. Serial radiographic examinations demonstrate improvement in the sagittal ratio; however, osteopenia may be observed. This dietary program is restricted in energy and protein (65% to 75% of National Research Council [NRC] recommendations) but maintains balanced vitamin and mineral intake (minimum, 100% of NRC recommendations). Vitamins A and E are provided at three times NRC recommendations, and selenium is supplemented to 0.3 ppm. Roughage is provided by pasture or low-quality (6% to 9% crude protein) Timothy hay. Dietary regimens are individually formulated according to the age and weight of the foal. Solitary stall confinement is recommended to minimize repetitive spinal cord compression because of dynamic instability. This program of dietary management and restricted exercise has been successful for preventing neurologic signs in foals with radiographic evidence of CSM and treatment of foals demonstrating clinical signs of CSM.[15]

Medical Management

Administration of glucocorticoids, dimethyl sulfoxide (DMSO), and nonsteroidal anti-inflammatory drugs (NSAIDs) may reduce edema and provide transient improvement in neurologic signs. Spontaneous recovery from CSM without dietary management or surgical intervention has not been reported.[21]

Horses with cervical pain and forelimb lameness as the result of cervical vertebral arthropathy may benefit from intra-articular administration of corticosteroids or chondroprotective agents, or both.[22] Arthrocentesis of the cervical vertebral articulations (facets) is performed under ultrasonographic guidance using a 15-cm (6-inch) 18-gauge spinal needle in the standing sedated or recumbent horse. The cranial facet of the caudal vertebra appears superficial to the caudal facet of the cranial vertebra. The articular space is accessed at the cranioventral opening of the articular facet, which is angled approximately 60 degrees from the ultrasound beam. The needle should be introduced 5 cm cranial to the facet and inserted at a 30-degree angle relative to the skin surface. Joint penetration should be confirmed by aspiration of synovial fluid. If the neck is extended, the transverse process of the cranial vertebra may obscure the path to the articulation. Intra-articular triamcinolone (6 mg per joint, not to exceed 18 mg total dose) or methylprednisolone (100 mg per joint) has produced a positive clinical response in more than 50% of horses with arthrosis of the articular processes. The goal of intra-articular anti-inflammatory therapy should be to decrease cervical pain or eliminate forelimb lameness. It is unlikely that intra-articular therapy will significantly improve clinical signs of spinal ataxia.

Surgical Management

Surgical treatment of CSM is discussed in detail in Chapter 52.

OCCIPITOATLANTOAXIAL MALFORMATION

Occipitoatlantoaxial malformation (OAAM) occurs most frequently in Arabian foals, although it has been reported in Morgan horses, Appaloosas, a Friesian, a Miniature Horse, and Thoroughbreds.[23-27] In Arabians, it is inherited in an autosomal recessive manner with no sex predilection.[23,24] In other breeds, a nongenetic congenital defect occurs at random during development of the vertebral column.[25-27]

In OAAM, the developmental error occurs during embryogenesis, before the end of the sixth week of gestation.[23-25] The atlas of affected foals is characterized by a small vertebral body, vertebral arches, and peglike transverse processes. The atlas is often fused to the occipital bone, resulting in synchondrosis or a barely detectable atlantooccipital joint (occipitalization of the atlas). The odontoid process of the axis (dens) is malformed and hypoplastic. In some cases, the axis may be luxated ventral to the atlas, leading to fracture of the dens. The fusion between the atlas and the occipital bones may be asymmetric, leading to the development of scoliosis.

Clinical Signs and Diagnosis

OAAM may be obvious at birth or detected within the first few weeks of life. Affected animals demonstrate marked weakness and ataxia when walking and may have an extended head and neck position. A clicking sound may be elicited by manipulation of the head, resulting from the dens slipping under the body of the atlas.[23-25] Clinical signs worsen over time as a result of repetitive or persistent compression of the spinal cord. The malformed vertebrae lead to the formation of excess fibrous tissue within the atlantoaxial joint and adjacent ligaments, which in turn creates additional compression of the spinal cord.

Pathologic changes of the nervous system associated with OAAM include a dorsoventral flattening of the medulla oblongata and cranial cervical spinal cord. Histolopathologic evaluation of affected neuronal tissue reveals gliosis, astrofibrosis, neuronal fiber degeneration, and perivascular fibrosis.[23]

Treatment

In Arabian foals, euthanasia is recommended because of the heritable nature of the condition. In other breeds, surgical intervention may be used to correct atlantoaxial subluxation and scoliosis. Young horses and foals with malformation of the

occiput, atlas, and axis should be managed conservatively if they do not demonstrate neurologic gait deficits.

ATLANTOAXIAL SUBLUXATION

Atlantoaxial subluxation (AAS) results from a developmental abnormality of the odontoid process of the axis (dens), such as malformation, absence, or separation from the axis. The excessive mobility of the atlantoaxial joint results in repetitive or constant spinal cord compression.[28] Congenital absence of the dens, the most extreme from of AAS, produces significant loss of stability between the atlas and the axis.

Clinical Signs and Diagnosis

The clinical signs of AAS include weakness, spasticity, and ataxia in all four limbs, consistent with focal compressive lesions of the cervical spinal cord.[28,29] Progression of signs is quite variable and depends on the degree of laxity between the axis and atlas. Clinical signs may slowly progress over weeks to months or may begin acutely, making spinal cord trauma important in the differential diagnosis. Severe compression may produce tetraplegia.

The diagnosis may be confirmed by evaluating clinical signs, manipulating and palpating the head and neck, and taking lateral radiographs of the skull and proximal cervical region. A lateral radiograph should be obtained with the horse standing, with little manipulation of the head and neck. Widening of the space between the dorsal portion of the atlas and the axis, and a rounded cranial end of the axis, indicate of an abnormally shaped or absent dens. A myelogram is normally not required for confirmation of the diagnosis.

The prognosis for AAS is poor. Surgical stabilization of the atlantoaxial joint may be attempted as a salvage procedure. Subtotal dorsal laminectomy of the impinging dorsal arch of the atlas has been described in foals with a developmentally malformed dens.[30,31] In addition, a ventral approach for stabilization of AAS resulting from a Salter-Harris type II fracture of the odontoid process has been described (see Chapter 53).[32,33] Medical therapy (glucocorticoids and hyperosmotic agents) should be administered before surgical correction to reduce spinal cord swelling and edema. Affected foals should be maintained in a quiet environment with minimal handling to avoid additional trauma to the spinal cord.

ATLANTOAXIAL INSTABILITY

Atlantoaxial instability produces clinical signs in older foals and young horses.[30] Subluxation of the atlas relative to the axis occurs during extension of the head and neck because of malalignment of the atlas, resulting in caudal ventral tilting of the atlas relative to the vertebral canal. The subluxation results from ligamentous laxity and malarticulation. A congenital anomaly has been suggested, although trauma may be an important pathophysiologic factor. This syndrome emphasizes the importance of evaluating this area when examining a young horse with clinical signs of spinal ataxia localized to the cervical region. Treatment is not recommended.

The differential diagnosis for OAAM, AAS, and atlantoaxial instability includes cervical trauma, vertebral osteomyelitis after neonatal septicemia, cervical vertebral stenotic myelopathy, and syringomyelia, a congenital malformation of the spinal cord.[2,3,34]

HEMIVERTEBRAE

Hemivertebrae are wedge-shaped vertebral bodies in which the vertebral body apex may point dorsally, ventrally, or medially, resulting in kyphosis, lordosis, or scoliosis.[2,3,35-39] Moderate to severe angulation of the spine may result in animals with hemivertebrae, and the severity of the malalignment typically worsens during growth as a result of compression by adjacent vertebrae. Hemivertebrae can result from a hemimetameric shift of somites during recombination of sclerotomes to form primordial vertebrae or from a defect of the vascular supply at this developmental stage that results in a failure of half of the vertebral body to ossify.[1,3] If metameric shift is abnormal, one right and one left hemivertebra will be adjacent to each other. The vertebrae resulting from an alteration in vascular supply may appear as unilateral, dorsal, or ventral hemivertebrae.

In horses, hemivertebrae have been observed at C5, although in most species T7 and T9 are most commonly affected.[36] Clinical signs of hemivertebrae in foals are observed before 1 year of age. Foals demonstrate progressive vertebral malalignment, leading to kyphosis or scoliosis, with or without neurologic gait deficits. Hemivertebrae are often weaker than normal vertebrae; a sudden jump or fall may result in acute worsening of clinical signs.

THORACIC VERTEBRAL MALFORMATION

Malformation and compression of the thoracic vertebrae has been described in two unrelated horses.[40] Both horses appeared normal for the first year of life, with ages of onset being 13 and 28 months. Clinical signs included acute-onset pelvic limb weakness and ataxia in one horse and severe spinal cord dysfunction and paralysis of the pelvic limbs in the other. Both horses demonstrated Schiff-Sherrington phenomena, characterized by hypertonicity of the thoracic limbs associated with focal thoracic spinal cord compression. This clinical sign has been described in humans and other animals with fracture of the thoracic vertebrae, although it is a rare finding in horses.[41] The malformation of the thoracic vertebrae was similar to malalignment and subluxation observed in horses with cervical stenotic myelopathy.

BLOCK VERTEBRAE

Block vertebrae are examples of improper somite segmentation and appear to be a result of incomplete separation of the vertebral bodies or arches, or both.[1] The length of a block vertebra is equivalent to the number of vertebral segments involved. Block vertebrae are generally stable and do not often result in clinical signs, although the rigid fixation may result in abnormal biomechanical forces on adjacent vertebrae. The sacrum is an example of a normal block vertebra. The most commonly reported location of block vertebrae in humans and animals is C2-3 (Figure 51-5).

BUTTERFLY VERTEBRAE

A butterfly vertebra is a rare anomaly of the equine vertebral column. The embryonic notochord is normally obliterated with the development of the vertebral bodies and remains only as the vestige of the intervertebral disc. If the notochord persists, a dorsal-to-ventral cleft results, which leads to vertebrae that

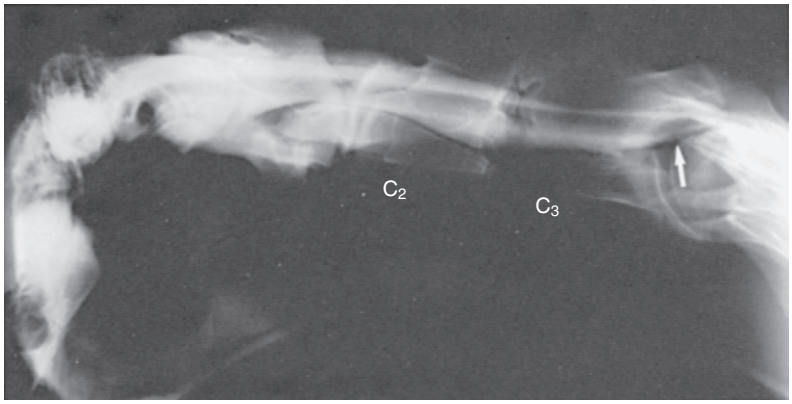

Figure 51-5. Block vertebrae involving C2-3. The block vertebrae are not associated with spinal cord compression, which is present at C3-4.

resemble a butterfly on a dorsoventral radiograph. Butterfly vertebrae typically do not cause spinal cord compression and do not require treatment, except in cases associated with meningeal cysts.[1,3] In one report, the butterfly vertebra was associated with a rare neurenteric cyst, which resulted in hindlimb ataxia, weakness, and frequent "dog-sitting."[42]

VERTEBRAL DEFECTS

Spina bifida results from incomplete fusion of the dorsal components of the vertebrae during the neurulation process. It is often associated with a protrusion of the meninges (meningomyelocele) through the vertebral defect. Spina bifida (C5 through C6) with an accompanying meningomyelocele has been identified in a Miniature Horse foal that was unable to stand at birth.[43] The meningomyelocele appeared as a 5-cm fluctuant mass of the dorsal aspect of the caudal cervical region. The colt also had hydrocephalus, which is present in the vast majority of children with meningomyelocele as part of the Arnold-Chiari malformation (protrusion of caudal cerebellar tissue through the foramen magnum with concomitant caudal displacement and elongation of the medulla oblongata, pons, and fourth ventricle).

Severe posterior ataxia as the result of a congenital cleft in the ventral aspect of a vertebral body (T5) has been described in an Arabian foal.[44] The foal had malformation and fusion of several thoracic vertebrae, with an intraspinal and extraspinal (intrathoracic) cyst. Therefore, a history of posterior paresis and ataxia present at birth should signal a need to perform a careful examination to search for malformation of the vertebral column in the thoracic region.

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CHAPTER
52 Surgical Treatment of Developmental Diseases of the Spinal Column

John Walmsley and Barrie D. Grant

Surgical treatment of developmental diseases of the spinal cord is focused on cervical vertebral stenotic myelopathy (CVSM). The goal of treatment is decompression of the spinal cord to allow healing of the compressive lesion and the return to full function. The etiology and diagnostic techniques have been discussed in Chapters 50 and 51, but comments on diagnostic techniques from a surgeon's perspective are appropriate here. Accurate diagnosis of the site of compression is fundamental to successful surgical treatment. A thorough clinical and neurologic examination should be performed, and if cervical spinal cord compression is suspected, survey radiographs of the neck are taken. These are evaluated for abnormalities, and the intervertebral and intravertebral ratios are measured (Figure 52-1).[1]

There are some caveats to note when performing this evaluation. Obvious abnormalities such as angulation or subluxation at C3-4 suggesting cervical spinal cord compression on plain films are not always confirmed as the site of compression on myelography. Osteochondrotic articular facets at C6-7 can be associated with spinal cord compression but they are also a common finding in horses without neurologic problems, and they may be present in horses with ataxia from another cause (e.g., protozoal myelitis) or from compression at another site. A longitudinal postmortem study of osseous changes in the cervical vertebrae of 87 nonataxic horses between 2 and 21 years old was performed by Whitwell.[2] Moderate to severe new bone formation both ventral to the cranial articular processes and on the caudal, lateral aspects of the caudal articular processes of C6-7 was recorded in 18% and 52% of horses, respectively. Equally there may be additional sites of compression not suspected on plain films, which can have a significant bearing on the feasibility of surgical intervention, the prognosis, and the eventual outcome.

The intravertebral and intervertebral ratios are useful (Figure 52-2), but they still require further validation. An intravertebral ratio of less than 0.5 was reported to increase the likelihood that cervical cord compression will be diagnosed by myelography at this site.[3] Hahn and colleagues measuring intervertebral and intravertebral ratios on eight horses with histological evidence of compression reported that these ratios were predictive of the site of compression.[4] Another study evaluating these ratios against myelographic results in 24 horses showed that the ratios were poor predictors of the site of compression but reasonably good predictors that the horse was likely to have

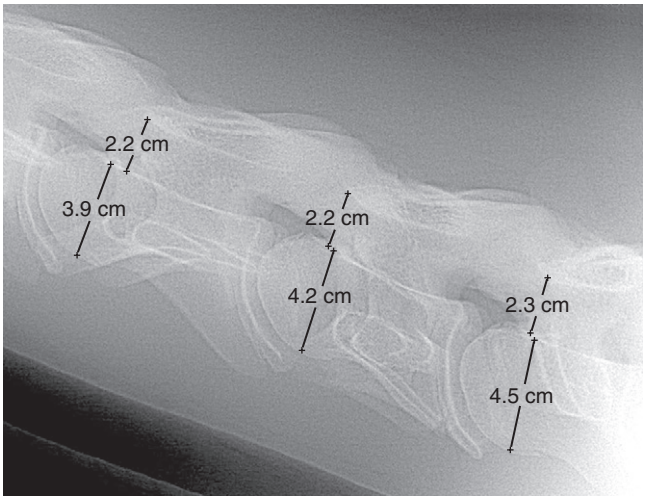

Figure 52-1. Survey radiographs of the cervical spine, showing normal measurements of vertebral body–to–canal ratios.

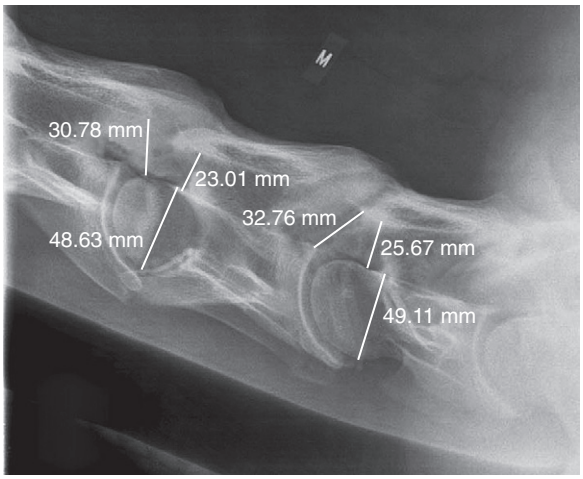

Figure 52-2. Survey radiographs of the cervical spine showing intervertebral and intravertebral measurements at C5-6 and C6-7.

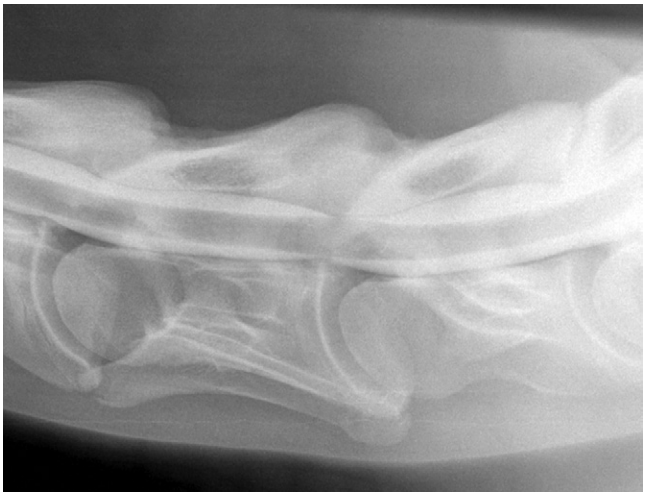

Figure 52-3. Myelogram showing severe cord compression at C6-7 in a 2-year-old Thoroughbred.

a cervical compressive lesion somewhere.[5] Sensitivity for the presence of compression but not specifically the site of compression was 42%, and the specificity was 79%. In six horses the site of compression was predicted but there were multiple false positives in each horse, and three horses had positive results on myelography but had normal ratios. With these comments in mind, it is essential to remember that successful treatment of localized cervical spinal cord compression depends on myelographic confirmation rather than interpretation of survey radiographs.

CASE SELECTION FOR SURGERY

Once cervical cord compression is confirmed, other factors considered important to the successful outcome of the case are (1) number of sites of cord compression, (2) whether the cord compression is static or dynamic, (3) severity of the clinical signs, (4) duration of clinical signs, and (5) the temperament, age, and intended use of the animal.[6] The supporting evidence for these parameters is incomplete but is discussed later.

Anecdotally, horses treated at multiple sites are considered to have a worse prognosis, although one study revealed no statistical difference in outcome between a group of 40 horses treated at one site and 22 at two sites.[7] One group described triple level treatment of 12 horses, out of which 9 improved two grades and 7 returned to ridden work; but all horses had a reduced lateral range of cervical motion postoperatively.[8]

Dynamic lesions (most commonly at C3-4) have been shown to carry a better prognosis after surgical treatment than static lesions at C6-7.[7,9] Another group reported return to use of 69% of horses with dynamic lesions and 59% with static lesions from a series of 45 horses.[10] From another series of 58 horses treated surgically, Walmsley reported that 11 out of 13 horses treated at C3-4 and 14 out of 27 horses treated at C6-7 returned to use, a statistically significant difference ($p = 0.035$, Fisher exact test).[9] The fatal complication rate in horses treated at C6-7 appears to be higher.[7]

The severity of clinical signs is thought to affect outcome because most successful cases improve up to two neurologic grades out of four (using the scale of grades 1 to 4 described by Mayhew and coworkers[11]), though Walmsley reported no statistically significant difference in outcome of surgery between 32 horses with neurologic grade of 1 or 2, and 15 horses with grade 3 or 4.[9] Some severely affected horses with short duration of clinical signs have returned to full use after treatment. Grant and colleagues have shown that the severity of myelographic compression has an impact on the outcome (Figure 52-3).[12] Horses with mild to moderate compression had a more favorable prognosis for future use than horses with severe compression. Only 50% of severely affected horses could be ridden after surgery, whereas 68% with moderate compression and almost 100% with mild myelographic compression were used in various riding disciplines after surgery.[12]

Duration of clinical signs was shorter for successful cases in the series of 63 described by Rush Moore and colleagues,[7] but Walmsley found no statistical effect of duration of signs on postoperative outcome in 58 cases.[9] Long duration of signs alone should not exclude horses from treatment.

The temperament, age, and intended use of the horse are subjective influences on recommendations for surgery. The temperament of the patient is important because there are personality changes associated with cord compression that are probably related to the anxiety of being ataxic. Animals may become intractable and sometimes self-destructive. Young animals that have had little handling (e.g., have never had their feet picked up and trimmed) before the onset of clinical signs are at great risk for traumatic complications resulting from the restraint needed during diagnostic testing, anesthetic induction, recovery, and postoperative treatment. Serious consideration should be given to delaying surgery for 4 or 5 days to accustom an unbroken adolescent horse to handling and restraint.

As for all elective surgery, procedures should not be performed on a patient heavily infested with parasites or suffering from a respiratory condition because these horses are more susceptible to pleuropneumonia from the stress of transportation and anesthesia. A thorough clinical examination, including hematologic tests if appropriate, is essential before embarking on myelography or surgery, and these should be postponed at any stage if adverse signs are noticed.

Because of the association of osteochondrosis with the pathogenesis of developmental cervical cord compression, it is important to ensure that other commonly involved joints (e.g.,

the stifle, tarsus, fetlock, shoulder) are free from significant pathology. If concurrent pathology exists, its significance should be carefully evaluated and its possible effects on the outcome discussed with the owner. Arthroscopic treatment of osteochondrosis lesions in the hock or stifle can be carried out concurrently with interbody fusion.

EXPECTATIONS OF SURGERY

It is important to understand the owner's hopes and expectations for the outcome of surgical intervention and to put them into the context of what is possible. Owner compliance and commitment before surgery is essential, and the owner must be a willing participant if surgery is to be undertaken. Owners must be completely informed of the risks, liabilities, and responsibilities of their decision. Their goals must be tempered with realism. Trainers and owners need to be patient because full recovery may take up to 1 year or more, and they will be required to condition the patient, using a slow, graded training program. In practice, the surgeon should be aware that some owners really hope to have a horse that will be able to compete at a high level, despite initially being emphatic that they will be satisfied with a pleasure horse or breeding animal as an outcome. However a long-term evaluation at 1 to 4 years after surgery did in fact reveal that as many as 90% of clients and referring veterinarians were satisfied with the outcome.[12] Owners should be informed that, if surgery renders an unsuccessful result, they may have a horse that has not recovered sufficiently to be handled safely or cannot be left in a large paddock without supervision. If the horse is to be used for breeding, a mare should be able to stand for service and a stallion should be able to cover a mare naturally with only moderate assistance.

Owners appreciate being informed of the chances of success based on available evidence. An analysis of long-term results of 126 horses that had undergone ventral interbody fusion using the Seattle Slew implant (see later) between 2000 and 2003 showed that 88% of the animals recovered at least one grade, 60% were ridden successfully, and 10% were used for breeding or were retired.[12] Another study revealed that 36 of 61 treated horses showed an improvement by two neurologic grades or a return to athletic use.[7] Sinha and associates documented a positive outcome in 28 of 45 horses (62%),[10] and Walmsley reported a return to full work, measured by return to expected use, in 31 out of 57 horses (53%).[9] Overall, the success rate of most surgeons is approximately 60%. As with any surgical procedure, owners should be warned of the possible complications (see later).

SURGICAL APPROACHES AND TECHNIQUES

Ventral Interbody Fusion

This surgical procedure requires a well-trained team and good facilities. Any delays caused by poor preparation are unacceptable and could increase the complication rate. The most frequently used technique today is a modification of Cloward's procedure developed for use in humans in 1958.[9,13-17] The modification uses a threaded or partially threaded titanium implant, the Kerf Cut cylinder (KCC), which has multiple holes and is open ended. The technique leaves an isthmus of bone in the center of the cylinder, the aim being to encourage faster fusion by increasing the blood supply to the graft material and to reduce the incidence of postoperative fracture and implant migration (Figure 52-4). Another technique using a ventral locking compression plate has been reported and is also described later.[18]

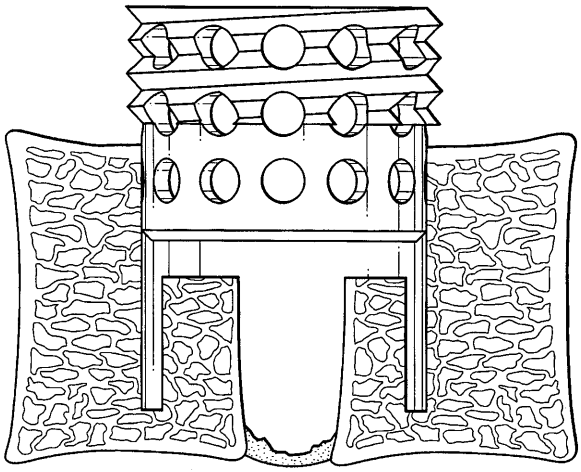

Figure 52-4. Drawing showing the Kerf Cut cylinder advancing into a prepared bone bed. Note the preserved peninsulas of bone, which remain inside the implant.

Preoperative Medication

Preoperative antibiotics consisting of procaine penicillin and gentamicin are administered to the animal before induction of anesthesia, after surgery and for the following 72 hours. Nonsteroidal anti-inflammatory drugs (NSAIDs) such as phenylbutazone or flunixin meglumine are administered at therapeutic levels before induction. They are continued for at least 7 days postoperatively or as long as necessary to reduce postoperative discomfort. Tetanus toxoid is administered to unvaccinated horses.

A preoperative endoscopic examination of the upper airways may be performed to detect laryngeal hemiplegia. The presence of this condition represents a risk for bilateral laryngeal paralysis should the right recurrent laryngeal nerve be damaged during surgery. The surgical approach should be made on the left side of the trachea under these circumstances (see later).

Anesthesia

Positive-pressure-assisted inhalation is the preferred method of maintaining anesthesia because it offers the best control while the surgeon is drilling close to the spinal cord. Common anesthetic problems include hypoxia, hypotension, and, if a facial artery is used, variable blood pressure readings. The latter is associated with the location of the surgical site; the right carotid artery can be compressed with retractors during the procedure, and we recommend using the left facial artery for blood pressure monitoring. Awareness of these possible complications allows early use of inotropic drugs. Frequent arterial blood gas analyses and high oxygen flow rates should enable the anesthetist to react appropriately.

Patient Positioning

The animal is placed in dorsal recumbency, with the neck in extension and the forelimbs tied in a moderately flexed position

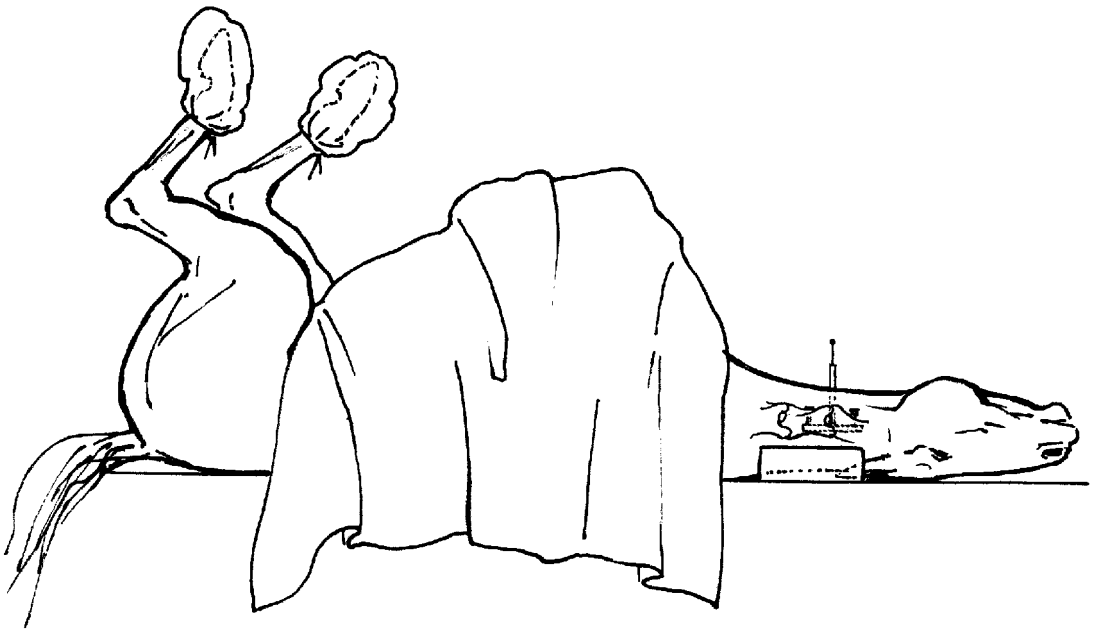

Figure 52-5. Schematic representation of a horse correctly positioned for surgery using a block of wood to extend the cervical articulations.

to allow adequate lateral stability without the excessive tension that could produce radial nerve dysfunction. A 30-cm block of wood or metal, which acts as a crutch for the neck, is placed under the affected area (Figure 52-5). This support assists the extension of the cervical articulations and at the same time stabilizes the neck, reducing excessive movement during drilling. It is very important to ensure that the neck is perfectly straight. At this stage the imaging system to be used (x-ray or image intensifier) should be set up to minimize time wastage during surgery.

Identification of the Surgical Site

Accurate identification of the affected cervical articulation is essential. The site can be determined by measuring one hand width for each vertebra retrograde from the easily palpable wings of the atlas (Figure 52-6). Markers such as 14-gauge needles or skin staples are then placed at the site and lateromedial radiographic projections are taken. The markers should be on the side of the neck closest to the x-ray cassette to prevent excessive distortion as a result of parallax, and they should be placed over the affected articulation as well as ventrally at the level of the incision. Alternatively, since C2 is clearly identifiable radiographically, markers are placed at several sites caudad from C2 and serial radiographs are taken identifying each articulation until the surgical site is reached (see Figure 52-11). Once the markers are verified to be at the correct level, aseptic preparation of the surgical site is completed and the entire horse is draped. The use of suction or electrocautery will reduce intraoperative time. Intraoperative imaging is a key part of the procedure, so the use of an image intensifier is recommended to prevent wasting time developing x-ray cassettes. If the latter are used it is vital to have the most efficient system possible for developing the images and returning them to the surgeon. Digital radiography is a very satisfactory alternative to an image intensifier, although it can be more difficult to identify the correct site because of the small cassette size.

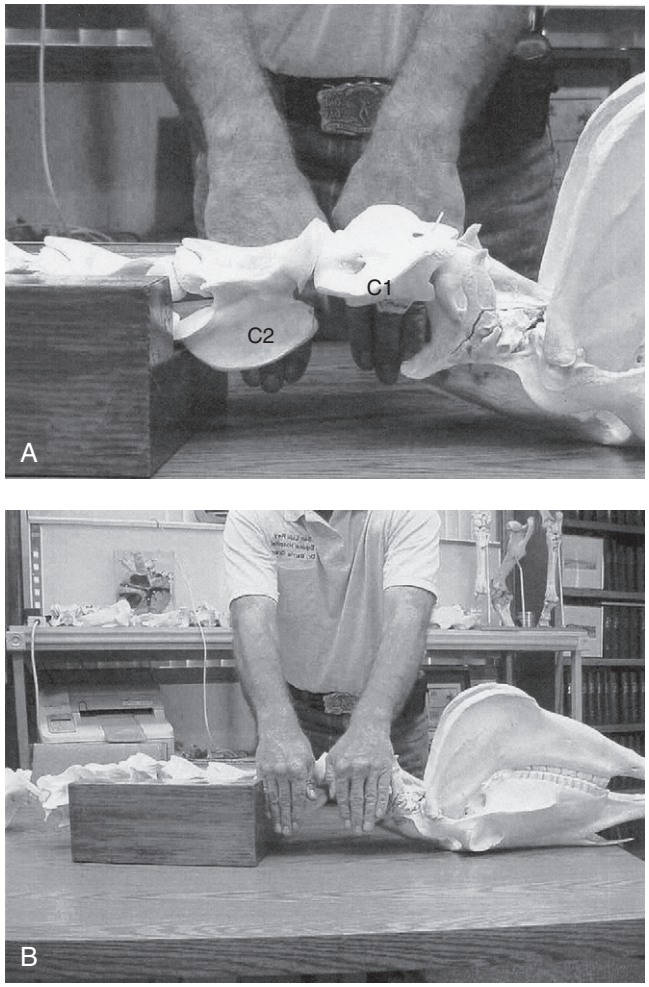

Figure 52-6. Demonstration on a horse skeleton of a technique for identifying the affected articulation. Using the wing of the atlas *(C1)* as a palpable landmark, each vertebral body length is determined to be about one hand-width. **A,** Hands placed behind the vertebrae. **B,** Hands placed in front.

Surgical Technique

THE KERF CUT CYLINDER

Dedicated instrumentation is necessary for implanting the KCC. The right-handed surgeon stands on the horse's left side. For a single-level fusion, a 20-cm longitudinal skin incision is made in the sagittal plane centered over the preplaced markers. A towel clamp placed at this level serves as a landmark and obviates the need for frequent palpation of markers through the drapes. The cutaneous musculature is incised with a scalpel blade or scissors, and hemorrhage is controlled as necessary. The sternothyroideus muscles are separated longitudinally to expose the trachea. Once the trachea has been identified, blunt dissection is continued dorsad down the right side of the trachea, separating it from the carotid artery and vagosympathetic trunk. Hand-held Deaver retractors can be used to retract the trachea to the left to expose the longus colli muscles. To reduce the risk of right-sided laryngeal hemiplegia postoperatively, the carotid artery and vagosympathetic trunk should be protected throughout the surgical procedure and should not be excessively retracted. The ventral spine (only rudimentary at C6-7) of the vertebral body cranial to the articulation to be treated can now be identified and dissected clear of its muscle attachments. Self-retaining Inge retractors are applied to gain access to the ventral spine and adjacent vertebral body for a curved osteotome, with which the ventral spine is removed. This creates a triangular platform on which to place the drill guide at the level of the ventral aspect of the vertebral body of the vertebra. Any hemorrhage is controlled with suction and sponges. The physeal line of the caudal aspect of the vertebra cranial to the articulation can be seen at the caudal edge of the platform in young horses. Since the intervertebral articular surface of the vertebral body is curved cranially, drilling starts just cranial to the ventral margin of the articulation so that the drill will equally bestride the articulation when it reaches a depth of 25 mm.

At this point, Grant proceeds as follows: A 16-mm drill guide is placed on the platform with its caudal surface on the physeal scar. Using a 16-mm drill bit, a test hole is prepared to a depth of 10 mm. For the sake of accuracy, a powered drill is essential. After the drill guide and drill are removed, it is usually easy to see the characteristic drab-white appearance of the intervertebral disc material. Two or three K-wires are gently tapped into the cranial and caudal aspects of the disc on the midline, using the spine of the anterior vertebra as a guide. An intraoperative image is then obtained to establish which K-wire is correctly placed so that the implant will be centered between the two vertebrae and to reconfirm that these are the correct vertebrae. The large centering pin is then placed over the chosen K-wire, and the drill guide is placed over the centering pin and hammered securely into the vertebra as close to perpendicular to the spinal canal as possible (Figure 52-7). This is used to guide subsequent drilling.

Walmsley prefers not to use the K-wires but places the 25-mm drill guide on the platform on the ventral surface of the vertebral body using radiographic control. Lines are drawn on the radiograph to ensure that the drill guide is placed so that the drill bit will equally bestride the articulation at a depth of 25 mm as described earlier (Figure 52-8). The 16-mm drill bit is then advanced to a depth of 10 mm to debulk the bone before using the 25-mm drill.

In both methods the implant site is now prepared by drilling with a series of drills starting with a solid 25-mm drill bit to a depth of 15 mm. If this is not done, the island of bone inside the implant may be too large, rendering it vulnerable to fracturing when reaming is performed with the Kerf Cutter later in the procedure. The drilling is continued with a narrow-core saw followed by a thicker-core saw (Figure 52-9), using radiography and depth measurements to monitor the direction and depth of the drill bit. Drilling should be performed only by one surgeon whose responsibility is to focus on the depth. The drilling depth should be 25 to 30 mm but must not be less than 10 mm from the spinal canal. It has been our experience that this shelf of bone should be left between the bottom of the drill hole and the spinal canal to provide overall strength to the articulation during the recovery process. Once the correct depth has been reached, the drill guide is removed and the implant site is initially widened with a No. 1 Kerf Cutter followed by the slightly larger No. 2 Kerf Cutter (Figure 52-10). The implant site is now tapped with the dedicated tap that matches the threads of the KCC implant. The implant is then inserted until firm resistance is encountered (Figure 52-11). A partially threaded implant is preferred, since this is less likely to cross-thread and drill a separate channel if the leading nonthreaded edge is toggled into place and gently twisted until it engages the already created thread cuts. The fully threaded implant is used in situations where a partially threaded implant fails to tighten. Care must be exercised because the self-tapping design of the fully threaded implant can permit the implant to be twisted into the spinal canal. A Hall drill or spoon curette is used to remove as much intervertebral disc material as possible to provide a bleeding surface of bone and encourage rapid acceptance of the bone graft. During the drilling an assistant collects bone from

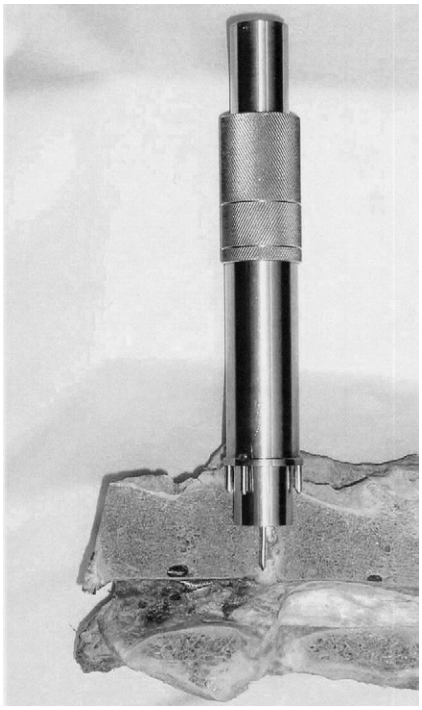

Figure 52-7. A cadaver specimen showing correct positioning of a solid cylinder placed over the guide pin, with a four-pronged drill guide placed over the cylinder. The guide pin and cylinder are removed after the drill guide is tapped into place. From this point the drill guide acts as the guide for the drilling processes.

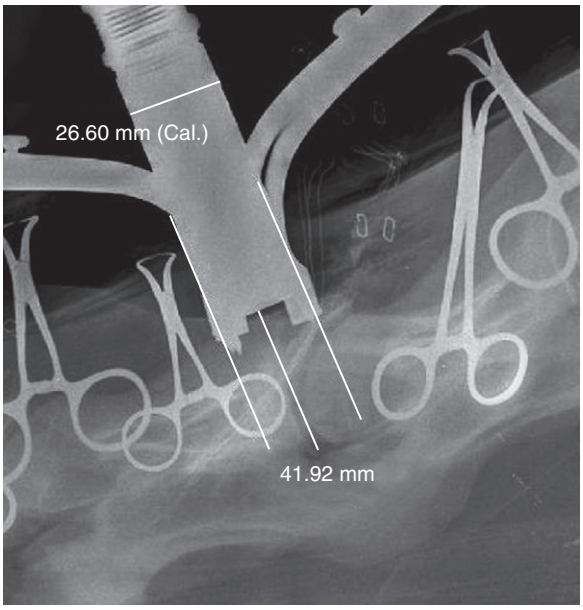

Figure 52-8. Intraoperative radiograph of drill guide in place with lines drawn on radiograph to confirm correct positioning. The distance to the spinal cord is also measured for the surgeon's reference.

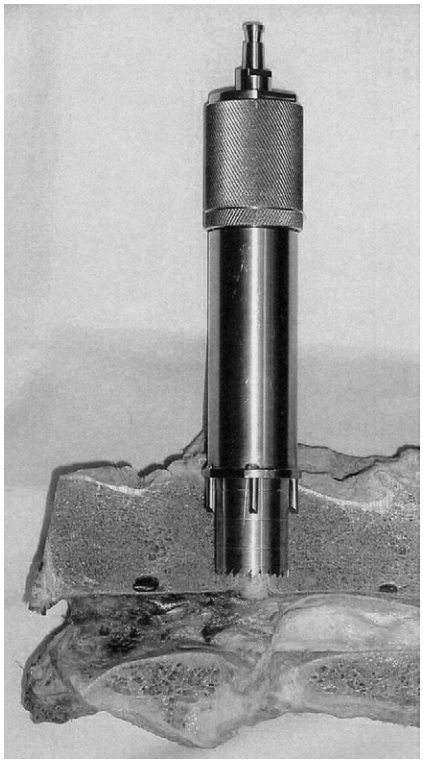

Figure 52-9. A cadaveric specimen showing the thin Kerf Cut saw advanced to the proper depth.

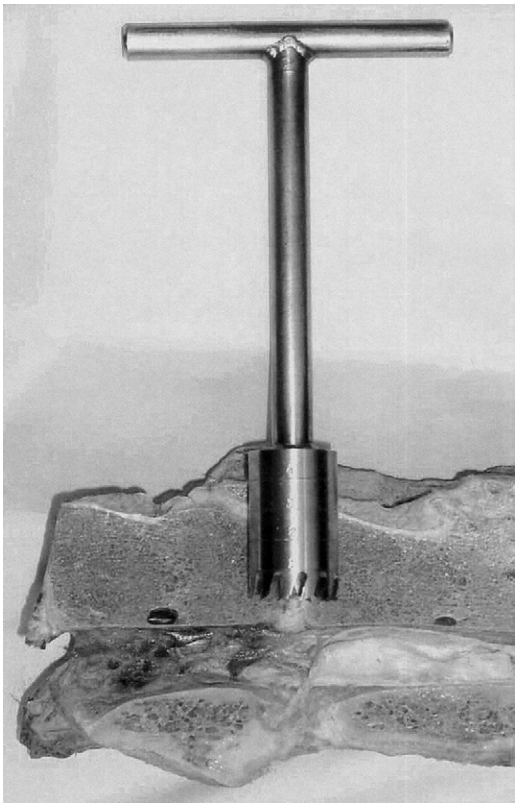

Figure 52-10. A cadaveric specimen showing the manual Kerf Cut widener and cleaner, which is used after the thin- and thick-cut saws.

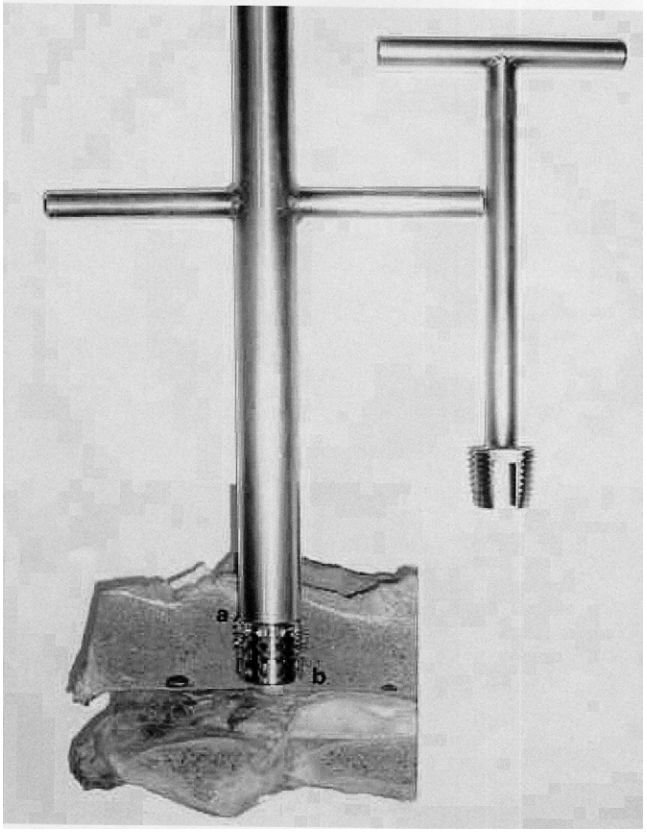

Figure 52-11. A cadaveric specimen showing the Kerf Cut cylinder (Seattle Slew implant) seated correctly and making complete contact with both the cranial *(a)* and caudal *(b)* vertebrae. The driver is still in place *(left).* The tap is shown *(right).*

the ventral spine and the drilling fragments to prepare a bone graft by dividing the dark red cancellous bone into small pieces and removing disc material and periosteum. The graft material should be covered with a blood-soaked sponge for optimal survival of the osteoblasts until it is required (see Chapter 77). Saline should not be used because its osmotic pressure may disrupt the osteoblasts. The graft is placed firmly inside the implant. A final radiograph is taken to confirm correct placement of the implant and for use as a reference before recovery from anesthesia (Figure 52-12). For a multilevel fusion, the whole procedure is repeated at each location.

After liberal flushing of the entire surgical area and a careful check for foreign materials (e.g., surgical sponges), the self-retaining retractors are removed. The longus colli muscle is closed with 0 polydioxanone suture (PDS) in a two-layer mattress pattern to reduce dead space. The remaining ventral cervical muscles are closed with 0 PDS in a simple-continuous or Ford interlocking pattern (the surgeon's choice). 2-0 PDS is used on the subcutaneous tissue and staples are used on the skin. Application of drains is not advised because they increase the risk of postoperative infection. In more than 600 cases, the cosmetic appearance of the surgical area has been very satisfying 4 to 6 weeks after surgery, without the need for aspiration or drainage of any seroma.[19] A sterile stent bandage can be sutured over the incision for recovery and replaced by a sterile dressing when the horse has returned to its stable; or the site can be recovered with an adhesive dressing protecting the incision. The use of a full neck bandage is a matter of the surgeon's choice.

LOCKING COMPRESSION PLATE

The use of a locking compression plate (LCP) for ventral fixation of the affected cervical articulation has been described.[18,20] Although the technique has not been thoroughly evaluated clinically, an *in vitro* study compared fixation using a KCC with fixation using a standard 4.5/5.0 broad 8-hole LCP at C4-5.[20] Testing the construct to failure showed a higher stiffness, moment to yield, and moment to failure with the LCP. Further evaluation is required, but the method may ultimately prove to be a useful alternative that does not require specialist equipment.

The first case reported was a 3-month-old Warmblood filly foal with a grade 3 neurologic deficit of several weeks' duration.[18] The dissection to the ventral aspect of C6-7 was as described for the KCC. The ventral surfaces of the vertebrae were flattened with an osteotome and as much intervertebral disc material as possible was removed using a spoon curette. The LCP was attached with five 5.0-mm self-tapping locking head screws placed under radiographic control. Because three screws pulled out during recovery, the surgery was revised on the following day using the longest possible screws and placing the central cortical screw across the intercentral articulation (Figure 52-13). The filly improved by 2.5 neurologic grades over 30 months. The second case report described fixation of C5-6 in a 2-year-old Standardbred colt showing grade 2 ataxia.[21] A 7-hole broad LCP with 5-mm self-tapping locking screws was used. The horse developed a seroma that was treated and resolved, and there was slight pullout of one screw. Seven months postoperatively the horse was in full training.

Recovery

A smooth recovery is important for a successful outcome in these patients. Some of these ataxic horses are unbroken, have had little handling, and will have difficulty standing up under the best of circumstances. Violent recoveries are associated with migration of the implants and, as with most equine surgeries, anything that anesthetists and surgical personnel can do to promote quiet, prolonged recoveries is beneficial. Hand recovery of cases with a tail and head rope is preferred by many surgeons. The head rope must not be overused because of the possibility that cervical traction will cause cervical fracture at the implant site. The head rope should be handled by an experienced person who has respect for the dynamics of the surgery.

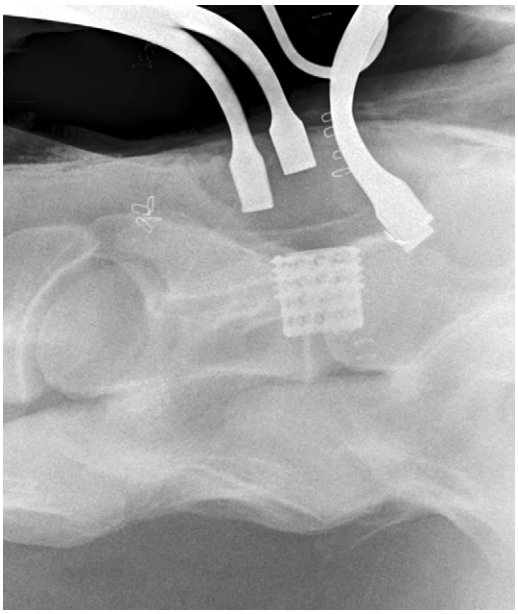

Figure 52-12. Intraoperative radiograph confirming correct placement of implant. Note the rows of skin staples used preoperatively to locate the affected articulation.

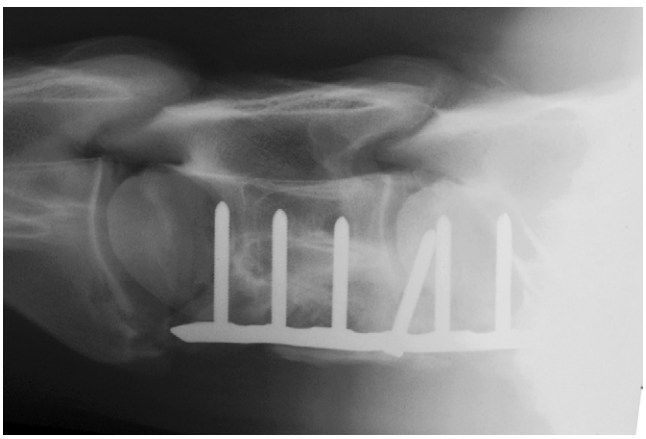

Figure 52-13. An x-ray of a locking compression plate implanted on the ventral surface of C6-7 in a Warmblood yearling taken 10 months after revision surgery.

An extended period of recovery in lateral recumbency (40 to 90 minutes) is usually beneficial. A combination of butorphanol and acepromazine in the early phase of the recovery to keep the horse comfortable and calm has proved to be an effective protocol. The onset of the effect of acepromazine is slow compared with that of xylazine, so early administration is important. It has the advantage of inducing less-severe ataxia than is produced by xylazine, though very small doses of xylazine may be more effective in controlling excessive early movements. Steroids can be useful if the patient's ataxia is greater than grade 3. Sling recoveries are sometimes necessary, especially for the patient with severe ataxia (grade >3.5). Placing these patients in the sling for 15 to 20 minutes a day for 1 to 2 days before surgery greatly facilitates the effectiveness of its use postoperatively. If a sling was required for recovery from the myelogram, it will probably be required after surgery. Pool recovery may be contraindicated since it requires extension of the head to avoid drowning, and this could dislodge the implant.

If there is a problem with stability and ataxia on standing, a second dose of NSAIDs can be administered at this time. The patient should not return to the stall until the surgeon deems the animal to be stable enough to do so. The floor leading to the stable should be dry and not slippery.

Postoperative Management

Penicillin and gentamicin are administered for 3 days postoperatively. Flunixin meglumine is usually given intravenously for 3 days to reduce swelling and postoperative discomfort. Administration of NSAIDs may be continued for a further 4 to 7 days depending on the clinical signs.

Most horses have good mobility and a good appetite postoperatively. Hay and feed should be presented at shoulder level to prevent unnecessary strain on the neck that would be caused by stretching to the floor. The animal should be managed in the stall in a quiet, gentle manner to prevent sudden movements or falls. If the animal objects to intramuscular injections, it can be medicated intravenously through an indwelling catheter or orally depending on the medication. The bandage may be left in place for the first 5 to 6 days postoperatively. It can be completely removed or replaced by a similar bandage if an early good cosmetic appearance is desired. Walmsley prefers not to bandage but to protect the incision with adhesive sterile dressings. The first 7 to 10 days after surgery are the most critical, and during this time the horse should not be walked out.

Postoperative radiographs should be taken 4 to 5 days after surgery to evaluate any change in position of the implant. If at any stage there is an increase in swelling, discomfort, or ataxia, radiographs should be taken immediately and the position of the implant compared with the immediate postoperative image. A change in position may be associated with fracturing of the implant bed, and even though any obvious fracture lines are not appreciated, this is often the cause of the increase in clinical signs. If the horse remains standing, fusion can occur with a satisfactory result; but if the patient is recumbent and cannot achieve sternal recumbency on its own, the prognosis is unfavorable.

At least 45 days of stall confinement is recommended to protect the implant, and the calorie intake is reduced. After 45 days, hand-walking can commence. To assess progress at 60 days, a follow-up radiographic and neurologic examination should be performed, if possible by the same surgeon to maintain continuity. An early study showed that the fusion process is well under way at 60 days, and postoperative myelograms are likely to have normal dye columns by this time, even on patients with static compression at C6-7.[19] Fusion can be substantially completed by 1 year (Figure 52-14). The owner and trainer should be warned that it may take at least 1 year before the clinical signs resolve and that a carefully executed rehabilitation program is of utmost importance for a good result. Exercise will assist in retraining the spinal tracts. After 60 days the horse should be turned out for at least 6 months or until clinical signs resolve. During this time some in-hand exercise may be beneficial for animals with grade 2 or greater ataxia. Horses with grade 1 ataxia can proceed to lunging, depending on progress.

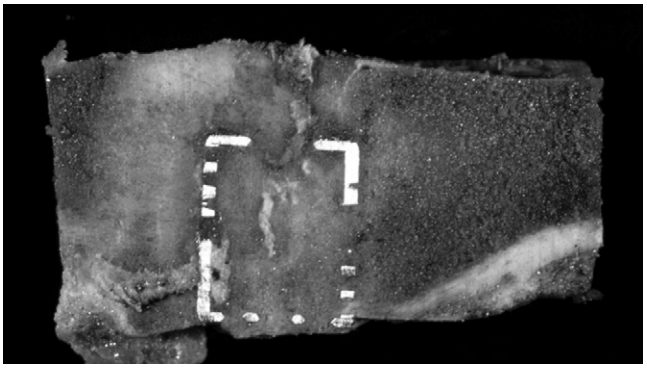

Figure 52-14. Postmortem specimen at 1 year after implantation of a Cloward Bagby Basket at C3-4 showing fusion of the vertebrae.

Complications

SEROMA FORMATION

Seroma formation has been reported in 4 of 126 cases in one study[12] and 9 of 63 cases in another,[3] but excessive seroma formation is rare. In our experience the development of postoperative seroma is usually associated with multiple-level fusions and older patients with heavier ventral cervical muscles that require more extensive dissection and retraction. It is preferable to avoid the use of prophylactic drains, since these could increase the chance of infection and most seromas resolve with time and without treatment. If the seroma develops into an abscess, open drainage will be necessary to avoid seeding the implant with bacteria. Effective control of hemorrhage during surgery, accurate tissue dissection, and closure of tissue layers will reduce the incidence of seroma formation.

INFECTION

Grant reports a relatively low incidence of postoperative infection of the soft tissues or the implant (less than 1%). Soft tissue infection usually induces a febrile response with a firm, painful enlargement at the surgical site, leukocytosis, and hyperfibrinogenemia. Treatment consists of drainage (best guided ultrasonographically), administration of appropriate antibiotics, and hot packing. Infection of the implant produces the same clinical response, but also the patient is noticeably unwilling to move the cervical area and may show worsening neurologic signs. In

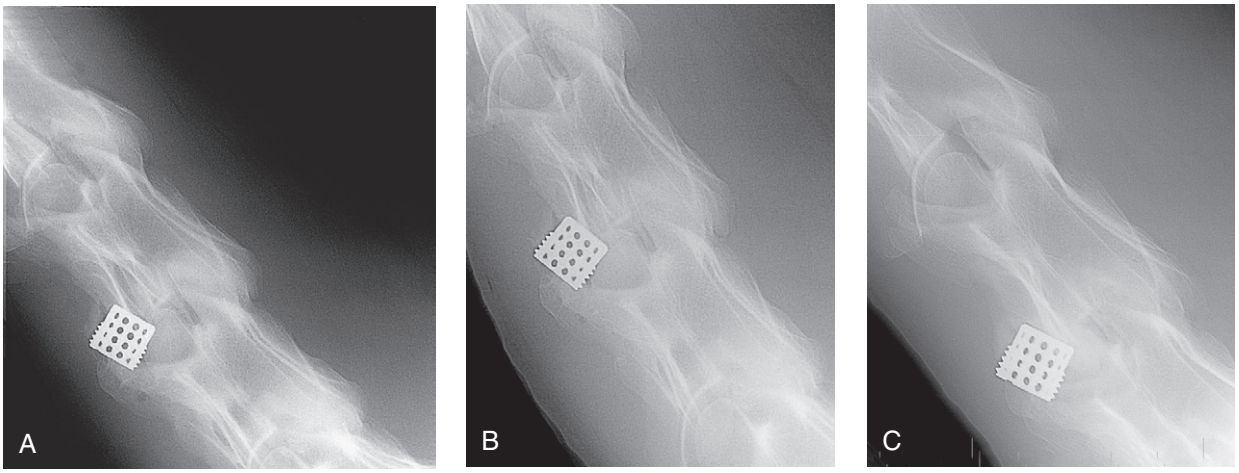

Figure 52-15. A, Cervical radiograph taken 1 day after surgery. This horse experienced a fracture cranial to the implant. **B,** The same fracture 1 week after surgery. **C,** The fracture healed after 8 weeks of stall rest.

one case of suppurative meningitis (from infection with *Klebsiella*), a postoperative myelogram taken when the patient was tetraparetic demonstrated a uniform narrowing of both dye columns of the entire cervical area. The signs resolved after retrieval of the implant and application of methyl methacrylate bone cement impregnated with cephalosporin antibiotic powder.

FRACTURES

Fracture of the adjacent vertebral body is a serious, often fatal complication that in most cases occurs during recovery (Figure 52-15). A fracture should be suspected if the horse shows a worsening of neurologic signs or appears to have excessive neck pain. In these cases radiographs should be taken. Conservative treatment is indicated because revision surgery or surgery to repair the additional fractures carries a high risk. Some horses recover, but if the horse becomes unacceptably ataxic or is unable to stand, euthanasia is usually indicated.

One study reported 3 fatal postoperative fractures in 63 horses.[7] From a series of 17 cases treated at the articulations between C5 and T1, two nonfatal fractures were recorded by Nixon.[22] There is some evidence that surgical treatment at C6-7 carries a higher complication rate.[7,22] In a review of 275 patients, only 3 had catastrophic fractures that resulted in euthanasia.[19] All the fractures occurred at C6-7 and the patients were all older than 15 years and had degenerate discs (narrowed and yellow) at the time of surgery. Two animals had violent recoveries. Walmsley reported 5 fractures in 27 horses treated at C6-7 compared with no fractures in 16 cases treated at C3-4, but the difference was not statistically significant.[9] However, these data showed a statistical trend for a lower chance of fracture when the KCC was used rather than the Cloward Bagby Basket (CBB).[9] Grant compared 69 horses treated with the CBB against 75 horses treated with the KCC and showed a significantly lower incidence of fracture with the KCC.[19]

OTHER COMPLICATIONS

Other complications associated with this procedure include ventral migration of the implant (Figure 52-16), laryngeal hemiplegia (LH), and Horner syndrome. Ventral migration was found on postoperative radiographs of 3.5% of patients and tended to occur within the first 7 days after surgery; but there is evidence that the risk of ventral migration is significantly less when the KCC is used.[17] The incidence of laryngeal hemiplegia appears to lessen with experience and has been variously reported in four studies as 1 out of 63,[7] 6 out of 75,[23] 3 out of 72,[24] and 3 out of 58.[9] Other fatal complications recorded include esophageal rupture and laryngospasm.

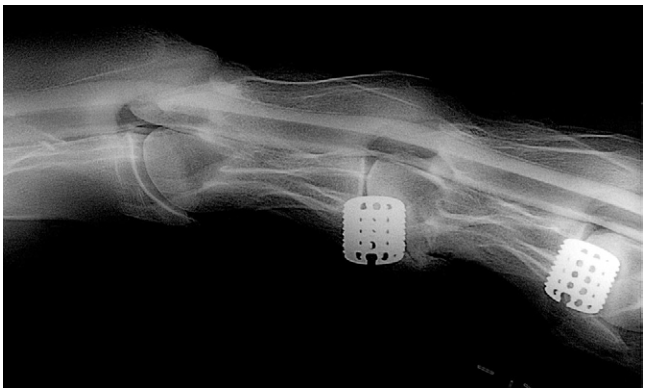

Figure 52-16. Cervical radiograph showing extrusion of the cranial implant, which occurred with a Cloward Bagby Basket implant (same patient as shown in Figure 52-15).

Because the use of the LCP to achieve ventral stabilization has yet to stand the test of clinical experience, the complication rate is unknown. One report of the use of a 9-hole 4.5/5.0 narrow LCP for stabilization of a dynamic stenosis at the C3-4 level demonstrated a serious potential complication.[25] The rostral part of the plate was not in contact with the bone at the completion of the procedure (Figure 52-17, *A*). Several months later the horse showed worsening ataxia; radiographs showed that the rostral plate was now in contact with C3 and the rostral locking head screws had penetrated the spinal cord (Figure 52-17, *B* and *C*). It was presumed that the normal neck movement had gradually forced the plate dorsad and with it the locking head screws. To prevent such complications, accurate contact between the plate and the bone is essential in this procedure.

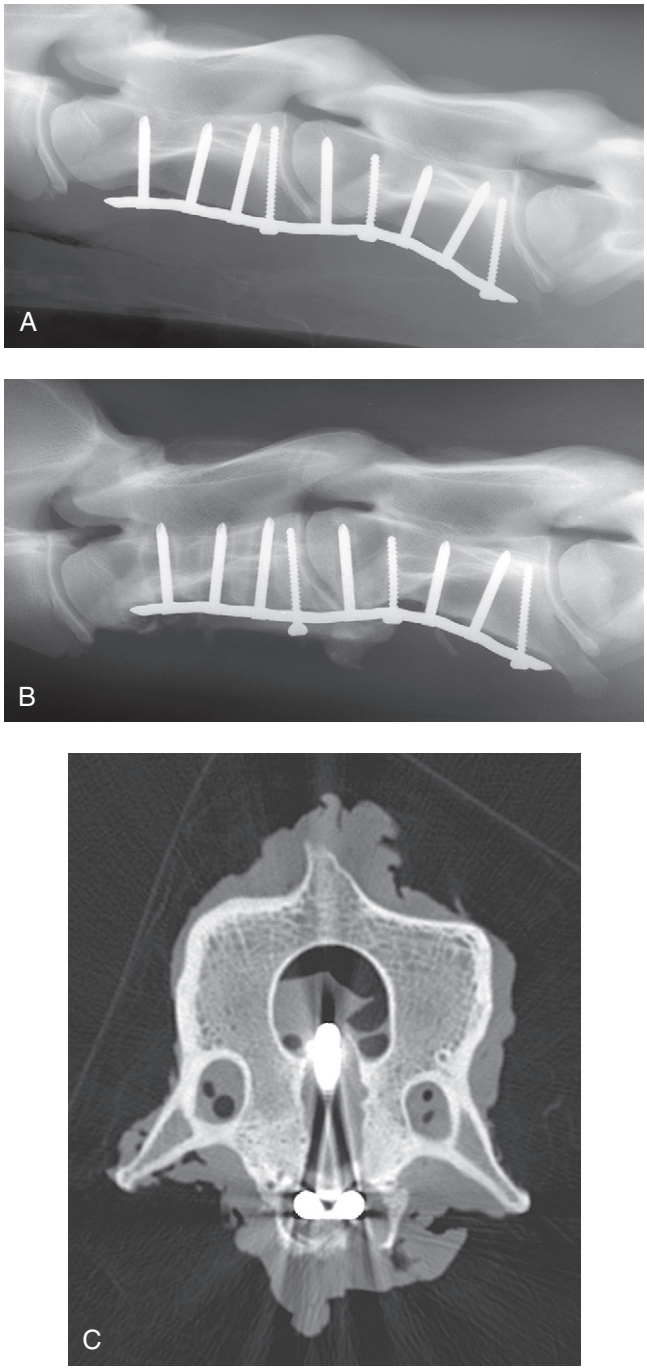

Figure 52-17. A, A 9-hole narrow locking plate was applied to stabilize the two vertebrae in neutral position; note the gap between the rostral aspect of the plate and the vertebra. **B,** A 9-month follow-up radiograph showing penetration of the two most rostral locking head screws into the vertebral canal with traumatization of the spinal cord. The rostral four locking head screws are loose and hold only in the plate hole; a halo can be seen around them. The gap at the rostral end of the plate is closed. **C,** Computed tomography cross-sectional image at the level of the most rostral locking head screw. The screw perforates the vertebral canal and protrudes into the spinal cord. (Courtesy A. Fürst, Zurich, Switzerland.)

Subtotal Dorsal Decompression Laminectomy

Indications

Subtotal dorsal decompression laminectomy, in which the dorsal caudal aspect of the cranial vertebra and the dorsal cranial aspect of the caudal vertebra are removed, is indicated in cases of intramedullary or extramedullary enlargement resulting in cord compression. These include meningeal infiltration of metastatic malignant melanomas, presence of excessive synovial villi from the osteoarthritic facets, and hypertrophic dorsal laminae.[26] Abscesses, parasitic granulomas, and intramedullary neoplasms may also be approached with this method if the diagnosis is reliable (Figure 52-18).

Subtotal dorsal decompression laminectomy is not indicated for the removal of dorsal arches that result from fractures or for the removal of dorsal arches of ankylosed hyperflexed joints that can occur at C2-3. There is even some question as to whether it is indicated for the treatment of a hypertrophic dorsal longitudinal ligament or a disc protrusion into the ventral aspect of the spinal canal.

Surgical Positioning

Once the decision has been made to perform a subtotal dorsal decompression, positioning of the patient is the most important factor contributing to the success or failure of the procedure. A number of surgical positions have been tried, including (1) placing the animal in ventral recumbency with all limbs flexed on a table and extending the head, (2) "hanging" the horse from the vertical surgical table under general anesthesia, and (3) placing the patient in a sling, inducing general anesthesia, and resting the sternum on the operating table. Currently, the third method and a lateral recumbency approach with the neck in full flexion are the most widely used.[27,28] The choice of position usually rests with the surgeon. Only experienced surgeons should attempt this surgery, and cadaver practice is essential before embarking on a clinical case.

Preoperative Preparation

Uneventful induction and recovery is facilitated, especially in younger animals, if 2 or 3 days before surgery the animal can be accustomed to the use of a sling. The usual standard preoperative preventive medicine, including administration of anthelmintic medication and vaccination against upper respiratory infections, is strongly recommended to reduce the incidence of postoperative complications. On the day before surgery the mane is clipped and the animal is given a bath with a medicated shampoo. In addition, scrubbing of the dorsal neck surface with a bacteriostatic detergent is indicated. The perioperative use of NSAIDs and antibiotics is strongly recommended. The use of a depilatory agent 1 or 2 hours before surgery further aids in the removal of small hairs in the many creases of the dorsal aspect of the cervical arch. Some animals show an inflammatory response to the depilatory agents, so the agent should be selected with caution.

Surgical Technique

After the animal is placed in the position favored by the surgeon, a large area on the dorsal surface of the neck extending from T4-5 to C2-3 is disinfected and draped. A long incision (30 to

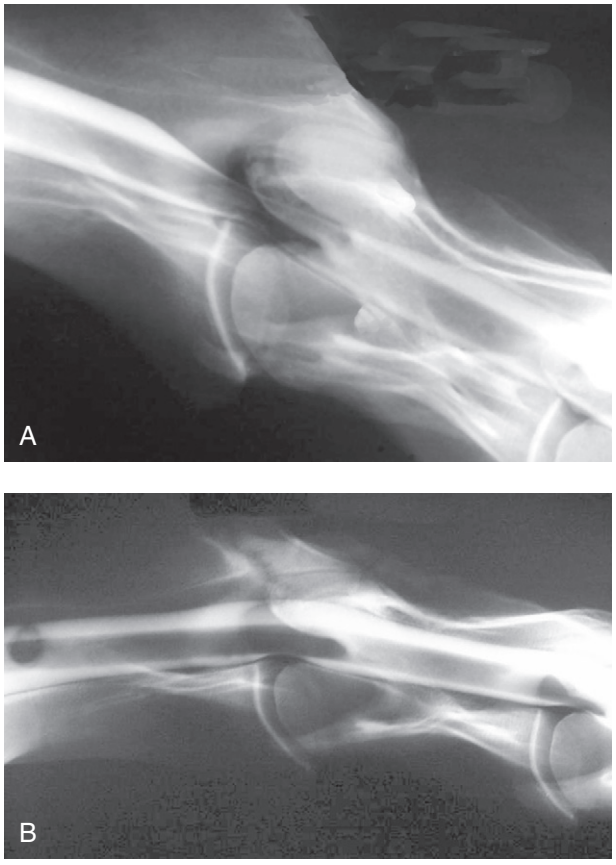

Figure 52-18. A, Myelogram of a 6-year-old Thoroughbred hunter that was falling during competition and was grade 2 (out of 5) ataxic. **B,** A 6-month follow-up myelogram performed after a subtotal dorsal decompression to remove a space-occupying mass later identified as a lymphoma.

40 cm) is made through the thick skin in the dorsal midline. After the initial and often profuse hemorrhage is controlled, the skin edges are draped. Although it is not always necessary, application of the towels and towel clamps helps reduce any further capillary hemorrhage. With a fresh scalpel blade, the subcutaneous tissue and fat overlying the funicular part of the ligamentum nuchae is sharply incised, exposing the dense white fibrous tissue of the ligament. Sometimes three or four small linear incisions are required to find the separation between the lamellar halves of the ligamentum nuchae, but as soon as this division is found the two halves can be separated by blunt manual dissection. The incision needs to be of considerable length because of the depth of the field and the inelastic quality of the ligamentum nuchae. One or two sets of large, self-retaining retractors of the Finochietto or Balfour type facilitate retraction in this area.

At this point it should be possible to palpate the small spinal processes on the dorsal aspect of the vertebra. The location of the dorsal decompression at C6-7 is facilitated by palpating the 3- to 4-cm dorsal spinous process that is often present on the first thoracic vertebra. Radiographic markers (needles) applied before draping in the manner previously described help confirm the proper location. The overlying epaxial muscles and soft tissues are removed from the dorsal arch with the combined use of periosteal elevators, large curettes, and rongeurs. The use of scalpel blades in this area should be avoided because incising the many small blood vessels produces hemorrhage that is often difficult to control, obscures vision, and increases surgical time. After the soft tissue is dissected from the vertebral arches, the white cortical bone is removed using a combination of a Hall drill, cranial drills, and rongeurs.

The original description advocated insertion of Gigli wire between one dorsal foramen and the next foramen. Grant reports that horses with stenotic hypertrophic vertebrae often have no discernible dorsal foramina because of encroachment by the hypertrophic bone, so attempts to pass a Gigli wire as described is not recommended. It is important to remember that the majority of these compressive lesions are the result of proliferation of dorsal bone, which is often much thicker than on normal specimens. Removal of this proliferative bone increases the surgical time, but it is important to be patient and careful. A rapid, forceful puncture through the dorsal arch can result in cord trauma that will exacerbate the already existing neurologic signs. A recommended technique is to identify the remains of any dorsal foramen and work craniad and caudad from this aspect with a Hall or cranial drill. After the inner cortical layer of bone appears, reverse-cutting rongeurs can be used to remove the dorsal arch while protecting the cord. Proliferative or hypertrophic soft tissue arising from osteoarthritic articular facets can be removed with rongeurs and long scissors.

The appearance of epidural fat over the bright, shiny dura indicates that decompression of the spinal cord has been completed. Removal of the bone over the affected site must be adequate, especially on the caudal aspect. If cord compression was caused by a metastatic melanoma, all the discolored tissue should be removed. Following removal of the bone and smoothing with a Hall drill, bone wax is applied to the bleeding edges of bone to reduce the amount of hemorrhage and the risk of clot formation around the spinal cord.

After careful inspection of the surgical site, all sponges and instruments are removed and a drain is secured into the tissues directly overlying the decompression site. The drain should exit through the lateral aspects of the cervical muscles. The drain is secured to the soft tissue surrounding the laminectomy with a 2-0 absorbable suture so that it can be retrieved with gentle traction after 4 to 5 days.

When the self-retaining retractors are removed, the laminar portions of the ligamentum nuchae fall together and need to be apposed only on the most superficial aspect with a continuous Ford interlocking pattern using absorbable monofilament suture material. The funicular portion of the ligamentum nuchae, the subcutaneous tissue, and skin are all apposed using absorbable sutures in a continuous Ford interlocking pattern. Finally, stent bandages are applied to protect the incision from excessive trauma that could occur during a violent recovery. Firmly applying bandages around the entire neck is discouraged because this produces pressure that is directly transmitted to the exposed cord and can result in noticeable discomfort to the patient and increased ataxia.

Postoperative Management and Aftercare

Immediate postoperative management consists of antibiotics for 36 hours or longer, depending on surgical time and the possibility of breaks of aseptic technique during the procedure. Because the procedure destabilizes the dorsal articulation of the vertebrae, fracture is one of the more frequent complications.

The continued use of NSAIDs appears to reduce the chance of a sudden fall, which may cause a fracture. The drain site should be continually bandaged or protected with antiseptic swabs and the drain left in place until there is a noticeable reduction in the amount of fluid obtained, usually 24 to 36 hours postoperatively. Hand-walking and resumption of physical therapy can begin within 10 days; the judgment of the surgeon or neurologist is the most important factor in determining this time.

Subtotal dorsal decompression laminectomy is not widely used because of its degree of difficulty, the amount of surgical time involved, surgical expertise required to perform it efficiently, and the significant incidence of postoperative complications, such as articular facet fractures, compressive hematomas, and suppurative meningitis.[27,29]

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CHAPTER 53

Traumatic Disorders of the Spinal Column

James T. Robertson and Valerie F. Samii

Trauma to the vertebral column in horses occurs throughout the cervical and thoracolumbar regions, and the reported incidence varies.[1-3] Vertebral fractures may be present with or without neurologic signs, depending on their location, the portion of the vertebra that sustains the trauma, and the degree and rate of compression of the spinal cord.[4] Confirming a diagnosis of spinal trauma is complicated by the substantial variation in clinical signs. Foals are more susceptible to vertebral fractures than adults, and the cervical vertebrae are more likely to be affected. The most common injuries in foals involve luxations, subluxations, and physeal separations of the cervical vertebrae.[2,3] In adult horses, the injuries tend to involve both the cervical and the thoracolumbar regions.[3,5,6]

Causes of spinal trauma include rearing and falling over backward, collisions with immovable objects, kicks from other horses, hyperflexion or hyperextension of the neck when falling, slipping on wet or muddy footing, and violently sitting backward on the ground or against solid objects.[1-3] Injury to the

spinal cord can occur without radiographic evidence of osseous damage, making the history of possible trauma important for arriving at a diagnosis.

This chapter describes the traumatic spinal injuries that are amenable to surgical correction, although in the horse the number is limited for many reasons. The size of the horse presents unique technical challenges from a diagnostic and surgical standpoint, and it is difficult to maintain vertebral fracture fixation during the anesthetic recovery and rehabilitation period. Innovation is required during the surgical repair to achieve success, and there certainly are not many case reports in the literature from which to draw information when formulating a surgical plan.

TRAUMATIC INJURIES TO THE CERVICAL SPINE

Fractures of the Axial Dens with Atlantoaxial Subluxation

This injury is most commonly seen in foals younger than 6 months, although it can occur in the adult.[3,7-10] The dens or odontoid process originates embryologically from the body of the first cervical vertebra and is attached to the axis by a physis. This physis closes by 8 to 12 months of age. Fracture occurs when a foal falls with the neck in hyperflexion (somersaulting) or when the fall is violent, as in halter-breaking incidents. The fracture most often occurs through the cranial physis of the axis, separating the dens from the body of the vertebrae (Figure 53-1). The dens remains attached by its ligaments to the atlas, and the axis moves ventrally. This results in cord compression at the atlantoaxial joint.

The degree of neurologic deficit depends on the degree and force of the spinal impact at the time of the injury. Neurologic deficits range from a stiff gait and splinted neck to total tetraparesis or even sudden death.[3,7-9] In most cases, the foal becomes ambulatory almost immediately, with signs of neck splinting, ataxia, and tetraparesis developing gradually. Manipulation of the neck produces pain as well as palpable and audible crepitation. There may be an obvious axial malalignment in the atlantoaxial area.[3] Lateral radiographs with the horse under sedation confirm the separation (widening) of the cranial physis of the axis and cranioventral luxation of that vertebra.[3,7-9] If ventrodorsal radiographs can be obtained, asymmetry in the atlantoaxial articulation may be noted.

The prognosis for recovery from this type of injury can be roughly correlated with the severity of the spinal cord damage. If there is motor function of all four limbs, with only paresis rather than paralysis, the prognosis for survival is favorable. If there is paralysis but the foal still shows conscious pain responses in all four limbs, the prognosis is guarded. If there is no conscious pain response, the prognosis is grave.

The method of treatment varies depending on the stability of the fracture and, to some extent, on the intended use of the horse. Nonsurgical management includes stall rest and the judicious use of anti-inflammatory drugs. Worsening of signs may become apparent if the foal falls and further displacement of the axis relative to the atlas results. With time, a fibrous union of the dens to the axis may develop and the neurologic deficits will decrease. However, late-developing neurologic deterioration in a patient that showed initial improvement indicates that a proliferative bony callus or soft tissue hypertrophy is compressing the cord.[3]

Surgical repair is warranted if the foal's clinical signs deteriorate, preferably before the foal becomes recumbent, or if it is a valuable foal intended for athletic competition. Although the prognosis is guarded to poor if the foal is presented in recumbency, it may be improved with surgery. The goal of surgery is to provide decompression at the fracture site by realigning the vertebrae and providing stability with some form of fixation. Surgical techniques for repair of fracture-luxations include external fixation using Steinmann pins, compression plating, ventral atlantoaxial fusion, and dorsal laminectomy of the caudal atlas.[3,7-9]

One report described the placement of two 6-hole, broad dynamic compression plates (DCPs) on the ventral surface of the atlas and axis.[7] The approach and exposure for this technique are the same as those described for ventral interbody fusion (see Chapter 52). After exposing the ventral surfaces of the atlas and axis, the alignment of the vertebrae is corrected and maintained with bone-holding forceps. The ventral spinous processes are removed using rongeurs to create a smooth bed for the two plates. One plate is placed on each side of the midline and anchored using short 4.5-mm cortex screws.

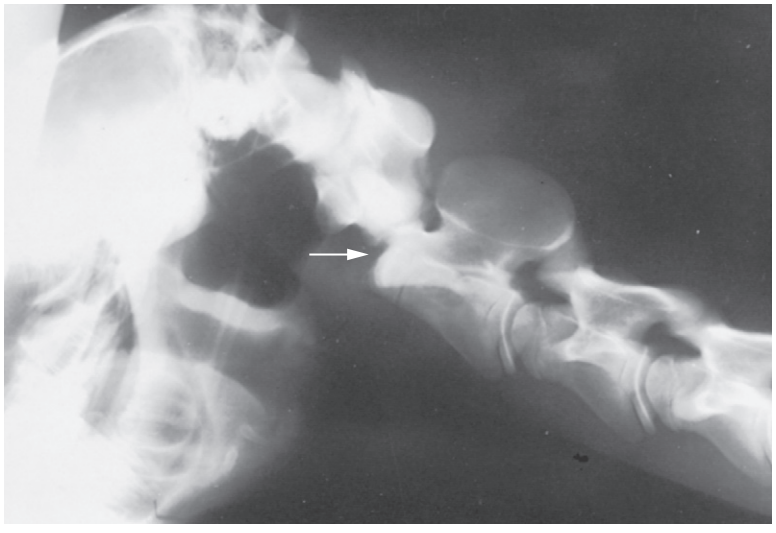

Figure 53-1. Fracture of the dens, which occurs through the cranial physis of C2.

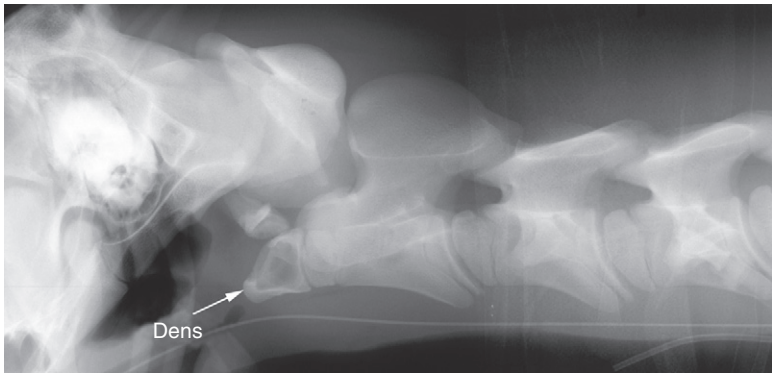

Figure 53-2. Lateral radiograph of the cranial cervical spine in a 1-week-old foal. There is luxation of C2 ventrally, relative to C1. The odontoid process *(dens)* is abnormally angled in a ventral direction. The body of C1 is rotated relative to the caudal cervical spine.

Another report described a ventral stabilization of the atlantoaxial junction by inserting two 6.5-mm cancellous screws across the articulation after removal of cartilage from the articular surfaces. Removal of the fractured dens before stabilization is optional. Care must be taken during drilling of the screw holes in the vertebral arches of the atlas to avoid the spinal canal axially and the vertebral arteries and first cervical nerves abaxially. A bone graft is placed in the articulation to promote fusion, and a polyvinylidine plate is applied to the ventral surface of the atlas and axis to provide additional stability.[3]

In the first of these two reports, ataxia was no longer apparent at 12 months after the surgery and radiography showed fusion of C1 to C2. One complication of surgery was right-sided laryngeal hemiplegia that did not resolve.[7] The author of the second report warns of reduced lateral motion of the cranial portion of the neck after atlantoaxial fusion.[3] Implants need be removed only if there is loosening or lysis of bone.

Various arrangements of Steinmann pins and variations in the application of Kirschner apparatuses in a through-and-through pattern have been reported. Four pins placed through the vertebral bodies of the atlas and axis, anchoring the dens with one pin and securing the pins with plates at the skin surface have been used. Care was taken to avoid the vertebral arteries. The animal recovered completely.[9]

With the introduction of locking plates and screws (see Chapter 76) it is logical that these implants would be preferred in the management of dens fractures. To date, no reports on the use of locking technology in the management of these fractures could be found in the literature.

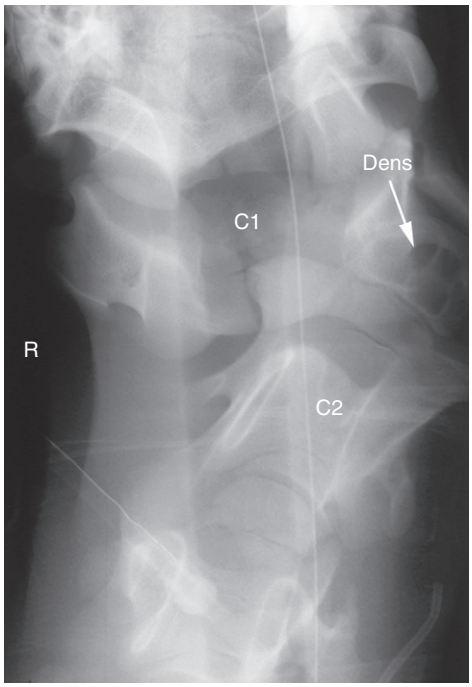

Figure 53-3. Ventrodorsal radiograph of the C1-2 articulation in a 1-week-old foal. There is leftward luxation of C2 relative to C1. The odontoid process *(dens)* is abnormally conformed and contains cystlike cavities.

Atlantoaxial Subluxation and Luxation

Subluxation of the atlantoaxial articulation, without fracture of the dens, is a rare condition seen in young horses up to 3½ years of age.[3,11-13] The condition can be congenital in origin or the result of a traumatic incident. A combination of stretching or tearing of the ligamentous attachments of the dens and the fibrous atlantoaxial joint capsule leads to instability and spinal cord compression that is exacerbated with neck extension and relieved with flexion. The neurologic signs can be acute or chronic, and they vary in severity from mild ataxia to recumbency.[13] The horse may have abnormal head and neck carriage and local swelling if the injury is acute, and it may resent neck manipulation. On plain radiographs, there may be a malalignment of the atlas and axis and, possibly, bony changes of the atlas secondary to trauma. On the extended views, further evidence of subluxation is a lengthening of the space between the articular surfaces of the atlas and the axis ventrally, whereas the space between the dorsal arch of the atlas and the spinous process of the axis is shortened.[3,8] The spinal cord compression is documented with myelography. Computed tomography (CT) scanning of the atlas and axis may help in further defining the nature of the luxation.

Complete luxation of the atlantoaxial articulation resulting in displacement of the dens so that it lies ventral to the atlas is very rare (Figures 53-2 through 53-5). For a complete luxation to develop, there needs to be complete disruption of the ligamentous attachments of the dens. Reportedly, horses with atlantoaxial luxation may have some stiffness of the neck but relatively few neurologic deficits.[3] The diagnosis is confirmed by radiography. With the horse under general anesthesia, efforts can be made to reduce the luxation with head and neck

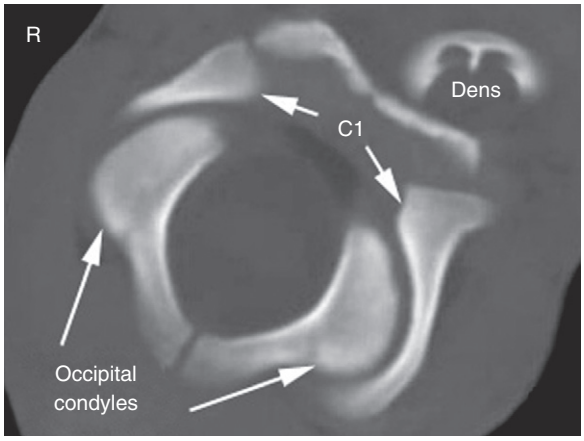

Figure 53-4. Transverse computed tomographic image at the level of the atlantooccipital articulation. The dens is displaced ventral to C1 and to the left, its shape is abnormal, and it contains cystlike cavities.

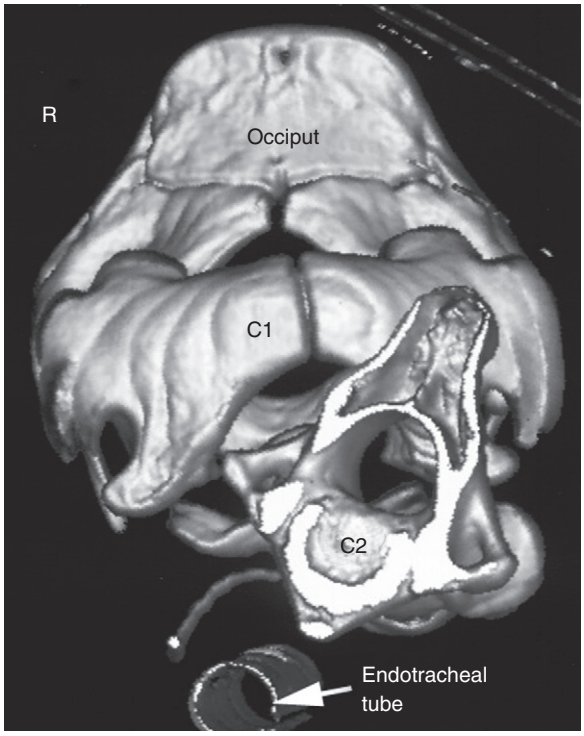

Figure 53-5. Caudodorsal, three-dimensional computed tomographic reconstruction demonstrating the relationship of C2 to C1.

manipulation, but this is likely to be successful only if the injury is acute.[3] In foals, there is also a significant risk of traumatizing the spinal cord during the reduction attempts.

Horses with atlantoaxial subluxation can be successfully treated with subtotal dorsal laminectomy (see "Subtotal Dorsal Decompression Laminectomy," in Chapter 52).[3,13] Surgery is certainly recommended if the horse is showing neurologic deterioration or if neurologic deficits persist despite conservative treatment. Removing the caudal two thirds of the dorsal arch of the atlas relieves the spinal cord compression and allows the range of motion of the atlantoaxial articulation to be maintained. A portion of the dorsal atlantoaxial ligament is left intact. Although the compression may be relieved, persistent instability represents a potential threat for several weeks after surgery. In a report of four horses with atlantoaxial subluxation treated with dorsal laminectomy, two horses recovered completely and a third showed satisfactory improvement. One was lost to follow-up. No surgical treatment has been reported for complete atlantoaxial luxation.

Fractures of the Atlas and the Axis

Fractures involving the atlas and the axis (other than fractures of the dens) occur occasionally. Fractures of the atlas produce neck pain and stiffness, but the degree of ataxia depends on the nature of the fracture and the degree of spinal cord impingement. In some patients, no neurologic deficits are associated with a fracture of the atlas. Standing radiographs, including a dorsoventral view, are used to make a definitive diagnosis. In the foal, the growth plates and their closure times must be taken into consideration when interpreting the films.[14]

Fracture of the ventral arch of the atlas and disruption of the ligaments that secure the dens can lead to the formation of a bony callus that impinges on the vertebral canal and induces ataxia later.[3] Myelography and, if available, CT scanning or an MRI study should subsequently be performed to further document and evaluate the nature of the compression. A dorsal laminectomy of the caudal half to two thirds of the dorsal arch of the atlas can relieve the spinal cord compression created by the fracture callus and may result in a long-term improvement in the neurologic status of the horse.[3] Fractures of the axis (other than those involving the dens) occur, but reports of surgical repair are rare.[2,3] There is a case report of a successful lag screw fixation of an oblique fracture of the body of C2 in a yearling Warmblood stallion that was accomplished with the aid of computed tomography.[15] An additional case report describes the treatment of a horizontal fracture of the body of the axis in a 5-year old French Saddle horse (Selle Français) with the help of a 14-hole DCP in combination with cancellous screws.[16] The horse acquired the fracture during a fall in an international cross-country competition. The partially threaded cancellous screws were used in lag fashion across the fracture and with fully threaded cancellous screws in C3. Part of the intervertebral disc was removed with a 13-mm diameter drill bit. The swath material was reintroduced into the defect. The fracture healed without complications and the horse was subsequently used for pleasure riding.[16] A spectacular vertical fracture occurred in a 7-year-old Swiss Warmblood gelding with history of an acute onset of neck stiffness 3 weeks before admission (Figure 53-6).[17] The owners did not observe a traumatic incident. The horse was anesthetized in sternal recumbency, and by placing a large Steinmann pin into each fragment and applying bone-reduction forceps, the fracture gap could be closed 2 cm. A 7-hole narrow 4.5-mm DCP was applied to the dorsal rim of C2 with three 4.5-mm cortex screws in each fragment (Figure 53-7). The horse made an uneventful recovery from anesthesia and 1 year later returned to athletic competitions.

Cervical Vertebral Fractures (C3 to C7)

Cervical spinal fractures in the adult horse are rare, but of the spinal fractures reported in the horse, approximately 50% involved the cervical vertebrae.[2,5,6,15] Compression fractures of the vertebral body are most frequently diagnosed, followed by

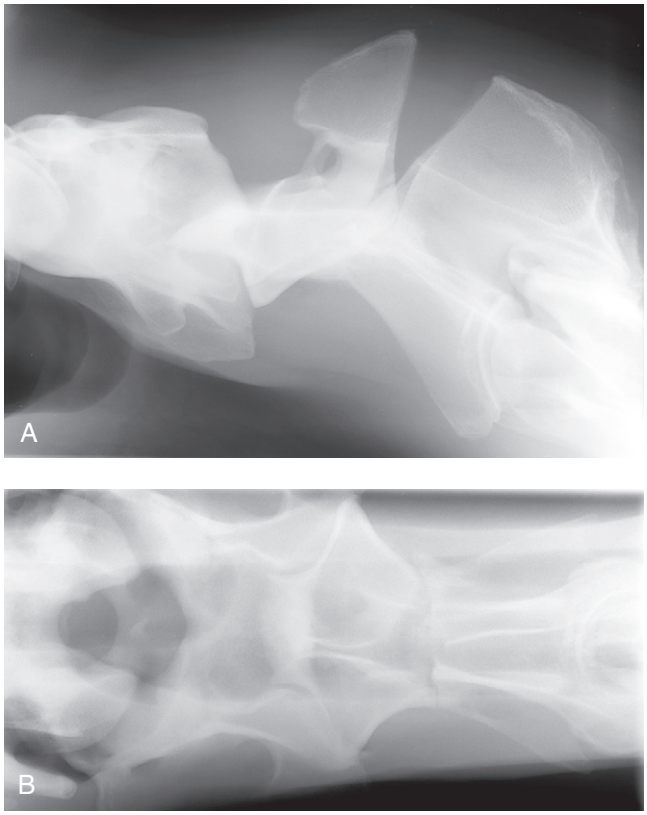

Figure 53-6. Laterolateral **(A)** and dorsoventral **(B)** radiographs of the cranial vertebral column, showing a complete transverse fracture in the cranial third of the axis with a marked deviation of the long axis of the second vertebra at the site of the fracture. (From Gygax D, Fuerst A, Piceck S, et al: Internal fixation of a fractured axis in an adult horse. Vet Surg 40(5):636-640, 2011.)

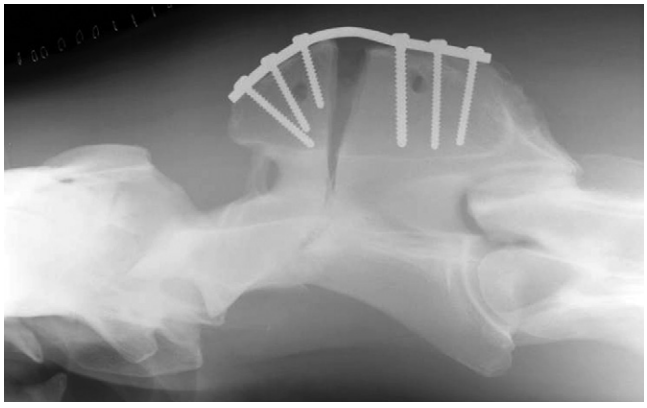

Figure 53-7. Laterolateral radiograph of the axis after fixation of the fracture with a 7-hole 4.5-mm dynamic compression plate (DCP); the hole over the fracture is left empty. Note the nearly normal long axis of the second vertebra when compared with the preoperative radiographs. (From Gygax D, Fuerst A, Piceck S, et al: Internal fixation of a fractured axis in an adult horse. Vet Surg 40(5):636-640, 2011.)

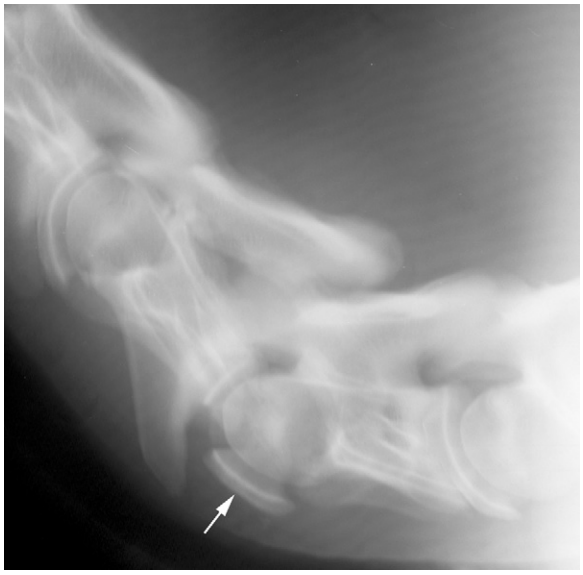

Figure 53-8. Standing lateral radiograph after an acute de-roofing injury at C4-5. The caudal endplate of C4 is fractured and there is fracture-subluxation of the C4-5 articular processes and C4 dorsal lamina, resulting in acute cervical hyperextension at this level. Note that the ventral portion of the caudal epiphysis of C4 remains attached to the C5 vertebra *(arrow).* Motion artifact, secondary to the horse's acute neurologic deficits, is evident on the image. The horse remained standing and subsequently recovered with minimal neurologic deficits.

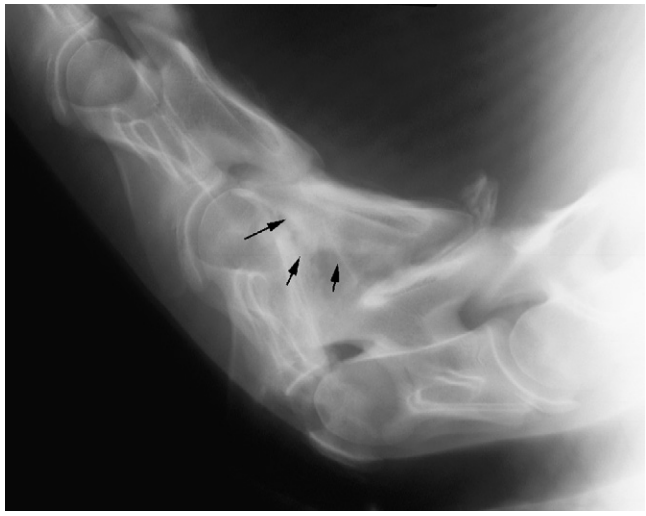

Figure 53-9. Standing lateral radiograph 40 days after injury. There is periarticular proliferative remodeling at the C4-5 articular processes representing an advanced stage of intervertebral fusion. Proliferative osseous remodeling is also present at the C4 dorsal lamina fracture *(arrows)* summating, and possibly extending into, the vertebral canal. The intervertebral disc space at C4-5 is wedged, and there is static hyperextension of the C4-5 articulation. The fractured ventral portion of the caudal epiphysis of C4 remains attached to the C5 vertebra.

articular process fractures.[2] These injuries result from head-on collisions or falls over jumps or obstacles. Falls in which the neck is severely hyperextended can result in fracture through the ventral half of the caudal end plate of the vertebral body. The ventral portion of the epiphysis remains attached to the caudal vertebra by the strong fibrocartilaginous disc (Figure 53-8). The caudal cervical vertebral body physes in horses are not closed until 4 to 5 years of age, although, practically, the cranial and caudal physes do not separate from the vertebral body in fractures or luxations after the horse is 2 years old.[2,3] If trauma is severe, pedicles of the caudal articular process fracture and elevate, effectively deroofing the spinal canal (Figure 53-9,

and see Figure 53-8). Although the fractures can produce severe angulation of the spine, the neurologic effects are diminished as a result of the deroofing. The dorsal portion of the intervertebral disc that remains intact is quite strong and is capable of providing enough support to maintain stability to the affected joint, despite the deroofing.[4,18]

Cervical vertebral body fractures can also occur after ventral interbody fusion using a Kerf Cut cylinder (KCC), particularly at C5-6 and C6-7.

Clinical signs of cervical vertebral fracture depend on the degree of bony disruption, instability at the fracture site, and the severity of the trauma to the cord. If the fracture is displaced and the spinal cord is compressed, neurologic signs vary from mild ataxia to quadriplegia and, in some cases, sudden death.[2] Because of damage to the central portion of the spinal cord with trauma, the innervation to the forelimbs is severely affected. When compression involves dorsal nerve roots, pain and torticollis are the principal signs. Sympathetic dysfunction, exhibited by Horner syndrome and skin hyperthermia, may be present.

The diagnosis of cervical vertebral fracture is confirmed by radiography. However, in thick-necked horses, even if they are positioned in lateral recumbency, it can be very difficult to get diagnostic radiographs in cases with only minimal disruption of the fracture fragments or with a great deal of swelling in the neck. Fracture lines may be poorly visualized. If the horse is standing, every effort should be made to get diagnostic standing radiographs. This allows a diagnosis without subjecting the horse to anesthesia and subsequent recovery, which may displace the fracture fragments, causing more damage to the spinal cord. In a recent case report, a number of imaging modalities including standard radiography, ultrasonography, nuclear scintigraphy, and fluoroscopy using a novel C-arm were used to diagnose a cervical articular process fracture of 3 weeks' duration in a standing horse.[19] If the horse is already recumbent, or there is progressive neurologic deterioration despite aggressive medical therapy, the horse can be anesthetized to obtain the radiographs. High-quality radiographs, including oblique and ventrodorsal views, and CT scans are necessary to gain an appreciation for the fracture configuration, particularly of vertebral body fractures where there can be multiple fracture lines and minimal displacement. The more distal portion of the neck (C5 to C7) in a large horse may not be accessible by the portal of the CT scanner because of size. If more than one vertebra is fractured, a myelogram may be useful to determine the site or sites of spinal cord compression. When the fracture is amenable to some form of fixation, the surgeon must be prepared to immediately follow the radiographic studies with the surgical repair. The risk of further spinal cord damage is too great if the horse is allowed to recover and undergo repair at a later date.

If the horse remains standing and is not showing progressive deterioration, conservative treatment is preferred. Some horses with pedicle fracture(s) experience further neurologic deterioration, with the formation of a healing callus that impinges on the dorsolateral aspect of the spinal canal causing cord compression (Figures 53-10 and 53-11).[3] Horses with an articular facet fracture can also develop torticollis that becomes permanent as a result of vertebral malalignment. There is a report of coaptation cast application used successfully to immobilize noncomminuted proximal cervical vertebral fractures in foals.[20] These types of casts can be difficult to apply properly and safely.

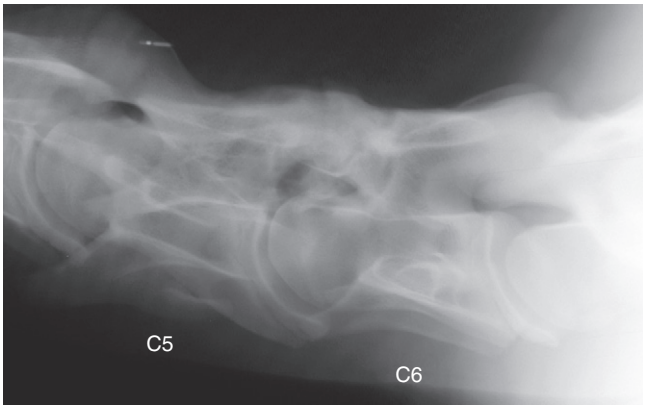

Figure 53-10. Standing oblique lateral radiograph of the C5-6 articulation in a yearling. There is severe proliferative remodeling of the articular processes at C5-6. Lateralization of the observed remodeling could not be determined radiographically.

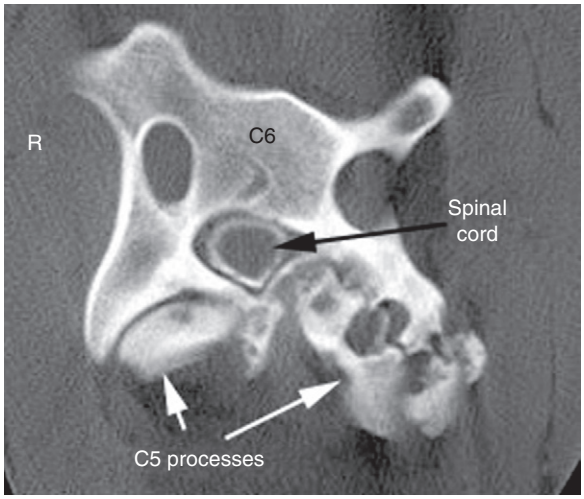

Figure 53-11. Transverse computed tomographic image through the cranial aspect of C6 after myelography. Severe degenerative remodeling and subchondral cystic change of the articular processes of C5-6 is seen on the left. Mild remodeling and subchondral cystic change is also seen at the axial aspect of the C5-6 articulation on the right. The spinal cord is outlined by a contrast-filled subarachnoid space. Note the left-sided, dorsal compression of the spinal cord secondary to the articular process remodeling.

Another problem that can be encountered in horses with cervical fracture and luxation that are managed conservatively is a subsequent worsening of neurologic signs after fusion of the affected intervertebral articulation, a result of the domino effect.[4] Spinal cord compression occurs at intervertebral sites adjacent to the fusion as a result of chronic malalignment and instability. An experienced author suggests that this domino effect occurs more frequently after injury-induced vertebral fusion (no surgery) than after surgical fusion for treatment of cervical vertebral malformation.[4] This should be kept in mind when deciding whether to attempt surgical repair. Successful realignment and stabilization of the affected vertebrae would likely prevent this from occurring.

If enough intact bone exists in the vertebral body to allow secure implant fixation, some types of recent fractures can be

realigned and stabilized with screws or with plate and screw fixation, possibly in combination with ventral intervertebral stabilization.[3] Each case needs to be considered on its own merits with regard to the type of fixation required for a successful repair. The greater the disruption of the vertebrae at the affected site(s), particularly if there is axial instability in conjunction with angular deviation in the sagittal plane, the poorer the prognosis. Intraoperative radiographic control of the drilling depth and screw positioning is critical to prevent penetration of the vertebral canal. It is difficult to get a secure screw purchase in vertebral bone, particularly in foals. There is a report of a successful repair of a cranial articular process and lateral neural arch fracture of C4 using a combination of an intervertebral fusion procedure followed by the ventral application of a broad DCP that spanned the bodies of C3 and C4.[3] In another report a 14-hole DCP was applied ventrally to a horizontal body fracture of C3 with articular involvement.[21] The plate was applied with 10 fully threaded and 3 partially threaded cancellous screws. Seven screws were implanted into C3 and six into C4, and the C4 joint was bridged.[21] A locking compression plate (LCP) can be used in lieu of the DCP for ventral application, without the use of a KCC. In a recent pilot study using the cervical spines of cadavers, the LCP construct was shown to have mechanical properties similar to the KCC fixation.[22] An advantage of the LCP over the DCP for ventral fusion is that the plate does not have to be applied tightly to the bone along the length of the plate, and therefore less bone from the ventral spine needs to be removed in preparation for plate application. An additional advantage is that the head of the locking screw is fixed in the plate and does not move, which results in superior stability. Also, no luting with methyl methacrylate is required to achieve plate contact, as is usually necessary with a DCP (Figures 53-12 and 53-13).

The prognosis for recovery from any form of surgical repair is related directly to the severity of the initial neurologic deficits and the long-term stability of the repair.[3] In horses that have pedicle fractures that develop exuberant bony callus impinging on the spinal canal or the lateral foramen, signs of spinal cord compression or nerve root impingement may develop. One author suggests that surgical intervention is indicated, in the form of either a dorsal laminectomy or a ventral stabilization.[3] Removal of the dorsal lamina and reduction of the bony callus at the compressed site produces immediate decompression, but the procedure is difficult and tedious, and there is a risk of refracture of the affected pedicle and worsening of the neurologic signs as a result of surgical trauma. Ventral intervertebral fusion leads to reduction in the size of the articular facets and the bony callus over a 3-month period after surgery.[3] Although there is not an immediate decompression, as achieved with dorsal laminectomy, there is much less risk associated with the surgery.

TRAUMATIC INJURIES TO THE THORACOLUMBAR SPINE

Thoracolumbar vertebral injuries are more common in adult horses than in foals.[5] Horses that engage in jumping activities are most often affected with life-threatening vertebral fractures.[5,23] The incidence of these types of lesions cannot be accurately assessed because some fractures are never identified. Stress fractures of the caudal portion of the thoracic and lumbar vertebrae in racehorses may go undetected as well.[24] Fracture of the thoracic dorsal spinal processes is often associated with the horse rearing, falling backward, and striking the withers. In a report of 22 cases of thoracolumbar vertebral fractures, 15 involved the vertebral body or neural arch and produced severe neurologic signs that necessitated euthanasia. The remaining seven horses had fractures of the dorsal spinous processes and exhibited no neurologic signs.[23] The more common sites of fracture of vertebral bodies include the first three thoracic vertebrae, around T12 (the area of greatest lateral bending and axial rotation), and the lumbar vertebrae.[25] Dorsal spinous process fractures tend to occur at or near T6. In a foal, a thoracic vertebral body fracture occurred at T11 after an electric shock.[26] Abscess formation in the vertebral body as a result of septicemia in foals can predispose to fracture.

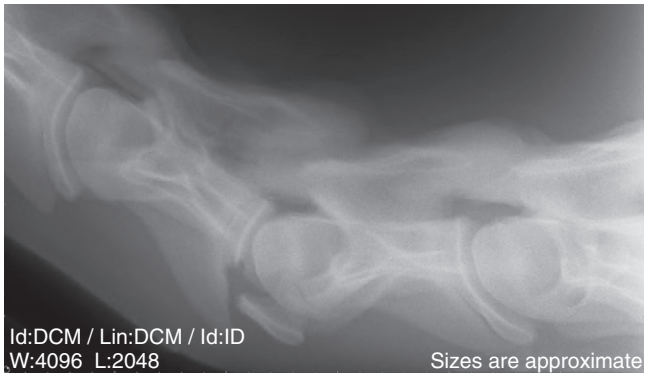

Figure 53-12. A lateral radiograph of a weanling Thoroughbred filly with fractures and angular deviation at C4-5. The filly was originally seen and radiographed 8 days before this examination. In the intervening time, she had deteriorated neurologically and the luxation angle had increased. Surgical repair was recommended. (Courtesy Drs. S. Reed, A. Ruggles, and B. Woodie, Rood and Riddle Equine Hospital, Lexington, KY.)

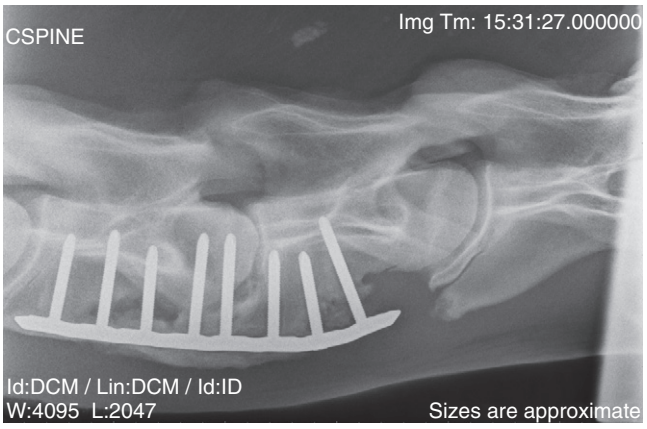

Figure 53-13. At surgery, vertebral realignment and fracture stabilization at C4-5 was achieved with a ventrally applied 8-hole, broad locking compression plate (LCP). Eight 5-mm diameter locking head screws were used, with the longest being 40 mm. This radiograph, taken 4 months after surgery, shows that the fixation has remained stable and vertebral alignment has been maintained. A substantial quantity of new bone formation had developed at the ventral aspect of C4-5 that will lead to bony fusion. (Courtesy Drs. S. Reed, A. Ruggles, and B. Woodie, Rood and Riddle Equine Hospital, Lexington, KY; Dr. Bob Hunt, Hagyard Equine Medical Institute, Lexington, KY.)

The clinical signs associated with a thoracolumbar vertebral fracture depend on the portion of the vertebra that is fractured and the degree of bony disruption. Severe compressive fracture of the vertebral body may result in paraplegia if the fragments displace, in delayed onset of paraplegia if the fragments do not displace immediately but with time through continued movement, and, finally, in no paraplegia if displacement remains minimal. Only the group with little or no displacement of fragments has a chance of total recovery with conservative management.

The diagnosis of a thoracolumbar vertebral body fracture in an adult horse is complicated by the fact that radiography of this area is generally unrewarding because of the massive musculature around the vertebrae. The clinician must often rely on the physical examination and neuroanatomic localization of the lesion to make a presumptive diagnosis. In the horse with a healing fracture, scintigraphy may be useful for localizing the affected vertebra. In foals, CT scanning can be used to assess the thoracolumbar spine, and myelography is used to document the extent of the spinal cord compression—information that is necessary for determining the length of decompression required if a dorsal laminectomy is being considered.[3]

Thoracolumbar laminectomy and fracture stabilization has been described in the foal and should be attempted only if the foal has evidence of deep pain recognition or some voluntary movement of the hind limbs.[3] A dorsal laminectomy of the affected area is performed with the foal in sternal recumbency. Steinmann pins are placed into the adjacent vertebral bodies, the vertebrae are manipulated into alignment, and polyvinylidine plates are applied to the dorsal spinous processes. Polymethyl methacrylate is used to connect the pins and produce rigid fixation. The plates and pin fixation are removed after 3 to 6 months. The author stated that the prognosis for foals with thoracolumbar fractures is generally grave, although surgical treatment may improve the outcome in selected cases.[3] Adult horses that are recumbent and have severe neurologic signs as a result of thoracolumbar spinal fracture are euthanized. In those horses where the signs are limited to back pain and rear limb incoordination, conservative management can be successful.[23]

The diagnosis of fracture of the dorsal spinous processes of thoracic vertebrae is not difficult. The height of the spine is decreased, because the most proximal aspect of the spine fractures and moves laterally. The area becomes swollen and painful to the touch. Radiographs are helpful in confirming fractures of the dorsal spines and the extent of the damage (Figure 53-14). Contrast fistulography is useful in horses with a draining tract to document communication with a dorsal spinous process.[27]

Fractures of the dorsal spine usually do not require surgery unless the bone fragments become sequestered or their position, after fracture, precludes the use of a saddle. A defect in the withers area may necessitate a special saddle pad. If surgery is required or elected, subtotal ostectomy of the dorsal spinous processes can be performed under general anesthesia or in the standing horse.[27-29]

If the procedure is performed under general anesthesia, the horse is placed in lateral recumbency with the displaced processes uppermost. A long, slightly curved incision is centered over the fragments. The incision extends through the skin, the subcutis, and the supraspinous ligament. Bone fragments are identified and removed by freeing them from underlying muscle and ligamentous attachments. If the bone has sequestered or if infection is present, appropriate exits are created to facilitate gravitational drainage. The area will heal enough to resume riding after 8 to 12 weeks; however, special saddle pads may need to be used to prevent abrasion by the saddle.

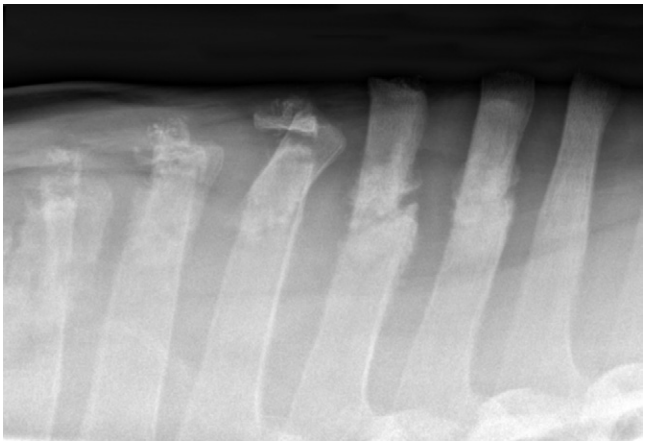

Figure 53-14. Lateromedial radiographic view of an 8-year old Arabian stallion suffering from six fractures of the thoracic dorsal spinous processes (Courtesy J. Auer, Zurich, Switzerland.)

If the procedure is done standing, the risks of general anesthesia are eliminated. In addition, there are other distinct advantages to performing the procedure standing, which include reduced hemorrhage, improved visibility, and better access to both sides of the dorsal spinous process(es), all of which facilitate dissection and separation of the muscular attachments. In a report of nine horses treated standing, thoracic spinous processes were resected for treatment of osteomyelitis, fracture, and impingement. The sites of involvement ranged from T2 to T18 in these horses.[27] With the horse sedated, the tissues surrounding the affected dorsal spinous processes are infiltrated with a local anesthetic. The surgical approach to the affected dorsal spinous process(es) is longitudinal and along the midline. The affected portion of the dorsal spinous process is dissected free, and it is resected using either an oscillating saw or an osteotome.

Endoscopic surgical subtotal resection of the dorsal spinous processes and interspinous ligament has been described.[30] Ten horses were treated for dorsal spinous process impingement in the caudal thoracic region and most were back to work by 8 weeks after this minimally invasive procedure. This technique allows the selective removal of the impinging bone on the spinal process, but it is unlikely that it could be effectively used in a horse with a fractured spinous process, particularly if there is a good deal of fibrosis or the bone is infected. Intraoperative hemorrhage greatly reduces the endoscopic visibility and complicates the procedure.

Fistulous draining tracts from an infected supraspinous bursa necessitate more extensive resection and débridement of infected tissues, including the affected nuchal ligament, the supraspinous bursa, and all infected bone of affected dorsal spinous processes. The supraspinous bursa lies between the funicular part of the nuchal ligament and the dorsal spinous processes of T2 to T5.[29] In one report of surgical treatment of supraspinous bursitis in 10 horses, paramedian incisions, either unilateral or bilateral depending on involvement, were made approximately 8 to 10 cm lateral to the dorsal midline and

dorsal to the scapula. The incisions were 15 to 20 cm in length, dorsal to the scapula, and started 5 cm cranial to the scapula. Ventral drainage was achieved by placing a large gauze seton drain between the ventral edge of the rhomboideus muscle and the dorsoscapular ligament, cranial to the scapula. The authors of that report stressed that the dorsoscapular ligament should not be penetrated. The procedure can be performed with the horse standing or under general anesthesia. There is a significant recurrence rate after surgical treatment of fistulous withers, and multiple surgeries may be required.[30,31] Because of the historical connection between fistulous withers and *Brucella abortus* (Bang disease), and the risk to humans, horses should be serologically tested before surgical treatment is undertaken.

Internal fixation of spinous process fractures with plates and screws has been performed, but the procedure carries the risk of longer time under anesthesia and a difficult recovery from anesthesia.[32]

TRAUMATIC INJURIES TO THE SACRAL SPINE

Trauma to the sacral area occurs when the horse suddenly dog-sits or falls over backward, landing on the croup area.[33] Subluxation where the sacrum articulates with the iliac crests causes chronic pain that may originate with one traumatic event or may be caused by repetitive jarring of the spine in this area during work. Compressive lesions of the sacrum involve the cauda equina with loss of function to the sacral and caudal nerve roots and the pudendal, caudal, and parasympathetic pelvic nerves that originate from these nerve roots.[34] The resultant clinical signs include pain and swelling over the croup and tail head, tail weakness or paralysis, decreased anal tone, retention of feces, bladder distention, and urine dribbling. There may be analgesia of the tail, anus, and perineal region and of the surface of the penis in males. Scintigraphy may be useful for initially localizing an injury to the sacrum. The diagnosis of a sacral compressive lesion is based on the clinical signs, palpation of the sacral area *per rectum*, pelvic ultrasonography, radiography, and, if the horse is small enough, CT scanning.[3,35] A positive contrast epidurogram may show compression in the sacrum.[36] More-extensive injuries that involve the caudal lumbar and sacral regions can produce marked hind limb weakness, ataxia, and neurogenic muscle atrophy of the hindquarters. This may be documented by electromyography and muscle biopsy.

Acute injury should be treated with anti-inflammatory drugs, and up to 10 days should be allowed for signs of recovery. During this time, evacuation of the rectum several times daily and topical treatment of the tail and perineal region to prevent scalding is part of the required management. If there is no improvement within 10 days, the prognosis is unfavorable for recovery, and either surgical intervention or euthanasia should be considered. Surgical decompression of the sacrum can be achieved by removing the spinous processes and dorsal laminae of the sacral vertebrae. Even with surgery, the prognosis is guarded. In acute cases, stabilization after decompression is necessary and can be accomplished by using plates and screws.[3] In foals where stabilization was accomplished using long plates attached to dorsal spinous processes, the plates must be removed within months of the repair to prevent development of lordosis in the lumbosacral region.[3] A recent report discussed the use of LCP fixation in three heifers suffering from fracture of the sacrum, with good results in restoring tail motility and defecation and allowing normal calving.[37]

Robertson examined a 6-week-old foal that had a compressive spinal lesion at L6 and S1 as a result of trauma at birth. The foal underwent a dorsal decompression of L6 and S1 and there was some improvement in clinical signs after surgery. A follow-up CT scan 4 months after surgery showed good decompression at the dorsal laminectomy site, but stenosis of several of the nerve root canals probably contributed to the ongoing muscle weakness. Five years after surgery, the horse had residual ataxia and muscle weakness but had learned to adapt remarkably well as a nonathletic companion animal. In horses with persistent tail paralysis, the tail may need to be amputated.[35]

TRAUMATIC INJURIES TO THE COCCYGEAL VERTEBRAE

Horses have an average of 18 coccygeal vertebrae. Trauma to the more distal vertebrae may well go unnoticed. Coccygeal fractures generally occur as a result of the horse falling back onto the tail or backing into an immovable object. Other causes of tail injury include entrapment in a door, bite wounds by other animals, and improperly placed tail wraps. If the tail distal to the site of trauma loses its blood supply, necrosis occurs, requiring amputation.

Trauma to the base of the tail (Ccy 1 to 3) may cause vertebral fracture, soft tissue injury, and neurapraxia of the motor nerves in this area. In most horses, the fractures are closed, but the fracture may be comminuted with displacement of the fracture fragments.[38] The nerve injury can result in an inability to move the tail and defecate properly. Urine (in females) and fecal scalds can develop. Muscle atrophy over the tail head is consistent with neurogenic atrophy. Manipulation of the tail produces pain, and there is palpable crepitus at the tail base. Distal to the lesion there is loss of motor function. Lateral or oblique lateral radiographs confirm the presence of a vertebral fracture. Nuclear scintigraphy may be useful for identifying concurrent bony trauma, such as a sacral fracture.

Most horses with fractures of the coccygeal vertebrae are managed conservatively with stall rest and the use of anti-inflammatory drugs. The fractures can be relatively slow to heal, and there may be permanent neurologic damage as well as conformational changes as a result of the muscle atrophy and callus formation.[38] In one report of a comminuted fracture of the first coccygeal vertebra with ventral displacement of the remaining coccygeal vertebrae, the fractures were stabilized using a combination of bone plates, wire, and bone cement. The implants were removed 4 months later with the fracture site being stable and pain free.[36] Other forms of stabilization or fixation, such as the placement of Steinmann pins across the fracture or the use of the pinless external fixator (see Chapters 76 and 102), might also hasten healing and reduce the irritation to nerves in the area, thereby allowing a quicker return of tail function.

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CHAPTER
54 Peripheral Nerve Injury

Robert J. MacKay

ETIOLOGY AND PATHOGENESIS

Mechanical injuries to peripheral nerves occur because of compression, entrapment, transection, laceration, ischemia, crushing, stretching, and chemical or burn damage.[1,2] These injuries are usually categorized according to the degree of damage to a single axon. However, compound nerves contain numerous fascicles, and each fascicle is made up of many axons within a connective tissue sheath. The functional effect of damage is seldom as straightforward as single-fiber classifications would suggest, and it usually reflects differing degrees of damage across a nerve.

Damage to individual axons fall into two major functional categories. Type 1 injury refers to loss of function *without loss of axonal continuity*. Type 2 injury represents damage to axonal structures such that *Wallerian degeneration of the axon ensues* distal to the site of injury; the proximal axonal segment and the cell body remain intact. Retrograde degeneration of the proximal nerve occurs if connectivity is not reestablished. A well-accepted classification system that divides the spectrum of injury into five categories is shown in Figure 54-1.[3]

Type 1 or *neurapractic lesions* are characterized by a failure of conduction of the action potential across the injured axonal segment.[4] Normal muscle action potentials are evoked by stimulation of the nerve anywhere distal to the lesion, but they are absent when the nerve is stimulated proximal to the injury. The pathologic basis for these *reversible* injuries could be nerve compression, damage to the myelin sheath, or alteration of the functions of the axonal cell membrane or its channel proteins.

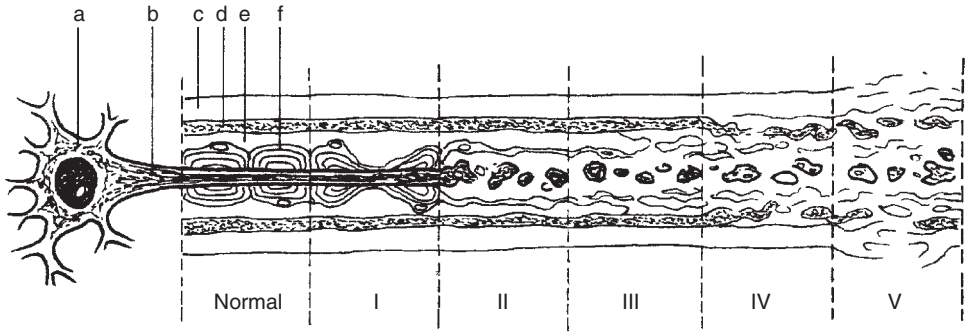

Figure 54-1. Sunderland classification of nerve injury. *a,* Cell body; *b,* axon; *c,* epineurium; *d,* perineurium; *e,* endoneurium; *f,* myelin sheath; *I,* first degree (neurapraxia); *II,* second degree (axonotmesis); *III,* third degree; *IV,* fourth degree; *V,* fifth degree.

Type 2 injuries are characterized by axonal interruption, or *axonotmesis*. This is equivalent to the Sunderland type II injury. If there is disruption of components of the connective tissue sheath and axon, the process is termed *neurotmesis*. According to the Sunderland classification system, there are three grades of neurotmesis (III, IV, and V), reflecting disruption of endoneurium, perineurium, and epineurium. Recovery by axonal regrowth is unlikely after neurotmesis.[5] Immediately after axonotmetic injury, the distal axonal segment is competent to conduct action potentials; however, within 3 to 8 days after injury, the distal segment degenerates and no longer responds to electrical stimulation. In this way, neurapractic (type 1) and axonotmetic (type 2) injuries of greater than 8 days' duration can be theoretically distinguished. Long-standing axonotmetic injuries result in retrograde degeneration of the neuronal cell body and secondary demyelination of the proximal axonal segment. After axonotmesis of motor nerves, muscle reinnervation occurs by two separate mechanisms: collateral sprouting and axonal regrowth.[1] If there is incomplete loss of axons to a muscle, surviving motor axons generate sprouts from their nerve terminals, which can establish competent junctions with adjacent denervated muscle units. Reinnervation of muscle units by this process occurs within days to weeks. However, if the motor axons to a muscle are severed, reinnervation occurs by growth of collateral sprouts from the proximal stump. Axonal sprouts grow at a rate of 1 mm per day (approximately 1 inch per month). Regeneration occurs under the influence of growth factors including neurotrophic growth factor, brain-derived neurotrophic factor, and ciliary neurotrophic factor.[6] Successful reinnervation thus may take many months. Because of progressive fibrous replacement of denervated muscle and retrograde degeneration of the proximal parts of affected motor neurons, it has been suggested that reinnervation may not be possible if more than 12 months have elapsed since the original injury. However, reinnervation surgery has been successful when performed on human patients 20 years or more after denervation.[7]

Large-diameter myelinated fibers are most sensitive to compression forces. The largest axons—sensory axons, including afferent fibers conveying touch-pressure, vibration, and proprioception—are usually damaged first (resulting in ataxia or inappropriate limb placement). The second group to be compromised are the large myelinated motor axons; and the smaller axons, which carry cutaneous and deep mechanoreceptor sensory information, are affected third. The last to be lost are the smallest diameter fibers transmitting pain sensation.

CLINICAL SIGNS

Peripheral nerve injuries are characterized by weakness of the innervated muscle, accompanied within 2 to 4 weeks by appreciable atrophy. When the injured nerve supplies important extensor muscles of the limbs (e.g., as is the case with radial, femoral, sciatic, or peroneal nerve injuries), there is obvious alteration of gait. Areas of cutaneous anesthesia occasionally accompany peripheral nerve injuries. Over the neck and trunk, cutaneous sensory innervation occurs in defined bands corresponding to the territories of the sensory nerves associated with segmental dermatomes. Damage to a peripheral spinal nerve or dorsal nerve root results in cutaneous anesthesia or hypalgesia over the supplied dermatome. With damage to the pudendal nerve or its sacral nerve roots, there is anesthesia or hypalgesia of the perineal area. In contrast, relatively small autonomous zones have been defined for sensory components of the ulnar, musculocutaneous, median, femoral, tibial, and peroneal nerves (Figure 54-2).[8,9] Sympathetic fibers are distributed with peripheral nerves, so denervated skin also may be evident as circumscribed spontaneous sweating. Rarely, pain or other unpleasant sensation (dysesthesia) is a sign of traumatic nerve injury. Neuropathic pain is characterized by exaggerated responses to modest stimuli like pinpricks (hyperalgesia) or painful responses to normally innocuous stimuli such as light touch (allodynia). *Complex regional pain syndrome* is the name given to a neuropathic pain disorder associated with the development of autonomic features, such as changes in skin temperature, color, and sweating and structural changes in soft tissues in and around the nerve's territory. A suspected complex regional pain disorder has been described in horses.[10] Neuropathic pain disorders in horses usually manifest as hypersensitivity to touch or attempted mutilation of a target area over the territory of the affected nerve.

The most common and important syndromes of mechanical injury to peripheral nerves of the limbs are described later.[9,11,12]

Suprascapular Nerve

The suprascapular nerve arises from the 6th and 7th cervical spinal cord nerve roots and passes laterad and ventrad between the supraspinatus and subscapularis muscles before coursing around the cranial edge of the scapula about 7 cm proximal to the scapular tuberosity and ramifying in the supraspinous and infraspinous fossae.

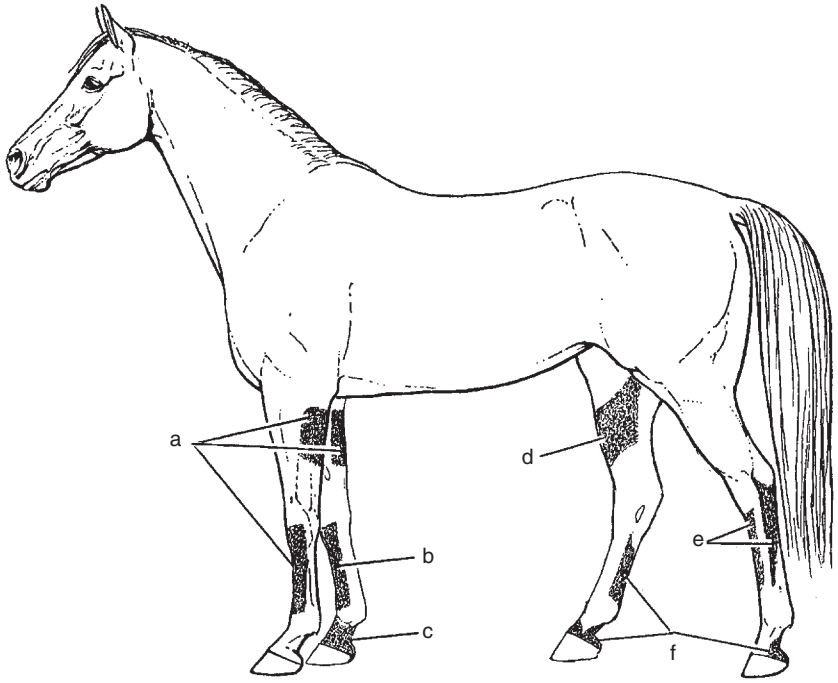

Figure 54-2. Areas of anesthesia/hypalgesia (autonomous zones) associated with injury to the major peripheral nerves of the limbs of the horse. *a,* Ulnar n. dermatomes; *b,* musculocutaneous n. dermatome; *c,* median n. dermatome; *d,* femoral n. dermatome; *e,* peroneal n. dermatomes; *f,* tibial n. dermatomes.

Suprascapular nerve injury occurs most commonly when a horse collides at speed with another horse or with inanimate objects, such as fences, trees, or doorways. It may also occur when a galloping horse stumbles over uneven ground. In each of these situations, there is likely to be stretching or direct compression (or both) of the suprascapular nerve as it curls around the cranial edge of the neck of the scapula under cover of the supraspinatus muscle. The muscles innervated by the suprascapular nerve, the supraspinatus and infraspinatus, normally provide lateral support for the shoulder joint. Consequently, when the nerve is injured, the immediate response is laxity and lateral instability of the shoulder joint, which bows out or "pops" as the affected limb bears weight. Although subluxation of the shoulder joint does occur after the suprascapular nerve is blocked at the scapular neck, it has been suggested that other nerves (e.g., axillary, pectoral, subscapular, brachial plexus roots) and muscles (e.g., pectoral, subclavius, subscapularis) are usually damaged with this type of injury, thus contributing to shoulder instability and subluxation.[11]

Within 2 to 4 weeks of injury, there is obvious atrophy of the supraspinatus and infraspinatus muscles. Wasting of these muscles becomes complete over the ensuing several months. The condition is known as *sweeney.*

Neurapractic damage to the suprascapular nerve should heal within several days to several weeks. After partial nerve damage, sprouts from the neuromuscular terminals of surviving fibers should find denervated muscle and restore function within several weeks. In either of these settings (neurapraxia or partial nerve damage), severe muscle atrophy should not occur before recovery begins. More complete axonal injury results in sweeney, and any recovery depends on regrowth of axonal sprouts from the proximal stump of the suprascapular nerve. Successful reinnervation is evident as recovery of muscle bulk in the ventral part of the supraspinatus muscle beginning within 3 months of injury. Maximal recovery takes an additional 3 to 12 months. Regrowth of the suprascapular nerve can be facilitated by surgery to resect scar tissue (external neurolysis) and reduce tension on the nerve as it crosses the cranial edge of the scapula (see Chapter 96).[13,14] The surgery can be performed either preemptively when soft tissue swelling has subsided or 3 to 6 months after injury when it is clear that spontaneous reinnervation is not occurring.

Radial Nerve

The radial nerve innervates a flexor of the shoulder and the extensors of the elbow, carpal, and digital joints. It arises chiefly from the first thoracic root of the brachial plexus. The nerve travels for a short distance with the ulnar nerve as it passes between the long and medial heads of the triceps. From there, it winds downward and outward around the humerus in the musculospiral groove, crosses the lateral aspect of the elbow joint, and ramifies in the extensor muscles of the digit.

The proximal portion of the nerve is well protected by surrounding muscles and therefore is not normally damaged unless there is severe injury to adjacent tissues. The T1 nerve root may be lacerated by fracture of the C7 or T1 vertebra or the first rib, or it may be compressed by remodeled arthritic intervertebral joints. Likewise, the proximal part of the nerve is commonly damaged as a result of humeral fracture.[15] The radial nerve (or the roots that form it) may be injured with other components of the brachial plexus by trauma to the shoulder region. The plexus or radial nerve, or both, is injured when the thoracic limb is severely hyperextended or abducted, such as, for example, when the upper leg gets wedged (e.g., in a tub or between the rails of a fence) and the horse struggles to release it. Injury may also occur during forceful traction of the forelimbs of a fetus during relief of dystocia. The radial nerve and triceps muscles are vulnerable to damage when horses are positioned in lateral recumbency, usually under general anesthesia. The risk is particularly high in large, muscular horses on poorly padded surfaces, and it increases with the duration of the procedure. Experimental studies have shown that ½ to 1 hour of ischemia produces neurapractic conduction failure, and greater

than 3 hours of ischemia causes permanent changes.[16] The lower part of the nerve is in close proximity to the lateral aspect of the elbow, under cover of the lateral head of the triceps, and it may be damaged by direct trauma to this site (usually by a kick from another horse) or by dislocation or fractures that involve the elbow.

Horses with complete radial paralysis stand with the shoulder extended and the rest of the joints flexed. The elbow is "dropped" (i.e., lower than normal) and the dorsum of the hoof rests on the ground. When forced to walk, the horse may partially protract the limb by exaggerated extension of the shoulder; however, the toe drags and the horse collapses on the limb during the weight-bearing phase of the stride. If the site of damage is distal to the branches to the heads of the triceps (middle third of the humerus), the shoulder is held in normal position and the elbow is not usually dropped. Such horses can bear weight if the hoof is placed flat on the ground. If reflexes can be tested while the horse is recumbent, it is expected that the triceps reflex will be absent but the flexor (withdrawal) reflex will be normal. Although the radial nerve has numerous cutaneous sensory branches, injury to this nerve does not result in any consistent area of cutaneous anesthesia.

Beginning at 2 to 4 weeks, there is progressive atrophy of the extensor muscles of the limb distal to the site of injury. The principles just described for neurapractic and axonotmetic injuries apply. With regrowth of axonal sprouts from the proximal stump of the radial nerve, the triceps muscle is expected to recover function before the digital extensors.

Musculocutaneous Nerve

The musculocutaneous nerve arises near the suprascapular nerve from the part of the brachial plexus supplied by the 7th and 8th cervical roots. It descends on the medial surface of the coracobrachialis muscle and forms a loop with the median nerve directly beneath the brachial artery before terminating in branches to the biceps brachii, pectoral, and brachialis muscles. The musculocutaneous nerve supplies flexors of the elbow.

Injury to the nerve is rare and causes only transient toe-dragging. The shoulder may be held in a flexed position and the elbow in an extended position. The diagnosis can be made by finding hypalgesia or anesthesia over the dorsomedial aspect of the carpal region and proximal metacarpus (see Figure 54-2), beginning immediately after the injury and by appreciating obvious atrophy of the biceps and brachialis muscles beginning 2 to 4 weeks after injury. Injury to the musculocutaneous nerve eliminates the biceps reflex, although this reflex is difficult to obtain even in normal horses. The flexor reflex should be intact, although elbow flexion may be weak.

Median and Ulnar Nerves

The *median nerve* is the largest branch of the brachial plexus and receives input from the 8th cervical and first thoracic nerves. After exchanging fibers with the musculocutaneous nerve, it descends along the medial aspect of the leg in association with major arteries and terminates in palmar and cutaneous branches. The *ulnar nerve* arises from the thoracic part of the brachial plexus, passes downward and backward over the medial epicondyle of the humerus (deep to the medial head of the triceps), and passes along the palmar aspect of the metacarpus under cover of the digital flexors to terminate in branches that contribute to the lateral palmar nerve and cutaneous sensation. These nerves supply muscles that extend the elbow and flex the carpus and digits.

Injury to either or both nerves can be caused by injury at the brachial plexus or along the medial aspect of the upper limb. Experimental section of the nerves causes a "tin soldier" gait, with decreased flexion and dragging of the toe during the protraction phase of the stride. Hypalgesia or analgesia of the skin develops over the caudal forearm, lateral metacarpus, and medial phalangeal areas. When flexor reflexes are tested by pinching the skin of an area with unimpaired sensation (e.g., over the lateral metacarpophalangeal [MCP] joint), there is an intact flexor response, but it is weak compared with the normal side. Even without recovery of nerve function, interference with gait at the walk decreases over time and is minimal by 3 months after the injury. The reason for this adaptation is unclear, although it has been suggested that fibers contributed by the musculocutaneous nerve may take over some of the functions from the median nerve. Atrophy of the carpal and digital flexor muscles is apparent after 2 to 4 weeks.

Femoral Nerve

The femoral nerve arises principally from the 4th and 5th lumbar nerve roots, with contributions also from the 3rd and 6th roots, courses backward and ventrad between the psoas major and minor muscles, and then runs between the sartorius and iliopsoas muscles lateral to the external iliac artery. It terminates as branches to the heads of the quadriceps muscle. The saphenous nerve, a branch with both motor and sensory function, joins the femoral nerve at the point of emergence from the psoas muscles. The femoral nerve innervates muscles that flex the hip and extend the stifle.

The nerve courses too deeply to be directly affected by an external blow to the pelvic limb; however, it can be damaged by ilial, femoral, or vertebral fractures. Ischemic injury caused by prolonged stretch or increased tissue pressure during anesthesia in dorsal recumbency may cause bilateral femoral neurapraxia.[17] A similar presentation, presumably caused by compression of nerve roots by the foal in the birth canal, is seen in mares after severe prolonged dystocia.

With unilateral paralysis of the femoral nerve, the pelvic limb adopts a flexed posture, usually with the foot flat on the ground. When the horse tries to bear weight, the limb buckles. During walking, there is obvious lameness, with a relatively brief and ineffectual weight-bearing phase over the affected limb. This can be quite subtle in horses with partial femoral nerve paralysis. In the case of bilateral involvement, the horse is either unable to rise or gets to its feet with difficulty, and then stands briefly and uncomfortably in a crouched position.

If the nerve injury occurs proximal to the saphenous branch, there is anesthesia or hypalgesia of the skin over the medial surface of the thigh (see Figure 54-2). Atrophy of the quadriceps muscle is evident within 2 to 4 weeks. The flexor reflex should be normal, but the patella reflex is weak or absent.

Sciatic Nerve

The sciatic nerve is the largest nerve in the body. It is derived principally from the 6th lumbar and 1st sacral nerves but also receives a branch from the 5th lumbar nerve. The nerve passes

caudad and ventrad on the sacrosciatic ligament before turning between the greater trochanter and tuber ischii. It courses ventrad in the thigh between the biceps femoris muscle laterally and the adductor, semimembranosus, and semitendinosus muscles medially. It continues as the tibial and peroneal nerves. The sciatic nerve supplies important extensors of the hip and flexors of the stifle.

Sciatic nerve damage is usually a result of deep injections into the caudal thigh ventral to the greater trochanter. Such injections may injure the nerve by direct laceration, by injection of material into the nerve, or through secondary inflammation or compression from scarring induced by previous deposition of the drug close to the nerve.[1] In most cases, the neurologic deficits are apparent within a few minutes. A delayed onset suggests the development of perineural or endoneural inflammation or fibrosis. The nerve may also be injured by fractures of the ilium or ischium or by sacroiliac and coxofemoral dislocations.

The posture of the affected limb is characteristic: it is held slightly caudal with the dorsum of the hoof resting on the ground. The stifle and tarsus are more extended than normal, whereas the distal joints are flexed. During walking, the leg is dragged forward by the actions of the quadriceps and biceps femoris muscles. These muscles, in concert with the reciprocal apparatus, allow the horse to bear some weight on the limb if the foot is first placed in normal position.

There is cutaneous hypalgesia or anesthesia over most of the limb except for the medial thigh. If the flexor reflex is tested by pinching the skin over the medial thigh, the response is intact but weak compared with the normal side.

Peroneal Nerve

The peroneal nerve arises from the sciatic trunk deep to the biceps femoris and descends on the lateral head of the gastrocnemius muscle. It crosses the lateral aspect of the femorotibial joint and terminates in superficial and deep branches, which course ventrally between the long and lateral digital extensors. The nerve provides motor innervation to the flexors of the tarsus and the extensors of the digit.

Paralysis results in extension of the tarsus and flexion of the distal joints of the pelvic limb. At rest, the limb is held slightly caudally, with the distal joints in flexed position and the dorsum of the hoof contacting the ground. During walking, the limb is moved erratically. The toe is dragged along the ground during the weak protraction phase and then pulled caudad as the horse attempts to bear weight. As is the case with sciatic involvement, the limb can support weight if the foot is placed in normal position.

There is progressive atrophy of the cranial tibial muscle and long and lateral digital extensors and immediate cutaneous hypalgesia or anesthesia over the lateral metatarsus. The flexor reflex is intact and usually appears normal.

Tibial Nerve

The tibial nerve represents the direct continuation of the sciatic nerve. It runs between the bellies of the gastrocnemius muscle and passes distad on the medial aspect of the leg immediately dorsal to the superficial flexor tendon before terminating in the medial and lateral plantar nerves. The tibial nerve innervates the gastrocnemius muscle (extensor of the tarsus) and digital flexors.

With tibial neuropathy, the resting limb is held more flexed than is normal, and although the foot contacts the ground in normal position, the MCP joint often partially collapses into a flexed position (i.e., "knuckles"). The foot is moved in stringhalt-like fashion, with exaggerated flexion of the tarsus and stifle during protraction, followed by sudden extension to the weight-bearing phase of the stride.

There is atrophy of the gastrocnemius muscle and cutaneous hypalgesia or anesthesia of the caudal metatarsal region and bulbs of the heels. The flexor reflex is tested by pinching the skin over the dorsal aspect of the metacarpophalangeal joint (MCP) joint. The reflex is present but weaker than on the normal side.

DIAGNOSIS

The diagnosis of peripheral nerve injuries is normally made by careful observation of clinical signs, including testing of reflexes and cutaneous sensation. It may not be possible to discern the effects of nerve damage until fractures are stabilized and soft tissue swelling subsides. The examination must carefully document the location and severity of weakness, atrophy, and sensory and reflex alterations. The findings should be documented thoroughly and recorded at each examination so that progression can be determined.

Beginning 2 to 4 weeks after injury, progressive atrophy becomes apparent in large muscles supplied by the damaged nerve. Beginning about 2 weeks after injury, the distribution of muscular denervation can be determined objectively by needle electromyography in the sedated or anesthetized patient. This is particularly helpful in cases of apparent suprascapular or radial neuropathy to determine whether there is singular involvement of a particular peripheral nerve or injury at the level of the brachial plexus.

Nerve stimulation can be used to assess location, severity, and recovery from nerve injury. With both neurapraxia and axonotmesis, nerve conduction across the lesion is affected. Thus, when the proximal segment of the motor nerve is stimulated electrically, the amplitude of the evoked muscle action potential is reduced in proportion to the severity of the injury. It is technically possible to distinguish signs of neurapraxia from those that are the result of physical interruption of the axons of the nerve. Because axonal injury leads to degeneration of the distal nerve segment, electrical stimulation of the nerve distal to the injury site fails to evoke motor action potentials, or they are of reduced amplitude, when tested at least 9 days after the injury.[1] In contrast, nerves blocked by neurapraxia evoke normal muscle responses after stimulation. It must be remembered that an injured nerve frequently has a mixture of neurapractic and axonotmetic lesions, and the findings may not clearly distinguish between the two types of injury. Sensory nerve conduction studies are performed in humans by electrically stimulating peripheral sensory axons at one point and recording sensory nerve action potentials at a more proximal point. This technique is useful for distinguishing lesions of the dorsal root from those involving peripheral parts of the nerve.

Nerve conduction procedures are not well established in the horse, but "proof-of-principle" studies have been published for selected nerves in normal horses.[8,18-20] High-resolution ultrasonography or magnetic resonance imaging may be used to help pinpoint the location of peripheral nerves and aid in precise stimulation and recording.[21]

MANAGEMENT

Nonsurgical Management

The objectives of medical treatment of peripheral nerve injury are (1) reduction of swelling and inflammation at the site of injury, (2) support and stabilization of the denervated area, and (3) maintenance of the general health of the animal.

In the case of lacerating trauma, the general principles of wound management apply. The wound should be cleaned, irrigated, débrided, and dressed and ventral drainage established. If severed nerve ends are visible, they can be tagged for later identification. Medical therapy for inflammation and swelling should be initiated, especially if there is obvious tissue swelling. This may include any combination of a cyclooxygenase inhibitor (e.g., flunixin meglumine, 1.1 mg/kg IV, IM, or PO once or twice daily), dimethyl sulfoxide (DMSO) (1 g/kg as a 10% solution IV or by nasogastric tube once or twice daily), and a glucocorticoid (e.g., dexamethasone, 0.05 mg/kg IV or IM once daily, or 0.1 mg/kg PO once daily). An additional anti-inflammatory effect can be provided by cold water hydrotherapy or local application of skin-permeant drugs such as DMSO or diclofenac. In the presence of an open wound or other risk for bacterial infection, broad-spectrum antimicrobial therapy should be initiated. This can be directed by culture results, if available.

Fractured or dislocated bones that have the potential to further injure nerves must be reduced and stabilized. Stall rest is essential to prevent further injury. In the case of paralyzed limbs, a protective wrap may be necessary to prevent damage caused by dragging or improperly placing the limb. A support wrap, splint, or light cast should be maintained on a leg affected with radial paralysis so as to prevent limb contracture. A heavy support wrap, possibly with additional fetlock support, should also be placed on the opposite limb to protect against breakdown caused by prolonged increased weight bearing. If the horse has difficulty alternating between recumbency and standing, it should be supported as necessary in an abdominal sling until it is able to rise unassisted. The newly developed Enduro-N.E.S.T. system (NASA equine support technology) (Figure 54-3), although restricted to referral facilities and rehabilitation centers, will obviate many of the problems associated with conventional slings and allow long-term support of valuable patients with multiple-limb or severe single-limb motor neuropathies. Because of the buoyancy provided by water, horses may be able to exercise affected limbs in swimming pools. Skin over bony prominences should be padded to prevent decubital injury; such injuries must be treated aggressively if they occur.

In foals, passive range-of-motion exercises should be performed on paralyzed limbs in an attempt to preserve joint flexibility and prevent contracture. Faradic or galvanic electrical muscle stimulation has been advocated for denervated muscles to help preserve muscle mass, and units designed for use in horses are available. This practice is controversial, however, and may possibly delay nerve regrowth into muscle.[22] Hyperbaric oxygen is an approved adjunctive treatment for acute traumatic ischemic reperfusion injury of humans and theoretically could help horses with postrecumbency neuropathies.

Surgical Management

Because neurosurgery in horses is at a rudimentary stage, it is difficult to provide precise rules for assessment and intervention after mechanical nerve injury. The following guidelines are based on comparable situations in human medicine.[1,2]

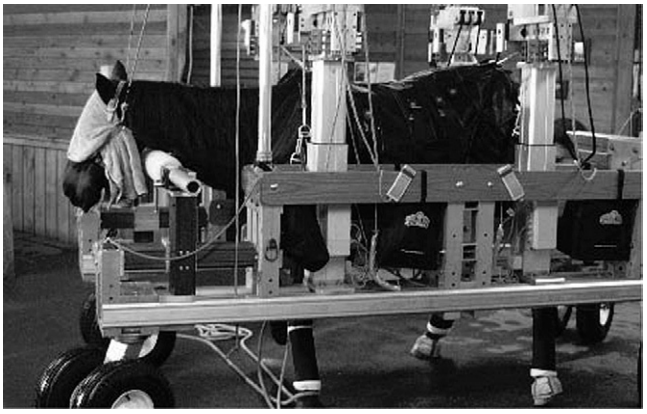

Figure 54-3. Fully anesthetized horse standing in the Enduro-N.E.S.T. sling system. Monoplegic and multiplegic horses can be safely supported indefinitely in this apparatus. The unit safely transduces friction energy to heat and can balance weight individually for each limb.

- In clean lacerating injuries in which the nerve ends are visible or when clinical examination reveals obvious motor and sensory deficits resulting from the injury at the laceration, primary repair may be indicated. In general, results of primary repair (within 3 to 4 days) are better than results of secondary repair (after 2 to 3 weeks).
- In nerve transections resulting from blunt lacerating trauma, secondary repair has a better neurologic result. This allows time for control of infection and resolution of soft tissue swelling.
- Injuries that do not demonstrate evidence of early spontaneous recovery should be explored surgically. This applies especially to traumatic injury of the suprascapular nerve or injection injury of the sciatic nerve. Empirically, the outer time limit for exploration can be estimated from the observations that muscle must be reinnervated within 12 months and that nerve regrows at the rate of 1 inch per month. According to these precepts, the length in inches from the injury site to the muscle supplied plus the time in months since the injury should not exceed 12. For example, if the peroneal nerve is injured 6 inches from its entry into the anterior tibial muscle, no more than 6 months should be allowed for spontaneous recovery of innervation.

Anastomosis

If the nerve is ruptured or severed, the retracted ends should be identified.[23] With the help of an operating microscope, the damaged portions of the nerve should be trimmed and the proximal and distal stumps aligned as accurately as possible by epineurial repair. Coaptation is achieved by simple-interrupted epineurial sutures (8-0 to 10-0 monofilament nylon or silk) around the circumference of the anastomosis. A perfect superficial alignment can be achieved using epineurial vessels as landmarks, but the internal orientation of fascicular bundles and individual fascicles may not be correct (Figure 54-4). The more technically difficult fascicular repair technique involves aligning and attaching individual fascicular groups. Epineurial tissue is resected from around the circumference of the nerve,

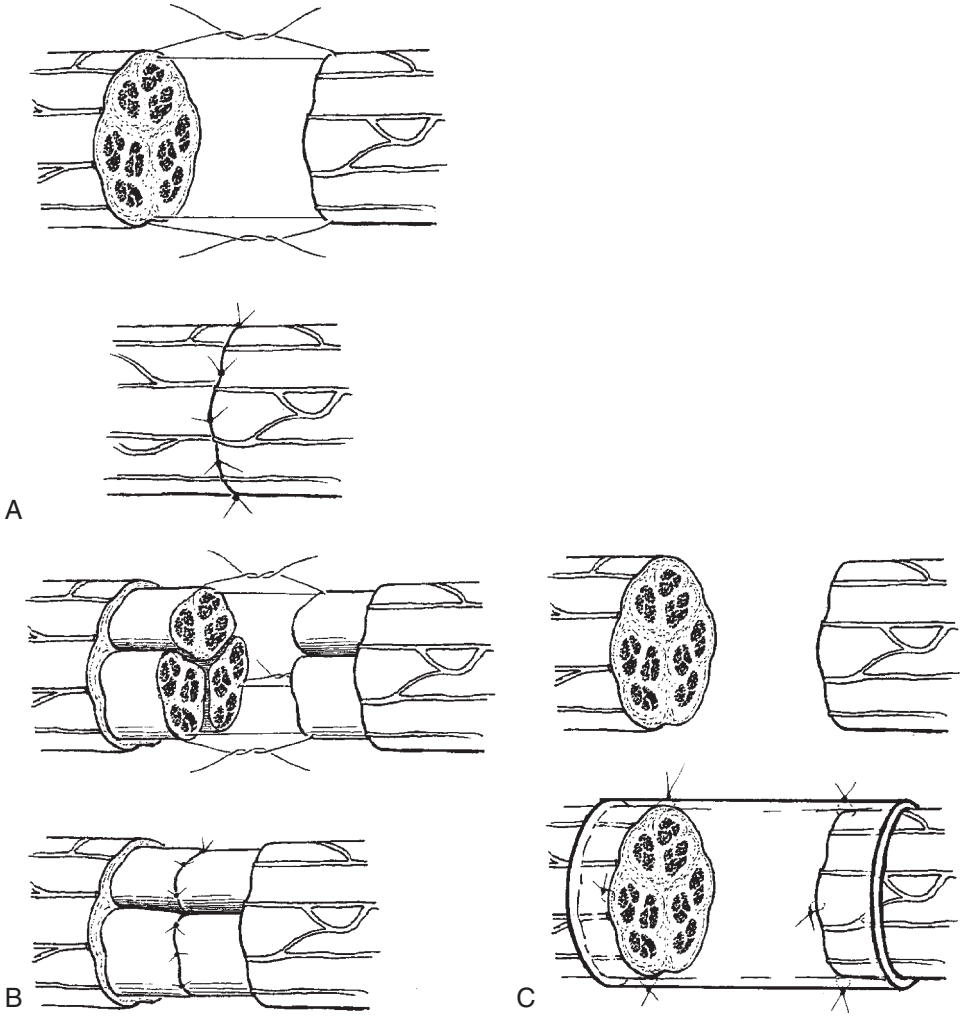

Figure 54-4. End-to-end repair of peripheral nerves. **A,** Epineural suture. Coaptation is achieved by single sutures in the epineurium around the circumference of the nerve. **B,** Group fascicular suture. Epineural tissue has been resected, and fascicular groups are coapted with single sutures in the perineurium or connective tissue surrounding groups of fascicles. **C,** Tubular repair. (Modified from Lundborg GA: 25-year perspective of peripheral nerve surgery: Evolving neuroscientific concepts and clinical significance. J Hand Surg Am 25:391, 2000.)

and fascicular groups are subsequently coapted with single sutures in the perineurium or connective tissue surrounding groups of fascicles. Nonsuture methods have been described and include wrapping the nerve, gluing the ends together with plasma clot or fibrin glue, or using a carbon dioxide laser to weld the nerve ends together.[24]

Tension at the anastomosis site must be minimized by mobilization of the nerve ends and postoperative rest and immobilization of the affected limb. If the gap between the ends is too large to permit anastomosis without tension, a conduit technique may be considered, wherein the defect is bridged in such a way as to support axonal growth toward the distal nerve segment. For this purpose, either a tube or a graft is used. Silicone tubes, freeze-thawed muscle tubes, and collagen-based nerve gap conduits have been used. Nerve autografts from a remote donor site (e.g., sural nerve) could also be used for bridging a long nerve gap. In nerve injuries in which the proximal segment cannot be used for grafting, the distal segment theoretically can be implanted in end-to-side fashion into an epineural window on an adjacent uninjured nerve. Similarly, the technique of direct neurotization of muscle has been validated experimentally in settings where nerve has been traumatically avulsed from muscle. A similar technique has been used in horses with left laryngeal hemiplegia. In this technique, a narrow strip of muscle with an intact nerve supply (the neuromuscular pedicle graft) is removed from the omohyoid muscle and inserted into the paralyzed dorsal cricoarytenoid muscle (see Chapter 45).[25] Over the subsequent months, the dorsal cricoarytenoid muscle becomes reinnervated and is stimulated to contract during exercise.

After surgical exploration of a nonregenerating nerve, the damaged section can be removed and the ends anastomosed using one of the techniques described earlier.

Neurolysis

External neurolysis refers to the dissection of the nerve from a bed of scar tissue.[1] This is indicated if there is obvious compression from fibrous tissue, and it is performed routinely in horses with suprascapular neuropathy (see Chapter 96).[13] Internal neurolysis involves an incision of the epineurium and resection of scar tissue from between nerve fascicles.

COMPLICATIONS

In response to trophic factors produced at the site of nerve injury, misdirected axonal sprouting and Schwann cell proliferation may produce bulblike neuromas, either at the proximal stump of a transected nerve (end-neuroma) or within a partially disrupted nerve (neuroma-in-continuity).[26] End-neuromas are a common complication of palmar digital neurectomy in horses treated this way for navicular syndrome (see Chapter 90).[27,28] Neuroma-in-continuity or fibrous entrapment at the site of injury or surgical repair is associated with a variety of pain syndromes in humans. On the basis of the documented painful local effect of neuromas after palmar digital neurectomy, it is likely that similar syndromes occur in some "nerved" horses. Neuropathic pain cannot be effectively controlled medically. Partial control may be achieved with nonsteroidal anti-inflammatory drugs, narcotic analgesics (morphine, butorphanol), antiepileptic analgesics (carbamazepine, gabapentin), or the tricyclic antidepressant imipramine.

In attempts to reduce neuroma formation in nerved horses, the proximal stumps of sectioned nerves have been exposed to CO_2 laser energy, injected with doxorubicin, heat-coagulated, entubulated with or without silicone, sealed with tissue adhesive or epineural caps, or anchored into the medulla of adjacent bone.[29] The most effective way to avoid postoperative nerve regeneration and associated clinical signs is reported to be guillotine transection of nerves under tension and removal of up to 10 cm of the nerve.[29]

In addition to permanent degenerative changes in muscle, fibrous replacement and contracture may result in reduced range of motion and permanent flexion of involved joints. Such an outcome is particularly likely after radial paralysis in the horse. Proper management by placement in a sling, prolonged stall rest, orthotic support, and passive range-of-motion exercises may help prevent such an outcome. Studies on the use of electrical stimulation of muscles have indicated that the function of denervated muscle can be preserved only if use-specific stimulation paradigms are developed.[22] Previous evidence suggested that electrical stimulation may reduce nerve regrowth into denervated muscle.

PROGNOSIS

With suprascapular neuropathy secondary to confirmed or suspected shoulder trauma, restoration of shoulder stability occurred within 3 to 12 months in seven out of eight horses treated only with stall rest.[30] Atrophy of the infraspinatus and supraspinatus muscles resolved in only two of these horses. Although there is no comparative study to show any additional effect of procedures to relieve entrapment of the suprascapular nerve, it is expected that these procedures should improve recovery of muscle mass.[13,14] Data for outcome of radial nerve neuropathy are not published; however, it is reasonable to assume that the prognosis for complete recovery from signs of radial paralysis induced by positioning in lateral recumbency, soft tissue trauma, and humeral fracture is good (greater than 80%), fair, and poor, respectively. Injection-associated neuropathy (usually sciatic) usually resolves in days.

It is reported that approximately 70% of end-to-end nerve repairs and 50% of graft repairs performed in humans achieve good functional results.[2]

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SECTION
VIII EYE AND ADNEXA

John A. Stick

CHAPTER
55 Surgery of the Globe and Orbit

Kenneth E. Pierce Jr. and Wendy M. Townsend

ANATOMY AND PHYSIOLOGY

The orbits are bony cavities that protect the eye and its associated muscles, blood vessels, and nerves.[1] Because of the myriad structures within and surrounding the orbit, one must clearly understand orbital anatomy to effectively diagnose and treat orbital disease. The equine orbit measures 62 mm in width, 59 mm in height, and 98 mm in depth.[1] The globe itself is 48 mm in width, 47 mm in height, and 44 mm in depth (Table 55-1).[1] The axes of the eyes and orbits are directed laterally 80 degrees off the midline, creating a panoramic field of vision.[1,2]

In horses, the orbital rim is a complete bony perimeter composed of the frontal bone dorsally, the lacrimal bone medially, and the zygomatic bone ventrally. The lateral border is formed by three fused bones—the descending zygomatic process of the frontal bone, the ascending frontal process of the zygomatic bone, and the zygomatic process of the temporal bone (Figure 55-1).[1] The supraorbital foramen, containing the supraorbital vessels and nerve, is located dorsolaterally within the zygomatic process of the frontal bone.[1-4] Along the medial rim of the orbit, the lacrimal sac is positioned within the lacrimal fossa, an indentation in the lacrimal bone.[2,5] The nasolacrimal duct then courses from the lacrimal sac through the lacrimal bone. Therefore, fractures or neoplasia involving the lacrimal bone can disrupt normal tear flow.

Caudal to the orbital rim, the lacrimal, sphenoid, and palatine bones form the medial orbital wall. The zygomatic arch forms the anterior aspect of the orbital floor. Periorbital fascia, orbital fat, and the pterygoid muscle form the remainder of the orbital floor. Multiple vessels and nerves course rostrad along the orbital floor to the pterygopalatine fossa rostroventrally. The fossa contains three foramina. The maxillary vessels and nerve pass through the maxillary foramen into the infraorbital canal. The sphenopalatine vessels and caudal nasal nerve pass through the sphenopalatine foramen toward the nasal cavity. The major palatine artery and nerve pass through the caudal palatine foramen to the palatine canal.[1,2] Therefore caution is required during surgical manipulations involving the orbital floor because of the potential for extensive hemorrhage and nerve damage.

The orbital apex contains four major foramina. The most dorsal is the ethmoidal foramen, through which the ethmoidal artery, vein, and nerve pass. Ventral and slightly caudal lies the optic foramen, through which the optic nerve and internal ophthalmic artery pass. The optic nerve (cranial nerve [CN] II) then travels within the extraocular muscle cone to insert on the globe.[2,3] The oculomotor (CN III), trochlear (CN IV), ophthalmic branch of the trigeminal (CN V), and abducens (CN VI) nerves pass through the orbital fissure, which lies ventral to the optic foramen. The oculomotor nerve innervates the levator

TABLE 55-1. Dimensions of Globes of Domestic Species

Animal	Meridional AP Axis of the Eye (mm)	Equatorial Axis (mm)	Horizontal Axis (mm)
Horse*	43.68	47.63	48.45
Mule*†	43.00 (42-44)	47.50 (47-48)	48.50 (48-49)
Donkey‡		44	52

*Values from reference 1.
†Values in parentheses from reference 2.
‡Values from reference 2.
AP, Anteroposterior.

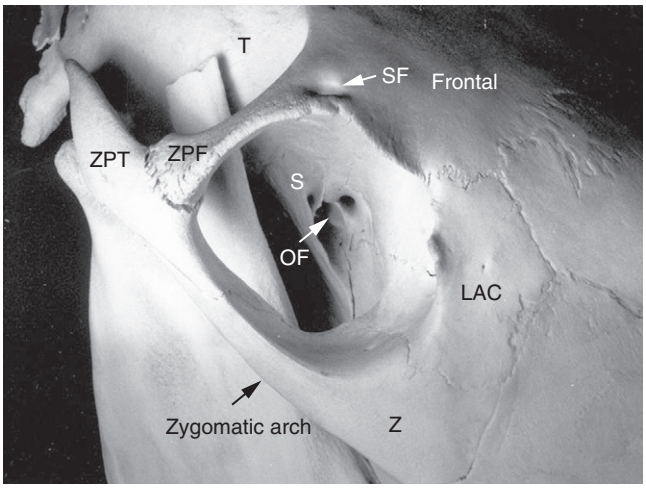

Figure 55-1. A normal skull, demonstrating the complete anterior bony orbital rim of the horse. The pertinent bones that form the orbit of the horse are labeled. *LAC*, Lacrimal bone; *OF*, orbital foramina including ethmoid, optic, orbital fissure, and rostral alar; *S*, sphenoid bone; *SF*, supraorbital foramen; *T*, temporal bone; *Z*, zygomatic bone; *ZPF*, zygomatic process of the frontal bone; *ZPT*, zygomatic process of the temporal bone.

TABLE 55-2. Extraocular Muscles, Scleral Insertions, and Innervation

Extraocular Muscle	Scleral Insertion*	Innervation
Dorsal rectus	9-13 (11.2) mm from limbus	Oculomotor n. (CN III)
Ventral rectus	8 (8.0) mm from limbus	Oculomotor n. (CN III)
Medial rectus	10 (14.2) mm from limbus	Oculomotor n. (CN III)
Lateral rectus	12 (4.0) mm from limbus dorsally, 5 mm ventrally	Abducens n. (CN VI)
Superior oblique	14 (14.2) mm from limbus dorsally, 23 mm ventrally	Trochlear n. (CN IV)
Inferior oblique	21 (4.8) mm from limbus dorsally, 3 mm ventrally	Oculomotor n. (CN III)
Retractor bulbi	37 mm from limbus dorsally 18 mm from limbus ventrally 18 mm from limbus medially 22 mm from limbus laterally	Abducens n. (CN VI)

CN, Cranial nerve; *n*, nerve.
*Extraocular muscle scleral insertion values are from references 2 (values in parentheses) and 3.

palpebrae superioris muscle; dorsal, medial, and ventral rectus muscles; and the ventral oblique muscle. It also provides parasympathetic innervation to the iris sphincter muscle.[2,3,6] The trochlear nerve (CN IV) innervates the dorsal oblique muscle.[2,3] The ophthalmic branch of the trigeminal nerve (CN V) provides sensation to the cornea, conjunctiva, and dorsal eyelid. The abducens nerve (CN VI) innervates the retractor bulbi and lateral rectus muscles. Caudoventral to the orbital fissure lies the rostral alar foramen through which the maxillary artery and nerve pass.[1,2,4,7] The majority of the orbital blood supply is provided by the external ophthalmic artery, a branch of the maxillary artery.[2,3,8]

The orbit contains the extraocular muscles responsible for globe movement. The scleral insertions and innervation of the extraocular muscles are listed in Table 55-2.[2,9] The dorsal, ventral, medial, and lateral rectus muscles originate from the orbital wall medial to the orbital fissure and move the eye as their names suggest.[2] The superior oblique muscle originates from the medial orbital wall, courses rostrad to pass through a trochlea at the medial canthus, and inserts on the dorsolateral sclera. The superior oblique rotates the dorsal aspect of the globe ventrad and mediad. The inferior oblique muscle originates from a shallow fossa close to the lacrimal canal, courses temporad, and inserts inferiorly adjacent to the insertion of the lateral rectus muscle. The inferior oblique muscle rotates the globe superiorly and mediad.[2] The retractor bulbi muscle, as the name indicates, retracts the globe. It originates from the orbital apex and inserts at the globe's equator.

A thin, tough connective tissue envelope (the orbital fascia) is also located within the orbit, which supports the globe, maintains anatomic relationships, and allows precise and coordinated globe movements.[1,3] The orbital fascia is divided into the periorbita, extraocular muscle fascia, and episcleral fascia (Tenon's capsule).[1-3] The periosteum that lines the orbital bones, optic foramen, and orbital foramen contributes to the periorbita and is continuous with the dural sheath of the optic nerve.[1,2,4] A portion of the periorbita separates from the periosteum and extends anteriorly to form the orbital septum, which merges with the tarsal plate at the eyelids.[1,2] The episcleral fascia, or Tenon's capsule, is the connective tissue layer between the bulbar conjunctiva and the sclera. Tenon's capsule is firmly adhered to the sclera at the limbus and extends to blend with the fascia of the extraocular muscles.[1-4] Orbital fat within and external to the extraocular muscle cone cushions the globe and fills dead space.

The lacrimal gland is situated within the periorbita dorsolateral to the globe and encased by orbital fat. The secretions form the aqueous phase of the tear film and are released into the dorsolateral conjunctival fornix via 12 to 16 ducts.[2,3] Caution should be used when incising the dorsolateral conjunctiva to ensure that the ducts are preserved and therefore tear flow is not jeopardized.

The nictitating membrane (third eyelid) is located within the ventromedial orbit. The third eyelid elevates passively as the globe retracts. Within this membrane lies a T-shaped cartilage with the gland of the nictitans surrounding its base.[2] This gland also contributes to the aqueous layer of the tear film. The third eyelid is frequently affected by squamous cell carcinoma.

Finally, numerous sinuses surround the bony orbit. The sphenopalatine sinus is located medial and ventral to the orbit near the optic chiasm.[10] The conchofrontal sinus is located medial to the orbit, extends dorsad and ventrad, and is composed of the frontal and dorsal conchal portions, which communicate.[5,9,11] The two maxillary sinuses are located ventral and axial to the orbit. The bony plate between the orbit and maxillary sinus is very thin.[4] Therefore processes may easily extend from the sinus into the orbit and vice versa. The landmarks for trephination of the maxillary sinus include (1) a vertical line tangential to the medial canthus, (2) the facial crest, (3) an oblique line drawn from the rostral facial crest to the infraorbital foramen, and (4) a line parallel to the facial crest intersecting the infraorbital foramen (see Chapter 43).[11] Trephination dorsal to a line drawn between the infraorbital foramen and the medial canthus may result in nasolacrimal duct damage.

RELEVANT OCULAR EXAMINATION TECHNIQUES AND FINDINGS

Initially a hands-off assessment of the head should be performed, including orbital symmetry, eyelid position, globe size and position, and pupil size. Abnormalities in globe position associated with orbital disease include strabismus (misalignment), exophthalmos (protrusion from the orbit), and enophthalmos (recession within the orbit) (Figure 55-2). The nares should be visually inspected for nasal discharge, air passing equally through both nostrils should be ensured, and the horse's visual status should be determined. Both globes should retain their normal range of motion. If the orbital rim is swollen or disfigured, digital palpation is indicated to identify potential displaced fractures or proliferative lesions. Additional clinical

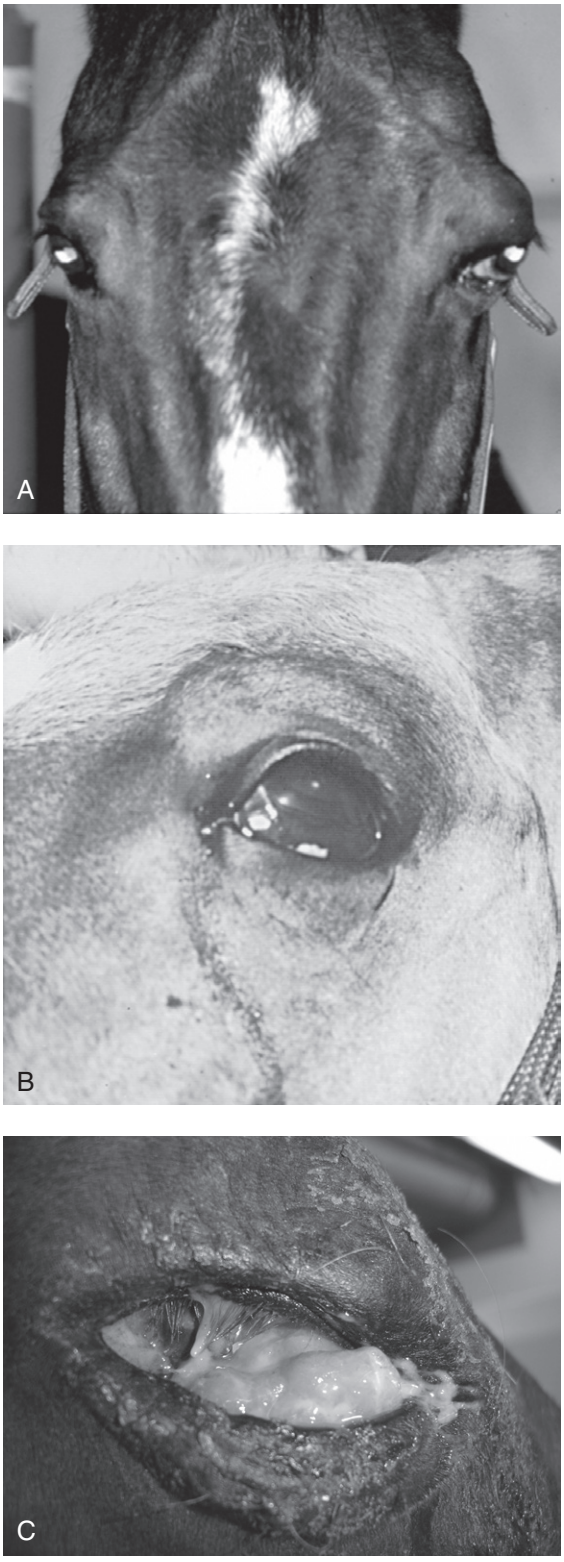

Figure 55-2. A, Exophthalmos in an aged gelding. Computed tomography revealed a sinus tumor to be the cause of the orbital disease. **B,** Dorsal strabismus caused by an orbital traumatic proptosis in this horse. **C,** Enophthalmos in an adult horse with orbital and adnexal squamous cell carcinoma.

signs may include epiphora, ptosis (drooping of the eyelid), reduced airflow from the ipsilateral nostril, reduced sinus percussion, bone or facial deformation, keratoconjunctivitis sicca, and supraorbital fossa distention.[12] The nictitating membrane may passively protrude with exophthalmos or enophthalmos. Corneal ulceration (exposure keratitis) may occur in severe cases of exophthalmos because the eyelids are unable to completely close over the cornea.

Orbital disease processes can occur within the extraocular muscle cone (intraconal), between the muscle cone and the periorbital sheath, or external to the periorbital sheath (subperiosteal).[13] The degree and direction of globe deviation depends on the size and location of the lesion. Intraconal lesions typically cause exophthalmos, whereas lesions of the medial orbit displace the globe laterally. Retropulsion of the globe through closed eyelids should assist in identifying space-occupying lesions.[14]

In the geriatric horse, orbital fat resorption will cause enophthalmos.[15] Fractures of the walls of the paranasal sinuses and orbit can result in enophthalmos, facial deformity, and orbital emphysema. Tooth root abscesses may cause sinus and orbital disease. It must be remembered that infectious,[16] traumatic,[17-19] inflammatory, parasitic,[20] cystic,[21,22] or neoplastic disease[23-36] processes involving the eyelids, paranasal sinuses, tooth roots, guttural pouches, or nasal cavity may extend into the orbit and produce orbital disease.

The globe size of the exophthalmic eye is normal and should not be confused with the globe enlargement (buphthalmos) noted in advanced equine glaucoma. Comparing the horizontal limbal diameter of the affected and contralateral eyes will aid in identifying buphthalmos. Corneal globosa (abnormal corneal curvature) reported in Rocky Mountain horses affected with anterior segment dysgenesis must also be distinguished from exophthalmos.[37] At the other extreme, one must differentiate phthisis bulbi (globe shrinkage) from enophthalmos (Figure 55-3).

Ocular motility must also be evaluated. Damage to the extraocular muscles, displaced fractures, and space-occupying lesions may entrap the globe within the orbit. The examiner should grasp the horse's halter and move the horse's head in a horizontal plane using gentle sweeping motions. The globes should remain relatively central within the palpebral fissures and move together in a yoked fashion. If the globe does not move normally, a topical ophthalmic anesthetic (0.5% proparacaine or

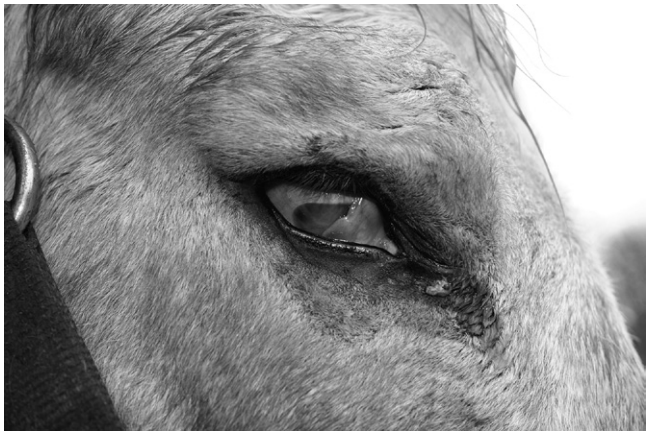

Figure 55-3. An adult horse with phthisis bulbi.

tetracaine ophthalmic solution) should be applied and the bulbar conjunctiva should be grasped gently with Bishop-Harmon forceps. The globe should be moved in the direction of interest to determine if a mechanical restriction is present. Simultaneously one should feel for globe movement initiated by the patient to confirm that the extraocular muscles are functional.

Sedation

Horses may require sedation, analgesia, and infiltration of a local anesthetic to complete a thorough ophthalmic examination. Typically the α_2-adrenoreceptor agonists detomidine or xylazine are used. Detomidine hydrochloride (10 mg/mL) administered at a dose of 0.02 mg/kg IV provides a steadier head position compared to xylazine and is useful for many minor ocular and periocular surgical procedures.[7,14,38,39] The addition of butorphanol (10 mg/mL) at a dosage of 0.01 to 0.02 mg/kg IV provides additional analgesia.[14,38-40] Confinement within stocks and application of a twitch may also aid in completing the examination.

Regional Anesthesia

Regional anesthesia greatly facilitates the ocular examination, particularly for painful conditions or a severely compromised globe. Lidocaine hydrochloride 1% or 2%, mepivacaine hydrochloride 2%, or bupivacaine hydrochloride 0.75% are typically used. Lidocaine has an intermediate potency and duration (45 to 180 minutes) with a rapid onset of action (5 minutes).[40-42] Mepivacaine is similar but has a slightly longer duration of action (120 to 180 minutes).[41,42] Bupivacaine is highly potent and has a long duration of action (180 to 500 minutes).[41-43]

Akinesia of the eyelids can be achieved by blocking the auriculopalpebral nerve, which innervates the orbicularis oculi muscle. By diminishing the ability to squint, the auriculopalpebral block greatly facilitates examination and is critical for globes in danger of rupture. The auriculopalpebral nerve, the palpebral branch of the facial nerve, can be blocked at three locations (Figure 55-4): (1) at the depression just anterior to the base of the ear, where the nerve emerges from the parotid salivary gland and becomes subcutaneous between the caudal border of the coronoid process of the mandible and the zygomatic process of the temporal bone; (2) lateral to the highest point of the caudal zygomatic arch where the palpebral nerve can be palpated or "strummed" under the skin over the dorsal border of the bone (Figure 55-5); and (3) just caudal to the bony process of the frontal bone where the palpebral nerve lies on the zygomatic arch.[14,44-46] At the location of choice, 1 to 2 mL of anesthetic should be injected using a 25-gauge, 1.58-cm needle, and then the injection site should be massaged to facilitate drug diffusion.[14,44-46]

Analgesia of the central two thirds of the upper eyelid can be achieved by blocking the supraorbital nerve, a branch of the frontal nerve of the ophthalmic branch of the trigeminal nerve (CN V). The nerve should be blocked as it emerges from the supraorbital foramen, which can be easily palpated as a small depression 2.5 cm above the free margin of the supraorbital process (Figure 55-6). A 25-gauge, 1.58-cm needle should be used to inject 1 to 2 mL of anesthetic. The use of a longer needle is discouraged because it could pass through the foramen and penetrate the sclera.

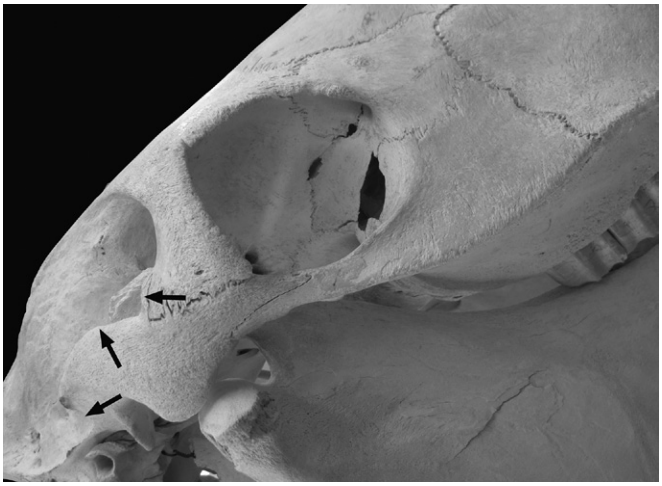

Figure 55-4. The three locations for auriculopalpebral nerve block indicated by the *black arrows*.

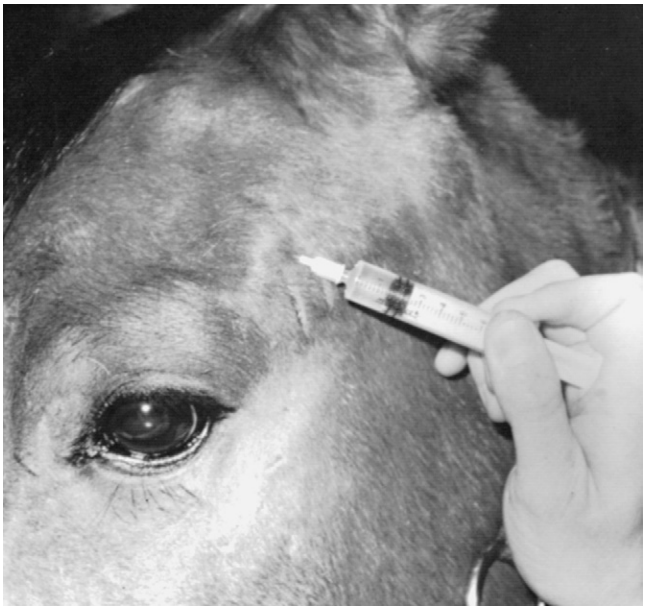

Figure 55-5. Auriculopalpebral nerve block anesthetic placement.

DIAGNOSTIC PROCEDURES

Retrobulbar Aspiration

Retrobulbar aspirates and biopsies allow collection of samples for culture, cytology, and histopathology from cases with space-occupying orbital masses. The site of aspiration varies according to the location of the lesion within the orbit. The needle or biopsy punch should be directed through the supraorbital fossa (see the description of retrobulbar nerve blocks in "Anesthetic Considerations," later) or through the conjunctiva. The procedure is often performed while the horse is standing and heavily sedated. Ultrasouznd guidance may facilitate placement of the needle within the mass and reduce the risk of postprocedural complications.[47] Care should be taken to prevent damage to the globe, orbital vessels, and optic nerve. Diagnostic imaging should be performed before collecting retrobulbar aspirates to avoid creating artifacts on the imaging study from the needle passage.

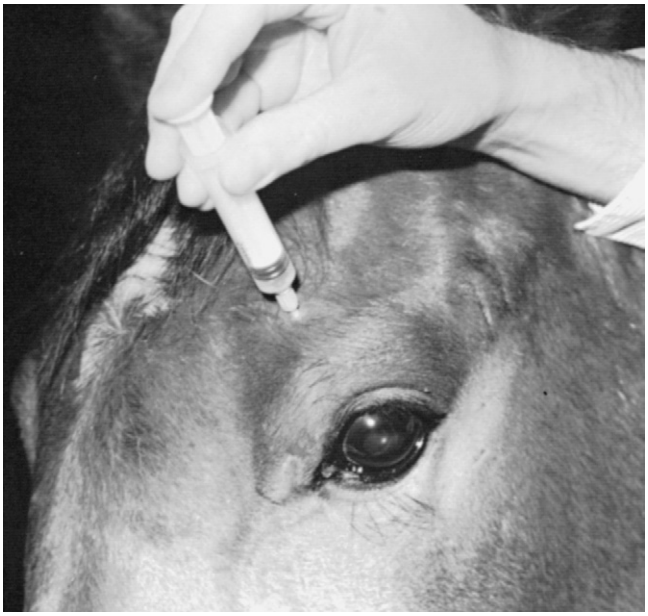

Figure 55-6. Supraorbital nerve block anesthetic placement.

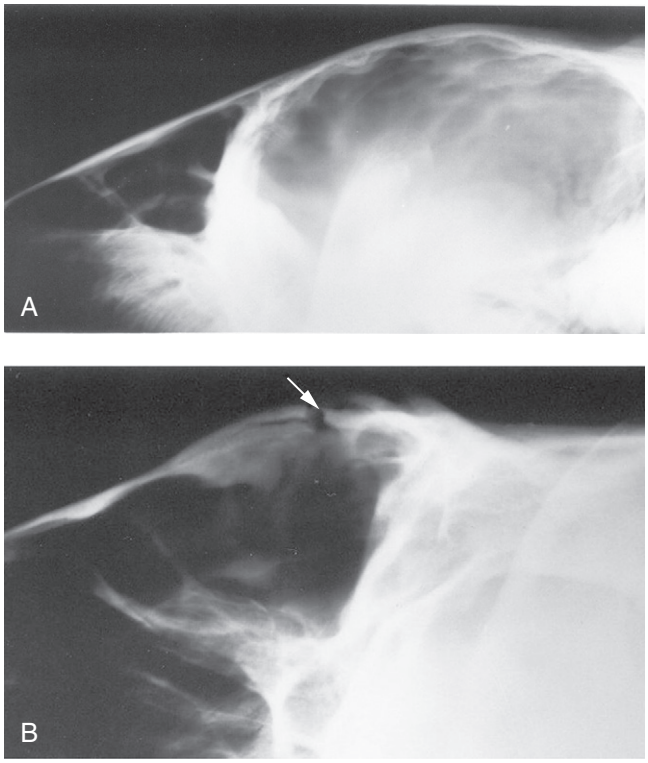

Figure 55-7. A, An orbital fracture is difficult to visualize on a lateral skull radiograph. **B,** The oblique radiographic view shows it well (*arrow*).

Diagnostic Imaging

Radiography

Radiographic examination of the orbit, paranasal sinuses, and nasal cavity can be an effective diagnostic tool (Figure 55-7). However, the superimposition of complex anatomic structures can make accurate diagnoses difficult to obtain.[48] Lesions involving the orbit warranting skull radiography include orbital and facial bone fractures, invasive bone-deforming neoplasia, a gas-forming abscess or cellulitis, exophthalmos, and metallic foreign bodies.[19,49-52] Multiple views, particularly oblique images highlighting the area of interest, should be obtained.[49-53] In the evaluation of unilateral lesions, the involved portion should be placed against the film cassette. Most lateral, dorsoventral, and oblique views of the equine orbit can be obtained with standing sedation. Ventrodorsal views of the orbit will require general anesthesia. A Flieringa ring, which is a stainless steel ring, can be placed at the limbus to help identify the globe within the orbit.

Ultrasonography

Ultrasonography is a useful, noninvasive diagnostic procedure that can usually be performed on the unsedated or sedated standing horse. It can be useful in evaluating exophthalmic globes; suspected retrobulbar or intraocular masses,[25,28,30,54,55] cysts,[22] abscesses,[56] or foreign bodies[57]; and globes obscured by marked eyelid swelling or with opacification of the ocular media (Figure 55-8). Brightness mode (B-scan) is most often used for clinical examination because it creates real-time two-dimensional, cross-sectional images.[58] Ultrasound probes of 6 to 10 MHz provide the best resolution and can penetrate the orbit to a depth of 3 to 7 cm.[57,59,60] The ultrasonic transducer can be placed in contact with the eyelids via a methylcellulose coupling agent to evaluate the globe and anterior orbit, or it can be placed in gentle contact with the cornea after instillation of a topical anesthetic to evaluate the posterior orbit.[61] The orbit can also be imaged through the supraorbital fossa. Abscesses appear as variably hypoechoic areas, whereas neoplasms usually appear more hyperechoic.[61] Both typically cause a markedly abnormal or asymmetric appearance of the affected area.[61] Foreign bodies often hyperechoic.[57] The opposite globe and orbit can provide a normal "control" for comparison.

Computed Tomography

Computed tomography (CT) creates cross-sectional, two-dimensional slices (i.e., tomograms) by measuring the attenuation of x-ray beams as they penetrate the tissue of interest from multiple angles.[48,62] These slices are then reconstructed in sagittal, dorsal, and oblique planes as desired.[48] The normal CT anatomy of the horse has been published,[63,64] and the reader is also referred to Chapter 70 for further information. Three-dimensional reconstructions of a CT study can be created, which are excellent for surgical planning and client education (Figure 55-9).[48] CT is an excellent modality for evaluating bone lesions and is superior to radiographs in evaluating soft tissues.[65] The large amount of orbital fat surrounding the globe, extraocular muscles, and associated nerves aids in identifying orbital soft tissue because it functions as a physiologic contrast agent.[57] Intravenously administered iodinated contrast medium accumulates in areas of increased perfusion and can therefore be used to delineate the margin between normal and abnormal tissues.[48,57,66] The drawbacks associated with CT imaging are its restricted availability and requirement for general anesthesia.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is another viable option for obtaining advanced imaging detail of the equine orbit, sinuses, and brain. MRI provides superior soft tissue detail of orbital

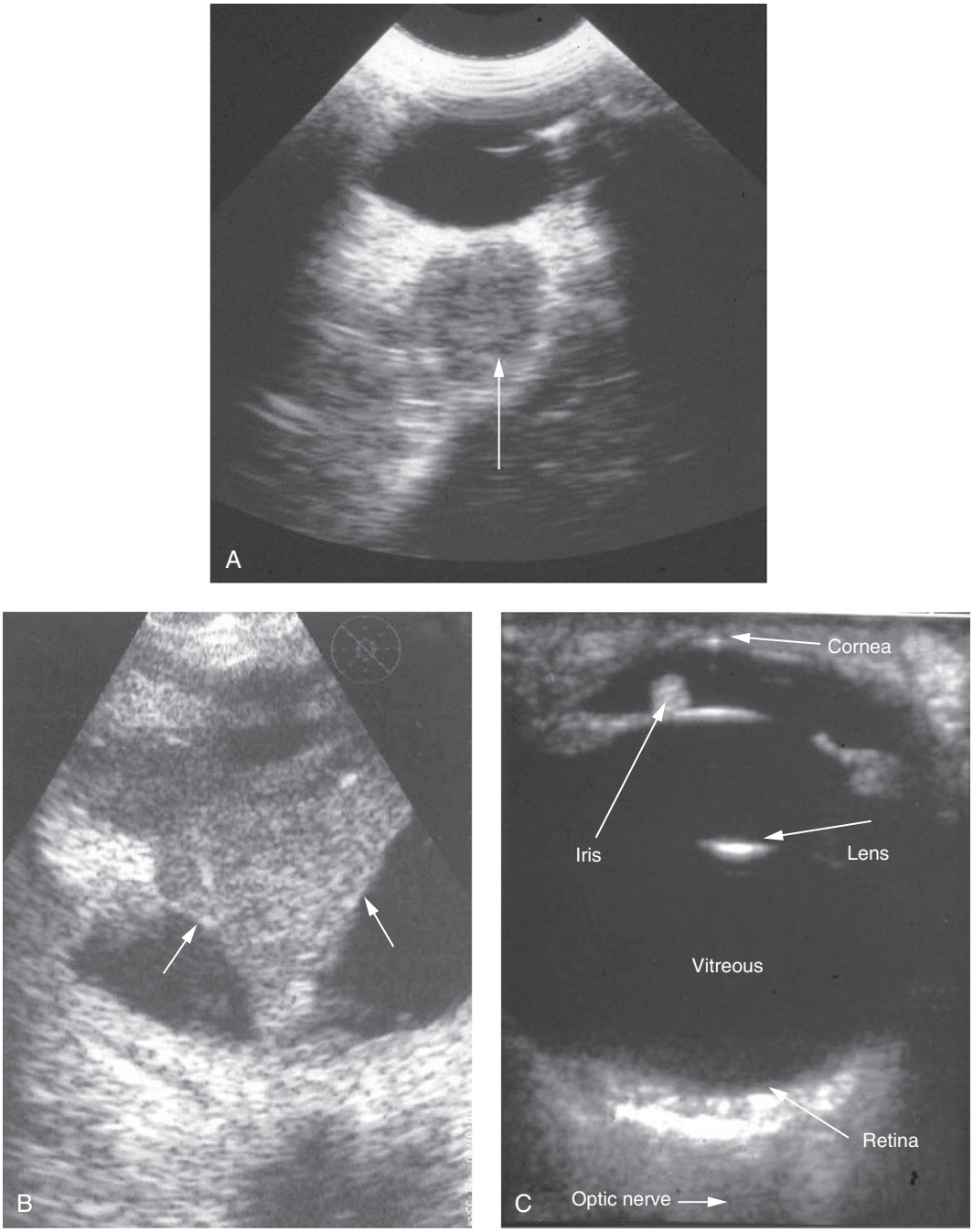

Figure 55-8. A, B-scan ultrasonographic view reveals an orbital optic nerve tumor (*arrow*). **B,** This ultrasonographic scan reveals a retinal detachment that resulted from severe eye trauma (*arrows*). The vitreous is opaque from hemorrhage. **C,** A normal B-scan ultrasonographic image is shown for comparison.

structures (Figure 55-10). MRI images are created by radiowaves emitted from hydrogen protons as they return to an equilibrium orientation within a magnetic field after being stimulated by a radiofrequency pulse.[67] A paramagnetic contrast agent can be used to identify increased vascular permability within the tissue of interest. Please see Chapter 71 for details regarding the various pulse sequences and their use. Similar to CT, MRI requires general anesthesia and has limited availability.[62,67] However, MRI provides better image resolution, anatomic detail, and tissue characterization of the orbital structures when compared to CT and does not rely on ionizing radiation.[67] The disadvantages of MRI include longer acquisition times, increased tissue slice thickness, poorer spatial resolution, and decreased ability to detect cortical bone defects or soft-tissue mineralization.[67] The reader should refer to the listed references for details of interpreting MRI images of the orbit, sinuses, and brain.[62,68-71]

ANESTHETIC CONSIDERATIONS

Equine anesthesia for ocular procedures remains challenging. Anesthesia for ocular surgery has a higher complication rate because of an increased incidence of movement under anesthesia and unsatisfactory recoveries associated with multiple attempts to stand.[72] The use of an anesthetic gas analyzer

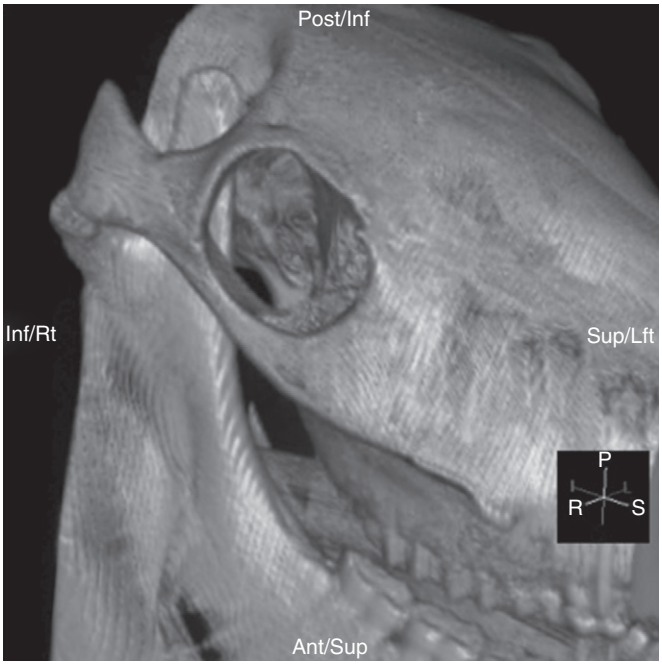

Figure 55-9. 3-D CT reconstruction of an adult equine orbit and skull.

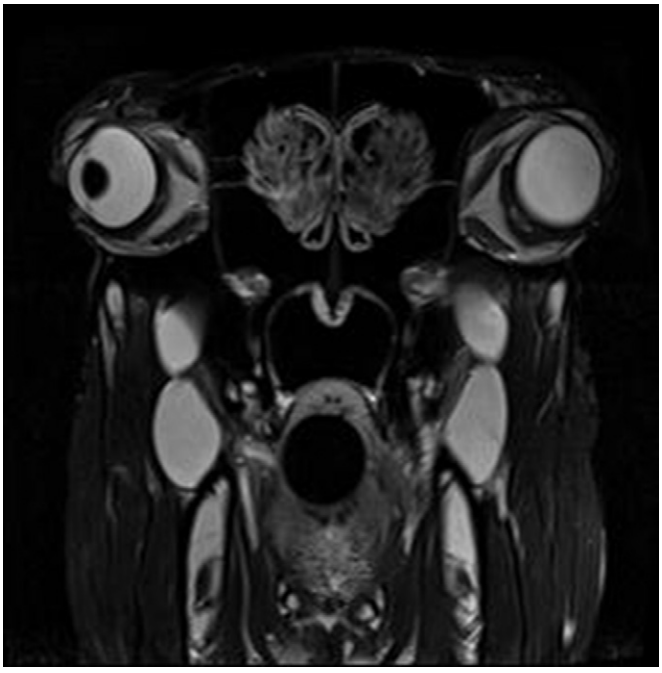

Figure 55-10. T2-weighted MRI image of a normal adult horse.

significantly reduces the incidence of movement.[72] Movement often occurs because of the persistence of palpebral and corneal reflexes and changes in globe position despite a deep plane of anesthesia. Administration of neuromuscular blocking agents can decrease these movements and thereby avoid a deep plane of anesthesia and its associated complications.[73] Retrobulbar nerve blocks can also be used to abolish the unwanted movement.[74] In our experience, even the minimal addition of a topical anesthetic agent (proparacaine or tetracaine ophthalmic solution 0.5%), a supraorbital block, and an auriculopalpebral block can relieve some of the unwanted movement.

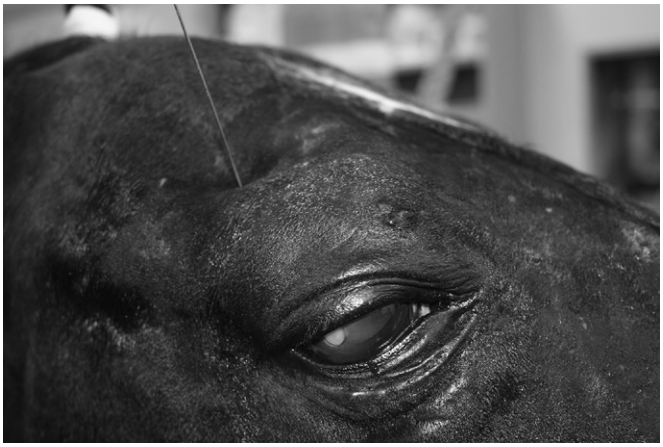

Figure 55-11. A retrobulbar nerve block placed in the supraorbital fossa immediately caudal to the posterior aspect of the dorsal orbital rim in a horse with a corneal stromal abscess.

Alternatively, procedures such as enucleation can be performed with standing sedation if the horse's temperament allows it. Flunixin meglumine (1.1 mg/kg IV) should be administered preoperatively.[75] The horse should be placed in stocks and sedated with detomidine (0.01 mg/kg IV) and butorphanol (0.03 mg/kg IV). Sedation should be maintained throughout the procedure with a detomidine constant-rate infusion (0.6 µg/kg/min).[75] The head should be suspended from cross ties after padding the halter or the chin can be rested on a padded table or dental rest. Regional anesthesia is crucial. A topical anesthetic should be applied to the corneal surface. Tetracaine 0.5% ophthalmic gel has been shown to provide superior analgesia for a longer duration when compared to proparacaine ophthalmic solution.[76] Auriculopalpebral, supraorbital, and retrobulbar blocks should be performed. The remainder of the eyelid margin can be desensitized through blocks of the infratrochlear and lacrimal nerves or placement of line blocks.

Several approaches have been described to perform a *retrobulbar nerve block*. For all, the injection site must first be prepared for aseptic surgery. Potential complications associated with retrobulbar blocks include orbital abscess formation; orbital cellulitis; laceration of the extraocular muscles, optic nerve, sclera, or ophthalmic artery; retrobulbar hemorrhage; optic neuritis; and elicitation of the oculocardiac reflex.[77]

In one approach, a 22-gauge, 6.35-cm (2½-inch) spinal needle is inserted into the supraorbital fossa immediately caudal to the posterior aspect of the dorsal orbital rim (Figure 55-11).[78] The needle should be oriented perpendicular to the skull and advanced until it reaches the retrobulbar muscle cone. A slight dorsal movement of the eye and/or a "popping" sensation should occur as the needle passes through the muscle cone. Once the needle is positioned, aspiration should be performed before injecting 10 to 12 mL of local anesthetic. Accurate anesthetic placement results in slight exophthalmia.

In a modification of the Peterson block an 18-gauge, 10.16 cm (4-inch) spinal needle is molded to a curvilinear shape.[74] The needle should then be inserted 1 cm lateral to the lateral canthal margin and directed in a ventromedial path in a line parallel to the contralateral medial canthus. The local anesthetic should be injected after needle placement and negative pressure aspiration.

A four-point nerve block can also be applied.[79] A slightly curved 20-gauge, 7.62 cm (3-inch) spinal needle should be used

to place 5 to 10 mL of local anesthetic in each quadrant of the orbit. The needle should be passed through the conjunctival fornix or eyelid and curved around the globe to the extraocular muscles and retrobulbar space. The ventral injection should be directed slightly nasally to avoid the optic nerve. Because of the large total volume injected, this technique can increase pressure on the globe and increase the risk of corneal perforation in a compromised globe.[79] Marked conjunctival swelling can occur if one inadvertently injects anterior to the orbital septum.[79]

REQUIRED SURGICAL EQUIPMENT

The instruments required for orbital surgeries include many of the standard instruments used for soft tissue surgeries. Magnification via head loupes may aid in visualization of tissues and suture material. The addition of ophthalmic instruments such as Bishop-Harmon tissue forceps, Stevens tenotomy scissors, and Castroviejo needle holders should facilitate manipulation of the conjunctiva and sclera (Figure 55-12). Osteotomes, an oscillating bone saw, and pneumatic drill will be required for resecting the zygomatic arch or other orbital bones (Table 55-3).

SURGICAL TECHNIQUES

For all ocular surgeries, the surgical site should be prepared with a 1:50 dilution of 10% povidone-iodine solution (Betadine), and the ocular surface should be flushed with this solution. The conjunctival fornices should be swabbed with sterile cotton-tipped applicators soaked in povidone-iodine solution. Alcohol, scrubs, and chlorhexidine are toxic to the corneal epithelium and therefore their use should be avoided. The surgical site should be draped routinely.

Evisceration and Intrascleral Prosthesis

An evisceration involves the surgical removal of the uveal tract, lens, vitreous, and retina from the fibrous tunic (cornea and sclera), which remains intact. Evisceration is a cosmetic surgical procedure for blind and painful eyes. It is not appropriate for eyes with septic endophthalmitis, intraocular neoplasia, or marked phthisis bulbi. Globes with preexisting corneal disease have an increased risk of complications postoperatively. A conjunctival advancement graft can be placed concurrently in cases with corneoscleral laceration.[80]

Surgical Procedure

A lateral canthotomy should be performed to improve exposure of the surgical field. Using tenotomy scissors, the conjunctiva should be incised parallel to and 68 mm posterior to the limbus to create a 180-degree peritomy (Figure 55-13).[81] The sclera should be exposed by bluntly undermining the conjunctiva, taking care not to undermine the insertions of the extraocular muscles (Figure 55-14). A No. 15 scalpel blade or No. 64 microsurgical blade should be used to make a 1.5-cm full-thickness incision in the sclera 4 to 5 mm from the limbus. Suction and cautery should be used to control extensive hemorrhage and visualize the incision. An evisceration spoon should then be inserted into the suprachoroidal space to separate the uvea from the sclera. The scleral incision should then be extended to 180

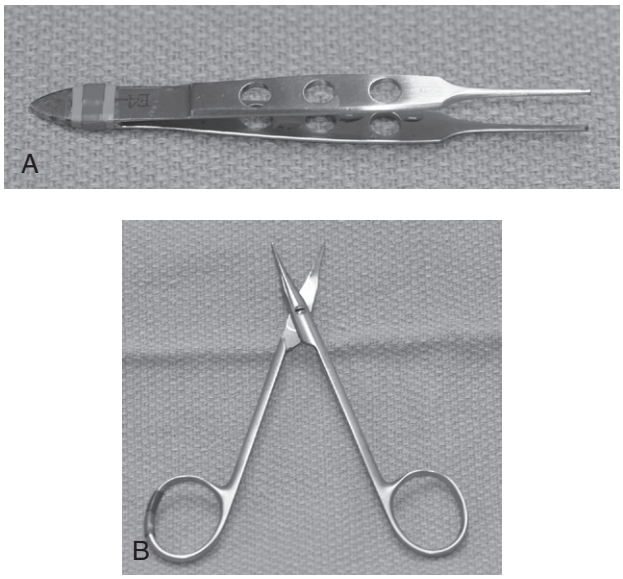

Figure 55-12. A, Bishop-Harmon tissue forceps. **B,** Stevens tenotomy scissors.

TABLE 55-3. Surgical Equipment per Procedure

Procedure	Instruments	Quantity
Evisceration	Backhaus towel clamps	4
	Eyelid speculum	1
	Bishop-Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	1
	Castroviejo or Jameson caliper	1
	Stevens tenotomy scissor (4 inches, straight or curved)	1
	No. 64 Beaver blade or No. 15 scalpel blade	1
	Beaver handle (5 inches) or scalpel handle	1
	Bunge evisceration spoon (5¼ or 5½ inches)	1
	Tissue forceps (6¼ inches)	2
	Silicone intraocular prosthesis (34 to 44 mm)	1
	Castroviejo needle holder (14 cm, straight or curved, with or without lock)	1
	Suture scissors (any type)	1
	6-0 polyglactin 910 (Vicryl)	1
	± Lens loop	1
	± Carter sphere introducer	1

Continued

TABLE 55-3. Surgical Equipment per Procedure—cont'd

Procedure	Instruments	Quantity
Enucleation	Backhaus towel clamps	4
	2-0 or 3-0 nylon suture (transpalpebral technique)	1
	No. 10 and/or No. 15 scalpel blade	1 of each
	Electrocautery	1
	Mayo scissors (serrated or nonserrated)	1
	Metzenbaum scissors (5 to 11 inches, standard or delicate, straight or curved)	1
	Suture scissors (any type)	1
	Adson-Brown tissue forceps (4¾ inches, 7 × 7 teeth)	2
	Bishop-Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	1
	Hemostatic forceps (standard or mosquito, straight or curved)	2-4
	± Jameson muscle hook	1
	± Rochester-Carmalt hemostatic forceps (20 to 25 cm, straight or curved)	2-4
	± Satinsky vascular clamp (27.3 cm)	1
	Silicone intraocular prosthesis (3 to 44 mm), equine conforming intraorbital implant: top hat or suture meshwork	1
	± Rongeurs and/or osteotome and mallet	1 of each
	Needle holder (5 inches, any type)	1
	3-0 or 4-0 absorbable suture	1-2
	4-0 nonabsorbable suture	1
Subconjunctival technique	Eyelid speculum	1
	Stevens tenotomy scissor (4 inches, straight or curved)	1
Exenteration	Backhaus towel clamps	4
	2-0 or 3-0 nylon suture	1
	No. 10 and/or No. 15 scalpel blade	1 of each
	Electrocautery	1
	Stevens tenotomy scissor (4 inches, straight or curved)	1
	Mayo scissors (serrated or nonserrated)	1
	Metzenbaum scissors (5 to 11 inches, standard or delicate, straight or curved)	1
	Suture scissors (any type)	1
	Adson-Brown tissue forceps (4.75 inches, 7 × 7 teeth)	2
	Hemostatic forceps (standard or mosquito, straight or curved)	2-4
	± Rochester-Carmalt hemostatic forceps (20 to 25 cm, straight or curved)	2-4
	± Satinsky vascular clamp (27.3 cm)	1
	± Periosteal elevator	1
	± Rongeurs and/or osteotome and mallet	1 of each
	Needle holder (5 inches, any type)	1
	3-0 or 4-0 absorbable suture	1-2
	4-0 nonabsorbable suture	1
Orbitotomy	Backhaus towel clamps	4
	No. 10 and/or No. 15 scalpel blade	1 of each
	Electrocautery	1
	Stevens tenotomy scissor (4 inches, straight or curves)	1
	Iris scissors (9.5 to 11.5 cm, straight or curved)	1
	Metzenbaum scissors (5 to 11 inches, standard or delicate, straight or curved)	1
	Suture scissors (any type)	1
	Adson-Brown tissue forceps (4¾ inches, 7 × 7 teeth)	2
	Bishop-Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	1
	Gelpi retractor	2-4
	Hemostatic forceps (standard or mosquito, straight or curved)	2-4
	± Jameson muscle hook	1
	± Oscillating bone saw	1
	± Pneumatic drill	1
	± Rongeurs and/or osteotome and mallet	1 of each
	± 20-gauge cerclage wire	1 spool
	± Wire cutter	1
	Needle holder (5 inches, any type)	1
	3-0 or 4-0 absorbable suture	1-2
	4-0 nonabsorbable suture	1

Figure 55-13. A conjunctival incision being performed 6 to 8 mm posterior to the superior limbus.

Figure 55-14. Superior scleral exposure after performing the conjunctival incision and blunt dissection.

degrees with scissors. The uveal tract should be grasped crosswise with a pair of blunt-tipped forceps and gentle traction applied with a rocking motion to deliver the uvea, retina, and vitreous. The lens can be removed with the evisceration spoon or a lens loop. Care should be taken to avoid scraping the corneal endothelium. The intraocular contents should be submitted for histopathologic examination if neoplasia is suspected. A suitably sized silicone implant (usually 34 to 44 mm in diameter) (Jardon Plastics Research Corporation, Southfield, MI) should be inserted into the sclera. A sphere introducer will facilitate implantation. The scleral incision should be apposed without undue tension. Preoperative ultrasonic measurement of the anterior-posterior dimension of the contralateral eye will aid in the selection of the proper-sized implant.[82] A simple-interrupted pattern of 6-0 absorbable suture should be placed in the sclera. A simple-continuous pattern of 6-0 absorbable suture should be placed in the conjunctiva. The lateral canthotomy should be closed routinely.

Aftercare

Topically applied antibiotics and systemically administered antibiotics and nonsteroidal anti-inflammatories should be used postoperatively. A temporary tarsorrhaphy should protect the cornea from exposure and mechanical abrasion while postoperative eyelid swelling subsides. The typical time for healing is 6 to 8 weeks.

Complications

Postoperative complications include corneal ulceration, endophthalmitis, and sugical site dehiscence.[12,82,83] Corneal perforation or incisional dehiscence can allow extrusion of the implant.[84] Enucleation is the only recourse if this occurs. Protective headgear should help prevent damage from head rubbing. Placement of a too-small implant will result in a cosmetically unacceptable eye. Owners should be warned that corneal opacification and vascularization occur in some animals and create a less-satisfactory cosmetic appearance. Tinted contact lenses can be placed for short periods to mask corneal opacification for show purposes.[83]

Enucleation

Enucleation involves the surgical removal of the palpebral margins, nictitans, conjunctiva, and globe.[85] This is one of the most commonly performed orbital surgeries. Enucleation is indicated for blind painful globes, severe corneal or intraocular infection, intraocular neoplasia, and traumatized globes not amenable to surgical repair. There are two surgical approaches: transpalpebral and subconjunctival.[85] An orbital prosthesis may be placed to improve cosmesis (Figure 55-15). If a visual eye must be removed, an opaque mask or full cup blinker should be placed for a day or two preoperatively to allow the horse to adjust to the vision loss and facilitate a smoother anesthetic recovery.

Transpalpebral Enucleation with Intraorbital Prosthesis

The conjunctiva, globe, and nictitating membrane are removed en masse. This approach prevents contamination of the orbit if neoplasia or severe ocular infection are present. However, it leaves a large orbital soft-tissue defect.[85,86]

The eyelids should be sutured together with 2-0 to 3-0 nonabsorbable suture in a continuous pattern (Figure 55-16, *A* to *D*). An elliptical, full-thickness skin incision should be created around and 5 mm posterior to the eyelid margins using a No. 15 scalpel blade. Using Metzenbaum scissors, blunt and sharp subcutaneous dissection should be performed posteriorly, taking care to avoid incising the conjunctival sac. The firm medial and lateral canthal ligaments should be transected with heavy gauge scissors. Care should be taken to avoid the angularis oculi vein at the medial canthus. Blunt dissection should be continued along the sclera with transection of the extraocular muscles at their tendinous insertions, thus minimizing hemorrhage. The retractor bulbi muscle should be blindly transected using curved, serrated Mayo scissors. Clamping of the optic nerve with either a Rochester-Carmalt forceps or Satinsky vascular clamp is optional. Curved, serrated Mayo scissors should be used to transect the optic nerve while avoiding incising the globe. Also, one should avoid excess tension on the optic nerve, which could traumatize the optic chiasm and result in vision loss in the contralateral eye.

At this point a silicone implant (equine conforming intraorbital implant) (Figure 55-17), polymethyl methacrylate sphere (34 to 40 mm in diameter), or suture meshwork can be placed

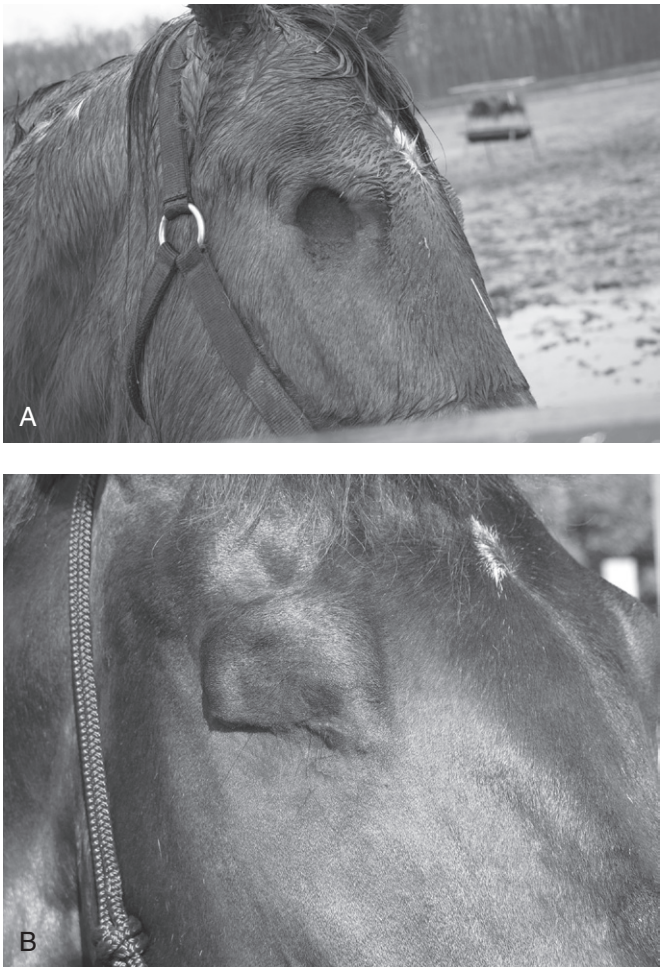

Figure 55-15. Image of two adult horses several years after enucleation. **A,** Without an intraorbital prosthesis. Note the degree of "pitting." **B,** With an intraorbital prosthesis. Note the more cosmetic appearance.

within the orbit to improve postoperative cosmesis.[80,81,87-89] An 8.6% complication rate with intraorbital infection and implant extrusion has been reported for intraorbital prostheses in dogs, cats, and horses.[84] An orbital implant should not be placed in the presence of infection or neoplasia involving the remaining structures within the orbit. The orbital septum should be closed in a simple interrupted pattern with 3-0 absorbable suture, which secures the prosthesis within the orbit and assists with hemostasis. The subcutaneous layer should be closed in a simple continuous pattern with 3-0 or 4-0 absorbable suture. The skin should be closed in a simple interrupted or continuous pattern with 4-0 nonabsorbable suture material.

Subconjunctival Enucleation

The subconjunctival approach is quicker, is associated with less hemorrhage, and removes less orbital tissue.[85] The subconjunctival technique maintains the eyelid margins and therefore must be used when placing a cosmetic shell. Patients with ocular disease confined within the globe should be excellent candidates for this approach.

An eyelid speculum should be placed and a lateral canthotomy performed to improve surgical exposure. The bulbar conjunctiva should be incised 5 mm posterior to the limbus using blunt-tipped Stevens tenotomy scissors and a 360-degree peritomy should be performed (see Figure 55-16, *E* and *F*). All four quadrants should be undermined posteriorly between Tenon's capsule and the sclera with tenotomy scissors and Bishop-Harmon forceps. The extraocular muscles should be isolated and transected at their tendinous insertions on the globe. The retractor bulbi muscle and optic nerve should be transected as described earlier (Figure 55-18). The orbit can be temporarily packed with sterile gauze to provide hemostasis. The third eyelid, gland, and associated conjunctiva should be completely excised with scissors, along with 3 to 5 mm of the lid margin and associated meibomian glands. The remaining conjunctiva should be excised to prevent mucocele formation. Placement of an intraorbital prosthesis and closure of the surgical site should be performed as described previously.[12,82,83,90]

Aftercare

All enucleated globes should be submitted for histologic evaluation. A pressure bandage should be placed immediately postoperatively to assist with hemostasis and reduce postoperative swelling. Slight epistaxis may occur postoperatively. Systemically administered antibiotics and nonsteroidal anti-inflammatory drugs should be beneficial. Most horses should be able to return to their original occupation after unilateral enucleation.[91]

Complications

Complications include intraoperative globe rupture, severe intraoperative and postoperative hemorrhage, orbital cyst formation resulting from incomplete removal of secretory orbital tissue (i.e., lacrimal glands and conjunctiva), and contralateral blindness. Rupture of an infected globe may result in orbital infection. In such cases, the orbit should be extensively irrigated, intraoperative cultures obtained, and broad-spectrum systemic antibiotics administered and modified in accordance with culture results. A drain should be placed if contamination is severe.

Surgical transection of the optic nerve may result in severe intraoperative hemorrhage. Wet-field cautery, application of hemoclips, or careful ligation should restore hemostasis. The surgical closure will tamponade blood within the orbit. Application of a pressure bandage should also assist with hemostasis and diminish trauma to the surgical site during recovery.

Scleral Shell Prosthesis

If exceptional cosmesis is required, a scleral shell or conformer of hydroxyapatite (HA), porcelain, or methyl methacrylate can be placed to cover a disfigured or phthisic globe.[87] If the globe must be enucleated, an HA orbital implant can be secured within the orbit and the scleral shell placed over or pegged to it. The surface of the shell is painted by an ocularist and the prothesis is placed into the conjunctival sac. Daily cleaning and high costs are the main limitations, but the results are quite extraordinary (Figure 55-19).[87] The owner should be aware of the long recovery period (3 to 5 months), frequent treatments, and repeated visits to the ophthalmologist and ocularists for examination and prosthetic fitting.[82] Contraindications to placement of a corneoscleral prosthesis include poorly developed conjunctival fornices, intraorbital infection, intraorbital

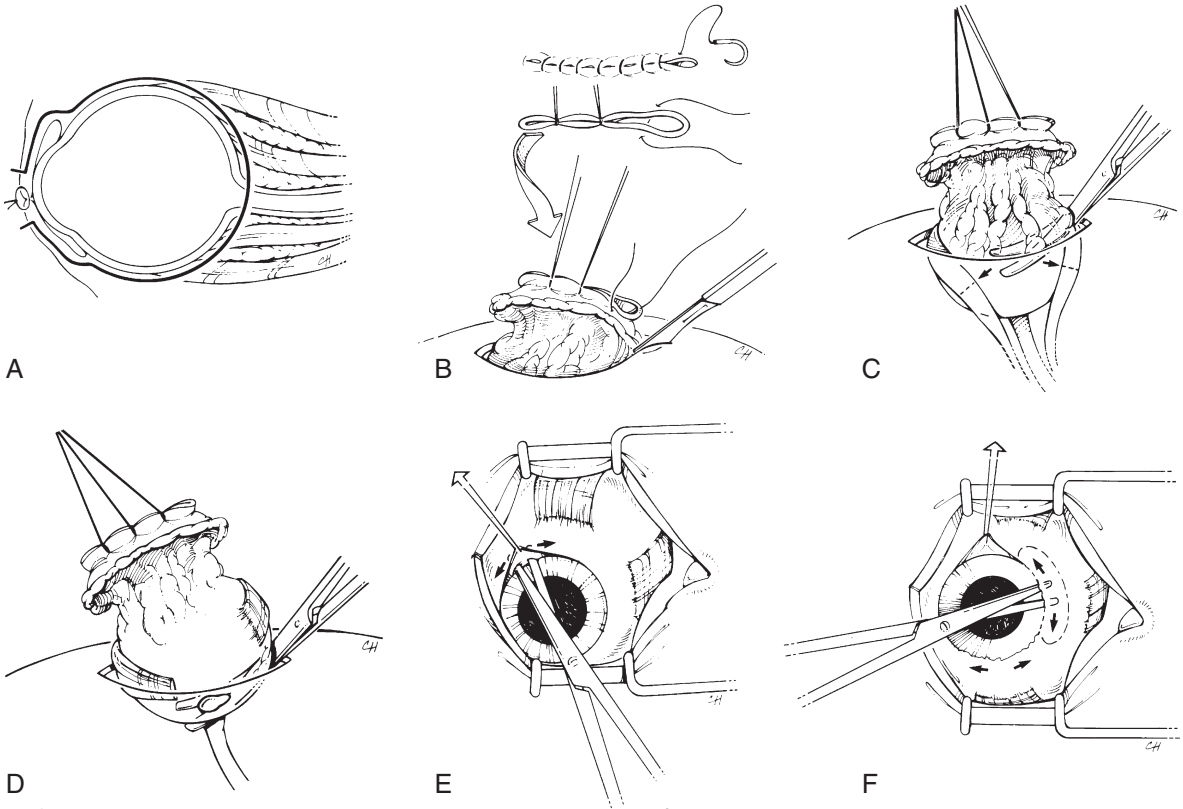

Figure 55-16. A, Enucleation by the transpalpebral method involves closure of the lids. **B,** An incision around the eyelid margin is performed. **C,** Blunt dissection posteriorly without breaking into the conjunctival sac follows. **D,** Transection of the optic nerve. **E,** The subconjunctival enucleation technique involves incising the conjunctiva and continuing the subconjunctival dissection posteriorly **(F).**

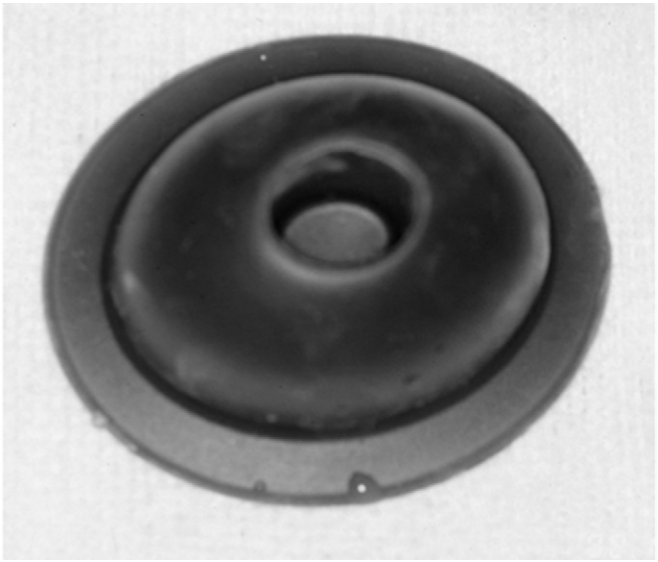

Figure 55-17. The top hat orbital prosthesis is designed to reduce "pitting" after enucleation.

neoplasia, and a horse that is difficult to treat.[82,88] If a globe will be enucleated with the intention of placing an HA orbital implant, the conjunctival fornices, extraocular muscles, and other orbital tissues must be meticulously preserved. The steps required to prepare the implant and place it within the orbit have been described.[87] Ocular conformers are available from Jardon Eye Prosthetics or can be custom made for horses by Dallas Eye Prosthetics (Dallas, TX).

Exenteration

Exenteration involves removing all orbital tissues (Figure 55-20). Indications for performing an exenteration include orbital and/or extensive adnexal neoplasia, medically nonresponsive orbital infections, or aggressive intraocular neoplasia with orbital extension. An exenteration is smilar to a transpalpebral enucleation. However, the posterior dissection and transection of the extraocular muscles and optic nerve should be performed along the orbital wall. A periosteal elevator may be used to strip the periosteal lining of the orbital wall if the disease extends to the periosteum. In the case of disease involving the bony margins, a curette should be used to remove diseased bone. If needed, rongeurs and an osteotome may be used to excise the diseased tissue, although this will be more traumatic and may damage adjacent healthy bone. The disease process may also extend through the orbital wall into adjoining sinuses. Adjunctive treatment via cryotherapy, radiotherapy implants, or chemotherapeutic agents may be warranted depending on the type of the neoplastic process.[12,85] Increased hemorrhage should be expected when performing an exenteration. Vessel ligation, electrocautery, manual tamponade, hemoclips, Gelfoam, and a LigaSure[92] device can all be used to aid hemostasis. Often, minimal periorbital fascia remain for closure. Orbital implants typically are contraindicated following exenteration. Partial dorsal orbital rim resection, mesh skin expansion, extensive undermining of

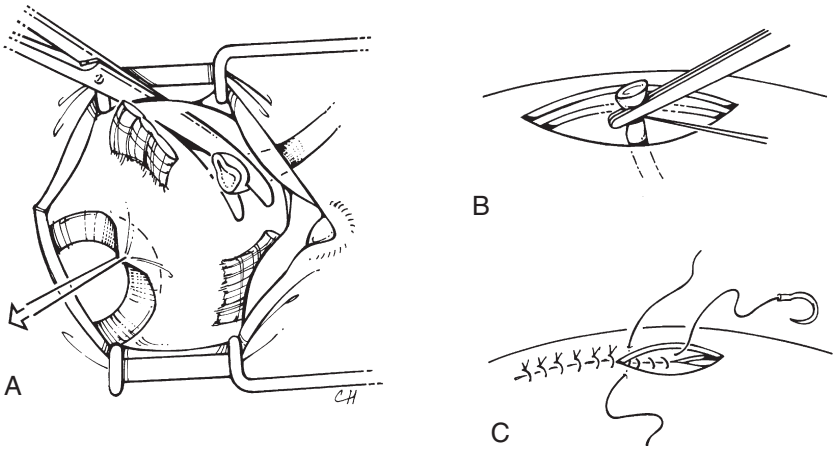

Figure 55-18. In both methods of enucleation (see Figure 55-16), the extraocular muscles are transected and the optic nerve is subsequently clamped **(A** and **B).** A three-layer closure is performed **(C).**

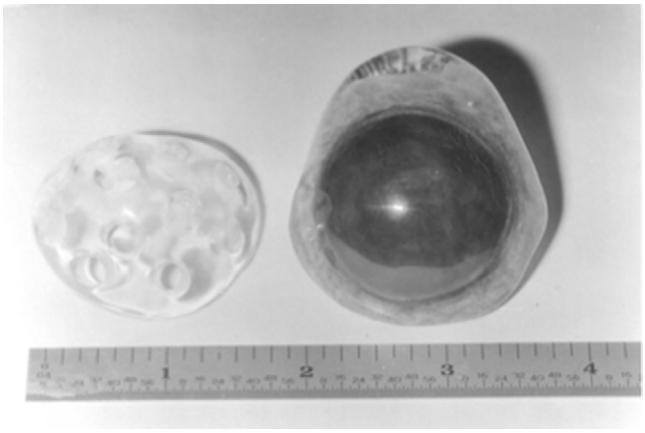

Figure 55-19. This methyl methacrylate cosmetic shell (*right*) was custom made to fit anterior to an orbital silicone implant in a horse. The impression tray (*left*) was used to make the initial mold for the shell.

surrounding skin, tension sutures, and/or second-intention healing may be required to close large orbital defects[93] (Figure 55-21). Placement of an active drain should help to evacuate the large potential dead space. The suction bulb should be secured to the halter after the bandage is placed. Broad-spectrum systemic antibiotics and anti-inflammatories are indicated perioperatively and postoperatively. Complications may include incomplete excision of neoplastic tissue, excessive swelling, and hemorrhage.[12,83]

Orbitotomy

Lesions necessitating orbital exploration include parasitic cysts or granulomas, encapsulated orbital abscesses, foreign bodies, circumscribed neoplasia, repair of some orbital fractures, and cystic dilation of the nasolacrimal or salivary system.[12,19,94] Advanced diagnostic imaging (CT, MRI, or both) should aid in selecting the best approach to the orbit by defining the lesion's location and extent.

A dorsal orbitotomy should be used for lesions in the dorsal orbit and supraorbital fossa.[23] A curvilinear skin incision should be made over the dorsal orbital region just lateral to the external sagittal crest of the parietal and frontal bones, curving laterally caudal to the zygomatic process of the frontal bone.[95] Lateral

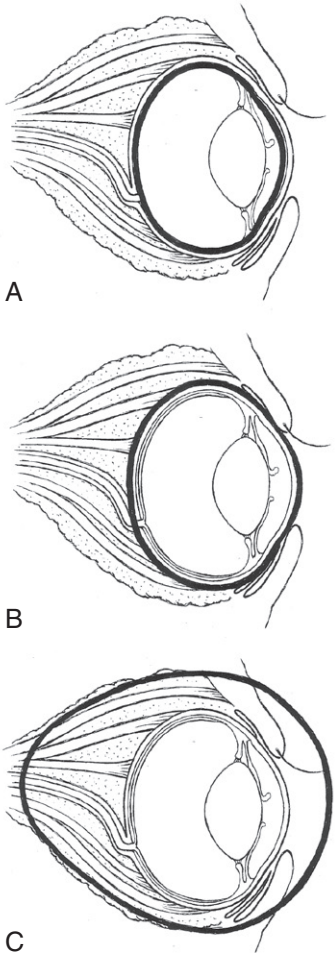

Figure 55-20. The surgical techniques for evisceration **(A),** enucleation **(B),** and exenteration **(C).**

retraction of the frontoscutularis, interscutularis, and temporal muscles exposes the extraocular muscle cone deep within the orbit.[12,23]

In an alternative dorsal approach, an S-shaped skin incision can be made parallel to the zygomatic process of the frontal bone.[94] The surgeon must avoid transecting the neurovascular bundle within the supraorbital foramen and nerve fibers

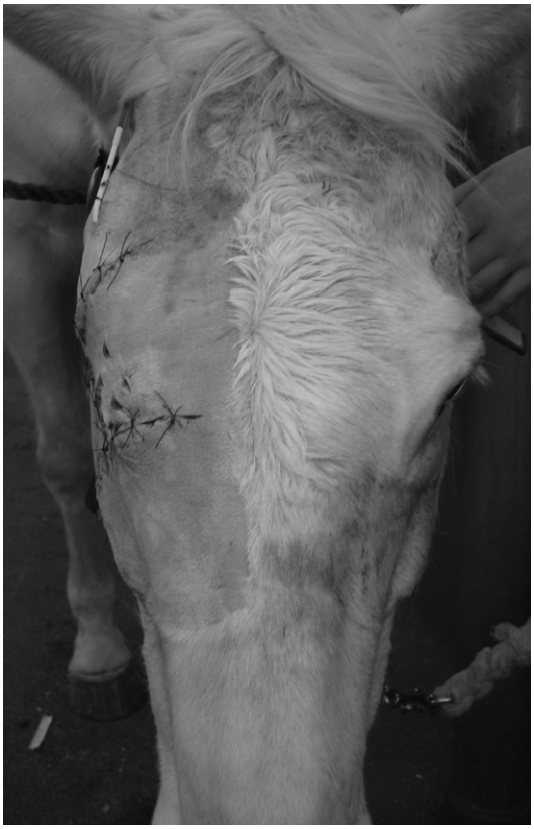

Figure 55-21. An adult horse after exenteration, dorsal orbital rim resection, meshed skin expansion, and placement of an active suction drain.

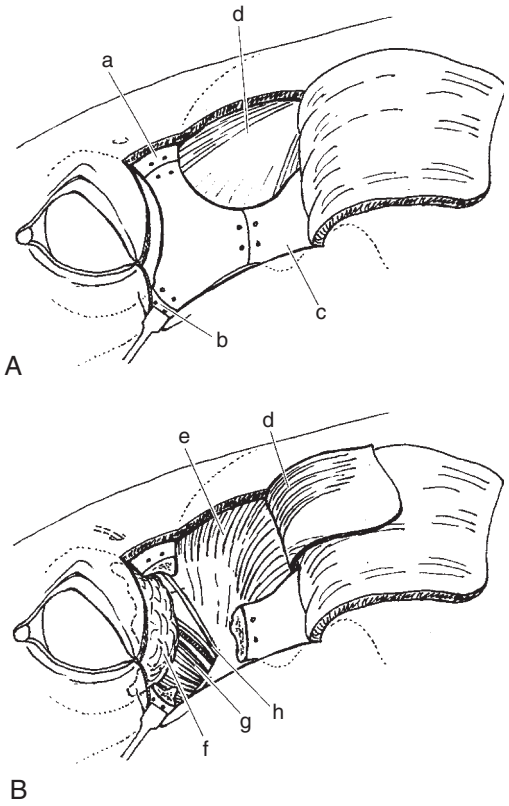

Figure 55-22. Diagrammatic orbitotomy technique involving zygomatic arch transection to remove orbital masses. **A,** The positions where the zygomatic arch and zygomatic process of the frontal bone are drilled before transection of the arch. **B,** The extraocular muscle cone is exposed after removal of the zygomatic arch and temporal muscle. *a*, Zygomatic process of frontal bone; *b*, zygomatic arch; *c*, zygomatic process of temporal bone; *d*, scutular muscles; *e*, temporal muscle; *f*, retrobulbar muscle cone; *g*, lacrimal gland; *h*, supraorbital nerve. (Technique modified from Goodhead AD, Vener IJ, Nesbit JW: Vet Comp Ophthalmol 7:96, 1997.)

entering the orbicularis oculi laterally. The periosteum should be incised anteriorly, reflected off the bone, and preserved for later closure. The zygomatic process of the frontal bone should be resected in section after predrilling 20-gauge holes within the bone for reattachment (Figure 55-22). Performing a lateral canthotomy may increase exposure. At closure, the zygomatic process should be replaced and fixed with 20-gauge stainless steel surgical wire. The periosteum should be closed in a simple interrupted pattern with 4-0 or 5-0 absorbable suture material. The subcutaneous tissue and skin should be subsequently closed.

A lateral approach has also been described.[22,24] A horizontal lateral canthal incision should be extended caudally 5 mm through the skin, followed by dissection through the levator anguli oculi muscle. Two centimeters of the zygomatic process of the temporal bone and its ventrolateral aspect should be resected after predrilling.[24] Closure should be performed as described earlier.

Orbital Fractures

Orbital fractures can threaten the globe's integrity through displacement, impingement, restriction, or laceration.[12] Orbital fractures may produce facial asymmetry, epistaxis, exophthalmos, proptosis, eyelid and conjunctival swelling, depression of the periorbital region, crepitus, and pain on periorbital palpation (Figure 55-23). Damage to the intraosseous nasolacrimal duct, globe, optic nerve, or paranasal sinuses can occur.[18,19] Damage to the poll after rearing or falling over backwards may cause blindness because of fractures of the basioccipital bone and the basisphenoid bone with involvement of the sphenoid foramina.[12] Although orbital fractures may be diagnosed through observation and palpation,[18,19] diagnostic imaging should be recommended.

Periorbital fractures should be repaired quickly before callus forms at 10 to 14 days, which makes manipulation difficult.[18,19] Nondisplaced or minimally displaced fractures of the orbital rim seldom require surgical intervention. Small displaced bone fragments, denuded of periosteum, should be removed surgically to avoid sequestrum formation.[96] Large bone fragments of the zygomatic process or dorsal orbital rim may be amenable to closed reduction.[19] Open fractures should be lavaged and débrided to remove necrotic tissues. Failure to repair large fractures may result in permanent facial deformity.[18,19] Fracture sites should be approached by slightly curved skin incisions adjacent to the fracture site.[18,19] Monofilament stainless steel wire suture (20- to 22-gauge), cerclage wire, small pins, orthopedic bone plates (Figure 55-24), and cancellous bone grafts should be used to stabilize bone fragments and immobilize and repair extensive orbital fractures.[18,19,94] Fractures that are

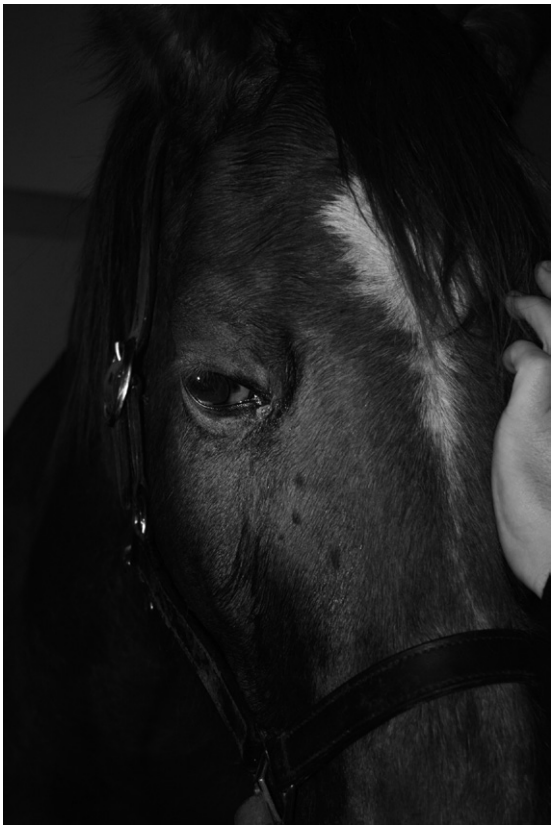

Figure 55-23. Medial orbital rim fractures of the frontal and lacrimal bones in a horse.

Figure 55-24. A 2.7-mm diameter 5-hole bone plate was used to repair this supraorbital process fracture.

difficult to elevate may be reduced by drilling trephine holes at the fracture periphery and using angled probes to elevate the fragment. Sinus fractures often cause epistaxis and emphysema. They should be considered open wounds and treated accordingly. The reader is referred to Chapter 102 for additional information on skull fractures. Broad-spectrum antibiotics and anti-inflammatories are indicated. Tetanus antitoxin or toxoid should be administered if appropriate.[12] Concurrent corneal ulcers, uveitis, lid lacerations, and other associated ocular problems should be treated at the same time.

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CHAPTER
56 Adnexal Surgery

Freya Mowat and Joshua T. Bartoe

Adnexa of the eye include the eyelids and conjunctiva, nictitating membrane, and nasolacrimal system. Each of these adnexa contributes to health of the eye by maintaining a normal physiologic environment. Disorders of each of these adnexa can have deleterious effects on health of the globe and vision, and so each is addressed separately in this chapter.

EYELIDS

Anatomy and Physiology

The different eyelid functions include protection of the globe, optimization of light transmission and refraction through the cornea, maintenance of globe position within the orbit, and musculature and motor control. These functions are discussed in detail later.

Protection of the Globe

The upper eyelid cilia (eyelashes; Figure 56-1) provide protection from light and foreign material. Reflex blinking is initiated by tactile sensation from the cornea, eyelid skin, cilia, or tactile hairs (vibrissae; see Figure 56-1, *A* and *B*). Blinking completely closes the palpebral fissure except at the medial canthus where the caruncle protrudes. The glandular secretions of the eyelids include those from the cilia-associated glands of Zeis (sebaceous) and Moll (apocrine), and the tarsal or meibomian glands (the meibomian gland orifices are visible clinically as the "gray line;" see Figure 56-1, *D*). These secretions provide components to the preocular tear film that is important for protecting the eye.

Optimization of Light Transmission and Refraction through the Cornea

The eyelids maintain optical clarity by distributing tears uniformly across the ocular surface. Loss of blinking immediately impairs corneal clarity. The physical action of blinking pumps tears down the nasolacrimal duct via the upper and lower lacrimal punctae (see Figure 56-1, *C*), promoting a uniform preocular tear film.[1]

Maintenance of Globe Position within the Orbit

The fibrous tissues and periocular muscles aid in globe retraction in combination with the extraocular muscles.

The eyelids consist mainly of skin, striated muscle, fibrous supportive tissue, and conjunctiva (Figure 56-2). The skin is thin and is covered with fine dermal hair, which is absent

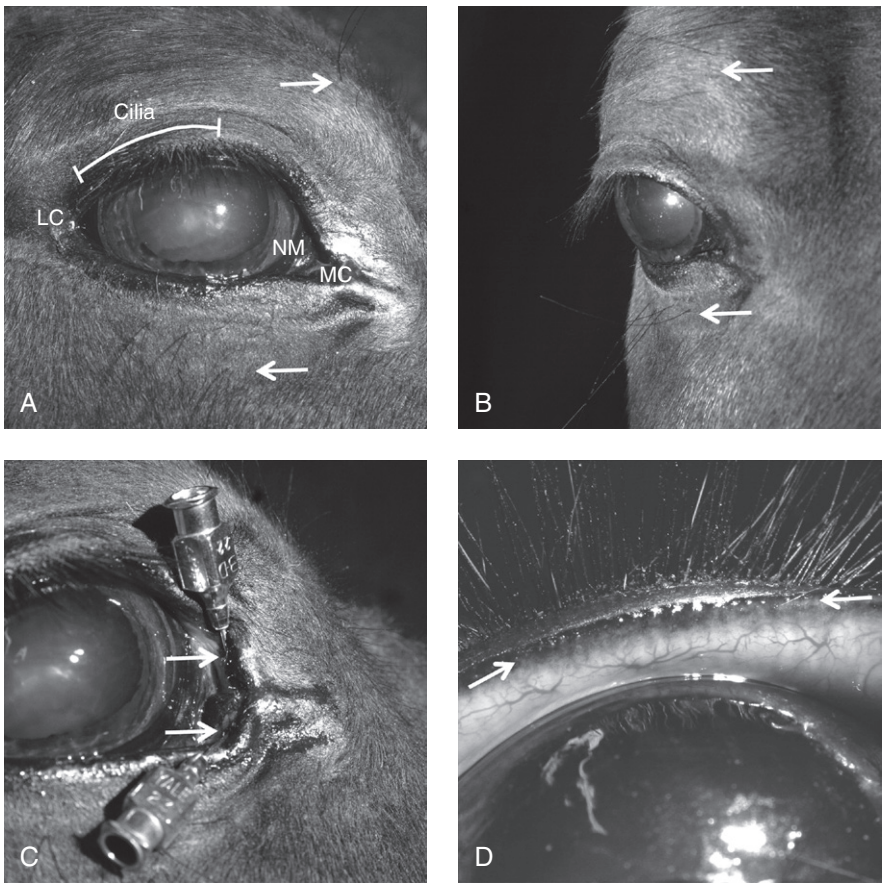

Figure 56-1. Overview of anatomy of the equine ocular adnexa. **A,** Equine eye, lateral view. Cilia (eyelashes) are present in the lateral two thirds of the upper eyelid. Lower eyelid cilia are poorly developed.[92] Upper and lower eyelid skin contains tactile vibrissae *(white arrows).* The palpebral fissures meet at the medial and lateral canthi, and the leading edge of the nictitating membrane is visible at the medial canthus. **B,** Equine eye, anterior view. *White arrows* delineate the position of the tactile vibrissae. **C,** Cannulation of the upper and lower nasolacrimal puncta to highlight their location. *White arrows* identify the puncta. **D,** Magnified view of the upper eyelid mucocutaneous junction. *Arrows* delineate the openings of the meibomian gland orifices (*the gray line*). *LC*, Lateral canthus; *MC*, medial canthus; *NM*, nictitating membrane.

adjacent to the eyelid margin. Sebaceous and apocrine glands are present within the skin. There is sparse subcutaneous loose connective tissue between the skin and the eyelid muscles, and this tissue does not contain any fat.[2] Connective tissue surrounds the muscles of the eyelid and blends with the fascial sheaths and orbital septum. Deep to the eyelid musculature is a region of dense connective tissue known as the tarsal plate. The tarsal plate is most pronounced close to the eyelid margins, blends with the fibers of the palpebral ligaments, and provides structural integrity to the eyelids. The tarsal plate is more extensive in the upper eyelid than the lower. The meibomian (i.e., tarsal) glands are embedded within the tarsal plate and provide the outer lipid component to the tear film, which is important in the maintenance of tear film stability.

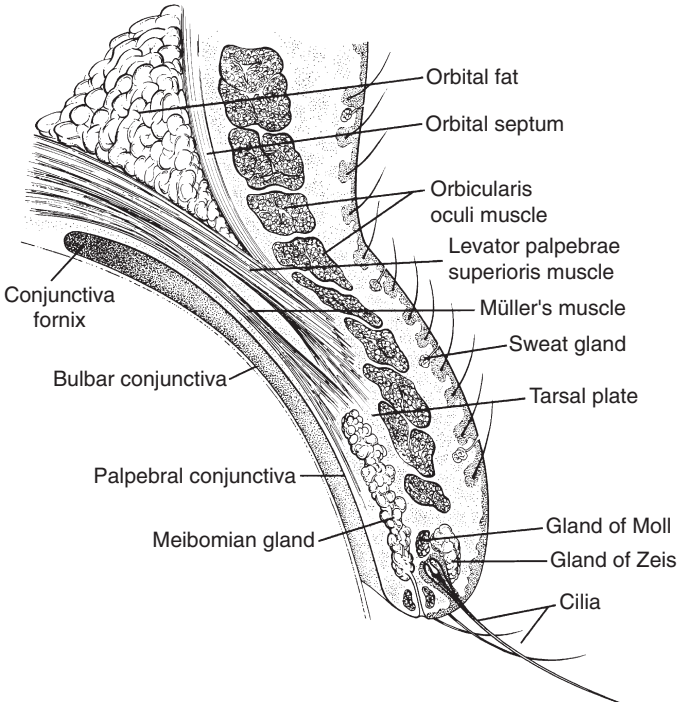

Figure 56-2. Cross-sectional anatomy of the normal upper eyelid.

Musculature and Motor Control

The eyelids are maintained in a roughly elliptical shape by the presence of the lateral and medial palpebral ligaments that restrict movement at the lateral and medial canthi (Figure 56-3). The ligaments originate at the bony orbital rim and insert on the orbicularis oculi muscle. The medial palpebral ligament is a defined fibrous band that overlies the lacrimal sac; the lateral ligament is a wider band containing muscle fibers.[3] The eyelids close in a lateral to medial direction by the contraction of the encircling orbicularis oculi muscle, (see Figure 56-3).[2,3] Opening the eyelids requires the combined action of multiple muscles that insert onto and antagonize the action of the orbicularis oculi muscle. The upper eyelid is larger and more mobile than the lower, and it has greater muscular control. The levator anguli oculi medialis (or corrugator supercilii) muscle inserts a third of the way from the medial canthus and contributes to the asymmetric ellipse of the eyelid aperture.[2] This asymmetry is enhanced during excitement by contraction of the levator palpebrae superioris muscle and levator anguli oculi medialis muscle of the upper lid and the malaris muscle of the lower lid (see Figure 56-3). Müller's muscle in the upper eyelid (in close association with the levator palpebrae superioris muscle) contains smooth muscle innervated by the sympathetic nervous system. The facial nerve (cranial nerve [CN] VII), via the auriculopalpebral and dorsal buccal branches, provides the majority of motor innervation to the eyelid musculature. Exceptions include the levator palpebrae superioris, which is innervated by the oculomotor nerve (CN III), and Müller's muscle, innervated by the sympathetic nervous system.

Sensory Innervation

The trigeminal nerve (CN V) provides sensory innervation to the eyelid skin. The upper eyelid and canthi are innervated by three branches of the ophthalmic division of CN V.[4] The *supraorbital nerve* (a branch of the frontal nerve) emerges from the palpable supraorbital foramen in the zygomatic process of the frontal bone. The *lacrimal nerve* emerges at the dorsolateral aspect of the orbital rim. The *infratrochlear nerve* emerges at a notch in the dorsomedial part of the orbital rim. This nerve also serves the lacrimal caruncle, third eyelid, nasolacrimal puncta, and ducts and areas of the conjunctiva.[3] The lower eyelid is

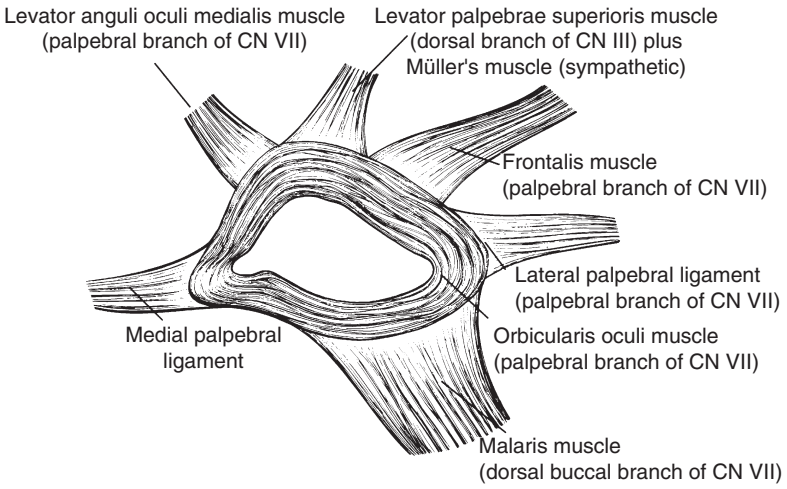

Figure 56-3. Muscles and motor innervation of the equine eyelids.

innervated by the zygomaticofacial branch of the maxillary division of CN V and emerges at the ventral orbital rim.

Vasculature and Lymphatics

The eyelids are well vascularized, which facilitates rapid healing of eyelid injuries. The major arterial supply is derived from the *angularis oculi artery* (a branch of the facial artery) and enters the region at the medial border of the medial palpebral ligament. Accessory arterial supply is provided by the rostral deep temporal artery and branches of the external ophthalmic arteries, including the supraorbital and lacrimal arteries and the malar artery.[2] The major venous drainage occurs via the *infratrochlear vein* (upper eyelids) and the *reflex vein* (lower eyelids and medial canthi), all joining the ophthalmic vein to drain into the internal maxillary vein.[3] The eyelids have significant lymphatic drainage, predominantly to the ipsilateral submandibular and parotid lymph nodes.

Ophthalmic Examination Techniques and Findings

In the investigation of eyelid abnormalities, a systematic approach to diagnosis should always be adopted, including a full bilateral ophthalmic examination and, where indicated, a systemic physical examination. This is particularly important when assessing trauma cases, because significant intraocular disease may be present, which may affect the choice of treatment. Initially a "hands-off" examination to evaluate comfort (epiphora, evidence of self-trauma, blepharospasm, etc.) should be performed. Subtle ocular discomfort or eyelid deformity can be noted by examining the angle of the upper eyelid cilia relative to the surface of the cornea; in an uncomfortable eye with blepharospasm, the angle formed between the cilia and the cornea is more acute.[5] Any asymmetry of eyelid position, swelling or depressions of eyelid or periorbital tissues and should be noted and examined further as necessary. Ptosis in combination with sweating is a consistent symptom of Horner syndrome in horses; enophthalmos and third eyelid protrusion may be less-consistent findings.[6] Vision, eyelid innervation, and eyelid function should be evaluated using the menace response. Both the palpebral and corneal reflexes can be evaluated; it should be noted whether the blink in each test is complete or incomplete. A complete ophthalmic examination should be performed using focal illumination and magnification; a bright light source (Finhoff transilluminator) and magnifying loupes, a direct ophthalmoscope, or a slit lamp biomicroscope are all appropriate choices.

Assuming good general health of the horse, standing sedation can assist with restraint for examination and can be an alternative to general anesthesia for minor surgical procedures. The patient is usually placed in restraint stocks, and cross ties or headrests can be used to achieve greater head stability for surgical procedures. Thoracic auscultation is recommended before sedation because of the cardiovascular suppression induced by sedative agents.[7] Short-duration sedation can be given via intravenous bolus. The α_2-adrenoreceptor agonists detomidine hydrochloride (0.01 to 0.02 mg/kg IV) and xylazine hydrochloride (0.4 to 1.1 mg/kg) each provide analgesia and sedation. Onset of peak activity of xylazine is within 3 to 5 minutes and lasts 30 to 40 minutes at the high end of the dosage range.[7] In fractious animals, a combination of detomidine hydrochloride (0.01 to 0.015 mg/kg IV) and the opioid butorphanol (0.02 to 0.03 mg/kg IV) may provide a deeper plane of sedation with minimal cardiopulmonary depression.[7] Sudden head jerking may occur with the use of opioids in horses, which can be minimized by using them in combination with α_2-adrenoreceptor agonists.[8] A longer duration of sedation may be achieved using detomidine infusions with a loading dose of 7.5 µg/kg followed by a constant-rate infusion of 0.6 µg/kg/minute (infusion rate halved every 15 minutes), providing sedation for up to 135 minutes.[9]

Topical anesthetic may be applied to the eye to facilitate examination and as an adjunct to minor procedures. Proparacaine hydrochloride 0.5% solution has an onset time of 5 minutes and effect duration of 25 minutes following instillation of 0.2 mL of solution.[10] However, even at maximal effect, some corneal sensation remains. Complete corneal anesthesia can be achieved using 0.5% tetracaine ophthalmic preparations (Tetra-Visc, viscous ophthalmic drops, 0.5%) when applied as 2 drops, 1 minute apart.[11] It has an onset time of 5 minutes and effect duration of 60 minutes. In addition to instillation into the conjunctival sac, local anesthetic eye drops can be first applied to cotton-tipped applicator swabs and held in apposition to areas where biopsies or surgical manipulation will be performed.

Regional nerve blocks can be used to provide both motor akinesia and sensory analgesia. Lidocaine hydrochloride 1% or 2% without epinephrine (1 to 2 mL per site[12]) has an onset time of 2 to 5 minutes and effect duration of 1 to 2 hours.[13,14] Mepivacaine hydrochloride has similar onset time but has slightly longer effect duration of 2 to 3 hours.[13,14] Bupivacaine hydrochloride without epinephrine is available in 0.25% to 0.75% concentrations and has an effect duration up to 10 hours.[13,15] After ocular examination, topical lubricants are indicated following instillation of local anesthetic drops and motor or sensory nerve blockade, because tear production and distribution will be impaired.

Motor Blockade

The *upper eyelid akinesis* provided by local blockade of the *auriculopalpebral nerve* facilitates ophthalmic examination and minor adnexal procedures by limiting the palpebral reflex (Figure 56-4). For additional details refer to Chapter 55.

Sensory Blockade

A number of local sensory nerve blocks can be used to anesthetize specific regions of the eyelids (see Figure 56-4).[5,12,14,16] The *central portion of the upper eyelid* is anesthetized using the *supraorbital* block. The supraorbital foramen is palpable clinically as a small depression within the supraorbital process of the frontal bone. The *lateral upper eyelid and lateral canthus* are anesthetized using the *lacrimal* block. A line of local anesthetic is injected along the lateral third of the dorsal orbital rim. The *medial canthus* is anesthetized using the *infratrochlear* block. Local anesthetic is injected through the bony trochlear notch on the dorsal rim of the orbit near the medial canthus.[12] The *lower eyelid* is anesthetized using the *zygomatic* block. A line of local anesthetic is injected along the ventrolateral orbital rim.

Diagnostic Procedures

Radiography, computed topography (CT), or magnetic resonance imaging (MRI) should be considered in cases of eyelid trauma when concurrent orbital fractures or draining tracts are suspected.[17] Further details on imaging techniques are provided

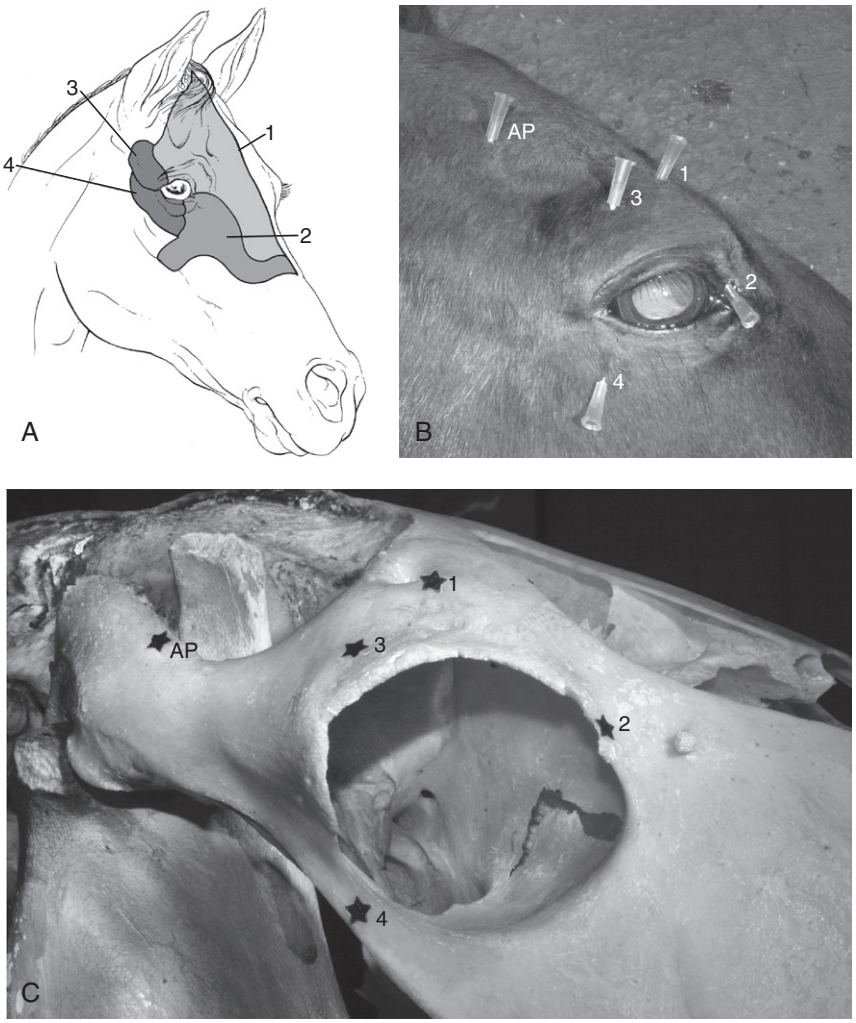

Figure 56-4. An overview of motor and sensory nerve block sites. **A,** Distribution of the sensory innervation of the periocular skin of the horse. **B,** The sites for sensory nerve blockade *(numbers)* and upper eyelid akinesia. **C,** The same sites superimposed onto the equine skull. *AP*, Auriculopalpebral nerve block: motor, upper eyelid; *1*, supraorbital nerve: sensory, central upper eyelid; *2*, infratrochlear nerve: sensory, medial canthus; *3*, lacrimal nerve (line block): sensory, lateral eyelid; *4*, zygomatic nerve (line block): sensory, lower eyelid.

in Chapters 67 to 71. When eyelid neoplasia is suspected, fine needle aspiration or surgical biopsy of masses, suspicious erosions, and regional lymph nodes should be considered to obtain a cell type diagnosis. Eyelid biopsy specimens are commonly fixed in 10% formalin (minimum 10 times the volume of the sample) before submission for histopathology. Suspected infectious eyelid diseases can be thoroughly investigated with impression smears or exfoliative cytology specimens and bacterial or fungal culture with antibiotic sensitivity assessment.

Neoplasia

The differential diagnosis for adnexal neoplasia in the horse commonly includes squamous cell carcinoma, sarcoid, and melanoma[18,19]; less commonly reported connective tissue tumors are fibroma, fibrosarcoma,[20-22] and lymphosarcoma.[23] Other reported tumor types include adenocarcinoma,[22] schwannoma,[24] neurofibroma,[25] plasmacytoma, mast cell tumor, papilloma,[26] and hemangiopericytoma.[27] Neoplasia should be differentiated from blepharitis induced by bacterial, fungal, or parasitic disease including ocular habronemiasis.[28]

Infiltrative periorbital neoplasms may require radical excision, potentially necessitating enucleation or exenteration, periorbital ostectomy, and skin grafting.[29] Although surgical therapy is the principal method of treatment for smaller eyelid masses, *adjunctive therapy* has been widely described in the treatment of periocular squamous cell carcinoma and sarcoids, and it can provide a better clinical response than surgery alone. The choice of adjunctive therapy depends on tumor size and location, availability of equipment, financial considerations, and the clinician's expertise. The choice of therapy should be made in conjunction with a histopathologic diagnosis.

Squamous Cell Carcinoma

The eyelid is a common site for development of adnexal squamous cell carcinoma (SCC) in horses,[24,30,31] and approximately 16% of cases have bilateral lesions.[30] The periorbital location carries a poorer prognosis than other locations on the body, and the eyelid location carries a poorer prognosis for recurrence than the nictitating membrane, nasal canthus, or limbal conjunctival locations.[30] SCC can spread locally, and a 6% to 10.2% rate of metastasis to local lymph nodes is reported.[24,32,33] Metastasis to the thorax and salivary glands has also been reported, although the frequency of this has not been indicated.[34]

Published studies have identified a predilection for draft breeds, Appaloosas, and Paints.[32,35-37] Incidence rises in middle-aged to old animals, with the ocular and skin forms occurring at a mean age of 13 years and 15 years, respectively, which is significantly earlier than penile, vulval, and perianal forms.[37]

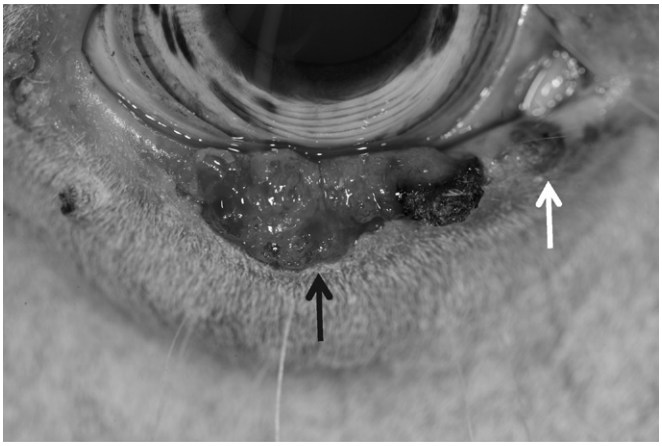

Figure 56-5. A squamous cell carcinoma (*black arrow*) and melanoma (*white arrow*) affecting the lower palpebrum in a horse with no eyelid pigmentation. (Courtesy Dr. Derek Knottenbelt.)

Exposure to ultraviolet light is a risk factor for the development of SCC, with greater incidence in sunny locations and higher altitudes.[35] Animals with minimal or no periocular pigmentation are at increased risk (Figure 56-5).[26,35] Solar-induced epithelial hyperplasia and squamous metaplasia often precede the development of carcinoma *in situ* (where the tumor has not yet invaded through the lamina propria of the epithelium) and overt SCC.[38] Tattooing of the equine eyelids and third eyelid to prevent the development of SCC has been described, lasting 2 to 3 years.[39] There has been no prospective study in horses to show the extent of protection that tattooing provides against solar radiation damage, although there are reports in humans of SCC forming within tattooed skin.[40,41]

Recurrence is a concern following surgical excision, and one study determined a recurrence rate of 68.2% with excision alone.[31] A large study conservatively estimated a survival time of 47 months for animals affected with adnexal SCC.[30] The prognosis is markedly reduced following one or more recurrences.[30] Lesions smaller than 1 cm in diameter and 0.2 cm in depth can be managed successfully using cryotherapy, radiofrequency hyperthermia, beta radiation, or laser ablation treatment, whereas lesions with a surface area of 1 to 2 cm and depth greater than 0.2 cm require cryosurgery, laser ablation, interstitial radiotherapy, or serial cisplatin injections.[19] Enucleation or exenteration should be considered for large, infiltrative lesions. Studies evaluating adjunctive therapies are detailed in Table 56-1. For information of SCC in other areas, please see Chapter 29.

Sarcoids

Although not classified as malignant, sarcoids are common, locally invasive fibroblastic neoplasms that frequently recur following therapy. Periocular sarcoids can cause ocular irritation and swelling of the eyelids, impairing function and abrading the cornea.[42] A previous study found that 14% of sarcoids occur exclusively in the periocular region, and of these, 78% are unilateral and 67% affect the upper eyelid.[42] Sarcoids can be divided into five broad categories: occult, verrucose, nodular (types a and b), fibroblastic (types a and b), and mixed.[42,43] Specific clinical features may indicate which type of sarcoid is present,[44] as outlined in Table 56-2. Biopsy or injury of sarcoids can result in the development of a more-aggressive tumor type.[44] Sarcoids usually develop in young horses between 3 and 6 years of age.[42] Quarterhorses, Appaloosas, Arabians, and Thoroughbreds may be at increased risk, and Standardbreds and Lipizzaner have a reduced risk.[45,46] Bovine papillomavirus infection has been associated with sarcoid development.[43,47-49] The prognosis for periocular sarcoid should be considered guarded because recurrence despite aggressive therapy is common.[5]

Summaries of the *adjunctive therapy* options are outlined in Table 56-3. Surgical debulking has been recommended before many of the adjunctive therapies described. Connective tissue tumors such as fibromas, fibrosarcomas, and neurofibromas should be treated in similar manner to sarcoids, because the success of surgical excision alone is poor.[19] For information on sarcoids located in other areas, see Chapter 29.

Anesthetic Considerations

In cases of trauma where eyelid lacerations have occurred, evaluation should be made regarding the presence and extent of orbital fractures that in extreme cases may involve the cranial cavity. This may affect the animal's demeanor and level of awareness, the timing of which can lag behind the initial injury.[50] Therefore sedation should be used with caution in cases where severe head trauma is suspected (to avoid iatrogenic cerebral edema). Trauma could also involve paranasal sinuses, and in these cases, airway inflammation, hemorrhage, or both could increase the potential for anesthetic complications. Anesthetic recovery should be as smooth as possible, and padded helmets can protect surgical sites during recovery.[51]

Required Surgical Equipment

Standard equipment useful for eyelid surgery is outlined in Table 56-4 and shown in Figure 56-6. Ophthalmic instruments should be kept in a protective container with plastic or foam compartments to protect the fine tips of these instruments. Instruments should be cleaned carefully using fine brushes.

Recommended suture materials are listed in the later descriptions of individual procedures. Most eyelid procedures can be performed using reverse cutting needles, and swaged suture materials minimize tissue drag. Firm monofilament sutures such as nylon should be used with caution close to the cornea to prevent abrasions.

Additionally, cellulose sponges (Weck-Cel, Medtronic, Jacksonville, FL) or cellulose sticks can be used to absorb fluids without damaging the ocular structures. Throughout surgery, regular application of balanced salt solution to the corneal surface is recommended to prevent corneal desiccation. Cautery is often beneficial because the eyelids are highly vascularized. Cryotherapy units used in general surgery can be used for ophthalmic interventions. Liquid nitrogen, carbon dioxide, or nitrous oxide units can be used. Magnifying loupes with or without a light source are useful, and are available with magnifications from ×1.5 to ×5. Loupes should have a comfortable working distance between 20 and 30 cm.

Relevant Pharmacology

Tetanus vaccination status should be established in all cases of trauma, and vaccine or tetanus antitoxin should be administered where the status is unknown or a booster is overdue. If

Text continued on p. 753

TABLE 56-1. Summary of Adjunctive Therapies Published for the Treatment of Periocular Squamous Cell Carcinoma

Treatment	Number of Periocular Cases	Stage of Disease	Protocol	Outcome	Reference
Surgical excision alone	22	Not specified	Not specified	68.2% recurrence over mean follow-up time of 313.9 days	31
Cryotherapy	1	2 cm × 2 cm	Not well specified, likely circulating freeze tip with liquid nitrogen. Double freeze-thaw to −20° C to −40° C	No recurrence in 6 months	93
	2	Not specified	Surgical debulking performed	100% recurrence	36
	1	Large tumor in multiple locations	Liquid nitrogen solid probe, 2 freeze-thaw cycles to −20° C, extending 2 to 5 mm beyond margin	Marked improvement within 4 weeks, no comment on resolution	94
	25	Not specified	Double freeze-thaw cycle	40% recurrence over mean follow-up time of 313.9 days	31
Hyperthermia	1	Large, extending into orbit from medial canthus	Initial hyperthermia (1.5-cm, 2-pronged probe in 3 sites for 30 sec) followed by implantation of 12 evenly spaced ^{198}Au seeds; entire treatment repeated after 9 weeks. Hyperthermia repeated 6 weeks later	No recurrence 3 years after treatment	95
	15 ocular or periocular	Not specified	6 surgically debulked	75% nonrecurrence 8 weeks in small tumors not excised, 33% regression in group that had surgery and hyperthermia	32
	8 including ocular, third eyelid, and eyelid	Not specified	Debulked tumors more than 3 mm depth; piercing probe or combination of piercing and surface probes for eyelid masses, using radiofrequency probe with a tissue temperature of 50° C	75% nonrecurrence for minimum 8 weeks	96

Continued

TABLE 56-1. Summary of Adjunctive Therapies Published for the Treatment of Periocular Squamous Cell Carcinoma—cont'd

Treatment	Number of Periocular Cases	Stage of Disease	Protocol	Outcome	Reference
Chemotherapy-intralesional	3	Not specified	Cisplatin 1 mg/cm^3 in an oil/water suspension every 2 weeks for 4 treatments	Mean nonrecurrence of 14 months, 1 year nonrecurrence of 65%	97
	13	Stage T2 (2 to 3 cm diameter involving full eyelid thickness)	4 times at 2-week intervals with slow release formulation of 1 mg/cm^3 cisplatin in oil/water emulsion	1 year nonrecurrence of 93%	33
	13	Stage T2 (2 to 3 cm diameter involving full eyelid thickness)	4 times at 2-week intervals with slow release formulation of 1 IU/cm^3 bleomycin in oil/water emulsion	1 year nonrecurrence of 78% (no significant difference in recurrence compared with cisplatin in same study)	33
	3	Not specified	Debulking of larger tumors and implantation of 3-mm degradable beads containing 7% cisplatin at 1.5-cm intervals, mean of 2 treatments	Two thirds had no recurrence for at least 2 years	22
	103	Not specified	Surgical debulking for larger tumors followed by cisplatin 1 mg/cm^3 in an oil/water suspension every 2 weeks for 4 treatments	Periocular cases not examined separately; **cisplatin alone:** 2-year nonrecurrence = 95%, 5-year nonrecurrence = 85%; **with surgery:** 2-year nonrecurrence = 79% to 100%, 5-year nonrecurrence = 78.5% to 100% (depending on timing of treatment and extent of residual disease); prior cryotherapy negatively affected prognosis	98
Immunotherapy	1	Highly invasive involving upper and lower eyelids	Intralesional injection of BCG cell wall fraction 6 weeks following unsuccessful cryotherapy, repeated 1, 3, 7, 14, 19 weeks later	Evidence of lymph node metastasis at final examination	99
Gamma radiation	52	15 = T1 (<2 cm diameter), 29 = T2 (2 to 5 cm diameter), 8 = T3 (>5 cm diameter)	Mean radiation dose of 62.7 Gy administered over 4.8 to 8.9 days using ribbons containing 1-cm spaced ^{192}Ir seeds placed at 0.8 to 1.2-cm intervals in a single or double plane	Mean nonrecurrence 46 months; 1-year nonrecurrence 81.8%, 5-year nonrecurrence 63.5%	100
	1	2 cm × 0.5 cm	Debulking of large tumors, implantation of 0.8-mm diameter ^{198}Au grains delivering total dose of 70 Gy	Lymph node metastasis evident 4 months following treatment	21
	20	Not specified	Surgical debulking followed by ^{90}Sr application 250 Gy extending at least 2 mm beyond edge of margin, repeated until no growth is seen	89% nonrecurrence at 1 year	101
Photodynamic therapy	10	Size range of 0.25 to 20 cm^2	Surgical resection, infiltration with HPPH, irradiation with 665-nm diode laser	Nonrecurrence interval of 25 to 68 months	102

BCG, Bacille Calmette-Guérin; *HPPH*, 2-[1-hexyloxyethyl]-2-devinyl pyropheophorbide-a.

TABLE 56-2. Clinical Characteristics of Different Types of Equine Sarcoids

Type of Sarcoid	Predilection Sites	Clinical Appearance
Occult	Head, neck, hairless skin	Circular, alopecic with small cutaneous nodules and/or mild hyperkeratosis
Verrucose	Face, axilla, groin	Hyperkeratosis, prominent scaling, resemble papillomas or fibropapillomas
Nodular type A	Not described	Firm, well-defined subcutaneous spherical nodules, **no** dermal involvement
Nodular type B	Not described	Firm, well-defined subcutaneous spherical nodules, **clear** dermal involvement
Fibroblastic type 1 (pedunculate)	Groin, eyelid, lower limbs, coronet, site of previous skin trauma or other sarcoid	1a: narrow neck with only superficial involvement 1b: narrow neck with deep and superficial involvement
Fibroblastic type 2 (sessile)	Groin, eyelid, lower limbs, coronet, site of previous skin trauma or other sarcoid	Broad, locally invasive base
Mixed (progressive state between verrucose/occult and fibroblastic/nodular)	Not described	Very variable appearance
Malignant/malevolent	Jaw, face, elbows, medial thighs	Often follows biopsy or trauma of fibroblastic mass. Invasive, metastasize readily to lymph nodes

Adapted from Knottenbelt DC: A suggested clinical classification for the equine sarcoid. Clin Tech Equine Pract 4:278, 2005; Knottenbelt D, Edwards S, Daniel E: Diagnosis and treatment of the equine sarcoid. In Pract 17:123, 1995.

TABLE 56-3. Summary of Adjunctive Therapies Published for the Treatment of Periocular Sarcoid

Treatment	Number of Periocular Cases	Type of Sarcoid	Protocol	Outcome	Ref.
Surgical excision alone	28	Superficial verrucose, well defined nodular type A	1-2 cm margins	82% local recurrence	42, 103
Cryotherapy	9	Not specified	–20° C for minimum 2 freeze-thaw cycles, freezing extending beyond margin. Multiple treatments, in some cases liquid nitrogen or nitrous oxide	56% local recurrence	104
	2 on head	Not specified	Surgical debulking, 2 freeze-thaw cycles to –25° C, 2- to 3-week intervals for 1-5 cycles, liquid nitrogen	No recurrence at 15 and 16 months	105
	23	Small verrucose or occult	Triple freeze-thaw to –25° C using liquid nitrogen unit	91% recurrence within 12 weeks	42
Hyperthermia	2	Not specified	64° C daily × 4 sessions. Followed procedure outlined in reference 105 but repeated at 4-day intervals	100% recurrence	42
Topical chemotherapy	9	Superficial occult or verrucose	5% 5-fluorouracil twice daily, tapering every 5 days	67% resolved	42
	146	Small superficial verrucose	AW4-LUDES (mixture of 10% 5-fluorouracil and rosemary oil) ointment once daily for 5 days	35% resolved	42

Continued

TABLE 56-3. Summary of Adjunctive Therapies Published for the Treatment of Periocular Sarcoid—cont'd

Treatment	Number of Periocular Cases	Type of Sarcoid	Protocol	Outcome	Ref.
Intralesional chemotherapy	12	Not specified	Cisplatin 1 mg/cm^3 in an oil/water suspension every 2 weeks for 4 treatments	Mean nonrecurrence, 21.6 months; 1 year nonrecurrence of 87%	97
	18	Fibroblastic, extensive nodular	Cisplatin 1 mg/cm^3 at 5- to 7-day intervals for up to 6 treatments	33% resolved (all nodular)	42
	4	Not specified	Debulking of larger tumors and implantation of 3 mm degradable beads containing 7% cisplatin at 1.5 cm intervals, mean of 2.4 treatments	75% had nonrecurrence for at least 2 years	22
	5 on head	Not specified	5-fluorouracil 50 mg/cm^3, 2 to 7 treatments	60% resolved	106
	103	Not specified	Surgical debulking for larger tumors followed by cisplatin 1 mg/cm^3 in an oil/water suspension every 2 weeks for 4 treatments	Periocular cases not examined separately; **cisplatin alone:** 2-year nonrecurrence = 97%, 5-year nonrecurrence = 93.7%; **with surgery:** 2-year nonrecurrence = 89% to 100%, 5-year nonrecurrence = 87.5% to 100% (depending on timing of treatment and extent of residual disease); prior cryotherapy negatively affected prognosis	98
Bacille Calmette-Guérin vaccine	26	Not specified	Surgical debulking for large tumors, 1 mL/cm^3 of 0.75 mg/mL BCG cell-wall fraction, 1 to 6 treatments	No recurrence in 24 months	107
	1 head, 1 eye	Not specified	BCG cell-wall fraction, 3 to 4 treatments	No recurrence in 20 months in eye case, 50% reduction in size in 11 months in head case	105
	3 on head	Not specified	Live BCG vaccine, 5 to 6 treatments	No recurrence in 66% cases in 12 and 18 months, respectively; 90% "remission" in 18 months in 1 case	105
	7, 3 with histological confirmation	Not specified	2 to 7 treatments of intralesional 0.75 mg/mL BCG cell wall fraction 1 to 10 mL	Nonrecurrence of 53 to 260 days	108
	1	Nodular type B	5 treatments of 10 mL intralesional every 2 weeks	No recurrence 2 years	109
	309	Occult, verrucose (26) Nodular, fibroblastic, mixed (283)	Intralesional 1 mL/cm^2 of tumor surface every 2 to 4 weeks	Verrucose, occult, mixed: 58% worsened, 39% static, remainder improved not resolved. Nodular/fibroblastic: 69% complete resolution	42

TABLE 56-3. Summary of Adjunctive Therapies Published for the Treatment of Periocular Sarcoid—cont'd

Treatment	Number of Periocular Cases	Type of Sarcoid	Protocol	Outcome	Ref.
Autologous tumor vaccine	6 on head	3 verrucose, 1 fibroblastic, 2 mixed	Surgical debulking, autologous vaccine made from minimum 10 g tumor tissue with tuberculin adjuvant, injected intradermally in both sides of neck 1 to 2 weeks following tumor excision, minimum 3 treatments, 4 weeks apart until healing is complete	No recurrence in 12 to 30 months in 3 horses treated for the first time, 9 to 48 months nonrecurrence in 3 animals treated with already-recurrent disease	110
Gamma radiation	2	Not specified	Debulking of large tumors, implantation of 0.8-mm diameter ^{198}Au grains delivering total dose of 70 Gy	No recurrence in 18 months and 3 years	21
	14 periorbital	Not well defined; 6 defined as T2 = 2 to 5 cm diameter or minimal invasion, 8 defined as T3 = >5 cm diameter or subcutaneous invasion	Large tumors debulked 52 to 76 Gy total dose administered over 4 to 14 days using ribbons containing 1-cm spaced ^{192}Ir seeds placed at 0.75 to 1.25 cm intervals in a single or double plane	No recurrence in 6 to 41 months, 1 year nonrecurrence 94%	111
	63 (27 recurrent)	Not well defined: 12 = T1 (<2 cm diameter), 27 = T2 (2 to 5 cm diameter), 24 = T3 (>5 cm diameter)	Mean radiation dose of 62.7 Gy administered over 4.8 to 8.9 days using ribbons containing 1 cm spaced ^{192}Ir seeds placed at 0.8 to 1.2 cm intervals in a single or double plane	Mean nonrecurrence of 49 months, 1 year nonrecurrence 86.6%, 5 year nonrecurrence 74%	100
	66	Nodular, fibroblastic, mixed	^{192}Ir rods delivering 70 to 90 Gy, *in situ* for 10 to 14 days	1 year follow-up: 100% no recurrence Long-term follow-up (3 to 14 years): 98% no recurrence	42
	8	Nodular, fibroblastic, mixed, verrucose	Implanted ^{192}Ir rods delivering 45 to 70 Gy	Median nonrecurrence rate of 14.5 months	112
Beta radiation	3	Single/superficial verrucose, occult	Strontium plaque wand delivering 100 Gy over 5 days (5 minutes twice daily)	1 to 4 year follow-up: no recurrence at treated site	42

AW4-LUDES, A Derek Knottenbelt homemade cocktail; *BCG*, Bacille Calmette-Guérin.

eyelid function is compromised, topical artificial tear supplement ointment should be applied regularly to the corneal surface to prevent corneal desiccation.[5] Antibiotics may be required as prophylaxis in the perioperative period or to address active or suspected infection. The hospitalization status and demeanor of the horse along with the results of cytological examination, cultures, and sensitivity testing, if available, will factor into selection of antibiotic class and route of administration. A subpalpebral lavage device is frequently used to deliver liquid topical medications and is particularly helpful in fractious patients. Further discussion of antibiotic selection is provided in Chapter 7 and in current ophthalmology texts.

Systemic anti-inflammatory medications should be considered in the treatment of adnexal disease, particularly following trauma and surgery. Perioperative flunixin meglumine can be administered intravenously, intramuscularly, or orally (maximum dosage 1.1 mg/kg every 12 to 24 hours for approximately 24 to 48 hours, followed by 0.5 mg/kg for up to 5 days).[52] Phenylbutazone can be administered intravenously or orally (maximum dosage 4.4 mg/kg every 12 hours for 24 to 48 hours, followed by 2.2 mg/kg every 12 hours for up to 5 days). Systemic nonsteroidal anti-inflammatory drugs (NSAIDs) have been associated with renal and gastrointestinal damage;[53] therefore, maximum doses should be used for a short duration, and

TABLE 56-4. Basic Equine Adnexal Surgery Pack

Instruments	Use	Quantity
Backhaus towel clamps		4
Mayo scissors		1
Metzenbaum scissors		1
Suture scissors (any type)		1
Stevens tenotomy scissors (10 cm, straight or curved)	Fine tissue dissection	1
No. 64 Beaver blade and/or No. 15 scalpel blade		1
Beaver handle (12.5 cm) and/or Bard-Parker scalpel handle		1
Mosquito forceps		4
Brown-Adson tissue forceps (16 cm)		2
Bishop Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	Fine tissue manipulation	1
Cilia forceps	Removal of ectopic cilia and distichia after treatment	1
Eyelid speculum (Barraquer, Guyton-Park, or Williams)	Eyelid retraction	1
Castroviejo or Jameson caliper	Measurement of lesions	1
Derf needle holder		1
Castroviejo needle holder (14 cm, straight or curved, with or without lock)	Manipulation of small needles	1
Irrigation cannula	Lubrication of the corneal surface	2-4
Jaeger lid plate	Protection of cornea during eyelid incision	1
Desmarres chalazion clamp	Lid immobilization	1

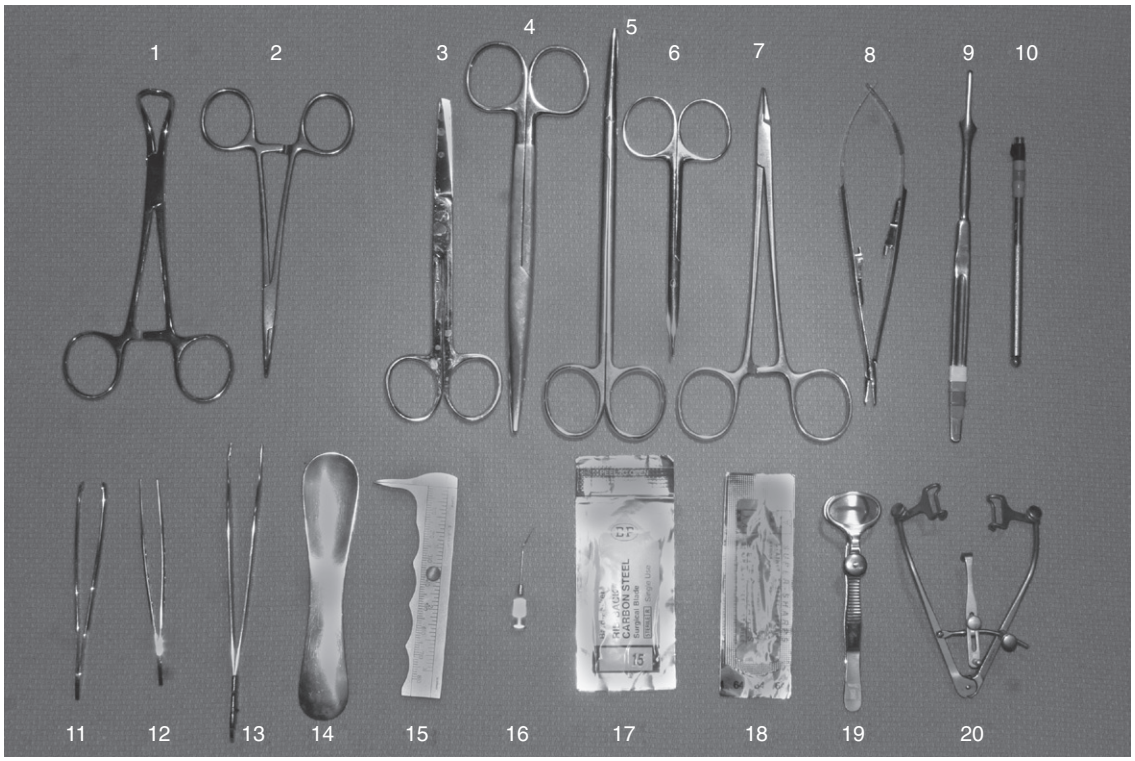

Figure 56-6. Recommended surgical instruments for eyelid surgery. *1*, Backhaus towel clamps (4); *2*, mosquito hemostats (4); *3*, suture scissors; *4*, Mayo scissors; *5*, Metzenbaum scissors; *6*, Stevens tenotomy scissors; *7*, Derf needle holder; *8*, Castroviejo needle holder; *9*, Bard-Parker scalpel handle; *10*, Beaver scalpel handle; *11*, cilia forceps; *12*, Bishop-Harmon forceps; *13*, Brown-Adson forceps (2); *14*, Jaeger lid plate; *15*, calipers (Jameson); *16*, irrigation cannula; *17*, Bard-Parker blade (No. 15); *18*, Beaver blade (No. 64); *19*, Desmarres chalazion clamp; *20*, eyelid speculum (Guyton-Park).

the doses should be reduced as soon as clinically indicated. Particular caution should be used in patients with systemic illnesses such as dehydration.

Surgical Techniques

For most adnexal procedures performed under general anesthesia, the horse is placed in lateral recumbency with the nose elevated using foam wedges or sandbags to maintain the palpebral fissure in a horizontal position. The periocular area is clipped and cleaned using a 1 : 50 dilution of 10% povidone-iodine solution (Betadine, Purdue Pharma, Stamford, CT). The conjunctival sac should also be rinsed with similarly diluted povidone-iodine solution and the area subsequently irrigated with sterile physiologic saline. Cotton or paper quarter drapes should be used surrounding the eye, and their size should be sufficient to prevent contamination from the surrounding surgically unprepared skin of the head and neck. A second paper or plastic adhesive fenestrated drape is placed over the quarter drapes.

Temporary and Permanent Tarsorrhaphy

A tarsorrhaphy is indicated to protect the cornea in cases of facial nerve paralysis or in other situations where blinking is impaired (e.g., severe eyelid trauma). Tarsorrhaphy may provide additional support to the cornea following intraocular surgery.

Temporary tarsorrhaphy is the most frequently used technique and can be performed under general anesthesia or using standing sedation and local anesthesia. The eyelids should be surgically prepared. Three or four horizontal mattress sutures using 2-0 or 3-0 monofilament nylon are usually placed, starting the sutures in the upper eyelid (Figure 56-7). The eyelid sutures should emerge at the meibomian gland orifices (the "gray line") so that neither eversion nor inversion of the eyelids occurs, both of which can predispose to corneal abrasion. The medial canthus can be left slightly open to facilitate the application of topical ophthalmic ointments. Plastic tubing or buttons should be used as stents to prevent the sutures from tearing through the eyelid.

Partial *permanent tarsorrhaphy* may be indicated in cases of prolonged facial nerve paralysis. In this procedure, a limited length of the eyelid margins is excised and sutured together, resulting in surgical apposition (Figure 56-8). The procedure otherwise is similar to performing a temporary tarsorrhaphy, although sutures can be removed after 10 to 14 days. If the underlying disease process resolves and the tarsorrhaphy is no longer required, the site of apposition is incised using scissors to restore a normal palpebral fissure.

Entropion

Entropion is an inward turning of the eyelid. It is uncommon in horses relative to other affected species, but is most frequently seen in foals.[54,55] Entropion can be *congenital* (secondary to microphthalmia or atypical development[5]) or *acquired* (secondary to trauma, scarring, dehydration or wasting, phthisis bulbi, or prolonged blepharospasm) and can affect one or both eyelids. The lateral margin of the lower eyelid is most commonly affected. Entropion can result in contact between the haired skin or cilia and the cornea or conjunctiva, causing irritation (trichiasis). The most common presenting signs include ocular discomfort (blepharospasm, lacrimation), keratitis, or conjunctivitis. Topical lubricants, used as a protective barrier for the cornea, may be sufficient to manage mild transient cases of entropion in foals,[54] although some require the placement of temporary everting sutures.

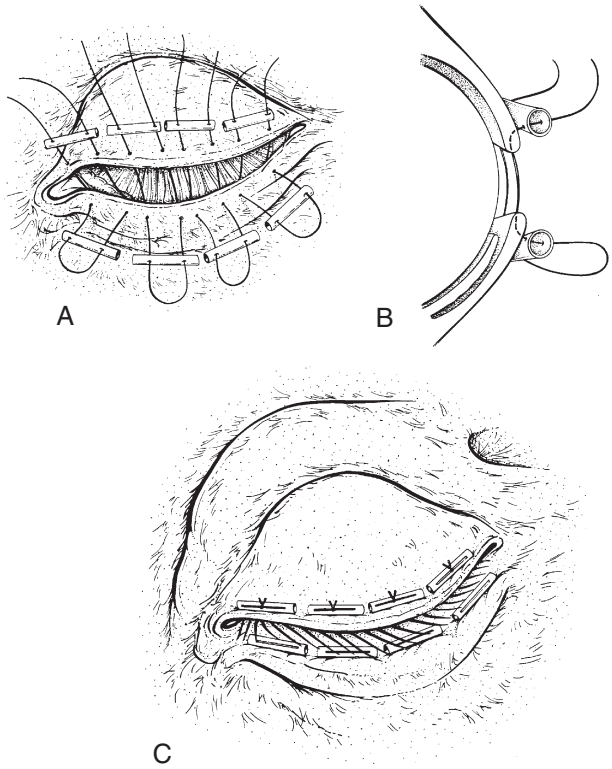

Figure 56-7. Temporary tarsorrhaphy. **A,** Sutures should be preplaced to distribute tension. **B,** Sutures should be placed at partial thickness, crossing the eyelid margin at the level of the meibomian gland openings. **C,** Sutures are tied. The use of stents reduces the risk of sutures cutting into the eyelids.

EVERTING SUTURES

This procedure will *temporarily* limit corneal irritation secondary to the entropion. If the entropion is persistent, permanent surgical correction may be warranted. Vertical mattress sutures are placed using monofilament nonabsorbable suture (e.g., 2-0 to 4-0 monofilament nylon). To accurately place the sutures, the entropion is everted to a normal position and a single vertical bite of eyelid skin is taken approximately 1 to 2 mm from the eyelid margin (Figure 56-9). A similar vertical bite of distal eyelid skin is taken. The distance between the bites determines the extent of eversion of the eyelid, so this bite should be positioned with care, and the suture must be replaced if insufficient or excessive eversion is achieved once the suture is tied. Sutures should be placed at approximately 0.5-cm intervals along the area of entropion. Care should be taken to ensure that the cut ends of the sutures are not at risk of irritating the cornea, and cyanoacrylate glue can be applied to the knots to ensure they remain in place.[52] Sutures are typically removed after 2 to 4 weeks.[52]

MODIFIED HOTZ-CELSUS PROCEDURE

This procedure should be performed when *permanent* correction is required. In foals, this procedure should be performed as late in life as possible because of the risk of overcorrection resulting

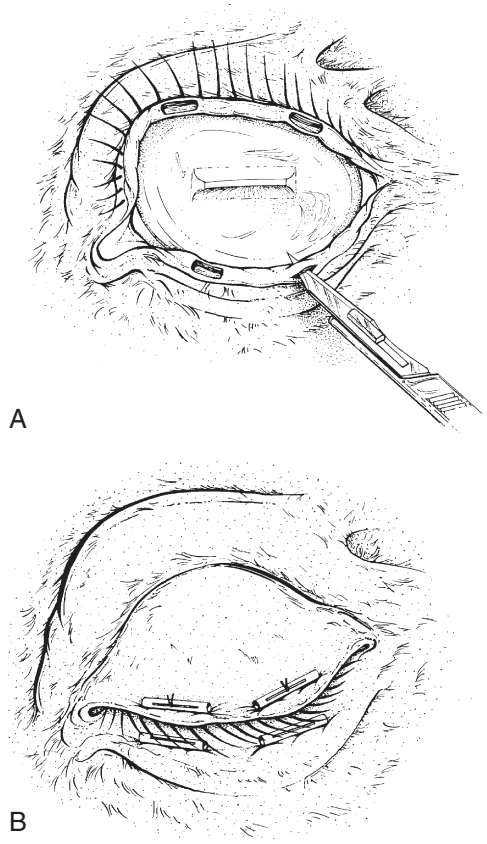

Figure 56-8. Permanent tarsorrhaphy. **A,** Opposing areas of eyelid margin (approximately 3 mm long) are excised with a No. 11 Bard-Parker scalpel blade. **B,** Sutures are placed in the areas of excised margin. Sutures should be left in place for 3 weeks to allow eyelid adhesion.

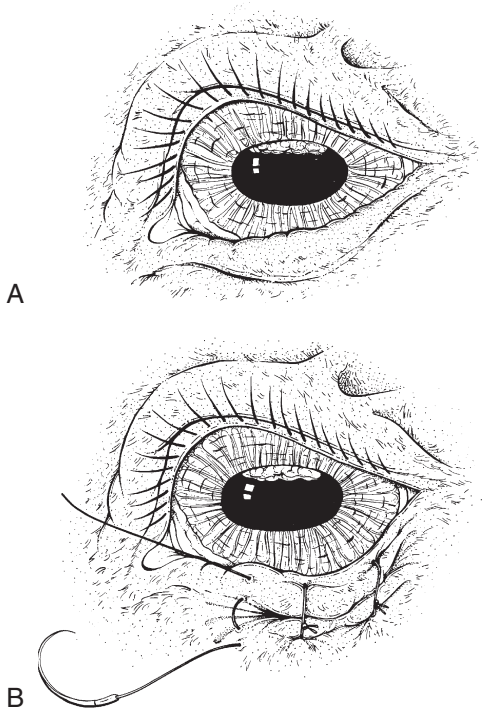

Figure 56-9. A, Entropion of the lower eyelid. **B,** Temporary stay sutures for correction of entropion. Vertical mattress sutures are placed perpendicular to the eyelid margin. The initial bite is taken close (2 to 3 mm) to the eyelid margin. Sutures should be preplaced to distribute tension equally. Knots should be placed away from the eyelid margin to avoid contact with the cornea.

from subsequent growth of the head and face. To effectively plan the extent of surgical correction required, topical anesthetic should be applied and a motor nerve block should be performed to eliminate any spastic entropion related to ocular pain and determine the true extent of the anatomic abnormality.[5,56]

A Jaeger lid plate can be used to support the eyelid, and helps to improve the accuracy of the incision. An incision is made through the skin and orbicularis oculi muscle, parallel to and approximately 2 to 3 mm from the eyelid margin (Figure 56-10). A second elliptical incision is made proximal to the initial incision, and the skin and orbicularis oculi muscle between the incisions is excised. The distance between the two incisions determines the extent of correction of the entropion. Care should be taken not to remove excessive skin initially, because further skin can be subsequently removed. A slight undercorrection is usually appropriate because scarring will increase correction.[5,56] Closure is performed using 4-0 to 6-0 polyglactin 910 suture in a simple-interrupted pattern, with sutures oriented radially away from the cornea. Sutures are placed initially at the widest central portion of the incision to determine the degree of correction. Suture tags should be oriented to prevent contact with the corneal surface. If too little tissue is removed, undercorrection will result in persistent entropion. Ectropion caused by overcorrection can cause exposure keratitis and ulceration.[56] Significant eyelid swelling is

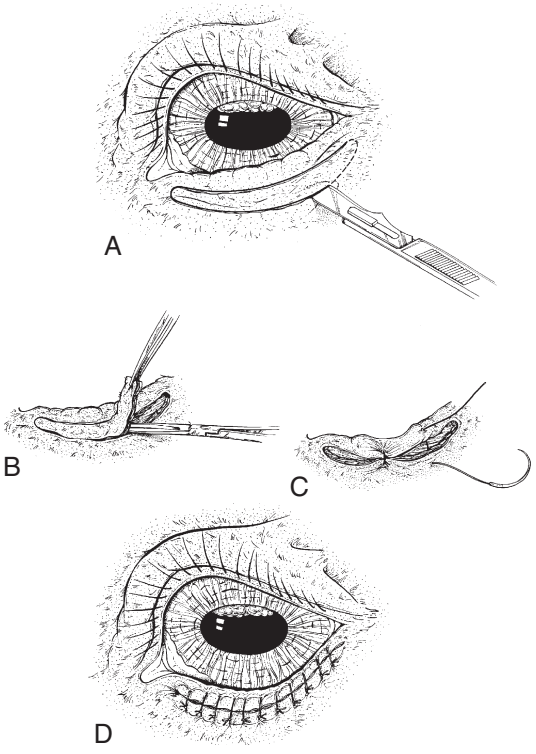

Figure 56-10. Modified Hotz-Celsus procedure for entropion repair. **A,** The initial incisions of the skin and orbicularis oculi muscle are made with a scalpel. **B,** The skin and superficial orbicularis oculi muscle are excised with scissors. **C,** A single-layer closure, starting at the center, incorporates both skin and orbicularis oculi muscle. **D,** Postoperative appearance.

common after surgery and generally resolves within the first 2 weeks postoperatively.

Ectropion

Ectropion is an out-turning of the eyelid. This results in increased exposure of the cornea and conjunctival surfaces that may result in corneal desiccation and inadequate distribution of tears. Ectropion is most commonly associated with scar formation secondary to trauma or previous surgery. The V-to-Y–plasty is useful to correct ectropion (Figure 56-11). A V-shaped incision is made in the eyelid skin, proximal to the eyelid margin, and underlying scar tissue is removed. The incision is then closed as a Y, causing the eyelid margin to roll in immediately adjacent to the surgical site, correcting the ectropion.

Distichia

Distichia are rare in horses and result from aberrant eyelid cilia that emerge from the eyelid margin, usually from the meibomian gland orifices.[57] They can cause corneal trauma and ulceration if untreated. Cilia can be removed under general anesthesia using an operating microscope. The cilia follicle is destroyed, preventing further regrowth using electroepilation or cryotherapy. If cryotherapy is used, a 4-mm probe is placed on the palpebral conjunctival surface, approximately 3 to 4 mm from the eyelid margin (at the base of the meibomian glands). Two freeze-thaw cycles should be completed to maximize destruction of the follicles. Cilia should not be resistant to epilation if effective follicular destruction has been achieved. Cilia too short to be visualized at the time of surgery cannot be treated; therefore the potential for repeated surgeries should be discussed with the owner. Other treatment options include partial tarsal plate excision[58] (although significant eyelid scarring can ensue) and for focal areas, wedge excision.

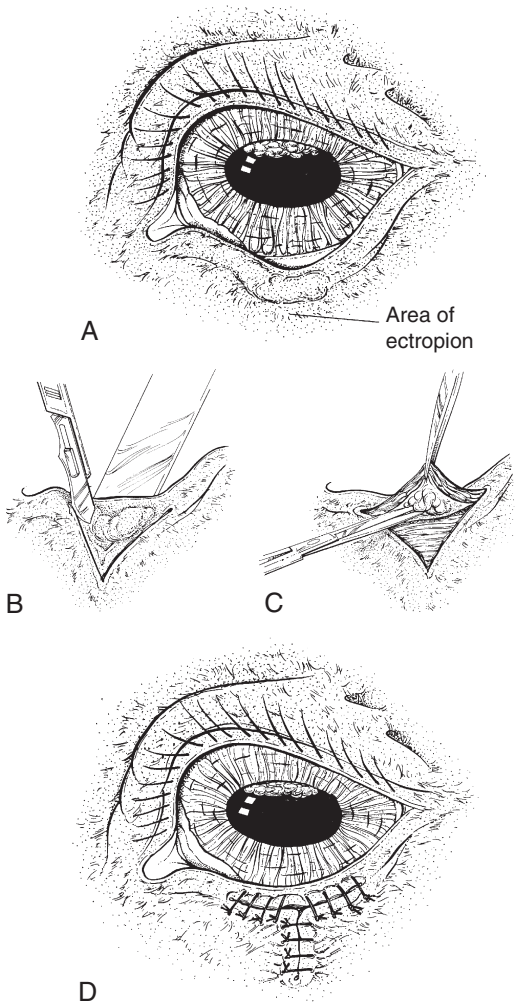

Figure 56-11. V-to-Y correction for ectropion. **A,** The area of ectropion is identified. **B,** The V-incision is made with a scalpel. **C,** The skin flap is elevated, and underlying cicatricial tissue is excised using scissors. **D,** The skin flap is advanced to relieve skin tension. Closure is performed in a Y pattern.

Ectopic Cilia

Ectopic cilia are also rare in horses and result from aberrant eyelid cilia emerging through the palpebral conjunctiva, causing considerable corneal irritation. In one series of seven cases, ectopic cilia were most common in the upper eyelid between the 10 o'clock and 2 o'clock positions.[59] For ectopic cilia, the treatment of choice is surgical excision.[59] The eyelids are immobilized using a Desmarres chalazion clamp, followed by sharp excision of the surrounding conjunctiva, meibomian gland, and cilia follicle *en bloc* using a Beaver blade (No. 6500). Other treatment options include cryotherapy and electroepilation.

Repair of Eyelid Lacerations

Eyelid trauma is relatively common because of the exposure of the wide-set equine eye and hazards of confinement management. It should be established whether lacerations are full or partial thickness. The eyelids are highly vascular and will bleed extensively and become edematous following trauma. Cold packs applied to the eye for 10 minutes, four to six times a day can help to reduce swelling if tolerated. Anti-inflammatory and prophylactic antibiotic drugs are frequently indicated. The globe should be carefully examined for other trauma, and careful palpation should be performed to evaluate any orbital or facial fractures. Radiography and other imaging methods (MRI, CT) should be considered for confirmation if fractures are suspected. A thorough evaluation for potential foreign bodies should be performed. Lacerations affecting the medial eyelids may involve the nasolacrimal apparatus. Nasolacrimal patency should be confirmed before closure, and nasolacrimal lacerations should be treated as described later.

Lacerations should be thoroughly flushed with a 1 : 50 dilution of 10% povidone-iodine solution followed by sterile physiologic saline before closure. All lacerations should be surgically managed using a two-layer closure, because healing by second intention can create eyelid abnormalities resulting from significant proud flesh and scar formation. Fresh lacerations can be closed immediately but long-standing, potentially infected lacerations should be treated with topical and systemic antibiotics for several days before débridement and closure. Closure can be performed using general anesthesia or with standing sedation and local anesthesia. Débridement of necrotic tissue or extensive granulation tissue is necessary, but excessive débridement should be avoided because of the limited tissue available for closure. Tissue-loss defects affecting less than one third of the eyelid margin can be closed using direct apposition, whereas

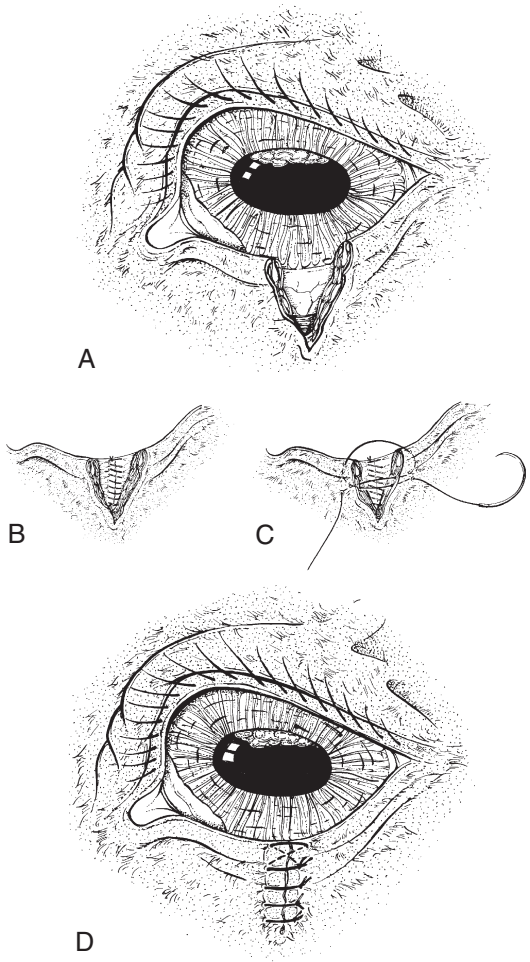

Figure 56-12. Repair of eyelid laceration. **A,** Minimal débridement is performed. **B,** Closure is performed in two layers, starting at the eyelid margin to ensure optimal alignment. **C** and **D,** Skin closure is accomplished with simple-interrupted sutures (4-0 or 5-0). A figure-of-eight suture pattern is useful for closure of the eyelid margin, because it allows suture placement on the eyelid margin, with placement of the knot away from the globe.

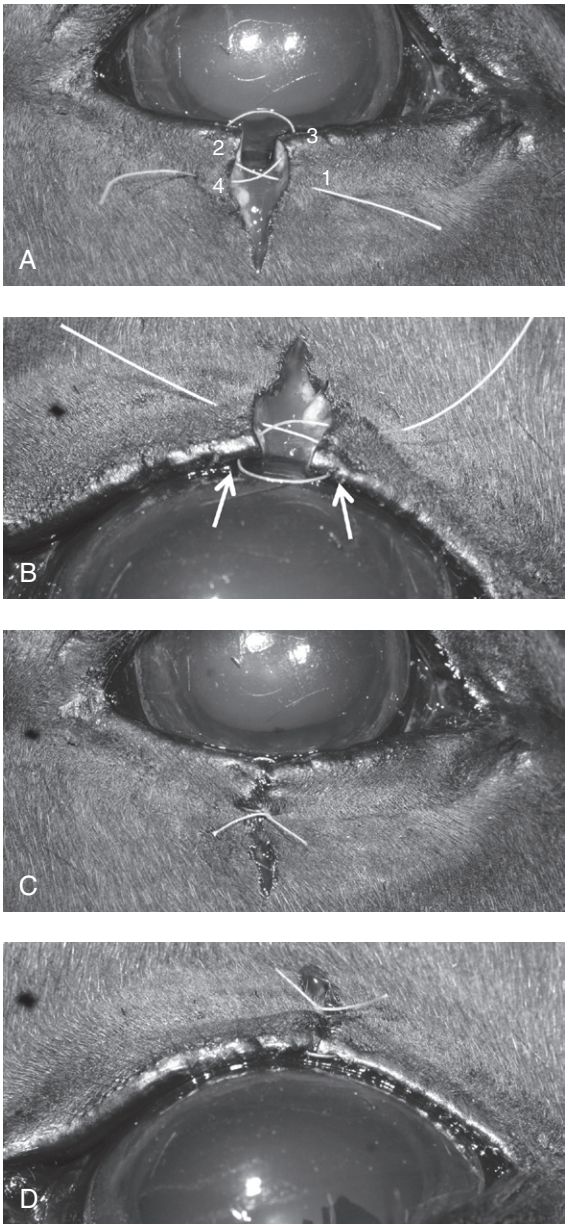

Figure 56-13. The figure-of-eight suture pattern for optimal eyelid margin apposition. **A,** Lateral view. The suture is placed in the numerical order shown, maintaining equal distances with each suture bite. **B,** View from above, illustrating the placement of the suture at the gray line in the eyelid margin (*white arrows*). **C,** Lateral view. The suture is tied and suture tags are positioned away from the corneal surface. The rest of the laceration can be closed using simple interrupted skin sutures. **D,** Shown from above, demonstrating apposition of the eyelid margin.

tissue-loss defects affecting more than one third of the eyelid margin or those close to the lateral or medial canthi may require more complex blepharoplasty procedures (see later). Closure should be performed in two layers, by closing the palpebral conjunctiva first, using a simple-interrupted or simple-continuous suture pattern with 4-0 to 6-0 absorbable suture material, such as polyglactin 910 (Figure 56-12). If the site is infected, nonabsorbable suture material should be used and sutures should be removed after 10 to 14 days. Knots should be positioned away from the palpebral surface of the conjunctiva. The eyelid skin is closed in a simple-interrupted pattern, starting at the eyelid margin to ensure appropriate margin apposition and healing. A figure-of-eight suture pattern (see Figure 56-12 and Figure 56-13) is useful to prevent the suture tags from abrading the cornea. The rest of the eyelid skin is sutured in a simple-interrupted pattern.

AFTERCARE

A protective eye mask with a hard cup may be necessary to prevent rubbing postoperatively. If an eye mask is not adopted, a fly mask should be used until healing is complete. Alternatively, fly repellent ointment can be applied to the skin adjacent to the surgical site.[5] Significant eyelid swelling is common after surgery (Figure 56-14) and generally resolves within the first 2 weeks postoperatively.

Reconstructive Blepharoplasty Techniques

More extensive eyelid lacerations or closure of large defects following removal of neoplasia (removal or loss of greater than one third of the eyelid margin) warrant reconstructive

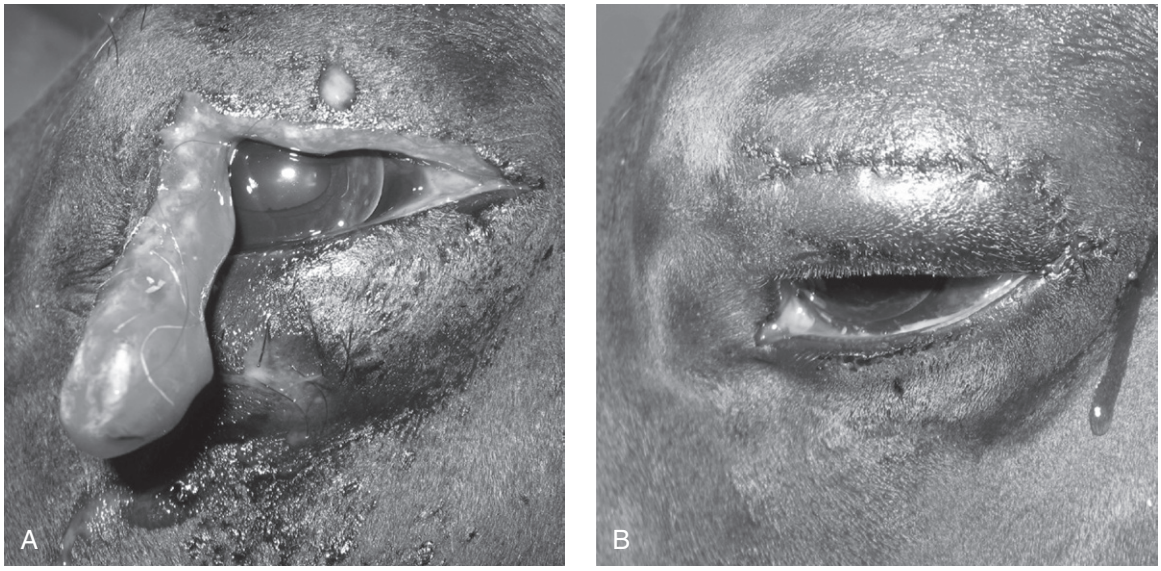

Figure 56-14. A, Example of an eyelid laceration before repair in a horse. **B,** A two-layer closure was performed in the upper palpebral conjunctiva and palpebral skin using absorbable suture material. Wound dehiscence is a risk because of extensive vascular injury.

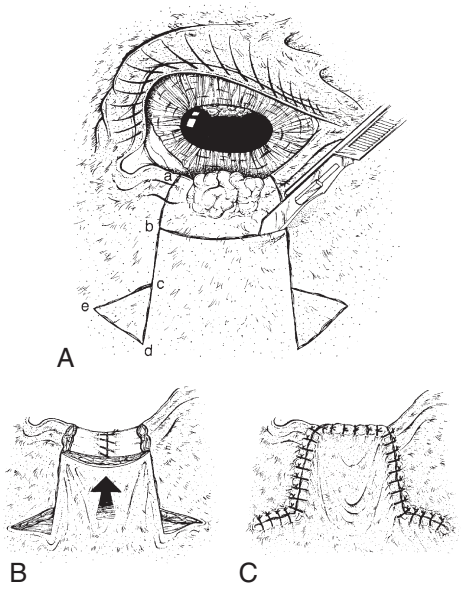

Figure 56-15. Sliding skin flap to repair eyelid defects. **A,** Proportions of incisions should be *ab* = *bc* = *cd* = *de*. **B,** Equilateral triangles of skin are excised, as is the affected portion of eyelid. The skin flap and adjacent skin are undermined with scissors. Adjacent conjunctiva is mobilized and closed with absorbable suture (polyglactin 910 [6-0 Vicryl]). **C,** The skin flap is advanced, and the leading edge of the flap is sutured to the conjunctiva and skin.

blepharoplasty to approximate normal eyelid conformation. This type of surgery frequently warrants general anesthesia.

SLIDING SKIN FLAP

A frequently used blepharoplasty technique is the sliding skin flap (Figure 56-15). Following neoplasia excision or débridement of lacerations, vertical incisions are made in the eyelid skin that extend in height approximately twice the width of the eyelid defect. Slightly diverging incisions compensate for some expected wound contracture. Small triangular portions of skin (Burow's triangles) are excised at the base of the vertical incisions (see Figure 56-15, *A*). These triangles allow closure without skin folds (dog-ears) and help to distribute tension; they should approximate half to the full height of the vertical incision. The surrounding skin, skin flap, and conjunctiva are undermined using blunt dissection, and the skin flap is advanced to the eyelid margin (see Figure 56-15, *B*). Wound contracture should be anticipated, and a slight initial advancement and fixation of the flap past the eyelid margin may provide a better ultimate cosmetic result. The flap is sutured to the conjunctiva at the eyelid margin and to the adjacent skin in a simple continuous pattern using 4-0 to 6-0 absorbable suture (polyglactin 910) (see Figure 56-15, *C*). A temporary tarsorrhaphy may provide additional support during healing.

CONJUNCTIVAL ADVANCEMENT FLAP

In cases of neoplasia or trauma with extensive conjunctival involvement, a conjunctival advancement flap from the opposing eyelid may be required (Figure 56-16). A sliding skin flap is created as detailed previously (see Figure 56-16, *A*). Palpebral conjunctiva from the opposing eyelid is incised approximately 2 to 3 mm from the eyelid margin, measuring the same width as the eyelid defect, and vertical incisions are made toward the conjunctival fornix (see Figure 56-16, *B*). The conjunctival flap is sutured to the remaining conjunctiva in the eyelid defect (see Figure 56-16, *C*). Closure of the sliding skin flap is as previously described. A temporary tarsorrhaphy is required to alleviate tension on the conjunctiva. A second procedure is performed to transect the base of the conjunctival flap and remove the tarsorrhaphy, approximately 1 month following the initial surgery (see Figure 56-16, *F*). This procedure can generally be performed on the standing, sedated horse using local anesthesia.

FULL-THICKNESS EYELID GRAFT

A full-thickness eyelid graft may be required for extensive lesions of the eyelid skin, where sufficient skin cannot be elevated using a sliding skin flap (Figure 56-17). This technique is

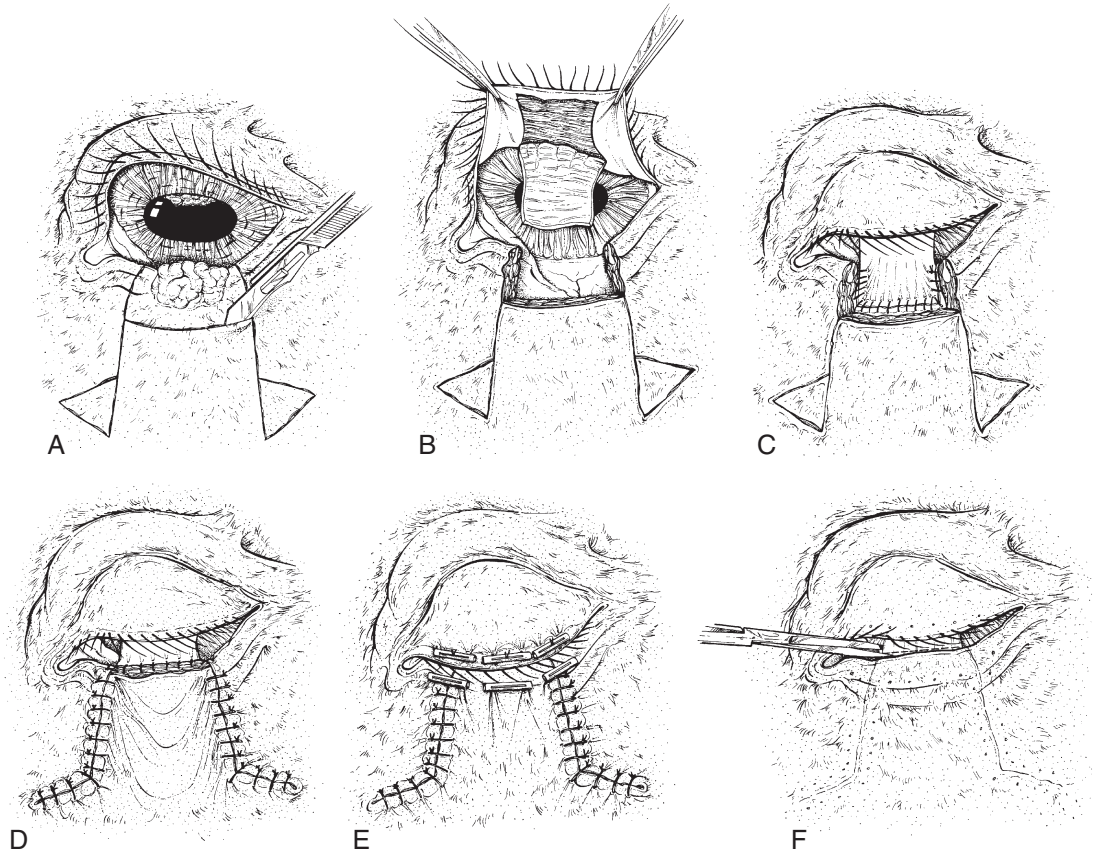

Figure 56-16. Tarsoconjunctival advancement flap. **A,** A skin advancement flap is prepared as in Figure 56-15. **B,** Conjunctiva of the upper eyelid opposite the defect is incised 3 to 4 mm from the eyelid margin and is undermined to create a flap. **C,** The conjunctival flap is advanced and sutured into the defect. **D,** The skin flap is advanced and sutured in place. **E,** A temporary tarsorrhaphy relieves tension on the flaps. The use of stents helps to distribute tension. **F,** After 4 weeks, the tarsorrhaphy is removed and the conjunctival flap is severed at the level of the eyelid margin. The conjunctiva and skin are apposed with a continuous pattern of 6-0 or 7-0 absorbable suture.

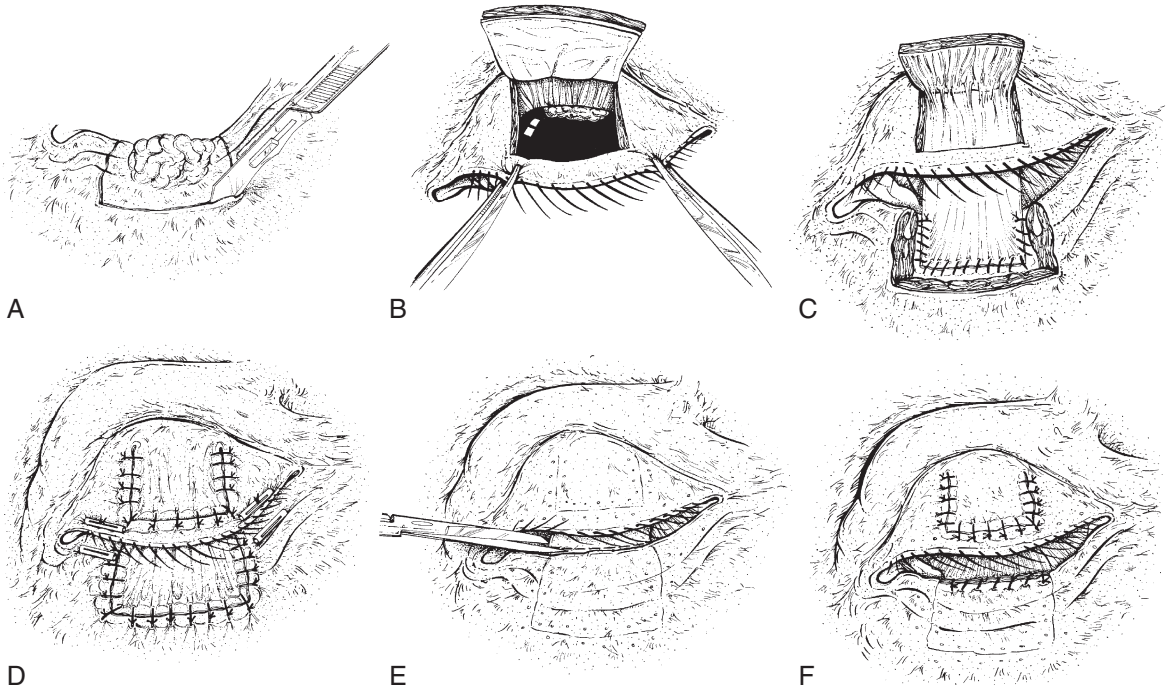

Figure 56-17. Full-thickness eyelid graft. **A,** The area of affected lower eyelid is excised. **B,** The upper eyelid is excised 5 mm above the eyelid margin opposite the defect. **C,** The graft is split into skin and tarsoconjunctival layers. The graft is advanced under the eyelid margin and sutured in place. **D,** The bridging eyelid margin is sutured to the graft. A temporary tarsorrhaphy alleviates tension on the graft. **E,** After adequate healing has occurred, the graft is severed along the intended eyelid margin. **F,** The conjunctiva and skin are apposed along the eyelid margin with a continuous suture pattern. The skin flap is sutured to the bridge to complete the closure.

easier to perform on a lower eyelid defect using the more mobile and extensive upper eyelid as the donor tissue. A sliding skin flap of the lower eyelid can provide partial closure of the defect to be grafted. The width of the graft should be 1 to 2 mm larger than the width of the defect in the opposing eyelid margin. The donor eyelid is incised approximately 5 mm from the eyelid margin (to spare the meibomian glands) (see Figure 56-17, *B*). The flap should be split into skin and muscle, and tarsoconjunctival portions to aid mobility of the tissue. The tarsoconjunctival portion of the graft is sutured to the conjunctival defect in the lower eyelid using a simple-continuous suture of 6-0 polyglactin 910 (see Figure 56-17, *C*). The skin portion of the graft is sutured to the lower eyelid skin defect using 4-0 nonabsorbable suture (e.g., monofilament nylon) (see Figure 56-17, *D*). The bridge in the upper eyelid is sutured to the graft to prevent retraction, and a temporary tarsorrhaphy is placed. In a second procedure, the flap is transected along the new eyelid margin (see Figure 56-17, *E*), and the lower eyelid conjunctiva and skin are sutured using 6-0 absorbable suture material (polyglactin 910) in a simple-continuous pattern (see Figure 56-17, *F*). The donor flap is sutured back within the upper eyelid.

RHOMBOID GRAFT FLAP

Rhomboid and modified rhomboid flaps are used to treat large periocular skin defects in human patients.[60] A large periocular or eyelid margin defect can be grafted by generating a rhombus, or equal-sided parallelogram, which can be rotated to cover the eyelid defect (Figure 56-18). The rhombus can be constructed as a square, or with sides at approximately 60 and 120 degrees. Once the defect is created, two incisions are made in the distal eyelid skin to form two additional sides of the rhombus (see Figure 56-18, *B*). The skin is undermined using blunt dissection and the rhombus is rotated 90 degrees to fill the defect (see Figure 56-18, *C*). Conjunctiva from the distal palpebral or bulbar surfaces should be advanced to the new eyelid margin. Simple interrupted or simple continuous sutures of 4-0 to 6-0 absorbable suture material are used to suture the conjunctiva to the new eyelid margin and to suture the graft in place (see Figure 56-18, *D*).

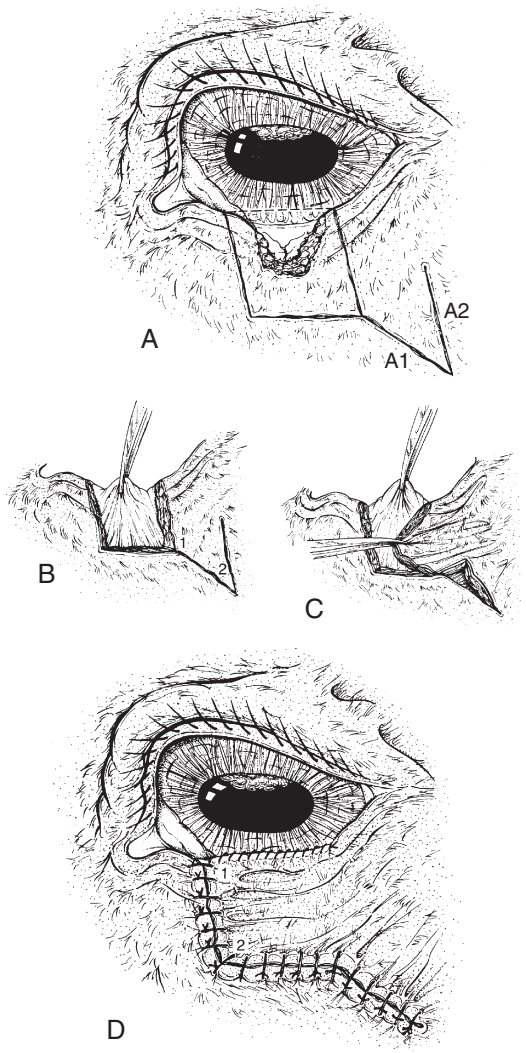

Figure 56-18. Rhomboid graft flap. **A,** The rhomboid is aligned with one side along the position of the eyelid margin. Sides of the rhomboid are equal. The replacement flap is incised on a line *(A1)* continuous with the diagonal of the rhomboid, for a distance equal to the sides of the rhomboid. The second incision *(A2)* is also equal in length and is placed parallel to the side of the rhomboid. **B,** The lesion is excised and conjunctiva is mobilized to cover the replacement flap. **C,** The flap is dissected free from underlying tissue and rotated into position. **D,** The flap is sutured in position with the leading edge forming the new eyelid margin. (Angles 1 and 2 are indicated on **B** and **D** to aid in orientation.)

SLIDING *Z* FLAP

Mass excision or tissue loss at the lateral canthus can be reconstructed using a sliding Z flap (Figure 56-19). The lesion should be fully excised or débrided, and the surrounding skin and tarsoconjunctiva should be separated using blunt dissection (see Figure 56-19, *B*). Triangles of skin are excised superior and inferior to the defect (see Figure 56-19, *C*). The skin is advanced to cover the defect. The new portion of eyelid margin is created by suturing skin and conjunctiva together using 4-0 to 6-0 absorbable suture material (polyglactin 910). The remaining skin is sutured in a similar manner (see Figure 56-19, *D*).

OTHER RECONSTRUCTIVE PROCEDURES

Numerous alternative blepharoplasty procedures with potential application to equine cases can be found in ophthalmology textbooks.[52,61-64]

NICTITATING MEMBRANE

Anatomy and Physiology

The third eyelid (i.e., nictitating membrane, membrana nictitans) is a mobile protective structure covered by conjunctiva located in the inferomedial orbit between the lower eyelid and the globe. This structure is well developed in the horse and moves passively in a temporal and superior direction to cover the anterior surface of the eye with the contraction of retrobulbar muscle (abducens nerve, CN VI) and retrograde movement of the globe. The third eyelid can protrude to cover the majority of the corneal surface.[65]

The third eyelid has a number of functions including the production of a portion of the aqueous layer of the tear film via the nictitans gland and the distribution of tears via passive movement across the globe following globe retraction. Others have suggested that the equine third eyelid provides globe protection, particularly while grazing,[3,63] and has a role in

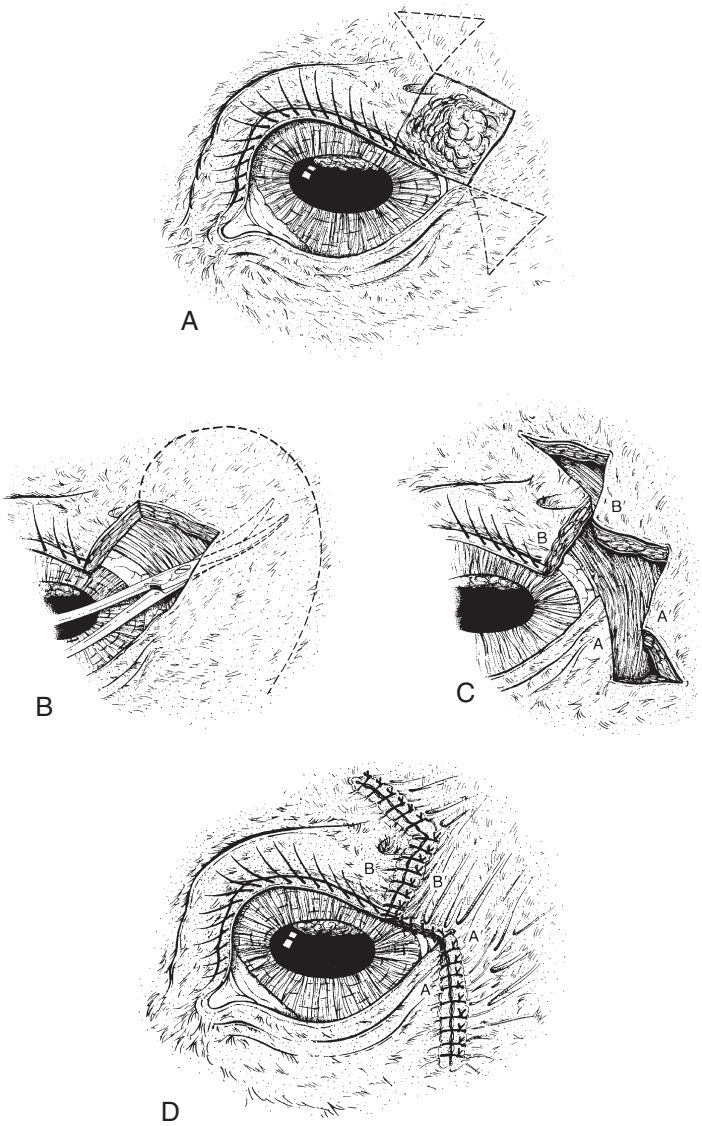

Figure 56-19. Sliding Z flap. **A,** Growths of the lateral eyelid can be removed *en bloc*. The triangular areas of skin to be removed adjacent to the defect are marked. Excision of these flaps facilitates skin mobilization. (The bases of the triangles align with the diagonal of the defect.) **B,** Adjacent skin is undermined. **C,** Equivalent triangles of skin are excised. (Cut edges *A, A′, B*, and *B′* are shown to aid in orientation for advancement of the flap.) **D,** The flap is advanced and sutured in place.

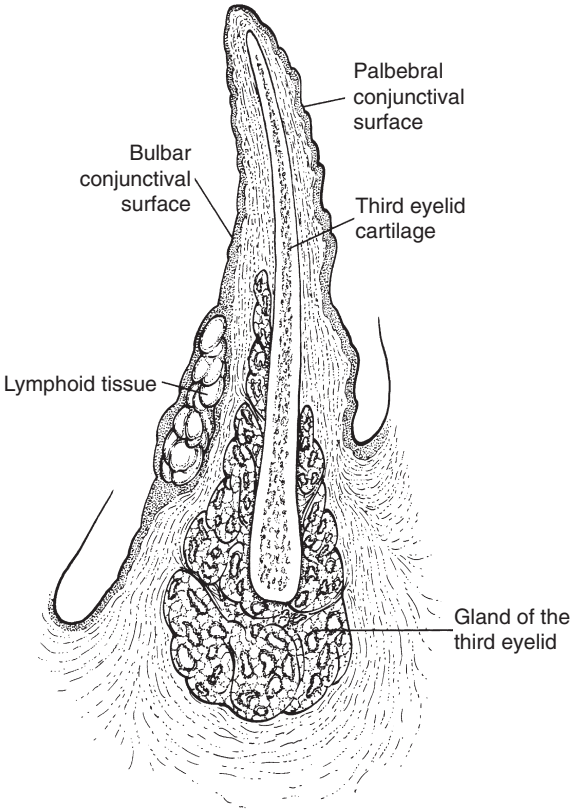

Figure 56-20. A schematic diagram of the histologic features of the third eyelid.

immunologic protection including the secretion of immunoglobulin A.[66]

Structural support of the third eyelid is provided by a T-shaped cartilage, in which the horizontal margin of the T is positioned close to the leading edge of the third eyelid (Figure 56-20), and both ends have a characteristic hook shape.[67] The cartilage contains a substantial elastic component[67] and its shape closely matches the curvature of the globe. The vertical base of the cartilage is surrounded by a large amount of fat and the seromucoid gland of the nictitans. The surfaces of the third eyelid are covered with conjunctiva, containing mucus-secreting goblet cells and intraepithelial glands at the base. The bulbar substantia propria of the third eyelid contains lymphoid tissue.

Sensory innervation is provided by the infratrochlear nerve, a branch of the ophthalmic division of the trigeminal nerve (CN V).[3,67] Retraction of the third eyelid is partially controlled by sympathetic tone in the orbital smooth muscles; loss of tone results in the protrusion that accompanies Horner's syndrome and associated enophthalmos. Vasculature is supplied by the malar artery, a branch of the internal maxillary artery.[3]

Ophthalmic Examination Techniques and Diagnostic Procedures

In the investigation of third eyelid abnormalities, a systematic approach to diagnosis should always be adopted, including a full bilateral ophthalmic and systemic examination. Particularly important is the palpation of regional lymph nodes if third eyelid masses are noted. In cases of corneal or conjunctival disease, the third eyelid should be carefully examined to rule out any contributing lesions. Manual retropulsion of the globe through the superior eyelid causes protrusion of the nictitating membrane, allowing examination of the palpebral surface and leading edge. Topical anesthetic drops and sedation restraint may be required to examine the bulbar surface closest to the globe (see "Eyelids," earlier). Forceps that cause minimal tissue trauma (such as Von Graefe fixation forceps) can be used to retract the third eyelid and examine the deep conjunctival fornices and the bulbar surface. The bulbar surface should be examined if a foreign body is suspected or in suspected cases of conjunctival parasites (e.g., *Thelazia* spp.). Digital palpation may also be necessary to fully evaluate the extent of any observed masses.

Diagnostic procedures such as cytology, fine needle aspiration, biopsy, and bacterial culture and sensitivity testing should be considered as necessary.

Anesthetic Considerations

Most third eyelid procedures can be performed on the standing, sedated horse, although general anesthesia can be performed if deemed necessary. The sensory infratrochlear nerve block provides anaesthesia to the third eyelid and can be used in combination with topical anesthesia. However, topical and local anesthesia will not desensitize the deep portions of the third eyelid; therefore, an additional retrobulbar block should be considered (see Chapter 55) when removing the third eyelid or making deep incisions or crushing tissues. A line block of local anaesthetic at the base of the third eyelid can provide additional anesthesia when performing removal or biopsy.

Required Surgical Equipment

Table 56-5 outlines some additional equipment that may be useful when performing third eyelid surgery.

Surgical Techniques

Third Eyelid Flap

There are limited indications for the use of a third eyelid flap. Unlike conjunctival pedicle grafts, third eyelid flaps cannot replace or support damaged corneal stroma and do not provide any serum-derived factors that assist in corneal healing. Corneal bullae can develop secondary to extensive corneal edema, and a third eyelid flap can provide tamponade of the cornea while facilitating treatment application to the conjunctival sac. Other indications include physically supporting a weakened cornea following a conjunctival graft, reducing contamination of an injured cornea, or reducing evaporative tear film loss secondary to exophthalmos or facial nerve paralysis.[56] Flaps are typically contraindicated in cases of deep ulceration or corneal melting, because they may prevent appropriate penetration of topical medications and promote the retention of inflammatory cells and bacteria adjacent to the corneal surface.[68] The flap also obscures visual examination of the cornea and prevents evaluation of progression or healing, unless sutures are placed to facilitate sporadic lowering of the flap.

A flap is placed either under general anesthesia or standing sedation and local anesthesia. A single suture of 2-0 to 3-0 nonabsorbable material is inserted from the haired skin of the upper eyelid into the upper conjunctival fornix. A bite is taken from the palpebral surface of the third eyelid, approximately 4 to 5 mm from the leading edge, taking care not to penetrate the full thickness of the bulbar surface. Because sutures can tear through conjunctiva, encircling the T-cartilage of the third eyelid helps to retain sutures. The final bite of suture enters the upper conjunctival fornix and exits the haired skin of the superior eyelid. Alternatively, three or four horizontal mattress sutures may be used. Stents of buttons or polyethylene tubing should be used to prevent damage to the eyelid skin. The suture tags can be tied and left long to allow the flap to be sporadically lowered. A subpalpebral lavage device can be placed at the same time the third eyelid flap is constructed.

TABLE 56-5. Additional Instruments Particularly Useful for Equine Third Eyelid Procedures

Instruments	Use	Quantity
Large curved hemostat forceps	Crushing of edges of third eyelid before removal	4
Allis tissue forceps	Third eyelid retraction	2-4
Sterilized buttons/ sections of plastic tubing	Stents for third eyelid flap	4

COMPLICATIONS

The cartilage of the third eyelid can become permanently deformed, causing corneal abrasion, and in severe cases this warrants third eyelid removal.[56] If stents are not used, ulceration of the skin around the sutures can be severe. If inappropriately placed, the suture can cause additional damage to the cornea.

Excision of the Third Eyelid

The third eyelid is a common site of adnexal neoplasia, and it is particularly susceptible to the development of squamous cell carcinoma.[24,30,31,36] Squamous cell carcinoma often initially appears as an area of hyperemia, becoming raised and in some cases developing a papillomatous appearance (Figure 56-21). Tumors of vascular origin, including hemangioma, hemangiosarcoma[69-71] and lymphangiosarcoma[72] have also been described. Hemangiomas and hemangiosarcomas often result in a hemorrhagic ocular discharge.[70,71] Other neoplasms reported include basal cell tumor[73] and lymphsarcoma.[23,74]

Excision is the treatment of choice for confirmed or suspected neoplasms affecting the third eyelid. A success rate of

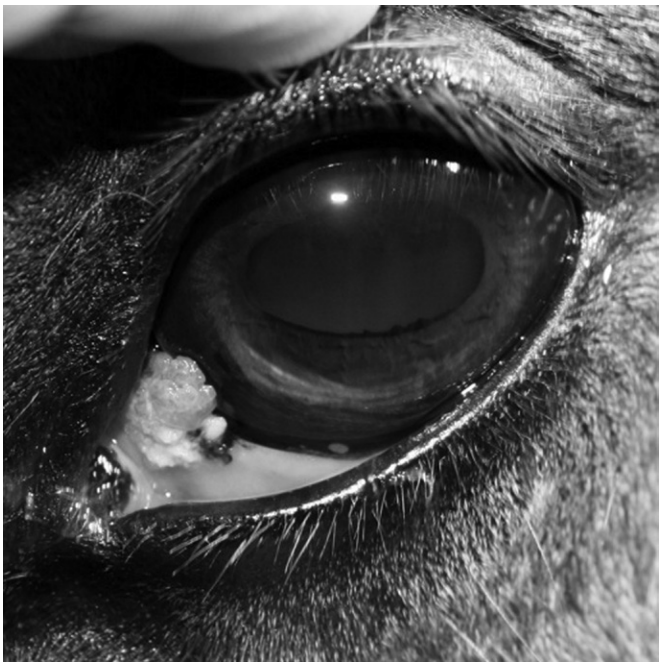

Figure 56-21. Typical verrucose appearance of a squamous cell carcinoma affecting the leading edge of the nictitating membrane.

approximately 90% has been previously reported using surgical excision alone,[30,36,75] although adjunctive therapies such as gamma- or beta-irradiation have been described in a small number of cases with limited effects on recurrence rates.[30] A recent study has shown that topical mitomycin C (0.2 mL of 0.04% formulation every 6 hours in 1-week cycles) can be effective alone or in combination with surgical excision.[76] Cryotherapy alone or as an adjunct to surgical excision should be avoided in cases of squamous cell carcinoma, because it has been shown to increase the risk of local tumor recurrence by 2.5 times.[30] If the tumor extends beyond the third eyelid, alternative or additional surgical and adjunctive therapies may be necessary. Partial excision is not recommended because of the recurrence risk[75] and the cut edge of the third eyelid cartilage can abrade the cornea. Excision can be performed under general anesthesia but is more commonly performed on the standing, sedated horse with local anesthesia.[75] A combination of auriculopalpebral, infratrochlear, and zygomatic nerve blocks; local infiltration; and topical analgesics usually provide adequate analgesia (Figure 56-22), although a retrobulbar block may be required to provide additional analgesia (see Chapter 55).

The third eyelid is grasped on the T-cartilage using Allis tissue forceps and elevated. Large hemostatic forceps are advanced from the medial and lateral edge along the base of the third eyelid until the tips are apposed proximal to the gland of the nictitans. All tissue superficial to the hemostatic forceps including the T-cartilage and gland are removed *en bloc* using scissors or a scalpel blade. The hemostats are removed after 1 to 2 minutes. The cut edges of conjunctiva can be sutured together using 5-0 or 6-0 polyglactin 910 in a simple-continuous pattern to reduce the risk of orbital fat prolapse.[56] All tissue removed should be submitted for histopathologic examination for determination of tumor type and surgical margins.

AFTERCARE

Topical antibiotic (with or without a steroid) ointment should be applied to the eye for 5 to 7 days following surgery. Flunixin meglumine administration for the first 3 to 5 days will reduce inflammation and discomfort. Although the nictitans gland is removed when excising the third eyelid, very few equine eyes develop tear production deficits,[75] and long-term lubricants are not usually required.

COMPLICATIONS

Complications are rare but include orbital fat prolapse, keratoconjunctivitis sicca, and superficial keratitis.[56] The absence of a third eyelid can impair globe protection and result in mild chronic ocular irritation and discharge.[56]

Lacerations

Lacerations affecting the third eyelid should be treated surgically to reappose the edges of conjunctiva and restore the margin of the third eyelid. Apposition of the margin should precede proximal sutures, because inappropriate apposition may result in scarring and corneal trauma. Sutures of 6-0 or 7-0 polyglactin 910 should be placed emerging from the palpebral surface. Sutures of the bulbar conjunctiva should be placed

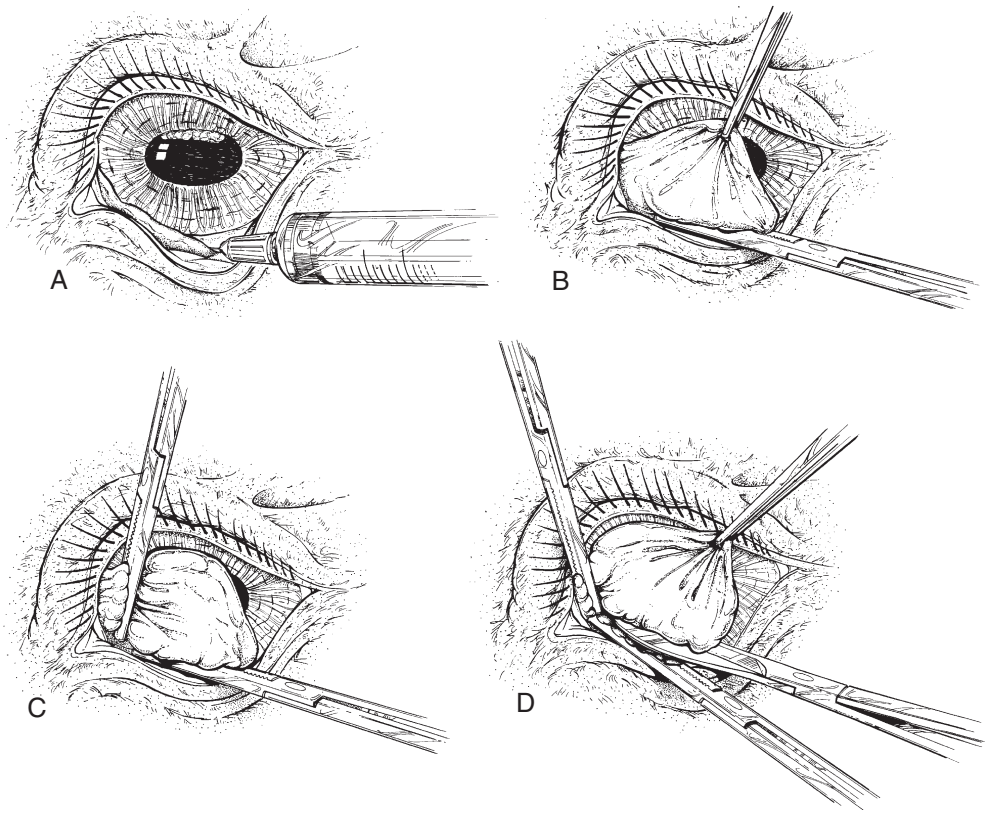

Figure 56-22. Surgical removal of the third eyelid. **A,** Local anesthetic is injected at the base of the third eyelid. **B,** The nictitating membrane is lifted from the fornix with forceps. **C,** Two hemostats are placed across the base of the third eyelid. **D,** The third eyelid is excised along the two hemostatic forceps.

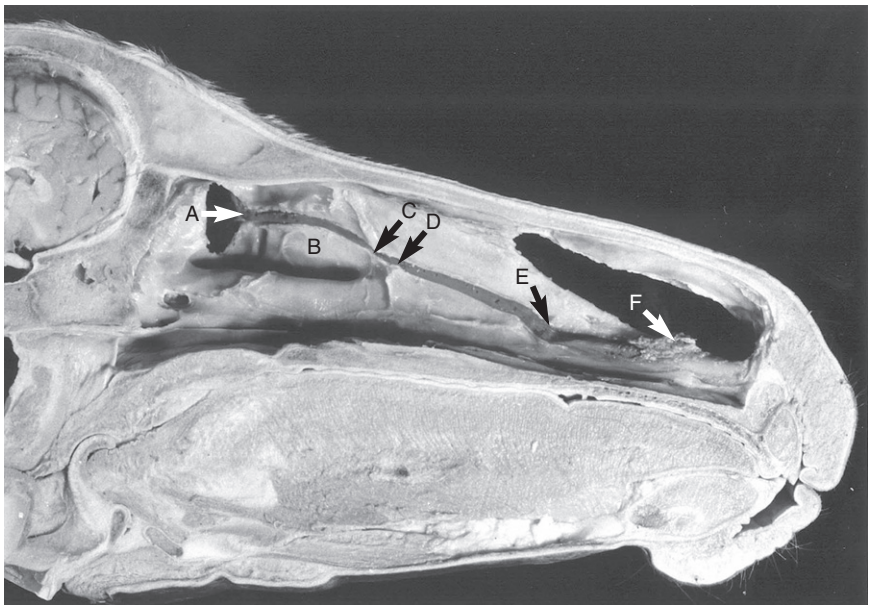

Figure 56-23. Methyl methacrylate cast of the left nasolacrimal duct of a horse. The medial bony orbit and medial wall of the lacrimal canal have been removed. **A,** Lacrimal sac. **B,** Course of the duct within the lacrimal bone. **C,** Narrowing of duct lumen before exiting lacrimal bone. **D,** Exit of duct from lacrimal bone. **E,** Compression of duct by cartilage within alar fold. **F,** Cast within the basal fold. (Reproduced with permission from Latimer CA, Wyman M, Diesem CD, et al: Radiographic and gross anatomy of the nasolacrimal duct of the horse. Am J Vet Res 45:451, 1984.)

carefully to avoid contact of suture material with the cornea. Any exposed cartilage should be covered by conjunctiva, particularly on the bulbar surface of the third eyelid.

NASOLACRIMAL SYSTEM

Anatomy and Physiology

Preocular Tear Film

The preocular tear film covers the cornea and conjunctiva and has three components. The *outer lipid* component is derived from the meibomian (i.e., tarsal) glands and the sebaceous glands of Zeis within the eyelids. The *middle aqueous* layer is predominantly derived from the lacrimal gland situated in the dorsolateral orbit (between the zygomatic process of the frontal bone and the eye). The secretions from the lacrimal gland enter the lateral part of the superior conjunctival fornix. Smaller contributions to this layer are produced by the gland of the third eyelid (i.e., nictitans gland) and the accessory lacrimal glands of the eyelids. The *inner mucinous* layer is derived from the conjunctival goblet cells and intraepithelial glands within the conjunctiva.

The tear film has many functions including flushing foreign material, lubricating the eye to facilitate the movement of the eyelids and third eyelid, maintaining a refractive corneal surface, delivering nutrients to the cornea and conjunctiva, and facilitating immune protection. In the normal equine eye, a complete recycling of tear volume occurs approximately every 7 minutes.[77] The lacrimal lake results from flow of tears across the cornea following blinking and forms along the leading edge of the lower eyelid and adjacent to the inferior edge of the lacrimal caruncle and third eyelid.

Nasolacrimal Drainage System

Horses possess both superior and inferior lacrimal puncta (see Figure 56-1, *C*). Each punctum is a horizontal slit in the palpebral conjunctiva approximately 2 mm in length. The lower punctum is generally larger, closer to the medial canthus, and farther from the eyelid margin.[78] Tear drainage into the puncta occurs via capillary action and blinking.

The nasolacrimal system begins with a pair of nasolacrimal canaliculi connecting the puncta with the nasolacrimal sac. The sac is often poorly defined in horses and lies within the lacrimal fossa of the lacrimal bone. The nasolacrimal duct measures approximately 29 to 33 cm long[79] and passes through the lacrimal canal within the lacrimal bone and maxilla. The duct is narrowest as it exits the lacrimal canal (Figure 56-23).[78,79] When performing maxillary trephination or evaluating trauma to this region, the course of the nasolacrimal duct may be predicted by drawing a line from the medial canthus to the infraorbital foramen.[78] After exiting the lacrimal canal, the duct continues within the lacrimal groove of the maxilla beneath the mucous membrane of the middle nasal meatus.[2] It continues within the basal fold of the ventral nasal concha, where the duct wall becomes irregular as it passes through the vascular plexus of the basal fold. It courses over the nasal process of the incisive bone to end near the mucocutaneous junction in the ventral nasal meatus at the nasal ostium, which is 3 to 4 mm in diameter and visible clinically (Figure 56-24). The nasolacrimal duct may contain blind-ending branches into channels that may not reach the nasal ostium.

Examination Techniques

In the investigation of nasolacrimal system abnormalities, a systematic approach to diagnosis should always be adopted, including a full ophthalmic and systemic examination. Keratoconjunctivitis sicca is rare in the horse. When it is present, the neurogenic form is most common and is often seen in combination with facial and/or trigeminal nerve paralysis.[80] A complete description of the investigation of dry eye conditions is beyond the scope of this text. However, when investigating corneal surface abnormalities, a Schirmer tear test (Schirmer tear test strips, Schering-Plough Animal Health, Kenilworth, NJ), fluorescein staining, and rose bengal staining should

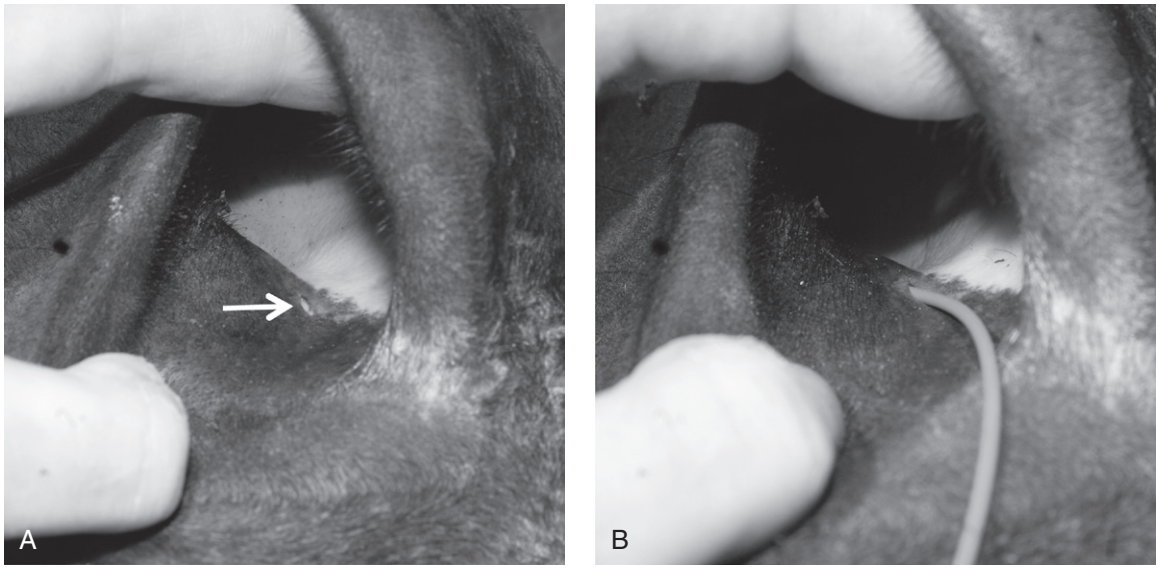

Figure 56-24. Identification and catheterization of the nasal ostium. **A,** The nasal ostium is visible in the ventral nasal meatus close to the mucocutaneous junction (*white arrow*). **B,** Distal catheterization using a 5-French rubber urinary catheter facilitates retrograde flushing of the nasolacrimal system.

be considered part of the ophthalmic diagnostic workup. A Schirmer tear test value of less than 10 mm/min is considered abnormal in horses and ponies.[81] The administration of any topical drops such as tropicamide affect Schirmer tear test values.[82] Cytology and bacterial culture and sensitivity testing should be considered in suspected cases of dacryocystitis.

The most straightforward method to evaluate nasolacrimal system patency is to assess the passive drainage of fluorescein to the nasal ostium by instilling fluorescein stain (wet fluorescein strips or single-use vials containing 2% fluorescein) into the conjunctival sac.[54] Positive drainage confirms nasolacrimal patency. A more-invasive method is to perform nasolacrimal cannulation via the upper and lower puncta (see Figure 56-1, *C*). Sterile disposable cannulas or reusable autoclavable cannulas can be used for cannulation, which can be performed under topical anesthesia on the standing, sedated horse if necessary. Minimal force should be required to achieve drainage through the nasal ostium. The use of fluorescein solution as the flushing medium aids in the positive diagnosis of patency. Retrograde flushing techniques can also be performed using a 5- to 6-French pliable urinary catheter (see Figure 56-24, *B*).

Diagnostic Procedures

Dacryocystorhinography

Radiographic methods of dacryocystorhinography have been described that involve cannulating the upper lacrimal punctum and injecting viscous contrast medium before radiography (Figure 56-25).[78,83] This technique has been described in the standing horse. Even in unilateral disease, it is advantageous to perform the technique on both nasolacrimal ducts to provide an internal control. Recent work has demonstrated that the nasolacrimal duct of the horse can be evaluated using CT dacryocystorhinography (Figure 56-26).[84] This technique requires general anesthesia and positioning the horse in dorsal or sternal recumbency. About 5 mL of iodinated contrast medium is injected retrogradely through the nasal ostium.

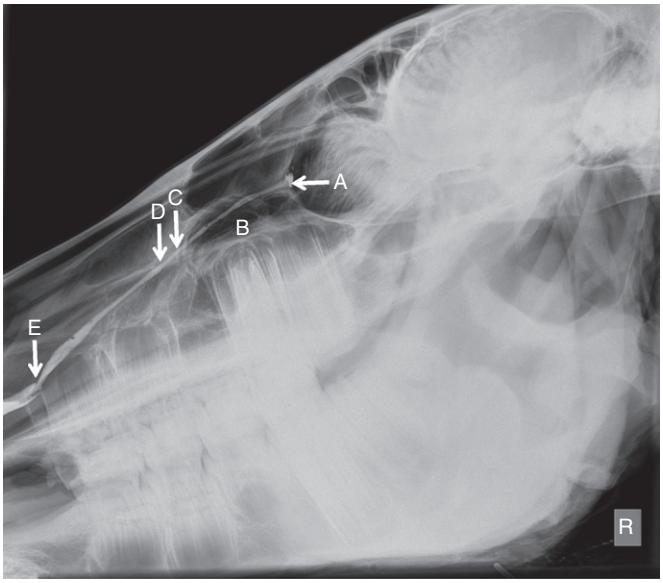

Figure 56-25. Normal right lateral equine dacryocystorhinogram illustrating the anatomical features shown in Figure 56-23. **A,** Lacrimal sac. **B,** Course of the duct within the lacrimal bone. **C,** Mild narrowing of duct lumen before exiting lacrimal bone. **D,** Exit of duct from lacrimal bone. **E,** Compression of duct by cartilage within alar fold. (Courtesy Dr. Anthony Pease.)

Endoscopy

Endoscopic evaluation of the nasolacrimal duct has been described in adult horses.[79] The portion of the duct within the lacrimal bone is difficult to examine using this method. This procedure may be useful for collecting samples in cases of dacryocystitis or aiding the diagnosis and removal of foreign bodies or dacryoliths.

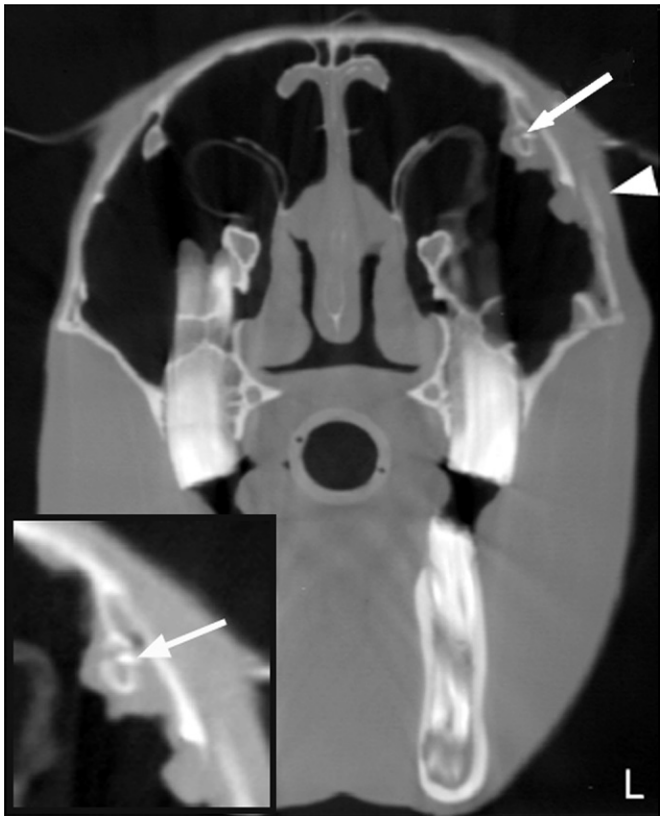

Figure 56-26. Transverse computed tomography dacryocystography scan of an equine skull at the level of the caudal maxillary sinus. There is a fracture of the left maxilla with adjacent soft tissue swelling (*arrowhead*). No disruption of the nasolacrimal duct is evident (*arrow*). The inset is a close-up of the nasolacrimal canal and duct. (Reproduced with permission from Nykamp SG, Scrivani PV, Pease AP: Computed tomography dacryocystography evaluation of the nasolacrimal apparatus. Vet Radiol Ultrasound 45:23, 2004.)

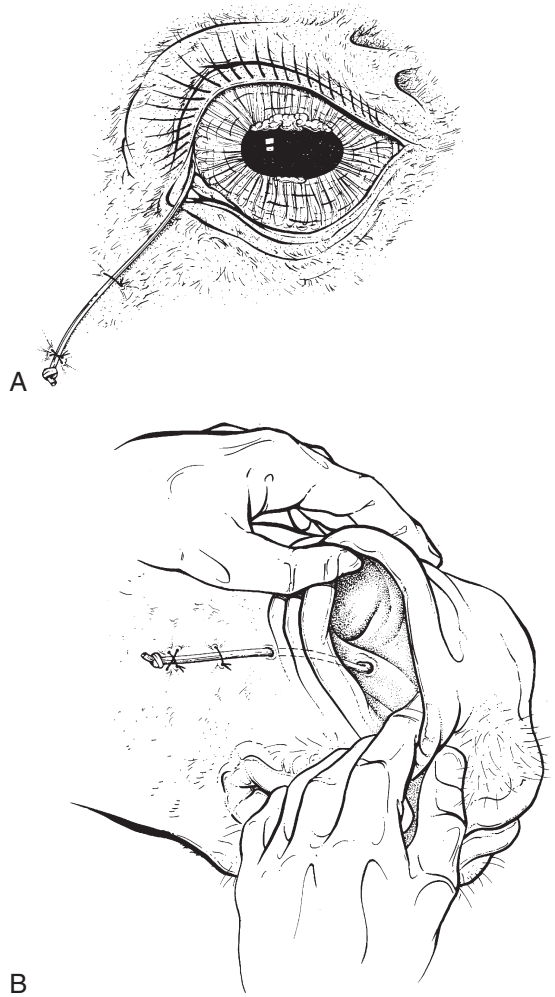

Figure 56-27. A catheter is sutured to the periorbital skin **(A),** and skin of the nostril **(B)** after cannulating and surgically opening an imperforate nasolacrimal duct.

Anesthetic Considerations

Topical anesthesia and standing sedation is useful as described earlier. For additional analgesia, a cotton-tipped applicator soaked in proparacaine can be applied directly onto the nasolacrimal puncta for 2 to 5 minutes before proceeding to its cannulation.

Required Surgical Equipment

Cannulas and catheters for cannulation of the nasolacrimal duct are useful. Standard single-use plastic cannulas and autoclavable steel cannulas are available. For cannulation of the nasolacrimal system to correct nasolacrimal punctal atresia, cardiac catheters of various sizes are useful (No. 4 to 6). A round-tipped, eyed pigtail probe (modified Worst probe) is effective in facilitating the placement of a silicone-tubing stent to treat lacerated nasolacrimal canaliculi.[85]

Surgical Techniques

Imperforate Puncta

The most common defect of the nasolacrimal system in the horse is an imperforate distal nasal punctum.[86] Other abnormalities include imperforate proximal lacrimal puncta within the eyelids and atresia or agenesis of the duct,[87] which should be ruled out by clinical examination, cannulation and flushing of the nasolacrimal system, and/or diagnostic imaging techniques. Clinical signs of epiphora or mucopurulent ocular discharge are typically first reported by owners in animals ranging from birth to 1 year of age.[54,86] Anomalous supernumerary openings of the nasolacrimal system have also been reported.[88]

Treatment of imperforate nasal puncta can usually be performed on the standing, sedated horse. The nasolacrimal duct can be filled with sterile physiologic saline with the addition of dilute fluorescein dye to aid visualization. The mucosa overlying the bleb of fluid within the nasal passage can be incised using a scalpel blade. Alternatively, a catheter (5- to 6-French silicone male urinary catheter[86]) can be inserted into the upper lacrimal puncta and passed down the duct, and the incision can be made over the palpable end of the catheter. The portion of catheter emerging from the lacrimal puncta should be sutured to the skin of the face (Figure 56-27). The distal portion of catheter can be sutured to the nose or lip or passed through the lateral wall of the nostril[86] (see Figure 56-27, *B*). The catheter is typically maintained in place for 2 to 4 weeks.

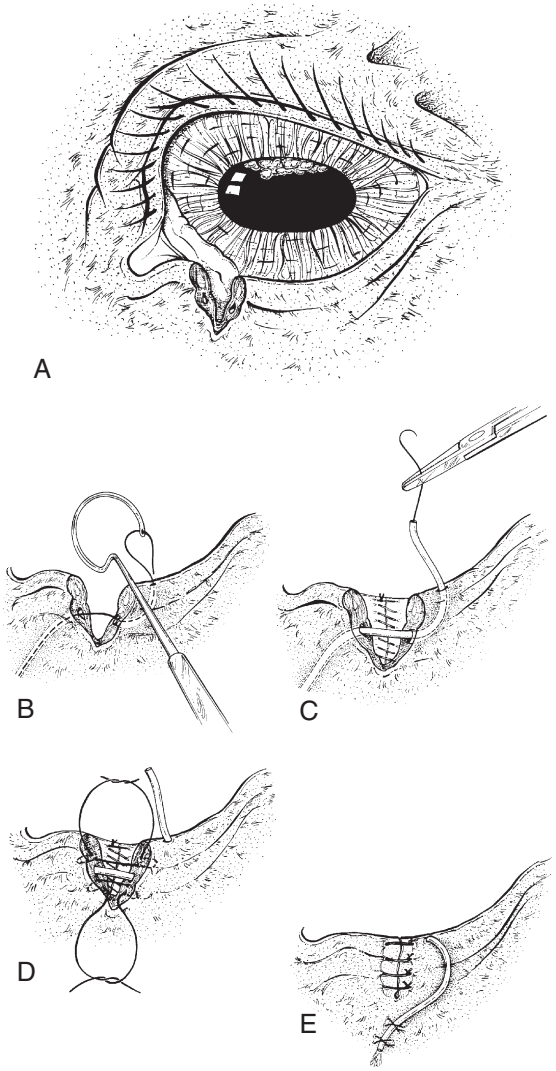

Figure 56-28. Repair of the severed lacrimal canaliculus. **A,** Laceration of the lower eyelid, severing the lacrimal canaliculus. **B,** A 2-0 nylon suture is passed through the nasolacrimal duct and exits through the wound. A Worst probe is passed through the ventral punctum and draws the suture through the distal portion of the severed canaliculus. **C,** A fine silicone tube is cut to a taper, tied to the suture, and pulled through the canaliculi. **D,** The canaliculus and wound are sutured with 6-0 nylon silk. **E,** The tubing is sutured to the skin of the eyelid and at the nasal end. It is left in place for 3 weeks.

Atresia of either the upper or lower lacrimal punctum can be treated by cannulation of the patent lacrimal punctum and using a scalpel blade to incise tissue over the bleb created. A silicone stent can be placed to aid healing.

Canaliculorhinostomy may be a surgical alternative for atresia or agenesis of substantial portions of the distal nasolacrimal duct.[89] An 18-gauge, 1.5-inch needle is used to drill into the rostral maxillary sinus, using the lower eyelid canaliculus as a guide; 60-gauge fishing line is used to create a retention stylette that remains in place for at least 1 month. Another method of conjunctivorhinostomy is to create a stoma between the inferior conjunctival fornix and the maxillary sinus.[90] With both of these procedures, there is a risk of strictures forming in the artificial stoma over time.

Lacerations

Lacerations of the nasolacrimal system occur most commonly at or near the eyelid margin. Surgical correction is indicated to prevent inappropriate healing or scarring, resulting in blockage of the canaliculus or punctum and resultant epiphora. The repair of nasolacrimal punctal lacerations have been well described in human texts, although no reports for equine nasolacrimal lacerations were found. A blunt-tipped, eyed pigtail probe is inserted into the unaffected punctum and fed until it exits from the severed end of the canaliculus (Figure 56-28).[85] A 6-0 polypropylene suture is placed in the eye of the probe, which is then retracted to draw the suture through to the unaffected punctum. The probe is passed through the punctum of the lacerated canaliculus and the suture is again drawn through to exit from this punctum. A silicone stent tube is passed over the suture (the tube cannulates both nasolacrimal canaliculi and puncta) from the unaffected side. The suture material can be tied, maintaining the silicone tubing in place, or it can be removed and the ends of the tube can be sutured together. The canaliculus and eyelid skin are sutured using 6-0 polypropylene. The tube is sutured in place and left for 4 to 6 weeks to maintain patency as the canaliculus heals.[91] Topical and/or systemic antibiotics should be administered in the postoperative period.

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CHAPTER
57 Surgery of the Ocular Surface

Matthew J. Annear and Simon M. Petersen-Jones

ANATOMY AND PHYSIOLOGY

The ocular surface consists of the cornea and conjunctiva. The clear cornea allows light to enter the eye and is continuous with the sclera, forming the outermost fibrous wall of the globe. The junction between the cornea and the sclera is termed the *limbus,* and at this site the epithelium of the cornea transitions into conjunctival epithelium. The bulbar conjunctiva overlies the sclera of the globe, over which it is relatively mobile. The conjunctiva is reflected at the fornices to line the third eyelid and the inner aspect of the eyelids where it is termed the palpebral conjunctiva (Figure 57-1). Beneath the bulbar conjunctiva lies loose connective tissue, Tenon's capsule (a thin fascia enveloping the eyeball), and the episclera.

Conjunctiva

Structure and Function

The conjunctiva is a thin mucous membrane, the bulbar component of which is typically pigmented because of the presence of melanin. It has a nonkeratinized stratified columnar to

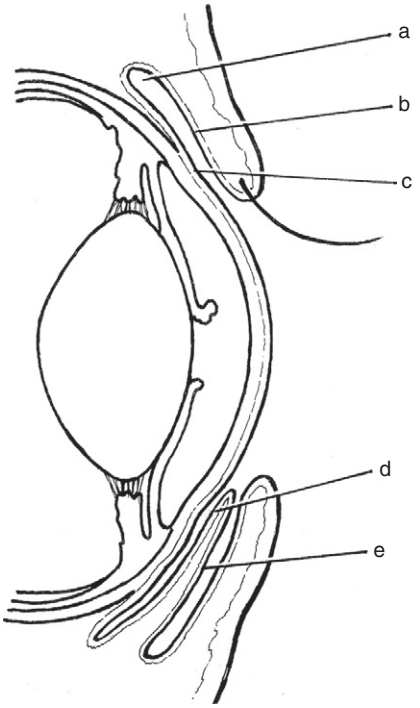


Figure 57-1. Areas of the conjunctiva. *a,* Fornix; *b,* palpebral conjunctiva; *c,* bulbar conjunctiva; *d,* bulbar conjunctiva of the third eyelid; *e,* palpebral conjunctiva of the third eyelid.

cuboidal epithelium, which is interspersed with goblet cells and surrounded on the palpebral and bulbar margins by a stratified squamous epithelium.[1] The substantia propria of the conjunctiva consists of two layers: the superficial adenoid layer that contains the lymphoid tissue and a deeper fibrous layer that contains the nerves and blood vessels. The conjunctiva plays three important roles. It facilitates movements of the globe and eyelids by the relatively loose attachments of its substantia propria to the underlying tissues. It provides an important line of defense through its role as a physical barrier, and more specifically by the presence of conjunctiva-associated lymphoid tissue that plays a significant role in the immune response to environmental antigens.[2] This conjunctiva-associated lymphoid tissue has also been shown to contribute to corneal immune protection.[3] Finally, goblet cells within the conjunctival epithelium produce mucus that is a principal component of the precorneal tear film, essential for maintenance of a clear cornea.

Lymphatics, Vasculature, and Innervation

The conjunctiva contains aggregates of lymphoid follicles, most numerous in the conjunctival fornices and on the bulbar aspect of the third eyelid. The lymphatics drain toward the commissures of the eyelids, with the lateral commissures draining to the parotid lymph nodes and the medial regions draining to the submandibular lymph nodes.

The conjunctival arterial supply is derived from the anterior ciliary arteries, which are branches of the ophthalmic artery, and the vascular arcades of the eyelids are derived from the ophthalmic artery medially and the lacrimal artery laterally. These vessels terminate as small radiating superficial conjunctival vessels. Venous drainage is via the palpebral veins and superior and inferior ophthalmic veins.

The bulbar conjunctiva is innervated by the long ciliary branches of the ophthalmic division of the trigeminal nerve. The superior palpebral conjunctiva is innervated by the frontal and lacrimal branches of the ophthalmic division of the trigeminal nerve, whereas the inferior palpebral conjunctiva is innervated by the both the lacrimal branch of the ophthalmic nerve and the infraorbital branch of the maxillary division of the trigeminal nerve.[4]

Response to Injury

Because of its dense vasculature and prominent lymphatics, the conjunctiva responds rapidly to ocular surface injury or disease by becoming hyperemic and edematous. Swelling of the conjunctiva can be so extensive that it causes the conjunctiva to balloon between the eyelids. This response to injury is nonspecific, consistent for surface irritation, infection, and neoplastic disease. Additionally, hyperemia of the conjunctiva frequently accompanies corneal lesions. With chronicity, the conjunctival epithelium can hypertrophy and lymphoid follicular hyperplasia may be observed. Most importantly for the surgeon, the conjunctival epithelium heals rapidly after injury, and often sutures are not needed if the defect is small.

Sclera

The sclera is the main protective wall of the eye. Along with the cornea, it encloses the intraocular structures to protect them from injury and is important for maintaining the structure of the globe and the intraocular pressure. It is composed almost entirely of collagen with a small amount of elastin and proteoglycans. Unlike in the cornea, the collagen fibers are irregularly arranged, so they scatter light and give the sclera its white color. The well-vascularized episclera is the outermost layer of the sclera, attaching to the fibrous Tenon's capsule that envelops the globe, and is adherent to the conjunctiva. The arterial blood supply is derived from the anterior and posterior ciliary arteries, arising from the ophthalmic artery. Anteriorly the intrascleral venous plexus receives aqueous humor via drainage through the iridocorneal angle. Sensory innervation to the anterior sclera is provided by the long posterior ciliary nerves, derived from the ophthalmic nerve. The posterior sclera is innervated by the short posterior ciliary nerves that branch from the ciliary ganglion to enter the sclera close to the optic nerve.

Limbus

The limbus represents the junction between the corneal stroma and the sclera, with the sclera obliquely overlying the cornea superficially. This scleral "shelf" obscures direct observation of the iridocorneal drainage angle dorsally and ventrally. Temporally and nasally the insertion of the pectinate ligaments, the thin fibers that span from the iris base to the peripheral cornea, can be viewed directly through the cornea (Figure 57-2). Superficially at the limbus the conjunctiva continues over the cornea as the corneal epithelium. At this junctional zone, limbal stem cells are present, located in the basal epithelium. These are important for healing extensive corneal lesions as well as for maintaining a healthy corneal epithelium under normal conditions.[5]

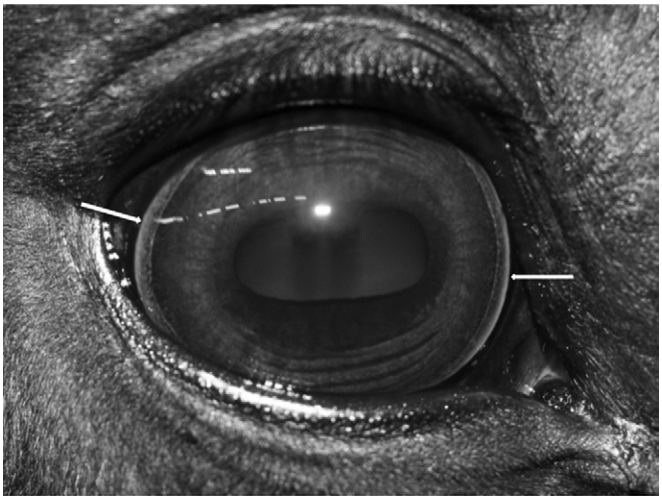


Figure 57-2. Blue-gray appearance of the drainage angle as seen through the normal equine cornea at the temporal and nasal limbus *(arrows).*

Figure 57-3. Photomicrograph demonstrating the four layers of the equine cornea. *a,* Epithelium; *b,* stroma; *c,* Descemet's membrane; *d,* endothelium *(arrow)* (hematoxylin and eosin, ×170).

Cornea

Structure and Function

The cornea is a transparent, highly specialized structure that transmits light and contributes to the anterior portion of the fibrous tunic of the globe. It is also the major refractive structure of the eye, so maintenance of corneal clarity is therefore essential. To this end, the cornea lacks blood vessels, has a nonkeratinized surface epithelium, and has regularly arranged parallel bundles of collagen fibrils organized into lamellae.

The shape of the cornea is a horizontal ellipse, measuring 29.7 to 34.0 mm horizontally and 23.0 to 26.5 mm vertically in the adult horse.[6] In the Miniature Horse, the cornea approximates 25.8 mm horizontally and 19.4 mm vertically.[7] Measurements of corneal thickness vary with the technique used for assessment but range from 0.77 mm to 0.89 mm centrally, with thickness increasing toward the periphery.[6-9] The cornea can be divided into four layers: epithelium, stroma, Descemet's membrane, and endothelium (Figure 57-3).

The most superficial layer is the *epithelium,* which is 8 to 10 cell layers thick[10] and serves two main functions. It provides a physical barrier to injury, and it is a smooth optical surface, aided in part by the overlying precorneal tear film. The most superficial layer is a nonkeratinized stratified squamous epithelium, and the middle layer consists of polyhedral wing cells. The basal layer of the epithelium is anchored to the underlying basement membrane and thus the corneal stroma by hemidesmosomes. The basal cells are columnar and are continually dividing. This activity is important for the healing of epithelial defects and the constant renewal of the surface epithelium, an important component of the ocular defenses.

The *corneal stroma,* located beneath the epithelium, makes up approximately 90% of the corneal thickness. It maintains transparency by the lack of vasculature and the narrow diameter collagen fibrils, predominantly type I and type V, that are uniformly spaced and organized into parallel sheets.[11] This natural arrangement facilitates separation of the corneal stroma along a plane parallel to the corneal surface, relevant for the surgeon performing a keratectomy. The stroma is relatively acellular but has keratocytes distributed throughout its structure that play an important role in wound healing. Following injury and formation of new reparative collagen fibers, the resulting irregular arrangement can scatter light and is appreciated as a corneal opacity or scar. The corneal stroma is hydrophilic and is maintained in a relative state of dehydration by the presence of an intact corneal epithelium and the action of the corneal endothelial pump mechanism (see later).

Descemet's membrane, lying beneath the stroma, is an exaggerated basement membrane that is produced continually by the corneal endothelium and thickens with age. This is often the last line of defense before perforation of a deep corneal ulcer and does not take up fluorescein stain.

Underneath Descemet's membrane is the *corneal endothelium.* This is a monolayer of roughly hexagonal cells that have tight cellular junctions that limit passage of fluid from the anterior chamber into the stroma. Additionally they have an active Na^+/K^+–ATPase pump that results in a net movement of Na^+ out of the cornea, with water following because of the osmotic gradient effect. Unlike other corneal tissues, the endothelial cells have little or no capacity for regeneration in the adult and their density decreases with age.[9,12-14] Following trauma or damage to the corneal endothelium, the remaining cells spread in an attempt to recreate a complete coverage of the posterior corneal surface.

Vasculature and Innervation

Since the cornea is normally avascular, nutrition is provided by the precorneal tear film and the aqueous humor. Normal corneal health therefore requires functional eyelids and a nictitating membrane for protection and distribution of the preocular tear film, as well as normal circulation of aqueous humor. The cornea is richly innervated by the long ciliary nerves, derived from the ophthalmic branch of the trigeminal nerve. These fibers are myelinated only at the corneal periphery,

and arborization density is greatest superficially. Those entering the epithelium terminate in naked nerve endings among the wing cells.

Response to Injury

The prominent position of the equine globe, along with the environment horses reside in, means that injuries to the ocular surface are common. Corneal responses to injury include development of edema, and with persistence of the insult, vascularization and pigmentation; in some instances, scar forms as the lesion resolves.

Corneal *edema* results in a blue-gray cloudlike haze to the cornea because of the accumulation of fluid separating the regularly arranged stromal collagen fibers. Fluid may enter the cornea from the tear film with loss of the corneal epithelium, or it can enter from the aqueous humor as a result of damage or dysfunction of the corneal endothelial cells. Corneal *neovascularization* develops in response to inflammation, the presence of polymorphonuclear (PMN) leukocytes, and cytokines such as fibroblast growth factor.[15] In disease states, this limbal ingrowth of vessels provides additional leukocytes and antibodies and helps combat infection and speed healing. Occasionally, persistent and prolonged irritation results in an exuberant vascular response. Drugs such as flunixin meglumine have been shown to delay corneal neovascularization. Despite this, they are often used because they play an important role in combating anterior uveitis and improving ocular comfort.[16] Fungi have also been proposed to produce metabolites that inhibit neovascularization, contributing to their persistence in the cornea.[17] Superficial corneal pigmentation can result from chronic corneal inflammation, where there is recruitment of epithelial cells from the limbus with migration of melanocytes from limbal and perilimbal tissue.

Wound Healing

Since the cornea is normally avascular, it requires growth factors, cytokines, and neuropeptides from the aqueous humor, tear film, and limbal vessels to coordinate wound healing. Factors such as epidermal growth factor increase mitosis and protein synthesis in the corneal basal epithelial cells and the stromal keratocytes.[18,19] Cytokines such as transforming growth factor-β (TGF-β) induce differentiation and infiltration of inflammatory cells.[20]

After damage to the corneal epithelium, the epithelial cells at the edge of the defect retract, losing their hemidesmosmal attachments (rivet-like structures that attach the basal epithelial cells to the corneal stroma); they then become thinner and migrate toward the center of the lesion in an amoeboid fashion. This occurs very soon after the initial insult, with basal epithelial cells sending out cytoplasmic processes within 1 hour of injury and PMN leukocytes arriving within several hours.[10,21] In the absence of infection, normal healing of a corneal defect is rapid and linear for 5 to 7 days before slowing.[19]

If the stroma is involved, the defect is filled with a fibrin clot and adjacent keratocytes are transformed by TGF-β into a myofibroblast-like phenotype that facilitates wound contraction. Depending on the chronicity, corneal vascularization occurs and can promote healing of more serious defects. Fibroblastic collagen is deposited in the defect, resulting in corneal scarring, which slowly remodels and becomes smaller with time. The mean healing time of an uninfected corneal wound of 30% stromal depth is reported to be 11 days.[10] However, normal strength of the injured tissue may not return for a considerable time. Preventing and controlling infection is essential to achieve healing with minimal scar formation. Following trauma to the conjunctival epithelium, with or without stromal involvement, the normal bacterial and fungal flora or contaminating organisms can colonize and then infect the wound.[22,23]

Full-thickness wounds resulting in aqueous leakage can seal as a fibrin clot forms. Larger defects that result in collapse of the anterior chamber may be occluded by the iris, forming an anterior synechia, which is an adhesion between the iris and cornea. With larger defects, there may be iris prolapse resulting in protuberance of the iris from the corneal surface, typically with a coating of clotted fibrin. In full-thickness defects that seal without iris involvement, corneal endothelial cells spread to fill the defect. The new layer of endothelial cells produce a new Descemet's membrane in that region. As already stated, endothelial cells in adults do not replicate so the cells adjacent to a defect spread to completely cover the inner corneal surface.

Corneal wounding stimulates the sensory fibers of the cornea and can result in a neurogenic anterior uveitis thought to be mediated by substance P.[24-26] Hence, painful conditions of the cornea can cause miosis, ocular hypotension, increased protein levels, and even hypopyon within the aqueous humor. Infected corneal wounds and ulcers can also cause an anterior uveitis and hypopyon, probably at least partly the result of the liberation of toxins.

Ocular Surface Microflora

The normal equine ocular surface microflora shows geographic variations, likely influenced by ambient temperature and humidity. Gram-positive bacteria are most commonly isolated from the normal equine ocular surface and include *Corynebacterium*, *Streptomyces*, *Staphylococcus*, and *Bacillus* species.[27-31] In disease states, gram-negative and gram-positive bacteria may be involved, with *Pseudomonas* and β-hemolytic *Streptococcus* resulting in more serious infections.[29,32-36] Fungi are also commonly isolated from the ocular surface of normal horses.[37] Most commonly recovered are *Aspergillus*, *Penicillium*, and *Cladosporium*.[30,31,38] In diseased corneas, the fungal species most commonly isolated are *Aspergillus*, *Fusarium*, *Candida*, and *Penicillium*.[39-43]

OPHTHALMIC EXAMINATION TECHNIQUES AND DIAGNOSTIC PROCEDURES

Examination of the Ocular Surface

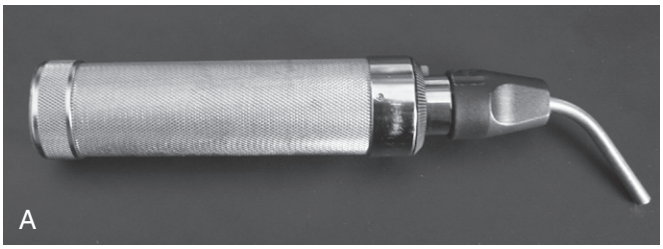
Causes of ocular surface disease can be broadly classified as infectious (bacterial, fungal, viral),[40,44-46] inflammatory (parasitic granulomas, pseudotumors, immune-mediated keratopathies),[47-50] anatomic,[51] traumatic,[52-54] and neoplastic (squamous cell carcinoma, melanoma, lymphosarcoma, mastocytosis, and vascular neoplasms).[55-65] The approach to the investigation of these conditions should begin with collecting data pertaining to signalment and history, followed by a preliminary evaluation of the eyes from a distance, before any manipulation of the globe or adnexal structures. The symmetry and position of the adnexal structures and the globe should be assessed,

with particular attention paid to any evidence of discomfort, appreciated as blepharospasm, epiphora, or photophobia. Variations in conjunctival or corneal coloration, such as red conjunctival hyperemia (engorgement of conjunctival vessels) or blue-gray corneal edema, and the presence of focal opacities or masses should be noted before further examination with illumination and magnification.

Cranial nerve function may be briefly assessed before sedating the horse. Of particular importance is assessment of the *palpebral reflex* and the corneal reflex. Ensuring that the palpebral blink reflex is present and that complete closure of the eyelid is possible is important, because this is required for the normal distribution of the precorneal tear film and thus maintenance of corneal health. The palpebral reflex can be assessed by lightly touching the medial or lateral canthus. In a normal horse, this may not result in complete lid closure; nevertheless, it is important to check that complete closure can be induced.[66] An abnormal palpebral blink reflex can result from deficits of the trigeminal (CN V) or facial (CN VII) nerves. Incomplete lid closure may also occur because of lid abnormalities, including congenital defects and scarring, or abnormalities of the globe's position or size, such as exophthalmos, proptosis, or buphthalmos. Assessment of the *corneal reflex* for any deficits in the ophthalmic branch of the trigeminal nerve will confirm corneal sensation. This test can be performed by lightly touching wisped cotton wool to the cornea and determining whether the response includes eyelid closure and retraction of the globe, mediated by the facial nerve. In performing this test, it is important to ensure that a menace response is not being induced and that contact with the long vibrissae is avoided.

For safe completion of the eye examination, it is necessary to restrain the horse, with the degree of restraint depending on the temperament of the horse and the diagnostic procedures being performed. Use of stocks and sedation may be helpful. When sedation is required, intravenous xylazine (0.5 to 1.0 mg/kg) is adequate for most examinations, though detomidine (0.02 to 0.04 mg/kg IV) will facilitate longer examination and maintenance of a steady head position.[67] Butorphanol (0.01 to 0.02 mg/kg IV) may be used if additional analgesia is required, but as with xylazine, this has been associated with small head jerking motions.[67] Additional information on chemical restraint can be found in Chapter 22. Horses have strong palpebral muscles that can make examination of the ocular surface difficult. Sedation may reduce the strength of the blink but an auriculopalpebral nerve block is often performed, particularly when painful lesions are present. The reader should consult Chapter 55 for details on performing an auriculopalpebral nerve block.

A complete and detailed examination of the ocular surface requires that the exam be performed in a dimly lit environment. Suitable lighting conditions can be achieved by darkening the stall or moving the horse to an area with low light. If this is not possible, many horses tolerate a blanket or towel draped partly over the head of both subject and examiner. Once the light level is reduced, a bright focal light source such as a Finoff transilluminator (Figure 57-4, *A*) can be used to facilitate much of the examination of the anterior segment of the eye. If available, magnification greatly improves the viewer's appreciation of a lesion; this can be via a slit lamp biomicroscope (Figure 57-4, *B*), a simple head loupe (Figure 57-4, *C*), or a direct ophthalmoscope with selection of a high positive diopter lens (+20D). A systematic examination should be performed so that all structures are evaluated and lesions are not missed; this includes an assessment of the adnexa and eyelids, abnormalities of which can contribute to ocular surface disease. The conjunctiva and the ocular surface should subsequently be examined, paying particular attention to the presence and location of corneal neovascularization, edema, stromal infiltrates, and proliferative lesions. It is equally important to assess intraocular structures; for instance, the presence of hypopyon in cases of ulcerative keratitis suggests a poor prognosis and may necessitate therapy with systemic antimicrobials. In instances of fungal



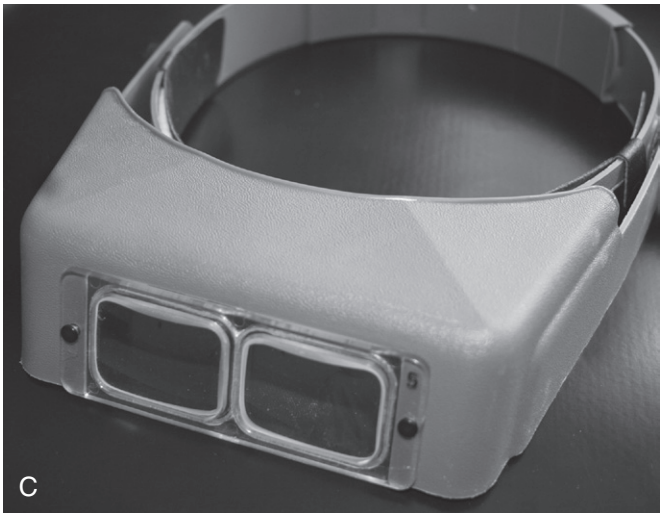


Figure 57-4. A, Finoff transilluminator. **B,** Slit lamp biomicroscope (SL-15, Kowa). **C,** Head loupe (Hongguang Optics).

keratitis, the organisms can traverse Descemet's membrane and persist in the anterior chamber, again indicating a poorer prognosis and altering the therapeutic plan.

Diagnostic Procedures

Ocular surface disease often manifests as a painful red eye. In this case, distinguishing engorged bulbar conjunctival vessels from injected episcleral vessels is important, because the former corresponds to surface ocular disease, whereas the latter is typically seen with intraocular disease. Conjunctival vessels branch extensively, can be moved relative to the underlying globe, and can be blanched by topical application of 2.5% phenylephrine. Other nonspecific signs of ocular discomfort include blepharospasm, photophobia, epiphora, and conjunctival hyperemia, which may indicate adnexal, ocular surface, or intraocular disease. However, approaching such an eye in a logical, systematic fashion yields a diagnosis in the majority of cases. To this end, the order in which diagnostic tests are performed is important. For example, if corneal sensation is to be evaluated, it must be performed before the application of topical anesthetics to the eye. It is also often recommended that samples for culture be taken before topical anesthetics and topical fluorescein are applied, because these agents may reduce the isolation rate of organisms in culture.[68,69] However, in practice the application of a topical anesthetic may be required to facilitate accurate and safe sampling of a lesion. Additionally, the clinician may need to apply fluorescein dye to confirm the presence of an ulcer before sampling for culture is considered.

Ophthalmic Stains

Fluorescein staining is primarily used to detect corneal defects, and this procedure should be performed on any inflamed or painful eye. In areas devoid of epithelium, the dye is able to pass into the hydrophilic corneal stroma, but it does not bind to Descemet's membrane. Hence, an ulcer extending to the depth of Descemet's membrane may be fluorescein negative over the deepest portion, surrounded by a doughnut-shaped area of fluorescein uptake. The dye is available as a single-use 0.5% solution or impregnated paper strips. A portion of the paper strip can be torn off and placed into a syringe containing a commercial eyewash solution to create a fluorescein solution to apply to the eye. This solution can be gently squirted directly onto the cornea from a hubbed syringe with the needle removed (Figure 57-5). Alternatively, the paper strip can be moistened with commercial eyewash and directly applied to the lower conjunctival fornix. Fluorescein peak excitation occurs under blue light, so addition of a cobalt blue filter to a Finoff transilluminator can make it easier to see areas of fluorescein uptake (Figure 57-6). Fluorescein can also be used to assess corneal integrity, for instance, after primary suture closure of a full-thickness corneal laceration. This is known as the *Seidel test*. The dry paper strip is applied directly to the site of injury, which is then monitored for the development of fluorescence, indicating leakage of fluid from the anterior chamber.

Rose bengal is a vital stain that adheres to dead and devitalized epithelial cells. As such, it is useful for detecting the punctate and dendritic patterned lesions seen with equine herpes viral corneal disease and early fungal keratitis.[36] If applied excessively, rose bengal can also stain healthy corneal epithelial cells and has a dose-dependent toxic effect on these cells.[70,71]

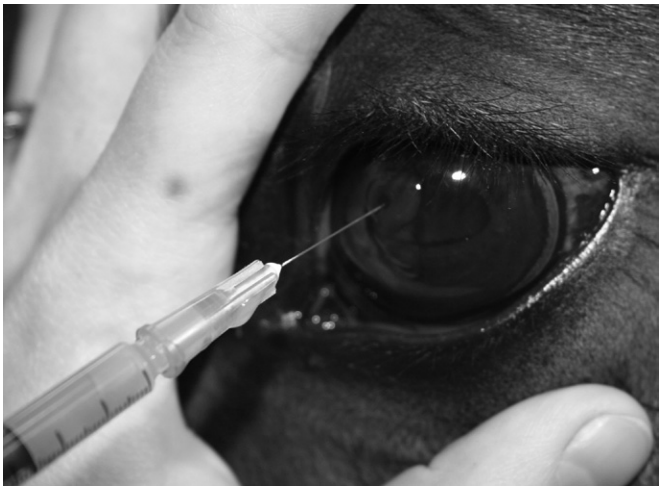


Figure 57-5. Application of fluorescein stain to the cornea using a hubbed syringe with the needle removed.

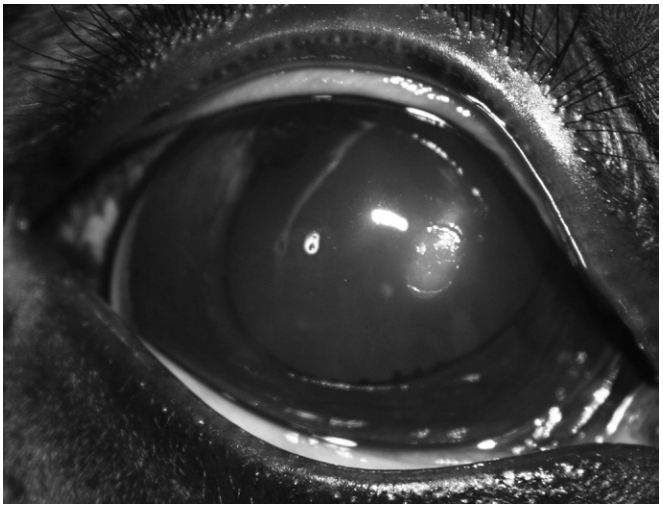


Figure 57-6. Superficial corneal ulcer stained positive with fluorescein.

Culture and Sensitivity

Pathogen culture and sensitivity testing provide useful information for guiding antimicrobial therapy and are important in the management of conditions such as corneal ulcers.[72] Indications for corneal or conjunctival culture include any corneal ulcerative disease, corneal stromal infiltrate, purulent ocular discharge, and proliferative masses. In cases of bacterial or fungal stromal abscesses, the pathogens may be located too deep within the cornea to allow collection of a diagnostic sample. In these cases the removal of epithelium overlying such a region may facilitate a diagnosis. Although topical anesthetics have been shown to inhibit the growth of microorganisms,[69] in many instances their administration is necessary to safely and accurately obtain a sample. To obtain a culture, the eyelids should be retracted and sterile Dacron-tipped culture swabs applied gently to the area (Figure 57-7). Collection of samples for aerobic bacterial and fungal cultures should be performed. In cases of deep or rapidly progressive ulcers, extreme care should be used and the edges rather than the center of the lesion should be sampled. Collection of samples for viral culture may also be

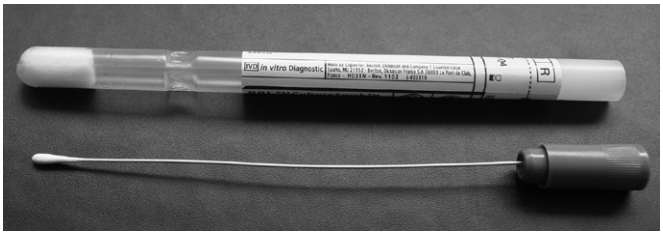


Figure 57-7. Dacron-tipped culture swab (Becton Dickinson).

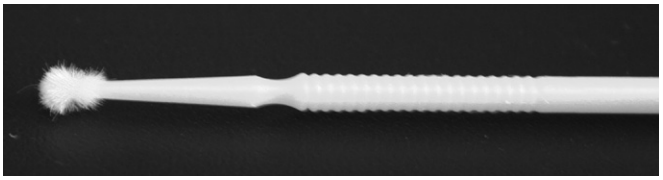


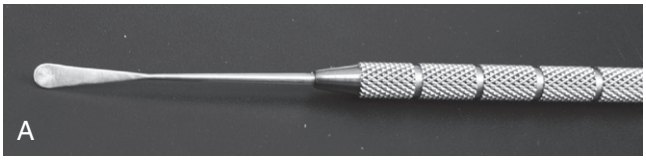
Figure 57-8. Cytobrush for cytology sampling (Microbrush).

indicated if equine herpesvirus 2 (EHV-2) infection is suspected.[45,73] In these cases, specific instructions for collecting and handling the sample should be obtained from the testing laboratory before sampling the lesion. Treatment should commence before receiving the results of culture and sensitivity testing, and for this reason, performing cytology (see next) at the time of the initial examination can be a very valuable guide to initial treatment options.

Cytology

The evaluation of cytology samples collected from the conjunctival or corneal surface can provide immediate characterization of the nature and severity of an inflammatory response, and it can confirm the presence of bacteria or fungal hyphae, thus guiding the initial therapy. Cytology is particularly recommended for corneal ulcers, corneal abscesses, and proliferative masses of the cornea or conjunctiva. In cases with suspected infection, samples are collected to attempt to identify causal organisms while waiting for more definitive results from culture and sensitivity testing. Samples can be collected using a cytobrush (Figure 57-8), a Kimura spatula (Figure 57-9), or the blunt end of a scalpel blade.[74] Of these implements, the cytobrush has been shown to yield superior diagnostic samples.[75,76] A possible exception is when a deep sample is needed for culture of a fungal abscess, in which case a Kimura spatula may prove superior. For deep ulcers, descemetoceles, and perforations, cytology samples should be collected with extreme care. With corneal perforations or other lesions that require treatment under general anesthesia, cytology sampling may be more safely performed while the horse is anesthetized. Usually smears are made on several slides, which can be stained with different techniques or submitted to a clinical pathologist.

After collection, the sample should be immediately subjected to a Romanowsky type of stain, such as Diff-Quik, and evaluated for the presence of inflammatory cells and infectious organisms. If bacteria are seen, another smear should be stained with Gram stain (Figure 57-10). Failure to identify fungal hyphae does not rule out a fungal keratitis because they can be difficult to identify on routine staining (Figure 57-11). Their identification can be facilitated by use of special stains such as Gomori methenamine silver (GMS), periodic acid–Schiff (PAS), and new methylene blue.



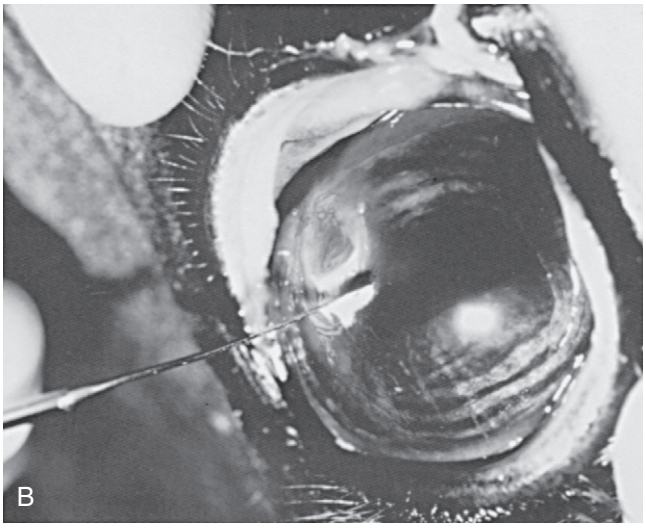


Figure 57-9. A, Kimura spatula. **B,** Technique for acquiring corneal samples for cytology and culture. The corneal surface is aggressively scraped with a Kimura platinum spatula.

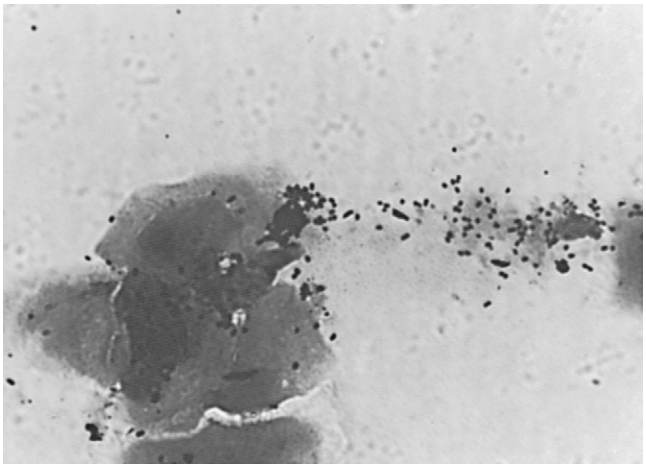


Figure 57-10. Cytologic appearance of corneal scraping from an eye with a bacterial ulcer. Numerous gram-positive organisms are visible (Gram stain, ×1100.).

RELEVANT PHARMACOLOGY

Application of Medication

Topical application of ophthalmic drugs provides a high concentration at the desired site of action for the treatment of ocular surface conditions, often negating the need for systemic dosing. Anti-inflammatories are a common exception to this,

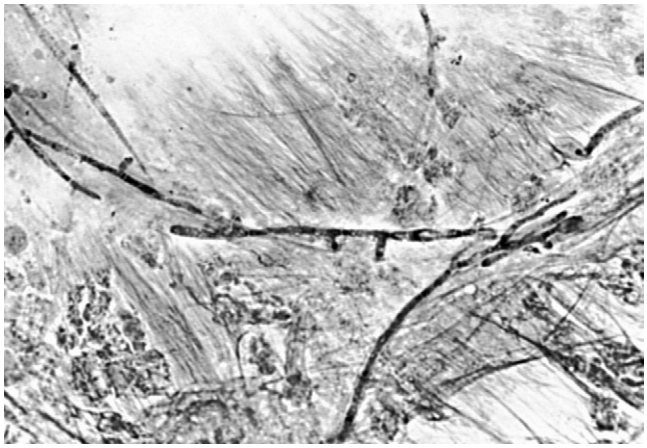


Figure 57-11. Cytologic appearance of corneal scrapings from an eye with a fungal ulcer. Branching hyphae are visible (modified Wright-Giemsa stain, ×700).

and drugs such as flunixin meglumine are often administered systemically in addition to or in place of topical application, both preoperatively and postoperatively, control to ocular inflammation. The retention and absorption of topical ophthalmic mediations depends on precorneal factors, such as lacrimation and nasolacrimal drainage, and characteristics of the drug formulation, which may be solutions, suspensions, or ointments. Topical ophthalmic ointments are often chosen because of their prolonged retention time and relative ease of administration to the equine eye, as compared to ophthalmic solutions. However, if the anterior chamber has been penetrated or is to be entered surgically, ointments should be avoided and an ophthalmic solution should be used. If a solution is used it is common practice to wait 5 minutes between applications of different medications to prevent diluting the previously applied drug. To achieve a persistent high concentration at the site of action, frequent administration may be necessary. This may be facilitated by placing a subpalpebral lavage line.

Subpalpebral Lavage Placement

Placement of a subpalpebral lavage line can be particularly valuable when frequent administration of topical medications is required and painful conditions are being treated. This can reduce the risk of trauma to the surgical site, because the horse often becomes refractory to frequent topical medication for painful conditions. Placement of a subpalpebral lavage may be performed immediately postoperatively before recovery from general anesthesia, or under standing sedation. If placed under sedation, an auriculopalpebral nerve block, topical anesthesia, and infiltration of local anesthetic into the upper eyelid can be performed. Subpalpebral lavage kits are available, comprising a silicone tube plus a circular baseplate with a hubless hypodermic needle for passage through the eyelid (Figure 57-12). Both superior and inferomedial placement have been described,[77] and the choice of site depends on the location of the injury; the goal is to maximize the distance from the tubing and baseplate to the lesion (Figure 57-13). In either instance, the planned entry site at the eyelid should be prepared with 1 : 50 povidone-iodine solution, which is also directly applied with cotton-tipped applicators to the conjunctival fornix at this same

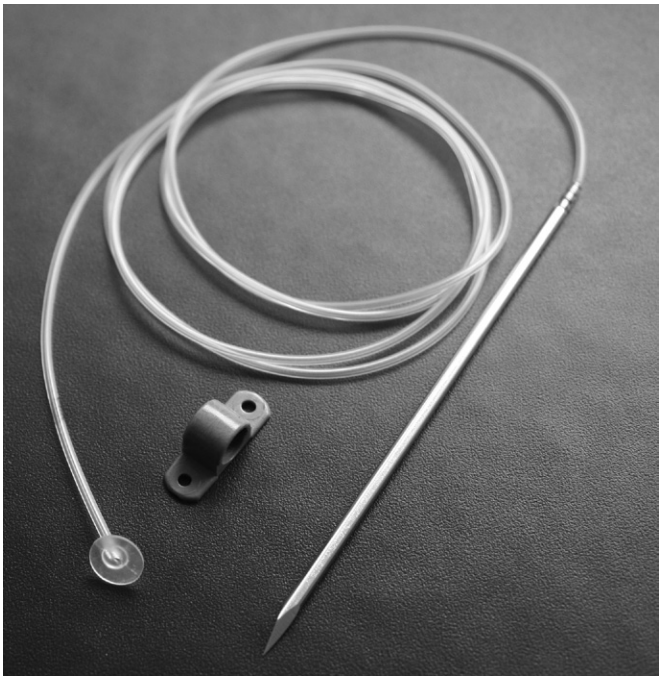


Figure 57-12. Subpalpebral lavage kit (Mila International).

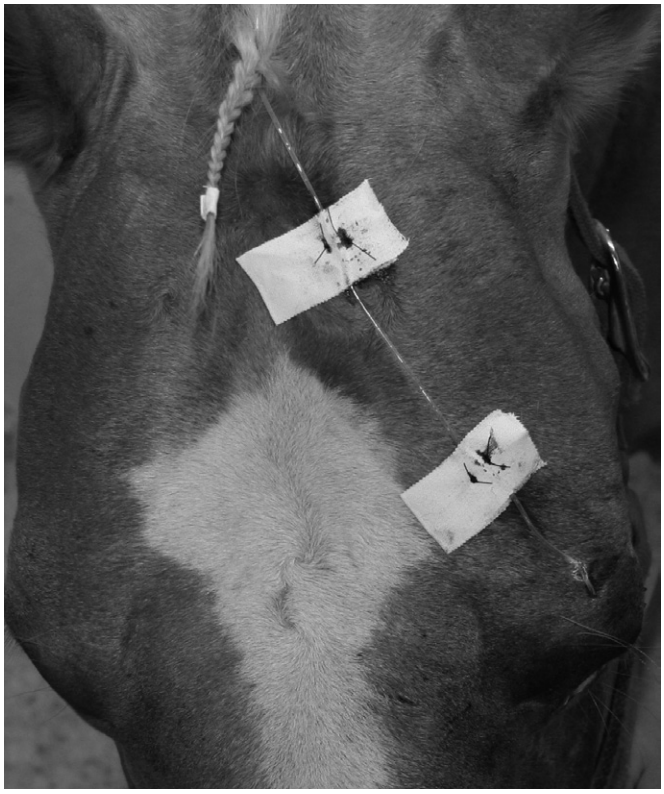


Figure 57-13. Subpalpebral lavage line placed superiorly with silicone tubing secured to the head with tape tabs.

site. Surgical scrubs containing detergents and alcohol preparations should not be used because they may result in epithelial loss and ulceration. The region of the eyelid through which the tube is to pass is anesthetized with subcutaneous 2% lidocaine, and 0.5% proparacaine is applied to the conjunctival surface

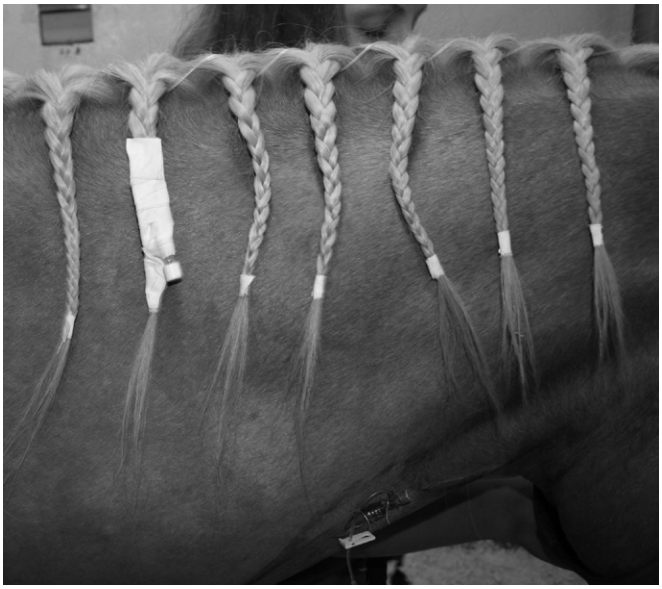


Figure 57-14. Subpalpebral lavage line threaded through braided mane, terminating in an injection port for medication administration.

topically. Alternatively, cotton-tipped applicators soaked in proparacaine can be held against the conjunctiva to ensure analgesia. The 12-gauge hubless needle is introduced into the deepest point of the conjunctival fornix and driven through the eyelid. This ensures placement of the baseplate deep in the conjunctival fornix and reduces the risk of corneal irritation and damage to the surgical site. The globe itself should be protected by keeping a finger between the needle and the eyelid. The lavage tube can then be sutured immediately adjacent to the exit site, via tape tabs secured to the line, and similarly secured at two other sites away from the eye, directing the lavage line into the mane. The line can then be threaded through the braided mane and an injection port placed to facilitate administration of medications (Figure 57-14).

Topical Antibiotics

The choice of topical antibiotics for the treatment of ocular surface infections should be based on the results of pathogen culture and sensitivity testing, with initial therapy based on the results of cytology while awaiting the culture results. The desired mode of administration, whether by topical application or via subpalpebral lavage line, also affects the choice of agent because many drugs are available as either an ointment or solution, but not both. Details of commonly used topical antibiotics are provided later.

Topical aminoglycoside antibiotics are often used in "triple-antibiotic" combinations, where neomycin is typically combined with polymyxin B and bacitracin or with polymyxin B and gramicidin. Triple-antibiotic preparations are a good choice for postoperative prophylaxis or treatment of minor ocular surface bacterial infections. They are available as solutions or ointments and are usually applied three or four times daily. For more serious confirmed bacterial infections, other drugs may be chosen and used in combination at an increased frequency. Gentamicin and tobramycin are bactericidal drugs and broad-spectrum aminoglycosides with an enhanced gram-negative spectrum, and they are available alone in solution. Allergic or irritant reactions have been reported with prolonged use of topical aminoglycosides.[78]

Other topical antibiotic preparations commonly used are the second-generation fluoroquinolones, ciprofloxacin and ofloxacin. They possess bactericidal, broad-spectrum activity and are available as ointments or solutions. Chloramphenicol, a bacteriostatic drug with a broad spectrum of action and good penetration, is also widely used. Because this drug has been reported to cause aplastic anemia in humans, it is prudent to warn owners regarding its potential toxicity and recommend the use of gloves when applying it.[78,79]

Topical Antifungal Medications

Because of the propensity of the horse to develop fungal keratitis, topical antifungals are often used prophylactically in cases of corneal injury. When fungal keratitis is suspected or confirmed with cytology or culture, aggressive medical management may be pursued before surgical intervention, as discussed later in this chapter. Briefly, medical therapy should target both the fungus and the reflex anterior uveitis that often increases in severity as the fungal organisms are killed. Of the available antifungal medications, natamycin ophthalmic suspension (Natacyn) is currently the only commercially available drug formulated for topical ophthalmic application. It is recommended for use four to six times daily and may be delivered via subpalpebral lavage. Other commonly used preparations include fluconazole and silver sulfadiazine.[44] For deeper fungal keratitis and corneal abscesses, access of the drug to the deeper cornea can be improved by débriding the corneal epithelium over the area of the lesion or adding DMSO to the preparation.[80,81]

Anti-Inflammatory Drugs

Topical corticosteroids and nonsteroidal anti-inflammatory drugs (NSAIDs) can be used to control inflammation of the ocular surface. Since topical corticosteroids delay wound healing and potentiate some infections, they are contraindicated in horses with corneal ulceration or overt infection. In other situations, for instance after routine surgery to the ocular surface, the use of a topical corticosteroid preparation has significant merit because of the potentially deleterious effects of ocular inflammation on vision. Topical prednisolone acetate 1% or dexamethasone 0.1%, with or without neomycin and polymixin B, are good choices. Topical NSAIDs can be used alone or in combination with topical corticosteroids. They do not have the potentially dramatic negative side effects of a topical corticosteroid but are best avoided in instances of corneal ulceration. Delayed wound healing and slowed corneal neovascularization should be weighed against their beneficial effects.[78] However, except in cases of overt infection, the benefits of their use generally outweigh these concerns. Flurbiprofen 0.03% solution and diclofenac 0.1% solution are commonly used examples. As already mentioned, systemic anti-inflammatories are often used to help control postoperative ocular inflammation.

ANESTHETIC CONSIDERATIONS

Topical Anesthesia

A topical anesthetic facilitates corneal and conjunctival manipulation for examination of the ocular surface and sample collection. Topical anesthetics can also be used in addition to sedation

or general anesthesia to provide corneal and conjunctival analgesia for procedures such as suturing and foreign body removal. Proparacaine hydrochloride 0.5% solution and tetracaine 0.5% solution are most commonly employed. They can be administered to the cornea via a 1-mL hubbed syringe with the needle snapped off, or they can be instilled directly into the lower conjunctival fornix. The duration of corneal anesthesia achieved by a single administration of either 0.2 mL of proparacaine 0.5% or 1 drop of tetracaine 0.5% is 25 to 30 minutes with maximal anesthetic effect occurring within 5 minutes of application.[82,83] For both agents, the application of multiple drops may increase the duration and intensity of the anesthetic effect.[83,84] Epitheliotoxicity and destabilization of the precorneal tear film are described side effects, and for this reason repeated application for therapeutic purposes should be avoided.[85,86] The antimicrobial activity of these agents also means that swabs for corneoconjunctival cultures should be collected before instillation of a topical anesthetic.[87,88] However, topical anesthetics are often applied first, because subsequent sample collection is safer and the desired site can be targeted more accurately.

Sedation

Sedation is often required for complete examination of the ocular surface. Additionally, minor procedures such as biopsies and cytologic sampling can readily be performed under sedation, topical anesthesia, and nerve blocks. For precision surgery of the ocular surface, general anesthesia is usually required. In addition to the agents described in, "Ophthalmic Examination Techniques and Diagnostic Procedures," a continuous intravenous infusion of detomidine has been successfully used for standing procedures anticipated to be of long duration.

General Anesthesia

General anesthesia is typically required for most surgical manipulations of the cornea; however, it should be borne in mind that the effects of general anesthetics on the eye are not insignificant. For surgery of the ocular surface, the cornea should be centrally positioned, all eye movements such as nystagmus should be eliminated, and the eye should have a normal intraocular pressure.

The effect of anesthetic agents on intraocular pressure is particularly important if the anterior chamber has been penetrated or is to be entered as part of the surgical procedure, such as for penetrating wounds and deep corneal abscesses. In such situations, any increase in intraocular pressure can be detrimental to ocular health and the success of the procedure. Sudden decreases in intraocular pressure have similar consequences and should be avoided by ensuring the globe remains inflated by the use of viscoelastic agents. Most anesthetic agents that are injected or inhaled will lower intraocular pressure. Ketamine is a notable exception; it elevates intraocular pressure, which is thought to be mediated by increased extraocular muscle tone.[89] Additionally, any prolonged respiratory depression or acidosis can elevate intraocular pressure.[90] Positioning of the globe is also important for maintaining intraocular pressure, ensuring that inadvertent pressure is not placed on the eye by the lid speculum or the surgeon's manipulations.

Achieving and maintaining a centrally positioned cornea will best facilitate surgery of the ocular surface, affording the surgeon maximal access to the cornea for surgical procedures. This must be actively addressed by the surgeon because the effect of most injectable and inhalational agents used for general anesthesia produce ventral and medial rotation of the globe. Strategies to overcome this problem include placement of stay sutures through the conjunctiva adjacent to the limbus with 4-0 silk. Alternatively, a neuromuscular blocking agent such as atracurium (0.2 mg/kg IV) can be combined with positive pressure ventilation to improve the position of the cornea. These agents typically relax the extraocular muscles within 1 minute of administration, causing the eye to return to a normal "primary gaze" position. These agents are also valuable to use in cases where the cornea is perforated, or surgical entry into the anterior chamber is planned, as for a penetrating keratoplasty. Neuromuscular blockade also relieves the tension exerted on the posterior globe by the extraocular muscles, which can increase the likelihood that the vitreous will push anteriorly and collapse the anterior chamber when the cornea is entered. Nystagmus may also be encountered and is clearly problematic for the surgeon. This can be rapid movements or a slow drift, and it is best controlled by achieving an appropriate depth of anesthesia and by using neuromuscular blocking agents.

Corneal drying during anesthesia should be prevented to minimize the risk of postoperative complications such as corneal ulceration.[32] To this end, the unoperated eye should be lubricated with an artificial tear ophthalmic ointment. Similarly, the operated eye should be frequently irrigated with a sterile saline solution such as balanced salt solution to prevent damage to the corneal and conjunctival epithelium.

For information on performing general anesthesia, please review Chapters 18 and 19.

SURGICAL EQUIPMENT AND SURGICAL PRINCIPLES

Performing surgical procedures on the ocular surface requires not only specialized techniques but also specialized instrumentation, smaller suture materials, and magnification where available. Magnification is strongly recommended to minimize damage to the delicate structures of the cornea and facilitate accurate placement of sutures. An operating microscope or head loupe can be used and affords an improved surgical outcome, but they also require some practice for the surgeon to adapt to the smaller field of view. Loupes providing ×2.5 or ×4 magnification are commonly used. For more advanced procedures, such as keratoplasty, an operating microscope is desired (Figure 57-15).

Instrumentation

The surgeon should have access to instruments suitable for handling the eyelids, for example to perform lateral canthotomies when indicated. More-delicate instruments for conjunctival manipulations and microsurgical instruments suitable for corneal surgery should also be available. For manipulation of the conjunctiva and sclera, Bishop-Harmon forceps are ideal, and conjunctival tissue is best incised with tenotomy scissors such as the Stevens or Westcott (Figure 57-16). To facilitate exposure, a lid speculum suitable for the equine eyelids is required. Several types are available. Heavy-duty models such as the Guyton-Park eyelid speculum provide good exposure of the ocular surface (Figure 57-17). For surgical procedures involving the cornea, microsurgical instrumentation should be

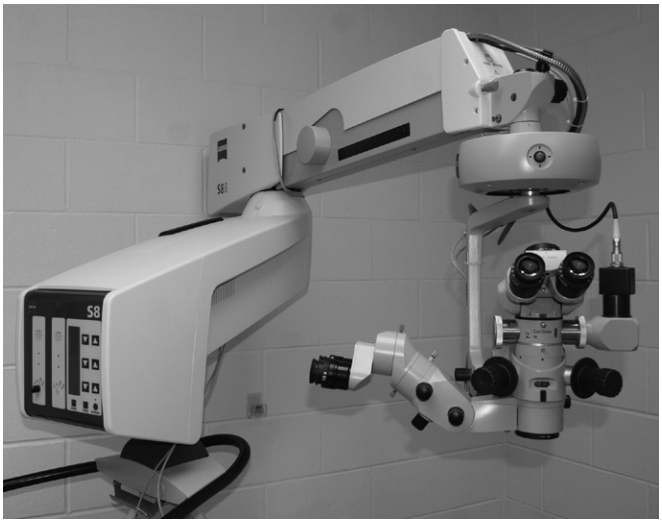


Figure 57-15. Operating microscope (Zeiss).

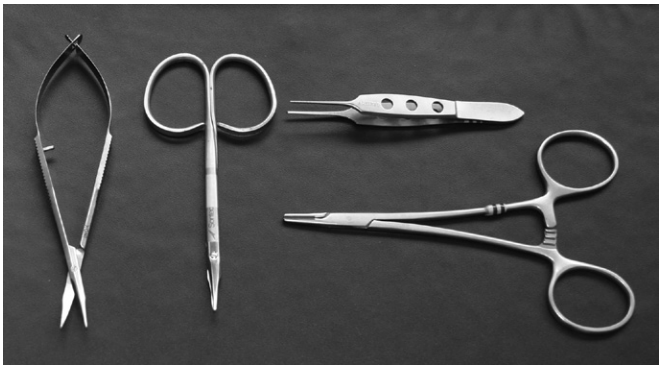


Figure 57-16. Instruments commonly used for conjunctival surgery, from left to right: Westcott tenotomy scissors, Stevens tenotomy scissors, Bishop-Harmon forceps, Derf needle holder.

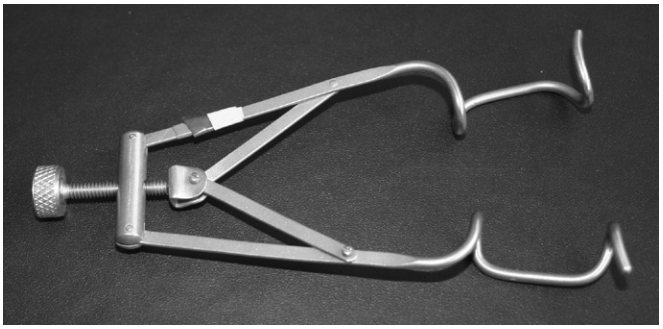


Figure 57-17. Guyton-Park eyelid speculum.

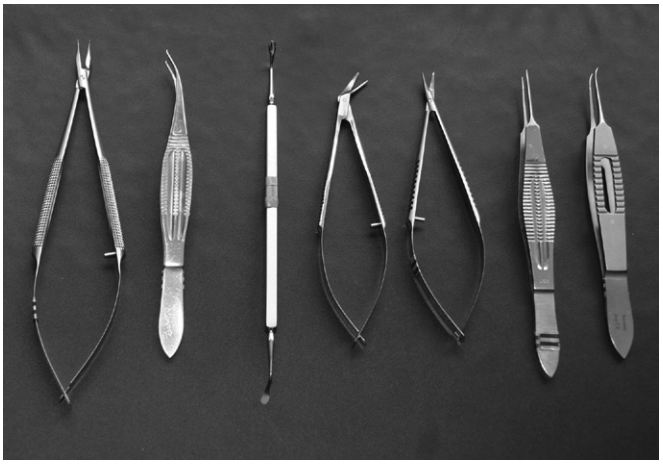


Figure 57-18. Instruments commonly used for corneal surgery, from left to right: Castroviejo needle holder, Colibri corneal forceps, Martinez corneal dissector, Castroviejo corneal section scissors, Castroviejo universal corneal scissors, straight suture-tying forceps, curved suture-tying forceps.

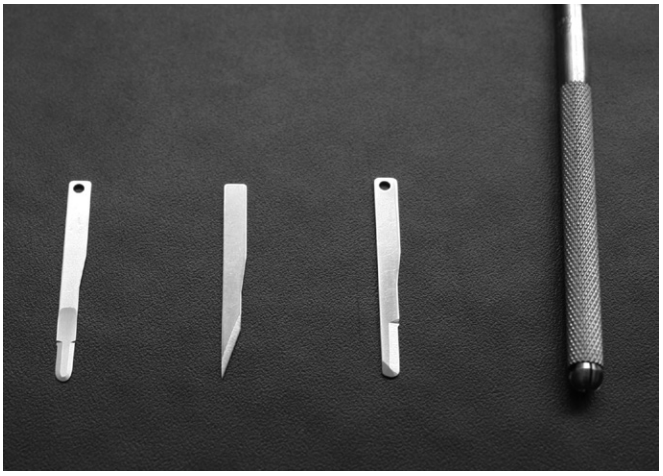


Figure 57-19. Beaver scalpel blades and Beaver handle, from left to right: No. 69 blade, No. 65 blade, No. 64 blade, blade handle.

used, with correspondingly smaller gauge needles and suture materials. The investment in such equipment is dictated by predicted frequency of use. Considered most important are Colibri or Castroviejo forceps; Castroviejo needle holders; Castroviejo universal scissors, curved with blunt tips; and a Beaver scalpel handle and blades (No. 64, 65, and 69) (Figures 57-18 and 57-19). For advanced procedures, such as a penetrating lamellar keratoplasty, a Martinez corneal dissector and corneal trephines may be used. Additional equipment that may be required includes fine-tipped disposable cautery units and cryotherapy units (liquid nitrogen or nitrous oxide). The investment in fine ophthalmic instruments should be protected by careful use, cleaning, and storage. A microsurgical instrument tray should be used for storing and sterilizing the instruments.

Suture Materials, Needles, and Suture Patterns

Fine suture materials with swaged needles are required for the ocular surface. Both absorbable and nonabsorbable suture materials have been described for corneal and conjunctival surgery. However, when operating on the cornea, absorbable 6-0 to 8-0 suture material, such as polyglactin 910, is recommended. For conjunctival and scleral procedures, 4-0 to 6-0 absorbable suture materials are most suitable. Reverse cutting or spatulated needles are typically chosen for reduced iatrogenic damage to the tissues.

The choice of suture pattern depends on the tissue being apposed and the nature of the surgical procedure. For instance,

if performing a penetrating keratoplasty or conjunctival pedicle graft, a simple-interrupted pattern using 7-0 or 8-0 absorbable sutures on a spatulated needle may be chosen. For closing a partial or full-thickness corneal laceration or surgical wound, a simple-continuous or bootlace pattern using the same suture material and needle just mentioned may be preferred. Apposition of bulbar conjunctiva may dictate the use of 6-0 absorbable sutures on a reverse cutting needle applying a simple-continuous pattern, whereas scleral defects may be best apposed with simple-interrupted pattern of 4-0 absorbable sutures on a reverse cutting needle.

Preparation for Surgery

Preparation for ophthalmic surgery is somewhat different from that for other surgical procedures. The head should be positioned for good access to the ocular surface. If an operating microscope is to be used, the head should be positioned securely with foam wedges so that the eyelids of the surgical eye are as close to horizontal as possible. The lashes and long vibrissae may be clipped before conjunctival or corneal surgery. The adnexa should be prepared for aseptic surgery with 1:50 povidone-iodine solution (diluted with sterile 0.9% saline solution). Surgical scrubs and alcohol should not be used because they are harmful to the corneal and conjunctival epithelium. After preparing the eyelids, the conjunctival fornices should be similarly prepared with povidone-iodine applied with cotton-tipped applicators, and the cornea should be flushed with the same solution. Cotton or paper drapes or aperture drapes are applied around the eye. Alternatively, a specialized disposable eye drape with an adhesive clear plastic center can be used. These drapes can be applied to the periocular tissue and an ellipse then cut out to expose the ocular surface (Figure 57-20).

Additional Exposure of the Globe

Before initiating surgery involving the ocular surface, additional exposure may be desired by the surgeon. In these instances, a lateral canthotomy may be performed. The procedure involves making a full-thickness incision 5 mm to 10 mm in length, extending laterally from the lateral canthus. Standard Mayo scissors are typically used for a full-thickness separation of the thick tissue at this site. Hemorrhage is typically minor and easily controlled with cellulose ocular sticks. After completion of surgery, the canthotomy should be closed in two layers. A simple-continuous pattern is used to close the subconjunctival layer, and the skin is apposed with a simple-interrupted pattern using 4-0 absorbable suture. Most important for achieving secure apposition of the lid margin, without risk of the suture tags causing corneal trauma, is the careful placement of a figure-of-eight suture at the lid margin. See Chapter 56 for more detail on the accurate placement of figure-of-eight sutures.

Intraoperative Hemostasis

For any surgery involving the ocular surface, hemostasis and removal of excess fluid should be performed with sterile cellulose ocular sticks rather than standard 4 × 4 swabs, because cotton swabs shed small cotton fibers and may be abrasive to the corneal epithelium. For full-thickness lacerations or in any procedure where the anterior chamber will be entered, such as a penetrating keratoplasty, Weck-Cel cellulose sponge spears should be used (Figure 57-21). If necessary, additional hemostasis can be achieved by direct application of 2.5% phenylephrine or 1:10,000 epinephrine, applied with moistened sterile cotton-tipped applicators.

Entering the Anterior Chamber

If the anterior chamber is penetrated, which may occur with a perforating corneal foreign body or penetrating keratoplasty, a viscoelastic agent should be administered directly into the anterior chamber to help maintain anterior chamber depth and protect the corneal endothelium. Commonly used agents are hyaluronan and 2% hydoxypropyl methylcellulose. If viscoelastic agents are to be used, an understanding of their properties within the eye and the potential complications associated with their use, such as postoperative ocular hypertension, is essential (described further in Chapter 58).

Postoperative Care

Before moving the horse into the recovery stall, a protective eye mask may be placed over the operated eye to prevent damage to the surgical site during recovery from anesthesia. The eye mask is then typically left in place for several days

Figure 57-20. Disposable ophthalmic drape with clear adherent window (Alcon).

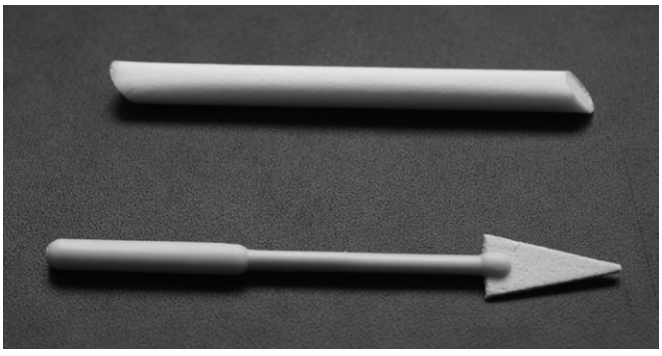


Figure 57-21. Cellulose ocular stick *(top),* and Weck-Cel cellulose sponge spear *(bottom).*

postoperatively to prevent self-trauma. An alternative is closing the eyelid following application of an ophthalmic ointment and covering the area with several 4 × 4 gauze swabs. The head is then wrapped with an Elastikon bandage, ensuring that vision from the unoperated eye remains unobstructed. After any surgical procedure, medical therapy is typically reinstated. This may consist of prophylactic antimicrobial therapy or therapy directed specifically at any organisms previously identified. If anterior uveitis is present or anticipated, topical atropine may be applied two to four times daily on a tapering course, and systemic NSAIDs may be administered twice daily.

ROUTINE SURGICAL TECHNIQUES

Conjunctivectomy and Conjunctival Biopsy

Conjunctivectomy has been described for removing foreign bodies such as burdock bristles embedded in the conjunctiva. Most foreign bodies can be removed without too much difficulty. However, burdock bristles migrate into and become embedded in the conjunctiva, causing chronic ocular irritation and corneal ulceration. They can be difficult to visualize and careful examination of the conjunctiva with magnification is necessary to locate them. Conjunctivectomy to excise the affected conjunctiva and foreign material can be performed under standing sedation and topical anesthesia. The affected region of conjunctiva is elevated with Bishop-Harmon forceps and excised with tenotomy scissors. Hemorrhage can be controlled with pressure, and if necessary, topical 2.5% phenylephrine can be applied directly to the site using a cotton-tipped applicator. A similar procedure is used to obtain a biopsy of conjunctival tissue for histopathologic examination. The conjunctiva heals rapidly, so unless the area of conjunctiva excised is large, the resulting defect can be left to heal by second intention. If sutures are used, care should be taken to ensure that they will not rub on the cornea and potentially cause ulceration.

Repair of Conjunctival Lacerations

Small lacerations can be left to heal by second intention after careful examination and possibly exploration of the wound. This is important since foreign bodies may be present and the underlying structures may also have been damaged. Stick injuries are common, and penetrating sticks can break off, leaving only a small amount or even none of the stick visible. If the foreign body has penetrated the periocular tissues or the globe itself, a complete ophthalmic examination will detect signs of penetration of the globe or laceration of the sclera. It is also possible for severe blunt trauma to the globe to result in scleral splits, the extent of which may not be immediately apparent but are usually accompanied by serious intraocular changes. In some instances, it may be necessary to surgically reflect the conjunctiva to reveal the full extent of underlying scleral lacerations. Scleral defects can be closed with 4-0 to 6-0 absorbable suture, such as polyglactin 910. When conjunctival defects require closure, a simple-continuous pattern of 4-0 to 6-0 absorbable sutures can be used.

Excision of Conjunctival Tumors

The primary approach to a conjunctival mass should be to obtain a histologic diagnosis. With small masses, this can be combined with an excisional biopsy. Larger masses can be sampled by impression smears or a small "snip" biopsy. The extent of the mass and its histologic diagnosis will influence the subsequent choice of therapy. Limbal squamous cell carcinomas (SCCs) are common and typically require a keratectomy to remove the corneal portion and additional excision to eliminate the conjunctival extension of the tumor. Areas of bulbar conjunctiva can be removed using tenotomy scissors to excise and undermine the mass. When excising a conjunctival mass, the inclusion of the underlying substantia propria and Tenon's capsule will help ensure complete excision. Small defects can be left to heal by second intention. If the lesion is larger, partial or complete closure of the resulting defect can usually be achieved by undermining the surrounding remaining conjunctiva to mobilize it and stretch it over the defect. For limbal and bulbar conjunctival SCC, where the recurrence rate has been reported to be particularly high, excision combined with adjunctive therapy has been shown to significantly lower the rate of recurrence.[91]

Superficial Keratectomy

Superficial keratectomy represents the surgical excision of a superficial layer of corneal tissue including epithelium and anterior stroma. Indications for keratectomy include presence of dermoids, typically hairy, skinlike tissue located on the cornea adjacent to the limbus,[92] and development of infection, particularly associated with superficial fungal keratitis, corneal malacia, or superficial corneal stroma abscesses.[44,93] Superficial keratectomy is also commonly performed to remove corneal neoplastic masses, the most common being limbal-based SCC.[94]

Superficial keratectomy is best performed under general anesthesia, with good magnification and illumination. An initial incision is made into the anterior stroma through normal cornea adjacent to the lesion to be removed. This is made using a No. 64 or No. 69 Beaver blade to the depth needed to remove the mass. Corneal forceps are used to grasp the incised edge of the cornea, and the corneal stroma can subsequently be separated along a lamellar plane to undermine the lesion. A blunt instrument can be used for this dissection, ideally a Martinez corneal dissector, which is specifically designed as a lamellar separator. Alternatively, the original blade can also be used for the purpose, taking care to only use it to separate the corneal lamellae rather than change the depth of the dissection. The resulting bed of cornea achieved by stromal dissection should be carefully inspected. If necessary, additional tissue can be removed by dissection along a deeper lamellar plane to ensure removal of all diseased tissue (Figure 57-22). The resulting corneal defect can be left to epithelialize. However, if the lesion is deep or the base of the lesion has been treated with an adjunctive therapy, a conjunctival hood graft or amniotic membrane graft can be used to cover the defect. These procedures are described later. They have the advantages of protecting the cornea and facilitating healing, but they have the disadvantage of potentially masking any tumor regrowth. Other than tumor regrowth, complications associated with superficial keratectomy are rare but include an excessive neovascular response, scarring, infection, and corneal perforation.

Adjunctive or Alternative Therapies for Ocular Surface Tumors

Several adjunctive therapies have been used to manage ocular surface tumors, such as conjunctival, limbal, and corneal

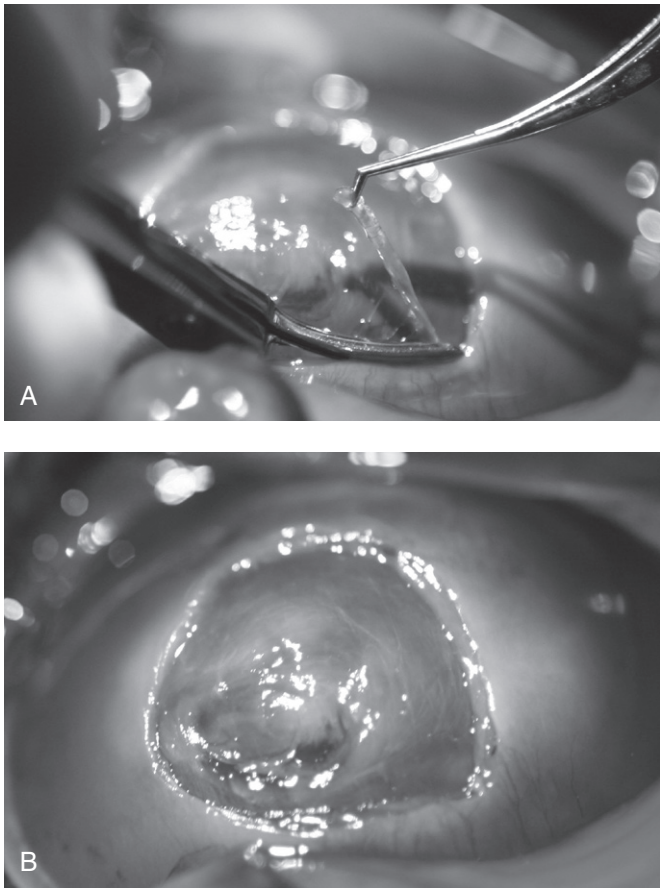


Figure 57-22. Intraoperative images of a superficial keratectomy. **A,** Following dissection along a lamellar plane with a Martinez dissector, the edge of the keratectomy is trimmed with Castroviejo universal corneal scissors. **B,** Final appearance of the completed keratectomy.

masses. These include beta irradiation, cryotherapy, radiofrequency hyperthermia, and CO_2 laser ablation. Most of these procedures can be performed on a horse under standing sedation with topical anesthesia and local nerve blocks. General anesthesia is typically reserved for cases where these therapies are used as an adjunct to more aggressive surgical intervention such as keratectomy.

Beta irradiation may be used as an adjunctive procedure for surface ocular lesions, such as SCC and epibulbar melanoma, by direct application of strontium-90 (^{90}Sr) to the surface ocular tissues. Because tissue penetration is 1 to 2 mm, this therapy is ideal for corneal lesions. A dose of 75 to 100 Gy can be delivered to multiple lesions, ensuring that a total dose of 500 Gy is not exceeded; at doses higher than this, there is a risk of permanent corneal endothelial damage and deep corneal necrosis.[56] Progressive corneal edema and bullous keratopathy are also reported complications.[95] When beta irradiation is combined with superficial keratectomy and permanent bulbar conjunctival grafts to treat corneal SCC, a recurrence rate of only 17% has been reported.[96] Beta irradiation can similarly be used for limbal and conjunctival masses.

Cryoablation may be used alone or adjunctively, in a fast freeze to −20° C to −40° C followed by a slow thaw for two cycles to achieve optimal cryonecrosis of malignant epithelial cells.[97] A nitrous oxide or closed liquid nitrogen probe can be used. Limbal and conjunctival masses are best suited to this treatment, and side effects are uncommon.

Radiofrequency hyperthermia and *CO_2 laser ablation* are additional modalities that have been described for the destruction of surface ocular lesions in the horse.[98,99] Intratumoral treatment with *cisplatin* has also been described for conjunctival lesions.[100] Most recently a report described treatment of ocular SCC that consisted of multiple topical applications of *mitomycin C* (Mutamycin), with or without surgical excision. Complete resolution occurred in 77% and 75% of cases, respectively, with no reported complications.[101]

Repair of Corneal Lacerations

Animals presenting with corneal lacerations should be examined carefully. It is important to ascertain if the injury is full-thickness, if the iris is involved in the wound, and if other intraocular structures such as the lens have been damaged. Involvement of the iris will complicate the repair. If the lens has been damaged, a cataract may ensue; if a large tear in the lens capsule is present, a severe inflammatory reaction to the liberated lens proteins may develop, significantly lowering the prognosis for achieving a comfortable, visual eye.[102]

Corneal lacerations typically require surgical repair using magnification with the horse anesthetized. Deep, uncomplicated corneal lacerations may be repaired by primary closure using simple-interrupted absorbable 6-0 to 8-0 sutures placed 1 to 2 mm apart. Small partial-thickness lacerations may be medically managed similar to a corneal ulcer, after ensuring that there is no foreign material lodged in the wound. If a foreign body is detected, its depth in the cornea must be accurately confirmed, because removal of a foreign body penetrating the entire thickness of the cornea may result in collapse of the anterior chamber. If a foreign body has penetrated the anterior chamber, or if its depth in the cornea cannot be assessed, it should be managed as a full-thickness laceration with the horse anesthetized and a viscoelastic agent available. Full-thickness lacerations may similarly result in collapse of the anterior chamber. If the wound is small it may seal with a fibrin clot, whereas larger wounds often show incarceration of the iris. In both situations, surgical repair is indicated. Typically, during the repair there is further leakage of aqueous humor, necessitating reinflation of the globe with a viscoelastic agent to maintain the shape of the anterior chamber during corneal suturing. Usually minimal débridement of the cornea is required, but the fibrin clot should be removed as the wound is prepared for suturing. A prolapsed iris is irrigated and, where possible, gently manipulated back into the anterior chamber using a blunt spatula or muscle hook. Any adhesions that have formed between the iris and the cornea are separated. This step should be done carefully because the iris is very vascular and hemorrhage may result, necessitating the use of topical epinephrine (1:1000 to 1:10,000). In instances where the laceration has been long-standing and the iris is incarcerated, it may be difficult to break down adhesions between the iris and cornea, and excision of the prolapsed iris may be necessary. In these cases, electrocautery may be useful to avoid excessive iridal hemorrhage (Figure 57-23).

In all cases where the anterior chamber has been penetrated, medical therapy should be prolonged and significantly more aggressive, including administering systemic antibiotics in addition to systemic anti-inflammatories and topical antibiotic solutions. Prognosis for healing with minimal scarring is good for

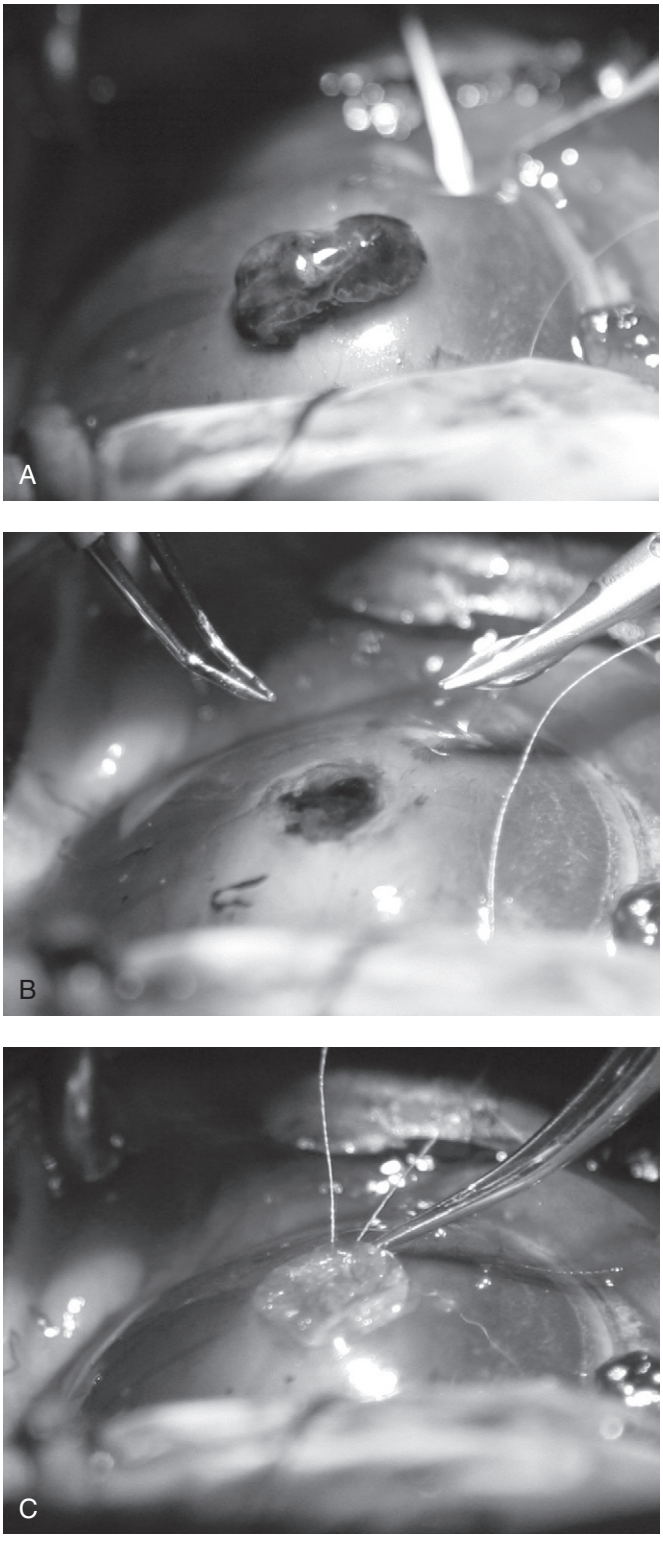


Figure 57-23. A, Preoperative appearance of a corneal laceration with prolapsed iris. **B,** Appearance of the corneal laceration after electrocautery of the prolapsed iridal tissue. **C,** Placement of porcine small intestine submucosa (SIS) in the corneal defect before placement of a conjunctival pedicle graft over the lesion.

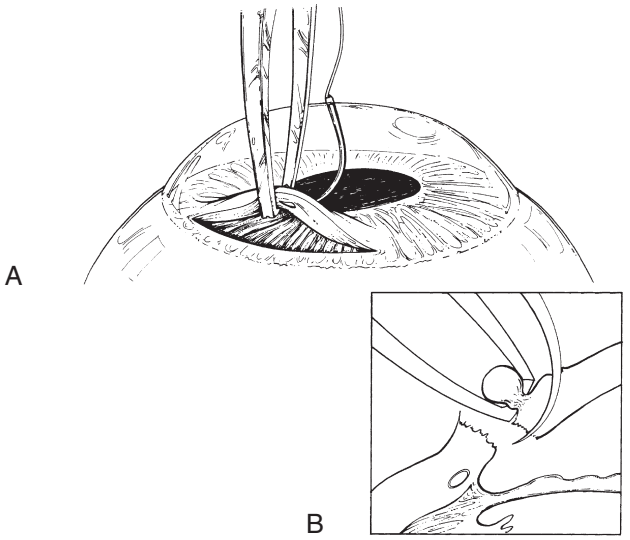


Figure 57-24. A, Correct placement of sutures to a depth of 80% to 90% of the cornea. **B,** Close-up view.

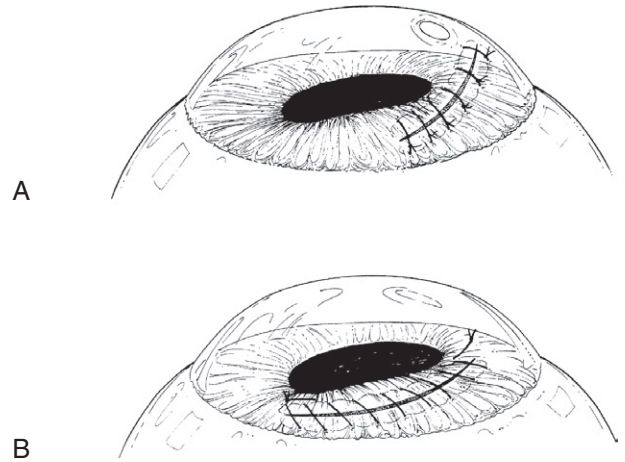


Figure 57-25. A, Closure of corneal defects using a simple-interrupted pattern. **B,** Closure using a simple-continuous pattern.

uncomplicated cases, but when grafting material is included, scarring will be more significant. Potential complications that can arise postoperatively include infection, excessive scarring, astigmatism, and suture dehiscence leading to aqueous humor leakage, iris prolapse, or globe collapse.

Considerations for Suturing the Cornea

Corneal sutures are best placed under magnification using 6-0 to 8-0 absorbable sutures, placed to a depth of approximately 80% to 90% of the cornea (Figure 57-24). Simple-interrupted, simple-continuous, and double bootlace suture patterns (Figure 57-25) are suitable. If sutures are placed too superficially, the posterior aspect of the wound will gape, jeopardizing wound integrity and increasing scarring. If sutures are placed full thickness through the cornea, the suture tracts may allow leakage of aqueous humor and increase the risk of contaminating material reaching the anterior chamber via the suture tracts. Suture

needle entry and exit sites should be at the same distance from the wound edges and be placed to the same corneal depth to avoid "steps" between the wound edges. If an arrowhead-shaped wound is present, a suture can be placed at the tip of the arrowhead first to prevent gaping at this part of the wound. Following the repair, a Seidel test should be performed with fluorescein dye to check that the wound is not leaking aqueous humor (see "Diagnostic Procedures," earlier). If corneal tissue is missing or a satisfactory repair is not possible, the surgeon should consider using a conjunctival graft. This may just be a temporary hood flap that is tacked to the cornea over the defect to give it some protection while healing, with the intention that it will retract unless it heals to the defect. If a larger defect is present, placement of a conjunctival pedicle graft or inclusion of material such as transplanted cornea, amniotic membrane, or porcine small intestine submucosa could be considered. These procedures are described in the following section.

ADVANCED SURGICAL TECHNIQUES

The procedures described here typically require general anesthesia, magnification, good illumination, appropriate instrumentation, and some practice to master. They are most commonly used for deep and perforating corneal ulcers or defects and deep focal corneal abscesses, although procedures such as conjunctival grafts and flaps can also be used to support and protect corneal repairs.

General Principles for Conjunctival Grafting

Conjunctival grafting procedures involve transposing a portion of conjunctival tissue to cover a corneal defect. This provides structural support and a vascular supply to the lesion and hence anticollagenases, fibroblasts, and growth factors that facilitate healing. Their main use is in cases of deep corneal ulceration, melting ulcers, descemetoceles, and corneal lacerations.[103] For the procedure, the conjunctiva should be incised and undermined with tenotomy scissors, and once mobilized it is sutured into position on the cornea, ensuring the needle passes first through the graft and then the cornea. Suturing should begin with placement of cardinal sutures at the tips or end of the graft.[104] A simple-interrupted, simple-continuous, or a combination of both suture pattern can then be used, ensuring that the sutures are placed to a depth of 50% to 65% of the cornea for adequate anchorage of the graft.

There are several types of grafts and flaps to choose from, and the selection largely depends on the position and size of the lesion. Defects close to the limbus can be covered by advancement or hood grafts, whereas more centrally placed lesions may be best covered with a pedicle graft. In some instances, a conjunctival graft may not sufficiently support a corneal defect; this is particularly the case for perforated ulcers but can also be true for larger, very deep ulcers and descemetoceles. In such situations, additional tectonic support for the cornea may be necessary and can be provided by scaffolding materials that can be sutured into the defect before graft placement. Materials that can be used for this purpose include transplanted cornea, either fresh or frozen; amniotic membrane; or commercially available products such as porcine small intestinal submucosa (SIS). The scaffolding material should be shaped to fit the defect and then sutured into the cornea, with the conjunctival graft sutured over the top of the site. Conjunctiva does not adhere well to porcine SIS, so removing a rim of epithelium from the cornea around the defect and suturing the conjunctival graft to the exposed corneal stroma helps the graft to heal into place. Additionally, if porcine SIS is used, a thicker four-ply preparation is preferred over the thin discs that are marketed for corneal use.

Some general principles to consider that may improve the outcome of conjunctival grafting procedures are described later. To reduce the risk of graft dehiscence and improve the cosmetic outcome, the conjunctival dissection should be kept thin, avoiding underlying Tenon's capsule. As a guide, dissection scissors should be visible through the graft as it is being harvested. The risk of dehiscence can also be reduced by ensuring the graft covers the defect without being under tension; if tension is present, additional conjunctiva should be undermined before suture placement. To ensure adequate coverage of the corneal lesion, the graft should ideally be slightly larger (1 to 2 mm) than the defect because it tends to contract. It is particularly important to realize that conjunctiva will not adhere to an epithelialized surface, so in instances where the epithelium has grown down the edges of an ulcer, it should be carefully removed to expose the corneal stroma to which the conjunctiva can adhere. A No. 69 Beaver blade can be used to débride the epithelium, or in some instances it can be peeled off using corneal forceps.

Conjunctival Grafting Techniques

Rotation Pedicle Graft

The rotation conjunctival pedicle graft is best suited for centrally located corneal defects, and it represents the most commonly performed type of conjunctival graft. Since a flap of conjunctiva is to be rotated onto the cornea, the surgery should be carefully planned, considering the site and size of the lesion and paying particular attention to ensure the vascular pedicles are not occluded at the base of the flap when it is rotated. The surgeon will find it is easier to harvest the lateral and dorsal bulbar conjunctiva rather than the medial or ventral conjunctiva because of the presence of the third eyelid. To begin the procedure, the bulbar conjunctiva is incised about 1 mm from the limbus using tenotomy scissors, and a combination of careful, precise blunt and sharp dissection is used to isolate the conjunctiva from Tenon's capsule. A finger-shaped strip of conjunctiva is fashioned, with the length determined by the distance of the lesion from the limbus (Figure 57-26). The recipient bed is prepared by removing necrotic or liquefied cornea and any epithelium that has grown into the defect. The graft can then be sutured into the recipient bed as described earlier, being sure that the conjunctival edges appose the corneal edges of the lesion. Finally, the defect created in the conjunctiva should be sutured closed in a simple-continuous pattern (Figure 57-27).

Advancement Pedicle Graft

An advancement conjunctival pedicle graft represents an alternative to the rotation conjunctival pedicle graft and is used for lesions located closer to the limbus. These grafts are fashioned using tenotomy scissors to create an incision in the bulbar conjunctiva, 1 to 2 mm longer than the width of the defect, adjacent to and parallel to the limbus. Subsequently, the bulbar conjunctiva is carefully undermined and extended up toward

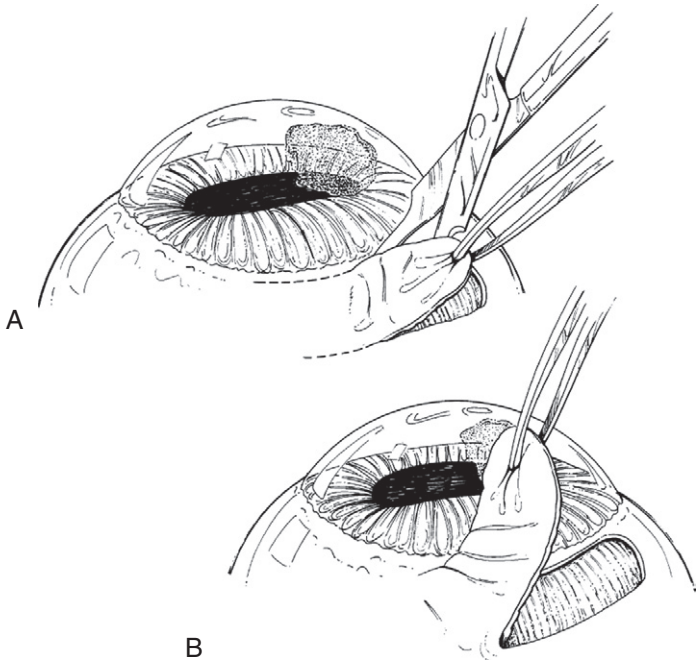


Figure 57-26. A, Conjunctival pedicle graft; the pedicle is dissected from underlying fibrous tissue with tenotomy scissors. **B,** The pedicle should lie over the corneal defect without undue tension.

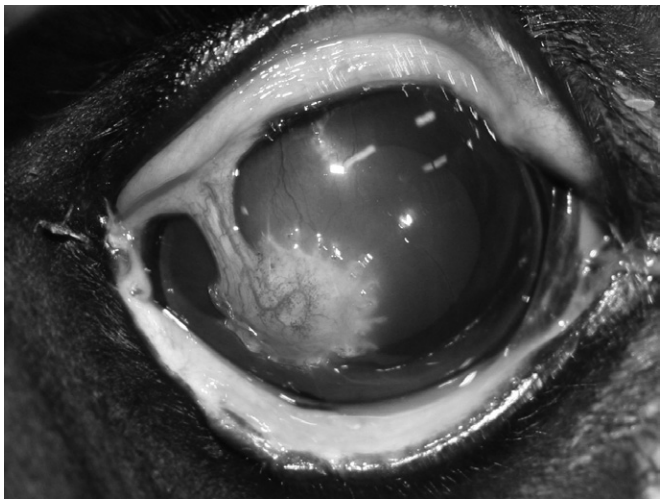


Figure 57-27. Appearance of a conjunctival pedicle graft 3 weeks after surgery showing a healthy, well-vascularized pedicle.

the fornix, again taking care to separate the thin conjunctival flap from Tenon's capsule. Two slightly diverging incisions are then made through the conjunctiva from the edge of the initial incision extending toward the fornix. The resulting strip of conjunctiva is sutured to the cornea after preparation of the recipient bed as described previously.

Tarsoconjunctival Grafts

Conjunctival grafts harvested from the palpebral conjunctiva of the upper eyelid have also been reported. Tarsoconjunctival grafts are created similar to a conjunctival pedicle graft but differ in that their base originates close to the eyelid margin. If this graft is performed, placement of a temporary tarsorraphy to reduce the risk of suture dehiscence has been recommended.[103]

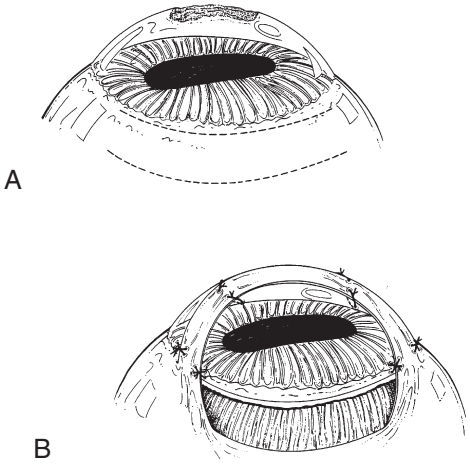


Figure 57-28. Bridge conjunctival graft. **A,** A strip of conjunctiva is incised and dissected from the bulbar conjunctiva. **B,** The bridge of conjunctiva is sutured to the cornea around the edge of the corneal ulcer.

Bipedicle and Bridge Grafts

Bipedicle or bridge conjunctival grafts have been described for linear corneal defects and large axially located lesions, with the advantage of providing additional blood supply. With this technique, the bridge graft carries a reduced risk of ischemia at the graft tip relative to the bipedicle graft.[93] The technique for creating a bipedicle graft involves creating two conjunctival pedicle grafts with their bases located 180 degrees apart, allowing the tips of the graft to meet over the lesion. A bridge graft involves creating two 140- to 180-degree bulbar conjunctival incisions, separated by a distance 1 to 2 mm wider than the lesion (Figure 57-28).

Hood Grafts

A hood conjunctival graft can be used to cover larger lesions close to the limbus. With this technique, a single incision is made 1 mm from the limbus, and the conjunctiva is undermined and advanced axially to cover the lesion (Figure 57-29). A hood graft can also be used to provide temporary additional protection of a peripheral corneal incision made for intraocular procedures.

Free Island and 360-Degree Grafts

Free island grafts have been described but are rarely useful, because their main disadvantage is the absence of a blood supply.[105] Also reported but rarely used are 360-degree grafts, because they preclude vision and prevent visualization of the globe. Their main use is for large lesions that cannot be effectively treated with one of the other grafting procedures. The 360- degree graft involves harvesting circumlimbal bulbar conjunctiva, which is subsequently undermined and advanced to cover the entire cornea (Figure 57-30).

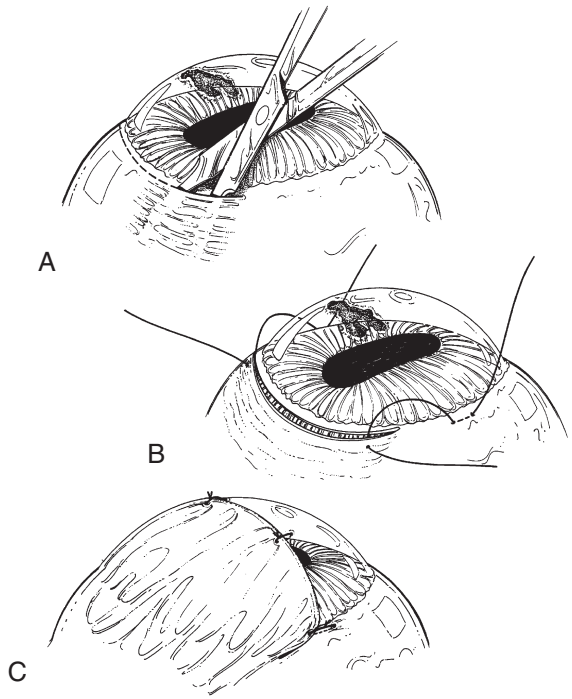


Figure 57-29. Hood conjunctival graft. **A,** An incision is made 1 mm from the limbus, and the bulbar conjunctiva is undermined toward the fornix. **B,** The graft is sutured to the episclera at the edges of the incision. **C,** The graft is sutured to the cornea.

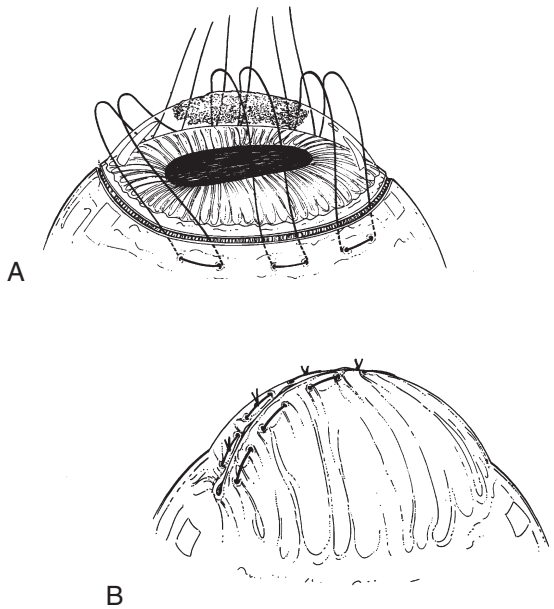


Figure 57-30. 360-degree conjunctival flap. **A,** The bulbar conjunctiva is incised circumferentially around the limbus and is undermined toward the fornix. **B,** The apposing sides of the flap are sutured together with mattress sutures.

Postoperative Management of Conjunctival Grafts

In the postoperative period, topical antibiotics and topical antifungals are typically indicated and may be administered with most ease via a subpalpebral lavage line placed at the time of surgery (see earlier). Systemic NSAIDs may also be indicated. The main complications associated with conjunctival grafting include graft dehiscence and infection. It should also be noted that although graft remodeling will reduce postoperative scarring, there can be a significant effect on vision, particularly if the graft is large or axially located.

Once pedicle grafts have healed in place and the corneal defect has healed, the attachment stalk to the conjunctiva can be cut, thus removing the blood supply. Trimming is typically performed 6 to 8 weeks postoperatively. Light sedation is recommended along with application of a topical anesthetic to the eye. For greatest effect, the topical anesthetic can be placed onto a cotton-tipped applicator that is held directly against the site to be trimmed. The stalk of the pedicle is then grasped with forceps, and one blade of the tenotomy scissors is placed between the cornea and the pedicle, close to the corneal lesion, and the stalk transected. Hemorrhage can be controlled by topical 2.5% phenylephrine if needed. The remaining pedicle will retract and remodel over time if left, or alternatively it can be trimmed close to the limbus. Removal of the blood supply to the graft helps to reduce its size and improves the visual outcome.

Amniotic Membrane Transplantation

Amniotic membrane transplantation can be used as an alternative to a conjunctival pedicle graft.[106-108] Used in cases of corneal ulceration, amnion has been reported to promote wound healing and epithelialization, reduce inflammation, and decrease fibrosis.[109] This may be through reduced expression of matrix metalloproteinases (MMPs) and increased expression of tissue inhibitors of metalloproteinases (TIMPs) in the cornea.[110] Equine amnion can be used fresh or can be stored frozen (Box 57-1). If frozen, the graft should be thawed and subsequently washed for 30 minutes in sterile saline before application. It can be sutured to the cornea with simple-interrupted sutures of 6-0 to 8-0 polyglactin 910, ensuring that the chorion side is oriented toward the cornea. Postoperatively, after reabsorption of the amnion (which may require several months), a new fibrotic stroma forms, which can reduce corneal transparency.[111] However, it has been proposed that amniotic membrane transplantation results in reduced corneal scarring compared to that resulting from a conjunctival pedicle graft.[112] A recent review describes the use of amnion as a graft, a "biologic contact lens," or its use in multiple layers as a "corneal filler." Included in the review is a series of 58 cases describing an excellent cosmetic outcome with corneal scarring classified as mild in 69% of cases.[113]

Box 57-1. Collection and Storage of Equine Amnion[122]

Source: Harvest fresh placenta after routine cesarean section

Harvesting: Separate the amnion from the vascularized chorion by blunt dissection

Cleaning: Rinse with phosphate buffered saline containing 2.5 g/mL amphotericin B, 100 g/mL neomycin, 50 g/mL streptomycin, and 50 g/mL penicillin

Preparation: Place with epithelial side up on 0.45-μm nitrocellulose paper, cut into 4 × 4 cm squares

Storage: Store up to 12 months at –80° C in 1:1 Dulbecco's modified Eagle's medium and glycerol, containing 2.5 g/mL amphotericin B, 100 g/mL neomycin, 50 g/mL streptomycin, and 50 g/mL penicillin

Sliding Lamellar Keratoplasty (Keratoconjunctival Transposition)

A sliding lamellar keratoplasty is suited for the repair of a deep corneal defect where the surrounding cornea is healthy and where achieving a clear axial cornea is desired.[114] Variants of this procedure include corneoscleral transposition and corneoconjunctival transposition.[44,115] The procedure for corneoconjunctival transposition is described here; this procedure is more commonly performed because it preserves the structural integrity of the sclera. Using the technique described for a keratectomy, necrotic corneal stroma is removed from the lesion and a square healthy recipient bed is created. A No. 64 or No. 69 Beaver blade is used to perform two diverging partial-thickness corneal incisions, extending from the site of the lesion 1 mm into the conjunctiva beyond the limbus. Using a Martinez corneal dissector or a Beaver blade, the superficial cornea delineated by the two incisions is separated from the underlying deeper corneal stroma at the required thickness. To facilitate dissection, the leading edge of the cornea can be grasped with corneal forceps, though handling should be minimized. When the dissection has passed the limbus, it is extended into the subconjunctival tissue, leaving the flap of cornea to be advanced attached to the conjunctiva. The conjunctiva is isolated from Tenon's capsule using blunt dissection with tenotomy scissors. The graft can then be advanced into the lesion and sutured in place with 6-0 to 8-0 polyglactin 910 in a simple-interrupted pattern (Figure 57-31). Postoperative care is as described for conjunctival grafting, with the exception of trimming of the conjunctiva, which should not be performed after a corneoconjunctival transposition. Dehiscence and infection are possible complications.

General Principles for Corneal Grafting

Deep corneal stromal abscessation is the main indication for the use of corneal grafting procedures, though these techniques have also been used for corneal endothelial disease and large corneal perforations.[114] The procedures place donor corneal tissue at the site of the excised tissue to facilitate healing and help reduce scar formation. Because they are allografting procedures, they do carry the risk of graft rejection and failure, despite the cornea's status as an immune privileged site.[116] Donor corneal tissue can be harvested fresh or stored frozen. For collection, the donor cornea should be placed epithelial side down on a Teflon block, and a button should be removed with a corneal trephine or disposable punch biopsy. If the tissue is to be used fresh, the epithelium should be completely débrided; otherwise the corneal button can be frozen. Fresh corneal tissue harvested within 48 hours of use is preferable, because freezing damages the corneal endothelial cells.[117] However, whereas fresh tissue is commonly used in human ophthalmology, it is rarely used in horses for practical reasons, so subsequent opacification of the graft is common. Placement of the donor corneal button in the recipient site should be performed with the endothelial side facing the anterior chamber. Cardinal simple-interrupted sutures of 7-0 to 8-0 polyglactin 910 should be placed at the 3, 6, 9 and 12 o'clock positions before securing the donor tissue with a simple-interrupted or simple-continuous pattern. The excised, diseased corneal button may be cultured and submitted for histopathology as indicated.

A number of surgical approaches are available, with the choice of technique depending on both the nature of the lesion and the surgeon's comfort and familiarity with the procedure. For instance, where a focal deep corneal abscess is present, which is typically at the level of deep stroma adjacent to and involving Descemet's membrane, the surgical approach is to excise the lesion. This can be achieved by excising full-thickness cornea with a penetrating keratoplasty (PK) or excising the deeper abscessed region of the cornea, while retaining the superficial corneal layers using procedures such as a posterior lamellar keratoplasty (PLK) or a deep lamellar endothelial keratoplasty (DLEK). All of these procedures use a corneal graft to fill the defect left in the cornea following excision of the abscess. However, the tendency has been to move away from using PK for deep corneal abscesses, because the overlying cornea is

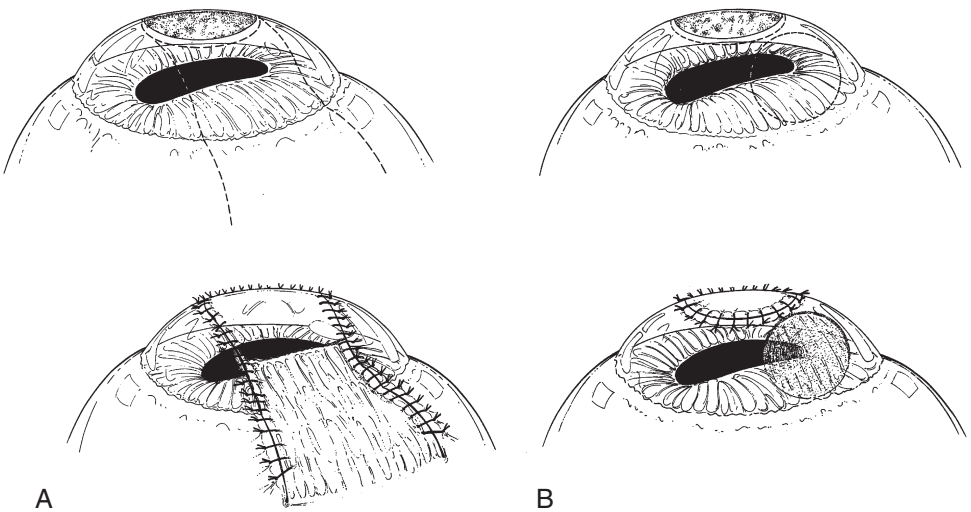


Figure 57-31. Sliding lamellar keratoplasty (corneoconjunctival technique), with diverging corneal incisions extending into the conjunctiva. **A,** A Martinez dissector is used to elevate the flap of cornea, and tenotomy scissors are used to undermine the bulbar conjunctiva. The graft is advanced over the defect and sutured along the edges of the incisions. **B,** Alternatively, the donor corneal tissue may be harvested from a site distant to the defect.

typically not infected and is usually in good condition. PLK and DLEK allow excision of the focal abscess with retention of the overlying cornea. Additionally, we have found that good results can be achieved without placing graft material if a careful dissection of the deep abscess is undertaken while retaining the unaffected overlying portion of the cornea.

Corneal Grafting Techniques

Penetrating Keratoplasty

A penetrating keratoplasty is the surgical placement of a full-thickness donor corneal button into the recipient tissue. Before commencing the procedure, the lesion should be measured with Jameson calipers and the prepared corneal donor button should be 1 to 2 mm larger in diameter than the intended recipient site. The corneal epithelium surrounding the recipient site should subsequently be débrided. A corneal trephine or disposable biopsy punch is used to incise the cornea to a depth of 75% of the stroma. The incision is then continued with a No. 65 or No. 69 Beaver blade to penetrate the anterior chamber. A viscoelastic agent is used to fill and maintain the anterior chamber, facilitating manipulations of the cornea and reducing the risk of hypotony. The incision can be completed with curved universal corneal scissors and the button of diseased cornea removed. The donor button is sutured into position as described earlier (Figure 57-32). Some surgeons choose to suture an amniotic membrane or conjunctival graft over the cornea for additional security. If a corneal graft is chosen, it will also provide a vascular supply and may decrease the risk of dehiscence and secondary infections.[46,118] The downside to their use is that they will increase the resulting scar and hence may limit vision; this may be less of a concern if amniotic membrane is used.[113]

Postoperatively, topical antibiotics and antifungals should be supplemented with a topical mydriatic, and systemic anti-inflammatories should be administered. Vascularization and subsequent scarring at the surgical site are to be anticipated.[119] However the major complication is dehiscence of the graft, and the risk of this is reportedly increased if the graft is larger than 8 mm in diameter or is applied close to the limbus.[115] However, the prognosis can be considered good with a visual outcome reported in 78% of horses with inflammatory keratopathy treated with a PK.[94]

Posterior Lamellar Keratoplasty

An alternative to a PK is a penetrating lamellar keratoplasty. It is suitable for abscesses involving the deep corneal stroma, Descemet's membrane, and underlying endothelium. The advantage of the PLK is that it leaves the healthy anterior corneal stroma and epithelium intact, because only the abscessed region is removed and an allograft applied to replace the diseased tissue.[120] Relative to a PK, a PLK may result in reduced postoperative scarring, it carries a decreased risk of dehiscence, and the surgical time is reportedly shortened.[118] The procedure involves using a No. 64 or No. 69 Beaver blade to create a three-sided corneal incision of approximately 50% corneal thickness over the lesion to be removed. This flap should be slightly larger than the lesion to be removed. A Martinez corneal dissector or Beaver blade is then used to separate the corneal stroma, allowing the created flap to be elevated and reflected to reveal the abscessed region. The diseased tissue is removed with a corneal trephine and curved universal corneal scissors. This excision involves the entire remaining thickness of the cornea and thus penetrates the anterior chamber, necessitating the use of a viscoelastic agent. Donor corneal tissue is subsequently sutured into the site as described earlier. For this procedure, the donor tissue should have the anterior half of the stroma removed by dissection so that only the posterior cornea, including Descemet's membrane, is used. As previously mentioned, we have also achieved excellent results by simply replacing the flap and not placing an allograft. In either instance, the anterior corneal flap should be sutured back into position with simple-interrupted sutures of 7-0 to 8-0 polyglactin 910 (Figure 57-33). Postoperative care is as for PK. Vascularization, scarring, graft dehiscence, and ulceration are among reported complications.[118] However, the prognosis is excellent with a positive visual outcome reported in 98% of cases.[94]

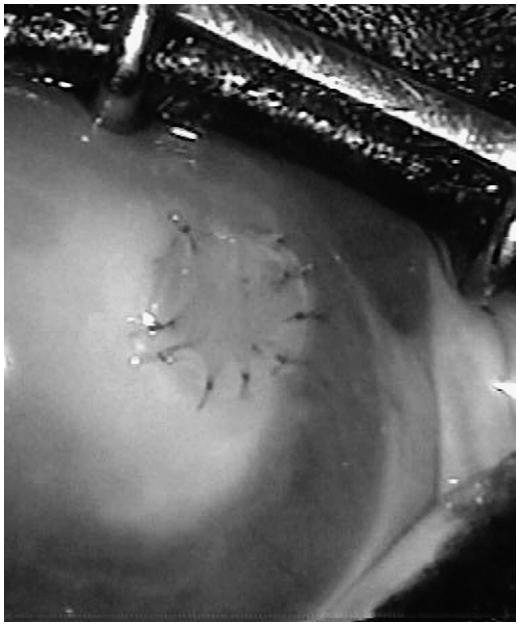


Figure 57-32. Postoperative appearance of a penetrating keratoplasty (PK).

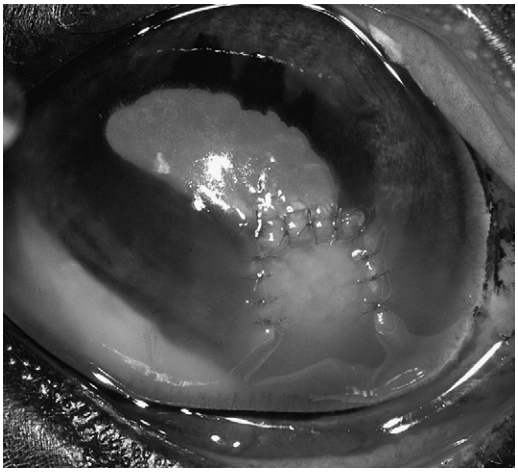


Figure 57-33. Postoperative appearance of a posterior lamellar keratoplasty (PLK).

Deep Lamellar Endothelial Keratoplasty

Deep lamellar endothelial keratoplasty represents an alternative to a PLK, designed to reduce corneal scarring by initiating the corneal incision at the limbus. The procedure is therefore particularly useful for lesions involving the peripheral cornea.[94] The procedure involves creating a pocket over the site of the lesion using a Martinez corneal dissector. This is initiated with a limbal incision using a No. 64 Beaver blade. Once the pocket has been created, a corneal trephine can be introduced, and surgery proceeds as described for a PLK (Figure 57-34). Postoperative care and potential complications are the same as those described for PLK. With this procedure a positive visual outcome was reported in 89% of cases.[94] A separate study has described a positive visual outcome with minimal scarring in 100% of cases.[121]

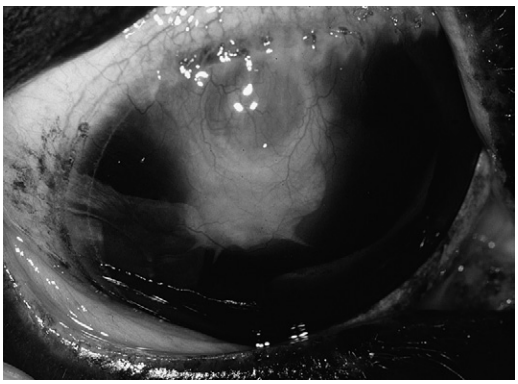


Figure 57-34. Postoperative appearance of a deep lamellar endothelial keratoplasty (DLEK).

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CHAPTER
58 Intraocular Surgery

Wendy M. Townsend

Equine intraocular surgery is performed primarily to treat equine recurrent uveitis, cataracts, and glaucoma. The relevant diagnostic procedures, anesthetic considerations, and surgical techniques are discussed for each condition within this chapter.

EQUINE RECURRENT UVEITIS

Equine recurrent uveitis (ERU), also known as moon blindness, is one of most common causes of blindness in horses. This immune-mediated condition has a prevalence of approximately 2% in the United States. ERU is characterized by repeated episodes of intraocular inflammation.

Anatomy and Physiology

The uveal tract is composed of the iris, ciliary body, and choroid (Figure 58-1). Damage to the uvea disrupts the ocular blood aqueous barrier (BAB) or the blood retinal barrier.[1] Breakdown of the BAB causes vascular dilation and increased vascular permeability, which leads to increased levels of protein and cells within the aqueous humor and vitreous. The increased protein within the aqueous humor scatters light (the Tyndall effect) when illuminated with a focused beam of light.[2] This so-called *aqueous flare* represents the hallmark of uveitis.

Prostaglandins and leukotrienes are the primary inflammatory mediators.[3] The prostaglandins cause hyperemia and a reduced intraocular pressure (IOP) (hypotony). Prostaglandins, particularly prostaglandin F2-alpha, constrict the iris and ciliary muscles, causing miosis and pain. Prolonged miosis combined with increased aqueous protein levels can create adhesions between the posterior iridal epithelium and the anterior lens capsule, called *posterior synechia*. Extensive posterior synechia can completely obstruct the pupil, causing vision loss. Complete seclusion (blockage) of the pupil can interfere with the

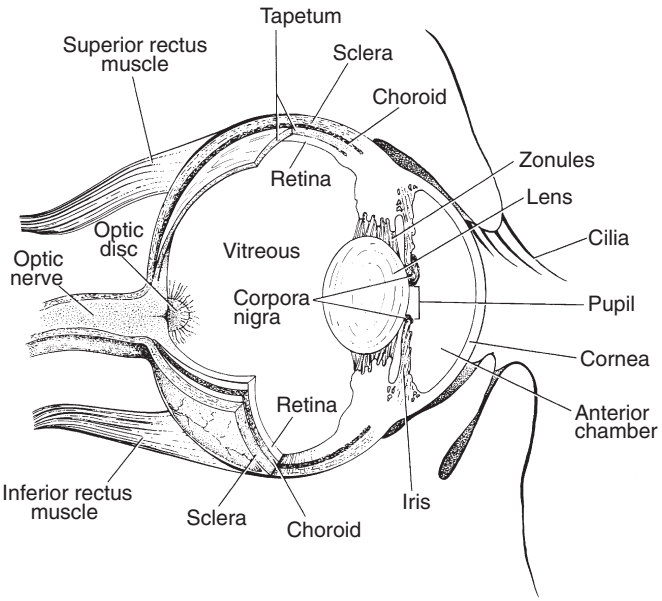

Figure 58-1. A cross-section of an equine eye.

flow of aqueous humor. Fluid can become trapped behind the iris and cause anterior bowing of the iridal face, called *iris bombé*. The altered fluid dynamics can cause *secondary glaucoma*.

Equine recurrent uveitis is characterized by recurrent or persistent bouts of ocular inflammation. ERU cannot be diagnosed after a single bout of uveitis. Immunologic studies have demonstrated the formation of autoantibodies directed at the corneal endothelium and retinal photoreceptors.[4-6] The most frequently implicated inciting cause is infection with *Leptospira interrogans*. In one German study, 75% of vitreous samples from horses with ERU were culture positive for leptospires and 100% were positive by polymerase chain reaction (PCR).[7] However, in chronically affected globes, leptospires were infrequently detected.[8] The association of ERU with leptospires may be based on antigenic homology between microbial peptides and autoantigens.[9]

Relevant Ocular Examination Techniques and Findings

A complete ophthalmic exam is critical for the diagnosis of ERU. Uveitis can be caused by corneal ulceration, stromal abscess, and ocular trauma. These conditions must be definitively excluded to determine the appropriate therapy. Sedation, an auriculopalpebral nerve block, and application of a topical anesthetic agent greatly aid the examination of a painful eye.

The visual status and level of discomfort must be ascertained. Fluorescein stain is then applied to the corneal surface to facilitate detection of corneal ulcers. Because topical application of corticosteroids is absolutely contraindicated in the face of corneal ulceration, this step is critical. The cornea should then be carefully examined, preferably with a slit lamp biomicroscope. A corneal stromal infiltrate suggests of corneal stromal abscess.

The anterior chamber should be examined with a slit lamp biomicroscope to detect aqueous flare. Fibrin can be seen as strands or clots within the anterior chamber. It is possible to see cells suspended within the aqueous humor; they appear as particulate material circulating within the focused beam of light. *Hyphema* (blood in the anterior chamber) and *hypopyon* (purulent material in the anterior chamber) may occur with severe inflammation.

The IOP should be assessed because hypotony is a subtle sign of uveitis.[10] Furthermore, glaucoma can occur secondary to chronic uveitis. Glaucoma may require additional therapy and alter the long-term prognosis. Pupil size and response to light should be noted because inflammation can induce marked miosis. The iris should be closely examined for anterior or posterior synechia. Fibrin may form a web across the corpora nigra, preventing pupillary dilation. A markedly miotic pupil may severely compromise vision.

The lens should be closely examined, preferably after pupillary dilation. Chronic inflammation induces cataract formation, likely because of diffusion of inflammatory mediators across the lens capsule.[11,12] The inflammatory products can also lyse the lens zonules.[13] Extensive zonular destruction allows the lens to luxate from the patellar fossa.

The anterior vitreous is examined with the slit lamp biomicroscope. The posterior vitreous is examined with an indirect ophthalmoscope and condensing lens. Liquefaction or condensation of the vitreous, an inflammatory cell infiltrate, and vitreal hemorrhage have been noted in patients with uveitis. The retina should be examined using indirect and or direct ophthalmoscopy. Areas of retinal detachment, subretinal exudates, retinal scarring, and optic nerve inflammation or atrophy should be identified. Posterior segment involvement influences pharmacologic therapy, potential success as a surgical candidate, and long-term visual prognosis.

Diagnostic Procedures

A full physical examination, complete blood count, and serum biochemical profile can assist in detecting underlying systemic disease. If the horse has received previous systemic nonsteroidal anti-inflammatory drugs (NSAIDs), renal parameters, and serum protein levels, an indicator of gastrointestinal mucosal health, should be established.[14]

Serologic testing for *Leptospirosis* spp. should be strongly considered in endemic areas, but interpretation can be difficult.[9] Serum antibody titers are significantly higher in horses with ERU.[15] However, 19% of normal horses have elevated serum antibody titers.[15] Also, the presence of elevated serum antibody titers does not correlate well with the isolation of *L. interrogans* from vitreous humor.[15] A positive titer for serovars at dilutions of 1:400 or greater is considered clinically significant,[16] and systemic antibiotic therapy (e.g., doxycycline, 10 mg/kg PO twice a day for 30 days) should be administered before considering surgical therapy.

One can perform *aqueous paracentesis* to obtain samples for pathogen culture, cytologic, and/or serologic evaluation. General anesthesia is recommended. The eye is routinely prepared with dilute povidone-iodine (Betadine) solution. An eyelid speculum is placed and a topical anesthetic agent is applied to the cornea and conjunctiva. The conjunctiva is grasped near the limbus with small-toothed forceps. A 25-gauge (or smaller) needle attached to a 1-mL syringe is inserted under the conjunctiva, through the limbus, and into the anterior chamber (Figure 58-2). The needle should be directed parallel to the plane of the iris. No more than 0.5 mL of aqueous should be removed.

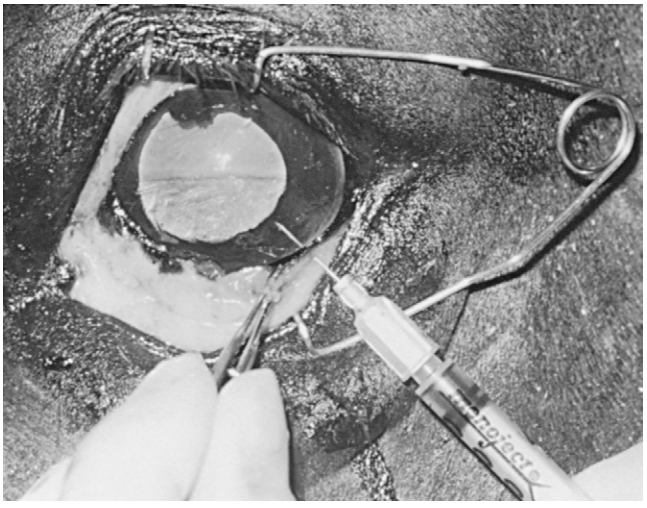

Figure 58-2. Paracentesis of the anterior chamber. A 25-gauge needle attached to a 1-mL syringe is inserted into the anterior chamber at the limbus, and fluid is withdrawn.

Anesthetic Considerations

The surgical techniques described are typically performed under general anesthesia. Retrobulbar anesthesia (see Chapter 55) or neuromuscular blocking agents may assist in globe positioning. For all ocular procedures, a smooth recovery from anesthesia prevents further trauma to the globe and decreases postoperative complications.

Required Surgical Equipment

For intraocular surgery to be successful, the surgeon should be trained in microsurgery and perform enough intraocular procedures to stay competent. Magnification, usually an operating microscope, and microsurgical instruments must be available. The specific instrumentation required for performing intravitreal injections (Table 58-1), suprachoroidal cyclosporine implants (Table 58-2), and pars plana vitrectomy (Table 58-3) are listed later.

Relevant Pharmacology

Corticosteroids, mydriatics, and NSAIDs are used to reduce inflammation and minimize permanent ocular damage at each episode of active uveitis. They do not effectively prevent recurrence of disease. Corticosteroids are applied topically or injected subconjunctivally together with topical 1% atropine sulfate to reduce inflammation and stabilize the BAB. Prednisolone acetate 1% ophthalmic suspension and dexamethasone 0.1% are the only topical corticosteroids that can achieve therapeutic concentrations in the aqueous humor.[17] NSAIDs are administered systemically, particularly if posterior segment inflammation is noted. Intracameral injection of tissue plasminogen activator may be used to dissolve organized fibrinous exudates in the anterior chamber. Aspirin and phenylbutazone may be used by some clinicians to prevent or decrease the severity of recurrent episodes. However, these medications have potentially detrimental side effects when used chronically in the horse.

TABLE 58-1. Surgical Instrumentation Recommended for an Intravitreal Injection

Instrument	Quantity
Solid blade eyelid speculum	1
Castroviejo caliper	1
Bishop-Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	2
Sterile cotton-tipped applicators	Package of 10

TABLE 58-2. Surgical Instrumentation Recommended for Suprachoroidal Cyclosporin (CsA) Implantation

Instrument	Quantity
Ocular adhesive drape	1
Eyelid speculum	1
Bishop-Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	1
Derf needle holder	1
Suture scissor (any type)	1
4-0 silk (stay suture)	1
Mosquito hemostatic forceps	2
Castroviejo caliper	1
Stevens tenotomy scissor	1
0.3-mm Colibri forceps	1
No. 64 microsurgical blade and handle	1
Castroviejo needle holder	1
5-0 to 6-0 polyglactin 910	1

Surgical Techniques

Intravitreal Injections

For a vitreal injection, a 22- to 23-gauge needle is used. General anesthesia is preferred. The needle is inserted 10 mm behind the limbus at the 12 o'clock position, through conjunctiva, sclera, and the pars plana of the ciliary body. The needle is directed toward the posterior pole of the eye to avoid traumatizing the lens. Because the needle often cannot be observed, intravitreal injections carry a greater risk of injury to the eye.

Intravitreal injections of triamcinolone acetonide are used to control inflammation in human patients with recurrent uveitis.[18] A study performed in normal equine eyes did not note any overt toxicity from intravitreal triamcinolone injections.[19] The ocular triamcinolone levels persisted for up to 21 days.[19] However, 4 out of 12 eyes (3 of which were controls) developed bacterial endophthalmitis. This finding highlighted the need for meticulous aseptic technique and the use of topical and/or systemic antimicrobials at the time of injection.[19]

I have injected 10 mg of filtered triamcinolone acetonide with 20 mg of ampicillin intracamerally in cases with intractable uveitis. Improvement in the uveitis was noted. Endophthalmitis has not been a complication. However, several horses developed persistent superficial corneal ulcers. Because of the complications associated with triamcinolone injections, other

TABLE 58-3. Surgical Instrumentation Recommended for Pars Plana Vitrectomy (PPV)

Instrument	Quantity
Ocular adhesive drape	1
Eyelid speculum	1
Bishop-Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	1
Suture scissors (any type)	1
Castroviejo caliper	1
Stevens tenotomy scissors	1
0.3-mm Colibri forceps	1
CO_2 laser	1
Irrigation port	1
Equine vitrector	1
Custom globe manipulator (recommended)	1
or	
Derf needle holder 4-0 silk (stay suture) mosquito hemostatic forceps	
Castroviejo needle holder	1
4-0 polyglactin 910	1
500 mL saline solution + 40 mg gentamicin	1 bottle/eye
Indirect ophthalmoscope	1
20D condensing lens	1

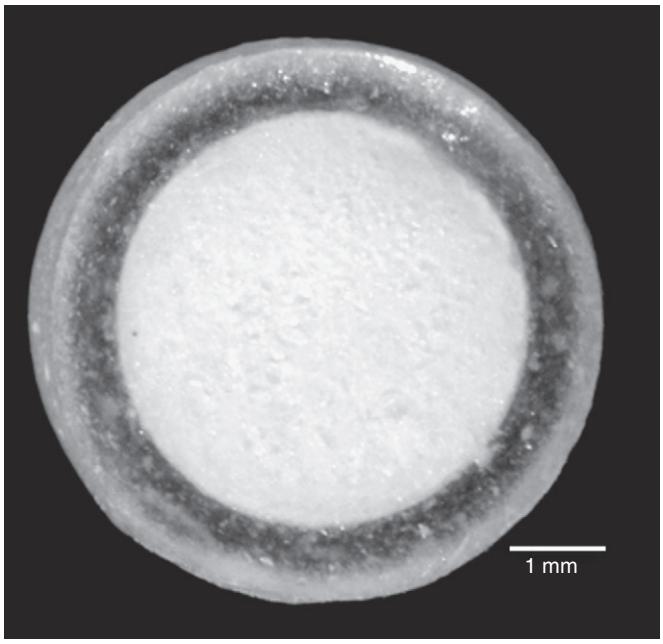

Figure 58-3. Cyclosporine-containing device for placement in the suprachoroidal space for long-term treatment of equine recurrent uveitis. (Courtesy Dr. Mike Robinson.)

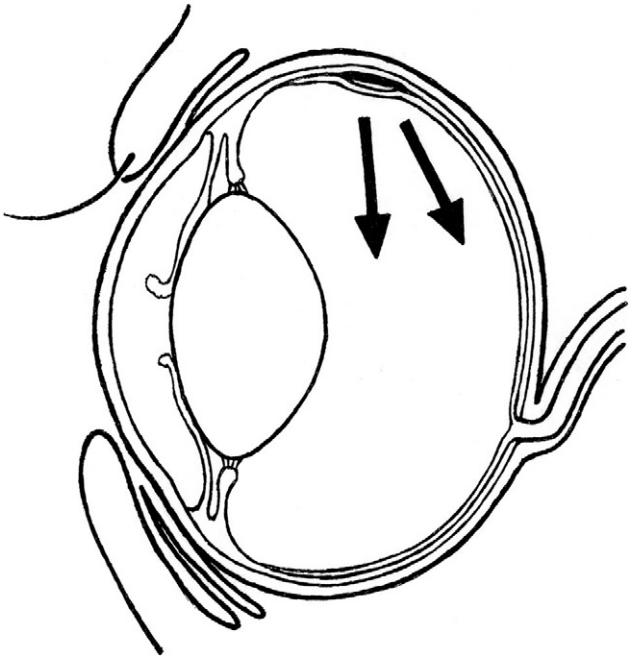

Figure 58-4. Location of the placement of a suprachoroidal drug-delivery device. Drug delivery occurs in the direction of the *arrows.*

agents such as rapamycin are currently under investigation. In preliminary studies, rapamycin injected subconjunctivally and intravitreally did not produce evidence of ocular irritation or toxicity.[20]

Suprachoroidal Cyclosporine Implantation

A device that allows constant release of cyclosporine A (CsA) was developed for placement in the suprachoroidal space directly adjacent to the ciliary body (Figure 58-3). The best candidates for CsA implantation are horses that have chronic ERU and experience frequent recurrences after stopping medication. The horse must have little or no active inflammation at the time of surgery. Horses whose inflammation cannot be controlled with anti-inflammatory medications are not good candidates for CsA implantation. Inflamed eyes are more prone to surgical and postsurgical complications. Equine patients with significant cataract formation and glaucoma are also considered poor candidates.

The horse is placed under general anesthesia. A 1-cm conjunctival incision is made in the dorsolateral bulbar conjunctiva. A 7-mm-wide scleral flap is prepared, exposing the uveal tract approximately 8 mm posterior to the limbus and just lateral to the insertion of the dorsal rectus muscle (Figure 58-4). The CsA-containing device is placed into the incision, in contact with the uveal tract (Figure 58-5). The scleral flap is closed over the implant using 5-0 to 6-0 polyglactin 910 or similar absorbable suture material in a simple-interrupted pattern (Figure 58-6). Recommended postoperative medications include flunixin meglumine (500 mg PO once a day) for 5 days, topical triple antibiotic ophthalmic ointment twice a day for 10 days, and topical atropine ointment once a day for 7 days. Some horses have a mild flare-up after flunixin meglumine is discontinued. If this occurs additional systemic NSAIDs may be needed.

Figure 58-5. A 7-mm-wide scleral flap is made exposing the uveal tract (the black uvea is just visible through the sclera) approximately 8 mm posterior to the limbus and just lateral to the insertion of the dorsal rectus muscle.

A long-term clinical trial of 133 horses with documented ERU demonstrated retention of vision in 78% of cases.[21] The complications resulting in vision loss were persistent uveitis, glaucoma, mature cataracts, and retinal detachment. The mean frequency of uveitis episodes was 0.09 episodes per month after

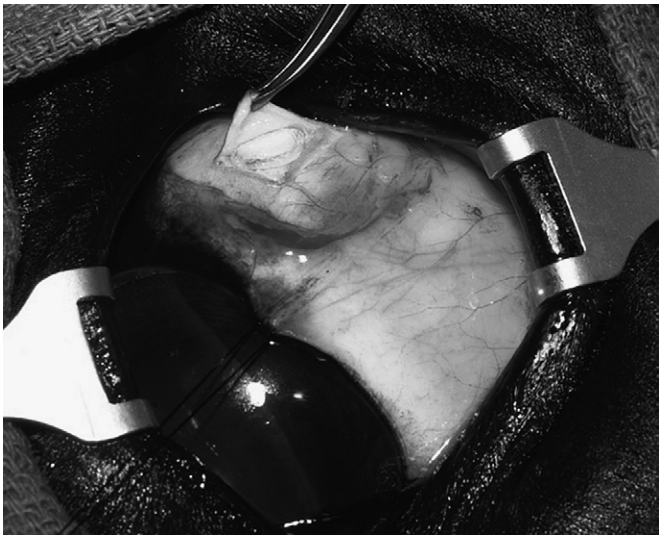

Figure 58-6. The cyclosporine-containing device is placed into the incision, in contact with the uveal tract. The scleral flap and conjunctival incision is closed with 6-0 polyglactin 910 suture material in a simple-interrupted pattern, followed by closure of the conjunctiva over the scleral incision.

placement of the CsA implant.[21] Inflammatory episodes increased 48 months after the initial surgery. Therefore, the authors suggest a second CsA implant might be required approximately 48 months following the initial surgery.[21]

Pars Plana Vitrectomy

First described in 1991,* pars plana vitrectomy (PPV) has been increasingly employed in the treatment of ERU, particularly in Europe.[22-24] The beneficial effect of PPV most likely results from the physical removal of resident inflammatory cells from the vitreous.[25] The most common complications are transient hypopyon, vitreal or retinal hemorrhage, retinal detachment, and cataract formation.

The potential complications of PPV necessitate careful patient selection and thorough owner education. Minimal inflammation should be present at the time of surgery. Transpupillary visualization of the vitrectomy probe is required. Therefore the ocular media must be transparent and the pupil must dilate maximally. Preexisting focal cataracts are likely to progress. Patients with secondary glaucoma, phthisis bulbi, or retinal detachment are poor candidates for PPV.

Topical neomycin, polymyxin B, and dexamethasone (NPDex) ophthalmic solution is administered four times a day for 1 week before surgery. Flunixin meglumine is administered beginning 3 days preoperatively. The pupil is dilated with 1% atropine drops on the day of surgery. Postoperatively, topical NPDex is administered 3 times weekly for 2 weeks and then tapered over 4 weeks. Systemic NSAIDs are continued for 1 week.

A standard two-port PPV is performed with the horse in lateral recumbency under general anesthesia. The eye is prepared for intraocular surgery. After draping, an eyelid speculum is inserted. A lateral canthotomy may improve exposure. With a custom-made globe manipulator (Figure 58-7) or limbal stay suture, the globe is rotated to expose the dorsal bulbar conjunctiva. A limbal-based conjunctival flap is prepared and the sclera exposed medial and lateral to the dorsal rectus muscle. Using a CO_2 laser in continuous mode at 50 W, a sclerotomy is performed 10 mm posterior to the limbus. A right-handed surgeon places this first entry to the left of the rectus muscle. The irrigation port is inserted and secured to the sclera with a 4-0 polyglactin 910 suture (Figure 58-8). The irrigation fluid consists of physiologic saline solution with 40 mg of gentamicin added per 500 mL. The fluid bottle is positioned 85 cm higher than the globe to maintain an intraocular pressure (IOP) of 40 mm Hg. The infusion is started at a flow rate of 20 mL/min.

A second sclerotomy is performed 10 mm posterior to the limbus and to the right of the rectus muscle (Figure 58-9). The vitrectomy probe is carefully inserted and advanced toward the central vitreous. A custom-made 55-mm oscillating vitrectomy probe is used at 6.5 Hz with an aspiration vacuum of 240 mm Hg (Figure 58-10). Care is taken to avoid touching the lens (Figure 58-11). The probe is held with the aspiration port facing the surgeon. The port can be visualized through the pupil using an indirect binocular ophthalmoscope (Figure 58-12).

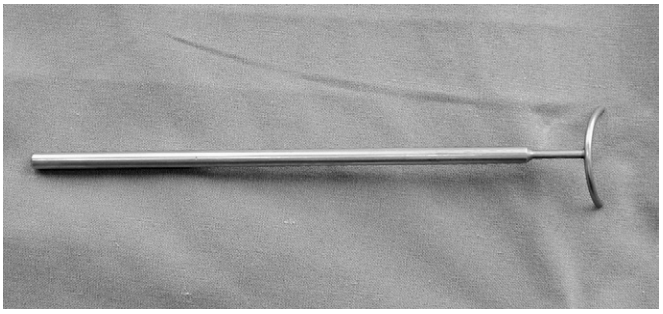

Figure 58-7. Custom-made globe manipulator to facilitate globe rotation during laser sclerotomy.

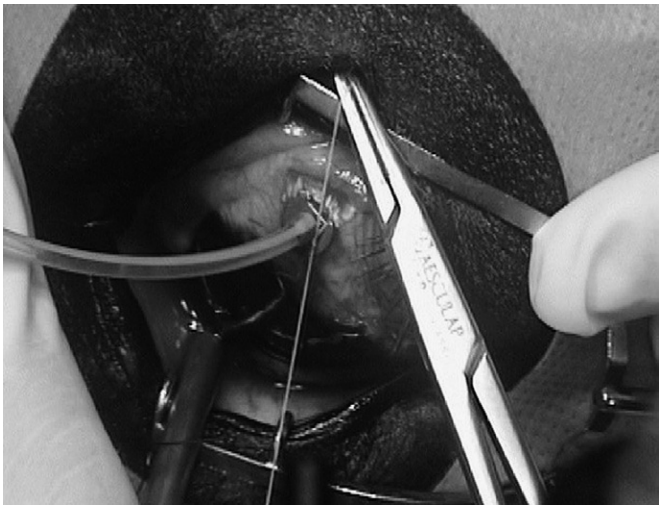

Figure 58-8. The irrigation port of the two-port vitrectomy unit is anchored to the sclera using 4-0 Vicryl suture.

*Editor's note: The first successful vitrectomy was performed in 1983 in the Equine Clinic in Wahlstedt, Germany, by L. Koehler and his associates.

Figure 58-9. CO_2 laser sclerotomy for the vitrectomy handpiece.

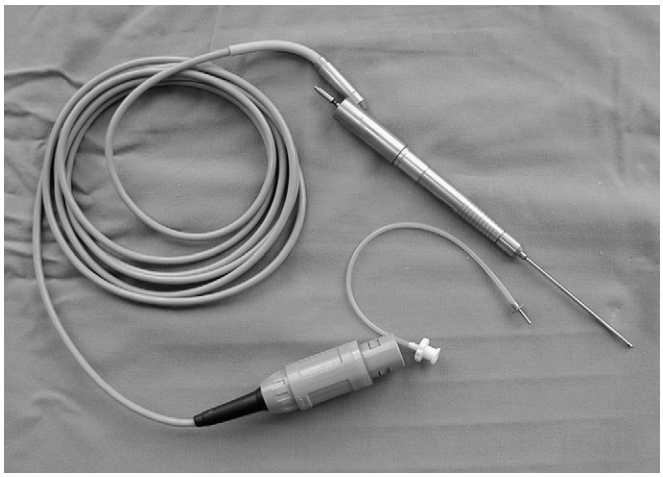

Figure 58-10. Custom-made vitrectomy handpiece with a 55-mm shaft and independent irrigation port.

Figure 58-11. The custom-made vitrectomy handpiece is advanced into the center of the vitreous cavity.

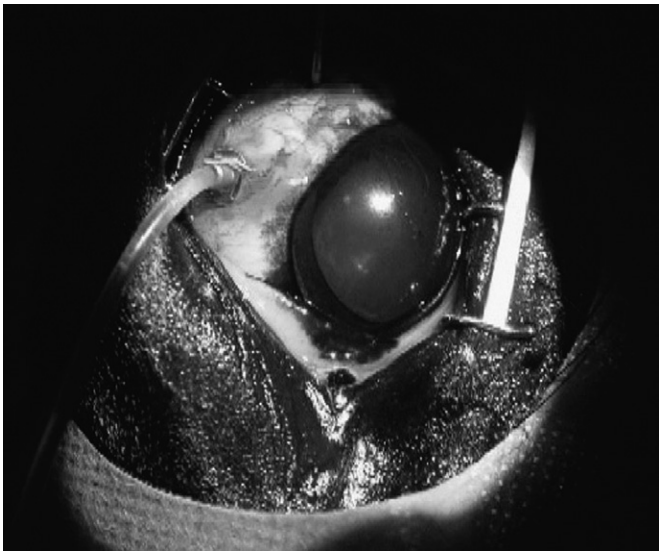

Figure 58-12. Vitrectomy is performed in a darkened operating theater using a binocular indirect ophthalmoscope and a 20D lens. The vitrectomy probe can be visualized through the dilated pupil. (Courtesy Dr. B. Wollanke.)

With a 20 diopter lens in the left hand and the vitrectomy probe in the right, the vitrectomy is started. The shadow cast on the retina by the probe assists the surgeon in estimating the distance between the probe and the retina. The IOP should be maintained at approximately 40 mm Hg. Slight wrinkling of the retina indicates the IOP may be too low. If this is noted, the surgery should be interrupted until a normal IOP is restored. The procedure is continued until all turbid vitreal material has been removed. Under continuous irrigation, the vitrectomy probe is removed and the sclerotomy is closed with one or two preplaced simple-interrupted sutures using 4-0 polyglactin 910. Subsequently, the irrigation port is removed. Remaining vitreous usually prevents fluid from escaping through this sclerotomy, which is closed with 4-0 polyglactin 910. The conjunctiva is closed with polyglactin 910 in a continuous pattern. The canthotomy is closed with a figure-of-eight suture using 4-0 nonabsorbable suture material. At the end of surgery, 20 mg of methylprednisolone is injected subconjunctivally into the inferior bulbar conjunctiva.

In one study of 38 cases, 33 eyes showed no recurrence during a follow-up period of up to 5 years.[24] Five eyes had a recurrence of uveitis between 10 days and 3 years postoperatively; two demonstrated marked loss of vision, and three maintained preoperative vision.[24] Vision remained stable in 28 eyes

and improved in 1 eye. The remaining eyes showed marked vision loss as a result of cataracts (3), phthisis bulbi (1), or unknown causes (1). In an earlier study of 43 eyes, 42 remained free of recurrent uveitis during the 67-month follow-up period, and of these eyes, 70% retained some vision.[26] The most common complication was cataract formation in 19 of 43 eyes, followed by phthisis bulbi in 6 eyes, and retinal detachment in 4 eyes.

CATARACTS

A cataract refers to any opacity of the lens. Cataracts may occur as a congenital lesion. In Morgan, Belgian, Quarter Horse, and Thoroughbred bloodlines, cataracts occur as a dominant trait.[27,28] Therefore breeding of affected individuals is not recommended. Cataracts also occur in Rocky Mountain horses with anterior segment dysgenesis (ASD).[29] In adult horses, cataracts often occur secondary to chronic ERU.[28] Blunt or penetrating ocular trauma can incite cataract formation. Older horses may develop senile cataracts.

Anatomy and Physiology

The crystalline lens is the transparent, biconvex structure that "fine focuses" light on the retina.[30] Zonular fibers suspend the lens behind the iris in the patellar fossa. The zonules extend from the ciliary body and insert onto the lens equator. Breakage of the zonules allows the lens to shift within the patellar fossa (lens subluxation), displace into the anterior chamber (anterior lens luxation), or displace into the posterior segment (posterior lens luxation). Lens luxations occur relatively frequently in horses.[9] Lens luxations may occur secondary to trauma, glaucoma with buphthalmos, resorption of a hypermature cataract, or chronic ERU. Rocky Mountain horses with ASD may suffer lens luxation because of inherited zonular abnormalities.

Relevant Ocular Examination Techniques and Findings

Mydriasis is required to completely examine the lens. Topical application of a rapid-onset mydriatic (1% tropicamide ophthalmic solution) will dilate the pupil within 30 minutes.[31] An opacity may be axial (along the visual axis), equatorial, capsular, subcapsular, cortical, or nuclear. The cataract can be classified based on the extent of lenticular involvement: incipient (less than 15%), immature (15% to 99%), mature (100%), and hypermature (100% with resorption of cortical material) (Figure 58-13). The degree of visual impairment depends upon the size, location, and density of the opacity. The remainder of the eye should be inspected closely for evidence of uveitis and other ocular abnormalities. A fundic examination should be performed if possible.

Congenital and traumatic cataracts typically have the best long-term prognosis for maintaining vision.[32] Rocky Mountain horses with ASD have a poorer prognosis because of the associated retinal dysplasia that predisposes to retinal detachment.[29,32] Horses with cataracts secondary to ERU have a guarded long-term prognosis for vision, because repeated bouts of uveitis often result in keratitis, retinal detachment, synechiae, or phthisis bulbi and blindness.[32] The temperament of the individual also should be carefully considered because restricted activity and the application of topical ocular medications are required for 4 to 6 weeks after surgery. Postoperative trauma incurred while applying medications or suture breakage because of excessive activity can diminish the success of the procedure.

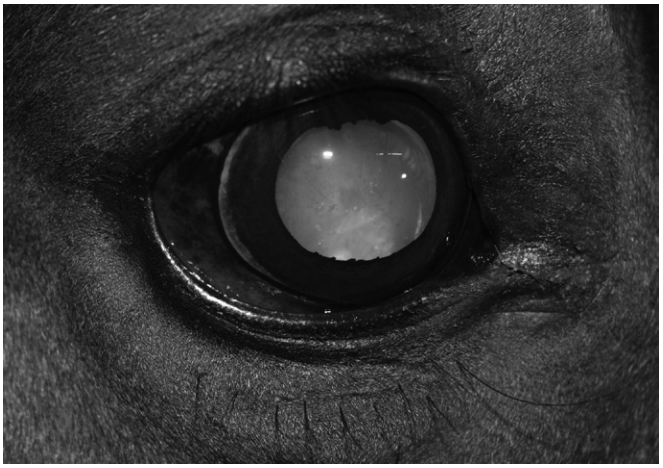

Figure 58-13. A horse with an immature cataract.

Foals with congenital cataracts should undergo surgery early if the cataract completely obscures vision.[32] Human infants with complete cataracts have a better success rate if surgery is performed in the first 6 weeks of life to allow proper development of the visual neural network.[33] Although the definitive age by which cataract surgery must be performed in foals is not known, current recommendations favor surgical intervention before 6 months of age.[32]

Diagnostic Procedures

A complete physical examination should be performed with special emphasis placed on the respiratory system. Occult respiratory infection, especially with *Rhodococcus equi*, can result in postoperative endophthalmitis. A complete blood count with fibrinogen levels should be performed to detect underlying systemic infection or inflammation. A serum biochemistry profile provides baseline renal values and ensures the normal organ function.

If the cataract obscures visualization of the fundus, an electroretinogram (ERG) and ocular ultrasonography should be performed. The latter assists in detecting retinal detachments, vitreal opacities, and remnants of the hyaloid vasculature. Retinal detachments preclude cataract surgery. The ERG evaluates retinal, particularly photoreceptor, function; it can detect diffuse retinal degeneration, especially in patients with evidence of trauma or chronic ERU. The ERG can also be used to rule out congenital stationary night blindness, which occurs in Appaloosas, Thoroughbreds, Paso Finos, and Standardbreds.[34-36]

Anesthetic Considerations

Intraocular surgery is performed under general anesthesia. Either retrobulbar anesthesia or systemic neuromuscular blockade is required to facilitate globe positioning and prevent compression of the globe by the extraocular muscles. Maintaining normal globe shape and IOP decreases the likelihood of posterior capsular tears, iris prolapse, and expulsion of ocular contents through the corneal incision. Retrobulbar anesthesia is performed as described in Chapter 55.

Figure 58-14. A standard phacoemulsification needle is shown in the lower aspect of the photograph. The significantly longer equine phacoemulsification needle is shown in the upper aspect of the photograph.

The neuromuscular blocking agent atracurium besylate (10 mg/mL, currently available only as a generic formulation, formerly available as Tracrium) or pancuronium (1 or 2 mg/mL, generic formulations or Pavulon) are administered at 0.12 to 0.2 mg/kg IV and provide a duration of action of 30 to 60 minutes.[37] Atracurium is preferred because of its reliable duration of action, lack of cumulative effect, lack of cardiovascular effects, and lower costs.[37] The degree of neuromuscular blockade should be monitored with a train of four stimulations from a peripheral nerve stimulator.[37] For ocular procedures, the superficial peroneal nerve is more accessible than the facial nerve.[38] The reversal agent edrophonium chloride (10 mg/mL, Tensilon, Enlon, or Reversol) is administered at 0.5 to 1.0 mg/kg, slowly by IV before recovery to ensure that residual neuromuscular blockade does not compromise the ability of the horse to stand. Recoveries after neuromuscular blockade are generally scored as good to excellent.[37]

Required Surgical Equipment

An operating microscope and phacoemulsification system are required to optimally perform equine cataract surgery. *Phacoemulsification* uses ultrasonic energy to break up lens material, which is removed through an aspiration port. Continuous irrigation maintains adequate pressures within the anterior chamber. Surgery can be performed through a small incision, which reduces the risk of iris prolapse and other operative complications. The degree of postoperative inflammation is significantly reduced. Phacoemulsification systems configured for equine use have longer needles, which greatly facilitate removal of lenticular material (Figure 58-14). The remainder of the required equipment is listed in Table 58-4.

TABLE 58-4. Surgical Instrumentation Recommended for Phacoemulsification

Instrument	Quantity
Ocular adhesive drape	1
Eyelid speculum	1
Bishop-Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	1
Derf needle holder	1
Suture scissors (any type)	1
4-0 silk (stay suture)	1
Mosquito hemostatic forceps	2
0.12-mm Colibri forceps (corneal forceps)	1
No. 64 microsurgical blade and handle	1
3.2-mm slit knife	1
Viscoelastic agent	1
Utrata forceps or high frequency capsulotomy probe	1
Phacoemulsification unit	1
0.3-mm Colibri forceps	1
±Lens introducing forceps	1
±Lens folding forceps	1
Castroviejo needle holder	1
Straight and curved tying forceps	2
Methylcellulose spears	Package of 10
7-0 polyglactin 910	1

Relevant Pharmacology

There are currently no systemic or topical medications that "dissolve" cataracts. Horses with focal opacities may have improved vision after pharmacologic mydriasis is achieved through application of 1% atropine ophthalmic solution or ointment. The only effective therapy for cataracts causing significant visual impairment is surgical removal.

The day before surgery, topical NPDex ophthalmic solution is applied every 6 hours to the ocular surface. Flunixin meglumine (1.1 mg/kg PO or IV every 12 hours) is administered. Gastric protectants such as omeprazole (2 mg/kg PO daily) are indicated, particularly for foals. Tetanus toxoid is administered if vaccinations are not current.

The day of surgery, topical NPDex and flurbiprofen 0.03% ophthalmic solutions are applied three times each to the ocular surface. Atropine 1% or tropicamide 1% ophthalmic solution is administered to achieve maximal pupillary dilation. Flunixin meglumine is continued. Systemic antibiotics (potassium penicillin 20,000 IU/kg IV every 6 hours, and gentamicin 6.6 mg/kg IV every 24 hours) are administered perioperatively.

Surgical Techniques

Phacoemulsification

After induction of general anesthesia, the horse is placed in lateral recumbency. The head is positioned such that the corneal surface is parallel to the surgery table. The ocular surface is prepared routinely with dilute povidone-iodine solution. The head is draped routinely and an eyelid speculum is placed. A lateral canthotomy is performed if needed to improve exposure. The anterior chamber is entered through a scleral tunnel or a two-step clear corneal incision. A highly cohesive viscoelastic material (hyaluronan; Acrivet Syn 2%, a 2% hyaluronic acid solution) is used to reform and maintain the anterior chamber. A continuous circular capsulorrhexis is performed.

The phacoemulsification needle is introduced and nuclear material is removed via phacoemulsification (Figure 58-15). The remaining cortical material is removed using irrigation and aspiration. After complete removal of lenticular material, a posterior capsulorrhexis may be performed to prevent migration of lens epithelial cells across the visual axis and thereby

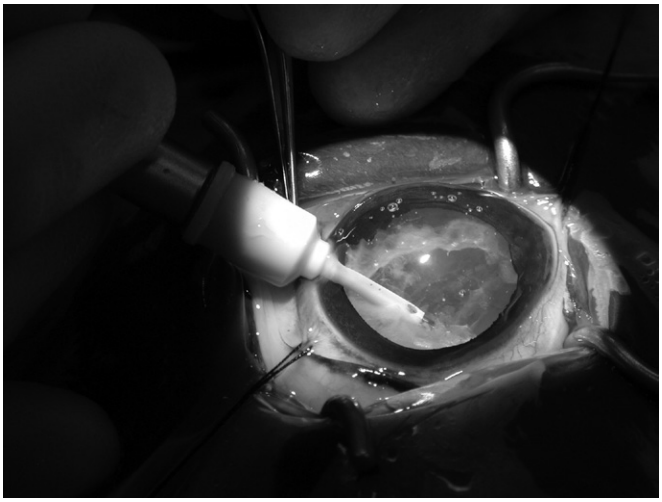

Figure 58-15. An intraoperative photograph demonstrating removal of nuclear material using phacoemulsification.

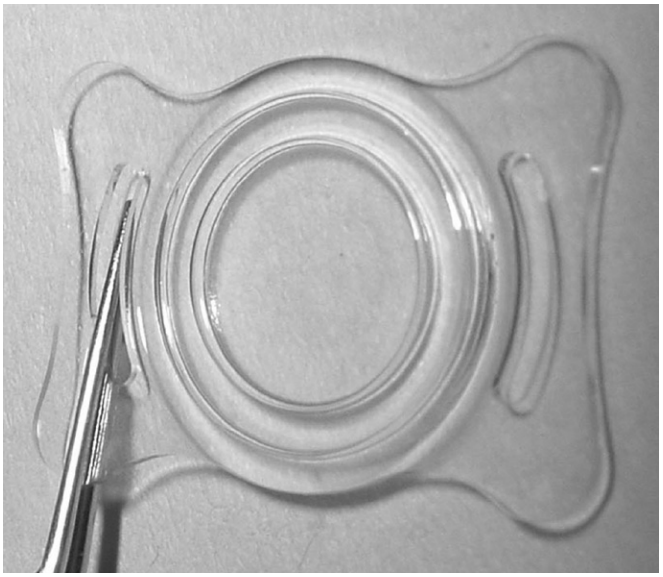

Figure 58-16. An equine intraocular lens with a 12-mm diameter optic and 21-mm overall diameter. This lens is appropriate for use in a foal.

maintaining a clearer visual pathway. If elected, an intraocular lens can then be placed. The cornea is closed with a simple-interrupted or simple-continuous suture pattern of 7-0 absorbable suture. Before the final suture is secured, the surgeon may choose to remove any remaining viscoelastic material (sodium hyaluronate) via irrigation and aspiration.

Unless a corneal ulcer is present, topical antibiotics and corticosteroids are applied every 4 to 6 hours to control postoperative inflammation. Topical atropine is administered as needed to maintain mydriasis. Systemic NSAIDs and gastric protectants are continued. A face mask with a protective eyecup prevents rubbing and suture breakage. Installation of a subpalpebral lavage (SPL) facilitates administration of medications to the globe. However, corneal ulceration is a risk if the SPL should dislodge. Activity is restricted initially to hand-walking only. A recheck examination is performed 7 to 10 days postoperatively. If ocular inflammation is controlled, medications are gradually reduced over 2 to 4 weeks. Once the corneal incision heals, typically 3 to 4 weeks postoperatively, the patient may resume normal activities. Possible postoperative complications include poor anesthetic recovery, systemic infection, suture breakage, endophthalmitis, ocular hypertension, corneal ulceration, inflammation, and retinal detachment.

Intraocular Lens Placement

Equine intraocular lenses (IOLs) are now available (Acrivet, Inc., Jordan UT) (Figure 58-16). They prevent severe hypermetropia (farsightedness), which results when a globe is aphakic (lacks a lens).[39,40] The predicted visual acuity of an aphakic horse would be 20/1200 on the Snellen chart.[41-44] Humans with this degree of uncorrected hypermetropia would be considered legally blind.[45] The lenses are made of foldable acrylic, which minimizes the incision size required for their placement. They are available at strengths of +14D and +18D and 21-, 22-, and 24-mm diameters. The +14D, 21-mm diameter lenses are typically implanted in foals. The lenses are inserted through the corneal incision into the capsular bag using lens forceps or an injecting cartridge. The corneal closure and postoperative management are the same as listed earlier.

Intracapsular Lens Extraction

If the lens luxates, an intracapsular lens extraction is required to remove the lens *en bloc*. This procedure should be reserved for potentially visual eyes. An intracapsular lens extraction requires a 180-degree corneal incision. Therefore, complications such as iris prolapse and marked uveitis are common, and the prognosis for vision is guarded. The postoperative therapy and additional complications are similar to those listed for cataract surgery. In an eye that has been blinded, an eye that has experienced multiple bouts of ERU, or an eye that has sustained marked trauma, enucleation or evisceration with intrascleral prosthesis is the preferred therapeutic option (see Chapter 55).

GLAUCOMA

Glaucoma is characterized by increased IOP resulting in damage to the optic nerve and retina.[46] Glaucoma is relatively rare in horses with an incidence of 0.07%.[28] However, as most equine practitioners lack the instrumentation to measure IOP and are unlikely to suspect the condition, glaucoma is easily missed or misdiagnosed.[47]

Anatomy and Physiology

Aqueous humor is formed by the ciliary processes.[48] Aqueous primarily leaves the eye through the trabecular meshwork within the iridocorneal angle. Aqueous also diffuses through the iris, ciliary body, and vitreous: the uveoscleral outflow pathway. Horses have a greater percentage of uveoscleral outflow than most other species.[49] Obstruction of aqueous outflow results in an increased IOP. The elevated IOP decreases optic nerve axoplasmic flow and eventually causes retinal ganglion cell death.[50] Although congenital and primary cases do occur, equine glaucoma is most often secondary to ERU.[51,52]

Relevant Ocular Examination Techniques and Findings

Horses with glaucoma may not present with overt signs of pain unless concurrent anterior uveitis is present. If the horse has secondary glaucoma with active anterior uveitis, the clinical findings may include chemosis, miosis, synechia, flare, and cataract formation.[47] Elevations in IOP cause corneal edema, mydriasis, and a decreased menace response. The mydriasis may not occur if synechia prevents movement of the pupillary margin. Horses with chronic elevations in IOP develop buphthalmos, corneal striae, corneal vascularization, lens luxation or subluxation, optic nerve degeneration, and retinal degeneration. The corneal striae represent breaks or thinning of Descemet's membrane.[53] However, corneal striae are not pathognomonic for glaucoma and can be present in otherwise normal eyes.[53] Horses with buphthalmos may retain some vision, unlike most other species. Nevertheless, vision is impaired and complete blindness often results.

Diagnostic Procedures

Measuring IOP is critical for diagnosing glaucoma. Use of an applanation (Tono-Pen) or rebound (TonoVet) tonometer is recommended. The normal IOP in unsedated horses is 17 to 28 mm Hg.[54,55] Sedation with xylazine decreases IOP by 23% to 27%.[56] An auriculopalpebral nerve block prevents false elevations in IOP that are due to pressure from the eyelids.[56] Lowering the head below heart level significantly elevates IOP (8 mm Hg).[57] Because many factors influence IOP measurement, a consistent protocol must be followed to allow meaningful interpretation of changes in IOP.

Required Surgical Equipment

Pharmacologic ablation of the ciliary body requires minimal surgical equipment (see Table 58-1). Cyclophototherapy requires a semiconductor diode or neodymium-doped yttrium aluminum garnet (Nd:YAG) laser (Table 58-5).

Relevant Pharmacology

If uveitis is present, medical therapy must address control of inflammation (see "Relevant Pharmacology" earlier). Topical atropine may benefit patients with ERU because it decreases discomfort and stabilizes the BAB.[47] However, the IOP must be closely monitored. In some patients the addition of topical atropine reduces IOP,[58] but in others, atropine may elevate it.[58]

Medical therapy to diminish IOP can be relatively successful early in the course of the disease.[50] However, long-term control of IOP is difficult to maintain with medical therapy alone. A target IOP of less than 20 mm Hg is a reasonable goal for therapy.[50] The most effective pharmacologic agents in horses reduce IOP by *diminishing aqueous humor production*. The topical β-adrenergic receptor blocker timolol maleate (0.5% ophthalmic solution; Timoptic) has been shown to diminish IOP by 17% in normal horses after administration of a single dose.[59] In clinical patients timolol is typically administered two or three times daily. The topical carbonic anhydrase inhibitor dorzolamide (2% ophthalmic solution, Trusopt, and generic formulations) also diminishes IOP through reduction of aqueous humor production.[60] However the reduction occurs through a different mechanism than that of β-adrenergic receptor blockers. Dorzolamide can be used alone or in combination with timolol. The typical frequency of application is two or three times daily.

Prostaglandin analogues are used extensively for glaucoma therapy in humans and small animals. They diminish IOP by *increasing uveoscleral outflow*. However, they have demonstrated little benefit following application to equine globes.[61,62] Adverse effects are frequently noted and include miosis, epiphora, blepharospasm, and blepharedema.[62] Therefore, prostaglandin analogues are not currently recommended for therapy of equine glaucoma.

TABLE 58-5. Surgical Instrumentation Recommended for Cyclophotocoagulation

Instrument	Quantity
Ocular adhesive drape	1
Eyelid speculum	1
Bishop-Harmon forceps (9 cm, 1 × 2 0.5-mm teeth)	1
Tuberculin syringe and 25-gauge needle	1
Sterile cotton-tipped applicators	Package of 10
Tono-Pen or TonoVet	1
Diode laser with glaucoma probe	1

Surgical Techniques

Transscleral Cyclophotocoagulation

Both Nd:YAG and semiconductor diode lasers have been used successfully in horses to reduce IOP.[28,63-65] Laser energy is applied to the ciliary body through the sclera to destroy the ciliary body epithelium and stroma, thereby reducing aqueous production.[50] A recent paper suggests that transscleral cyclophotocoagulation (TSCP) aids in control of IOP and maintenance of vision but does not diminish the need for topical glaucoma medications.[64] Therefore, patients with the potential for vision are the best candidates. Individuals with chronically blind eyes are better candidates for enucleation or evisceration and placement of intraocular silicone prosthesis (see Chapter 55).

When using the Nd:YAG laser, 55 to 60 applications are placed 5 to 6 mm from the limbus with a power of 10 to 11 W for a duration of 0.4 seconds per site.[28,66] For the diode laser, settings are 1500 mW and 1500 msec per site.[64] The number of sites varies from 30 to 80, placed 4 to 6 mm from the limbus.[28,64] The nasal region should be avoided because of the proximity of the retina.[67] Aqueous paracentesis is often performed immediately following TSCP because of rapid elevations in IOP.[64] Hyphema, corneal ulceration, conjunctival hyperemia, increased corneal edema, and aqueous flare have all been reported following TSCP.[50,64,66] Postoperatively, the glaucoma medications are continued. Topical anti-inflammatory therapy is used unless corneal ulceration occurs. In addition, systemic NSAIDs are administered to control the postoperative inflammation. The IOP should be evaluated 24 hours after surgery. The anti-inflammatory therapy is tapered over several weeks. The glaucoma medications often continue indefinitely. A decrease in IOP is typically not seen for 2 to 4 weeks after diode TSCP.[64]

Gonioimplants (Anterior Chamber Shunts)

Gonioimplants are considered experimental in the horse, but they have been used successfully to improve aqueous drainage.[28] The implants provide an alternative outflow pathway for aqueous drainage to the subconjunctival space. The implants typically have a short life span because of fibrosis of the filtration bleb.[50] There are no commercially available equine gonioimplants.

Pharmacologic Ablation of the Ciliary Body

Pharmacologic ablation of the ciliary body is a salvage procedure and should only be performed in eyes that are permanently blind. Owners should be warned that the procedure can induce phthisis bulbi.[50] The procedure is typically performed under a brief episode of general anesthesia or heavy sedation with regional and topical anesthesia. The injection consists of 25 to 40 mg of intravenous gentamicin to ablate the ciliary body and 1 mg of dexamethasone to control inflammation. The injection is performed with a 22-gauge needle attached to a 3-mL syringe. The needle enters the globe 7 to 8 mm posterior to the dorsotemporal limbus at a 45-degree angle toward the optic nerve.[50] The needle must be directed away from the lens. Rupturing the lens capsule with the needle is a catastrophic complication that incites marked inflammation (phacoclastic uveitis). Vitreous can be aspirated before injection or aqueous paracentesis can be performed after injection to reduce the elevation in IOP produced by the increase in volume of globe contents.

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SECTION
IX REPRODUCTIVE SYSTEM

Jörg A. Auer

CHAPTER
59 Testis

James Schumacher

ANATOMY AND PHYSIOLOGY

Scrotum

The scrotum of the horse is a prepubic diverticulum of skin that contains the testes, their associated ducts, and the distal portion of the spermatic cords.[1] It is divided on the midline by the scrotal raphe, which is continuous with the raphe of the prepuce, penis, and perineum.[1,2] The skin of the scrotum is thin, is sparsely covered with fine hair, and contains an unusually high number of sweat glands.

Intimately adherent to the scrotal skin is a layer of connective tissue and involuntary muscle, the *tunica dartos.*[1,3,4] This muscle relaxes with heat and contracts with cold to regulate testicular temperature, thus varying the size of the scrotum.[1] At the median plane, the tunica dartos sends a sagittal septum into the scrotal sac, dividing the scrotum into right and left pouches, each of which contains a testis.[1,3]

The vascular supply to the scrotum originates from the external pudendal vessels.[1,3,4] The nervous supply descends into the scrotum on the outer surface of the tunica vaginalis via the genitofemoral nerve.[1,3] Lymphatic vessels from the scrotum drain to the superficial inguinal lymph nodes.[4]

Epididymis and Testis

The testes produce spermatozoa and hormones.[5] The hormones govern spermatogenesis, sexual differentiation, secondary sexual characteristics, and libido.[6] The testes are oval and, depending on the age and breed of the horse, weigh between 150 and 300 g each.[1,2] The left testis is usually larger, suspended more ventrad, and situated further caudad than the right testis.[1] The long axis of the testis is nearly horizontal but raised slightly craniad.[1-4] The epididymis attaches to the dorsolateral border of the testis (i.e., the attached or epididymal border) and overlaps the lateral surface slightly (Figure 59-1), forming a testicular bursa between the testis and epididymis. Opposite the attached border is the free border, and opposite the lateral surface is the medial surface.

The epididymis is an elaborately coiled tube consisting of three parts: the head (caput), body (corpus), and tail (cauda).[1] The head lies at the cranial pole of the testis, and the body is positioned on the lateral surface of the testis. The tail projects beyond the caudal border of the testis, wraps around to the medial side, and continues proximally as the ductus deferens.[1,3,4,7] The epididymis receives immature sperm, which enter it through the efferent ducts of the testis. At ejaculation, peristaltic contractions of the epididymis force sperm into the ductus deferens.

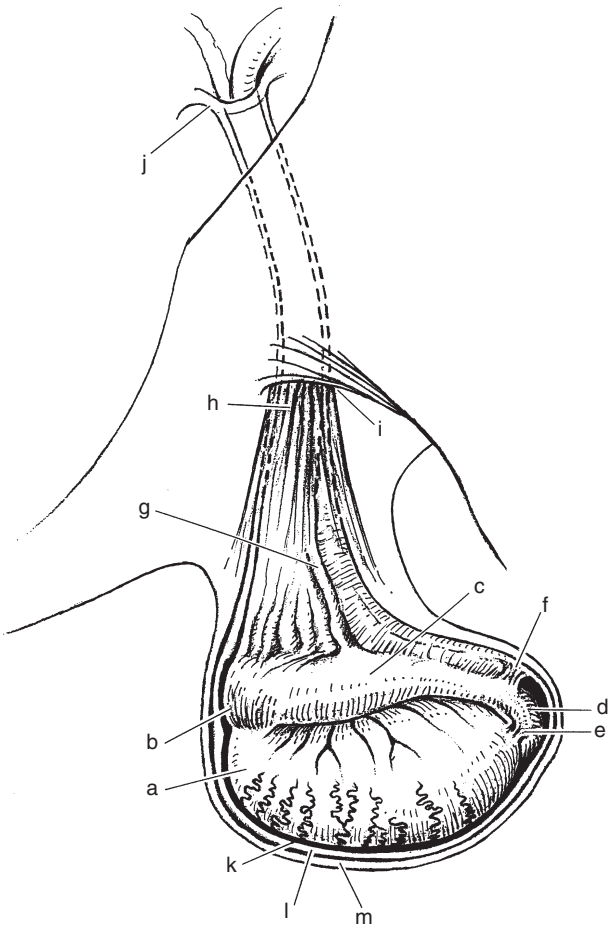

Figure 59-1. Right testis and epididymis of a stallion, lateral aspect. *a,* Testis; *b,* head of epididymis; *c,* body of epididymis; *d,* tail of epididymis; *e,* proper ligamentum testis; *f,* ligament of the tail of the epididymis; *g,* spermatic cord; *h,* cremaster muscle; *i,* external inguinal ring; *j,* vaginal ring; *k,* visceral tunic; *l,* vaginal cavity; *m,* parietal tunic.

The testis is covered by the *tunica albuginea,* a tough, inelastic capsule of dense fibrous tissue.[1,4] Trabeculae and septa of connective tissue from the tunica albuginea penetrate the testis to subdivide the parenchyma into lobules. Each lobule consists of

convoluted seminiferous tubules lined by spermatogonia, from which spermatozoa arise, and *Sertoli cells* (also called sustentacular cells), which supply mechanical and nutritive support for the developing spermatozoa.[1,5] The primary spermatogonia are attached to the basement membrane of the tubule, and the more mature spermatozoa are pushed toward the lumen. Follicle-stimulating hormone (FSH) produced by the hypophysis stimulates spermatogenesis. The seminiferous tubules make up over 70% of the parenchyma of the testis.[8]

Located between the seminiferous tubules are interstitial cells, known as the *cells of Leydig*, which produce androgens in response to interstitial cell–stimulating hormone (which is similar or identical to luteinizing hormone), produced by the hypophysis.[5] The testis of the stallion produces an unusually high concentration of estrogen in comparison to other domestic species, and the source of this estrogen is the Leydig cells.[9]

The seminiferous tubules converge to form the tubules of the rete testis, which pierce the tunica albuginea at the dorsocranial border of the testis to become the efferent ductules.[3] The dozen or more efferent ductules unite in the head of the epididymis to form a single coiled tube, over 70 m long, that continues to become the body and tail of the epididymis and ascends with the testicular vessels as the ductus deferens.[1,2] Spermatozoa mature in the epididymal duct, and the duct's tortuous course allows the storage of a large number of spermatozoa.[5]

Inguinal Canal

The inguinal canal is an oblique passage in the abdominal wall through which traverse the spermatic cord, genitofemoral nerve, external pudendal vasculature, and the efferent lymphatic vessels from the superficial inguinal lymph nodes.[10] The internal opening of the inguinal canal, the deep inguinal ring, is a dilatable slit, about 16 cm long in the average-sized horse, bordered cranially by the caudal edge of the internal abdominal oblique muscle, ventromedially by the rectus abdominis muscle and prepubic tendon, and caudally by the inguinal ligament.[1,3] The external opening of the inguinal canal, the superficial inguinal ring, is a 10- to 12-cm-long slit in the external abdominal oblique muscle.[1,10]

The medial border of the superficial inguinal ring lies directly below the medial border of the deep inguinal ring, making the medial wall of the inguinal canal very short.[3] The superficial inguinal ring is directed craniolaterally, and the deep inguinal ring is directed dorsolaterally, making the lateral angles of the rings widely divergent. The length of the canal in a medium-sized horse is about 15 cm when measured along the spermatic cord.[7] The wall of the inguinal canal is lined by peritoneum, which forms the tunica vaginalis.[10]

Tunics

The *tunica vaginalis,* derived from the abdominal peritoneum, continues through the inguinal canal to line the interior of the scrotum and envelops the testis and its associated ducts and spermatic cord[1,11] (Figure 59-2). The tunica vaginalis consists of a visceral tunic (tunica vaginalis propria) and a parietal tunic (tunica vaginalis communis). The visceral tunic adheres firmly to the tunica albuginea and covers the testis and associated ducts, except at the dorsal border of the testis, where vessels and the epididymis enter or leave the testis.[1,3,4,11] The parietal tunic is continuous with the parietal peritoneum of the abdomen at the deep inguinal ring and forms a sac that lines the scrotal cavity.[1,4,11] This sac is referred to as the *vaginal process* or *vaginal sac,* and its opening at the deep inguinal ring is the *vaginal ring.* The vaginal ring is found, during examination *per rectum* in the average-sized horse, 10 to 12 cm abaxial to the linea alba and 6 to 8 cm cranial to the iliopectineal eminence.[1] The diverticulum of the peritoneal cavity between the parietal and visceral tunics is the vaginal cavity.[1,11] This cavity communicates with the peritoneal cavity and normally contains only a small quantity of serous fluid, which serves as a lubricant to facilitate movement of the testis. The right and left vaginal cavities do not communicate.

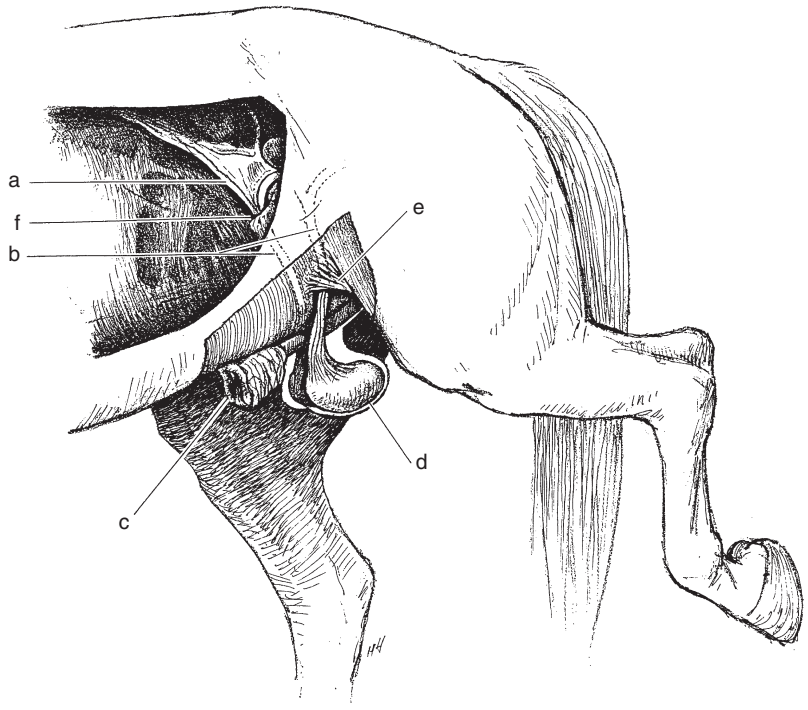

Figure 59-2. Graphic representation of the reproductive tract of the stallion, left-sided view. *a,* Testicular artery and vein; *b,* spermatic cords with their inguinal canal; *c,* external lamina of the prepuce; *d,* testis within scrotum; *e,* external inguinal ring; *f,* internal inguinal (vaginal) ring.

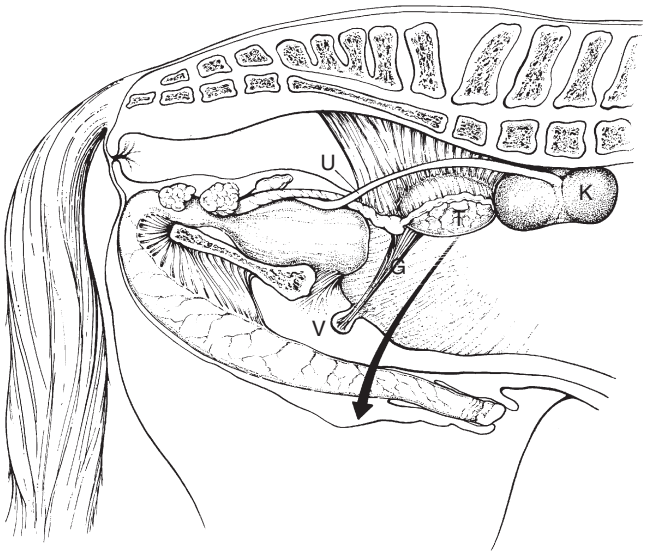

Figure 59-3. Descent of the testis. *G,* Gubernaculum; *K,* kidney; *T,* testis; *U,* ureter; *V,* vaginal ring.

Gubernaculum

The gubernaculum testis is a fetal, retroperitoneal, mesenchymal cord that extends between the caudal pole of the fetal testis and the scrotum and guides the fetal testis in its descent from the ventral surface of the kidney to its final position in the scrotum[10] (Figure 59-3). The gubernaculum testis can be divided into three parts: the cranial part, which lies between the testis and the epididymis; the middle part, which lies between the epididymis and the point at which the gubernaculum penetrates the abdominal wall at the inguinal rings; and the caudal or scrotal part, which extends from the abdominal wall at the site of the future inguinal canal to the scrotum. The cranial part becomes the *proper ligament of the testis* and connects the tail of the epididymis to the testis.[3,4,10] The middle part becomes the *ligament of the tail of the epididymis* (also known as the caudal ligament of the epididymis) and connects the tail of the epididymis to the parietal tunic. The caudal part becomes the *scrotal ligament* and connects the parietal tunic to the bottom the scrotum. The scrotal ligament of an abdominally retained testis is sometimes referred to as the inguinal extension of the gubernaculum testis.[12] These ligaments may be abnormally long if the testis fails to descend.[13] Improper function of the gubernaculum may result in abdominal or inguinal retention of a testis.[14,15]

Descent of the Testis

The gonads arise retroperitoneally from the gonadal ridges caudal to the kidney and differentiate into testes or ovaries at 40 days of gestation.[16,17] By gestational day 55, the testis is suspended cranially by the cephalic (suspensory) ligament and dorsally by the mesorchium. The interstitial cells begin multiplying at 6 weeks of gestation, causing the testis to hypertrophy until, at 5 months of gestation, the testis is nearly as large as that of a mature stallion and contacts both the kidney and the deep inguinal ring. This phase of testicular hypertrophy corresponds with a period of high serum concentration of estrogen in the mare.[18]

The gubernaculum extends from the caudal end of the testis to the inguinal canal and ends in a knoblike expansion between the differentiating internal and external oblique abdominal muscles.[15,16] On gestational day 45, the peritoneum invades the subperitoneal tissue around this extra-abdominal expansion of the gubernaculum to form the vaginal process. At about 5 months of gestation, the cephalic ligament atrophies, and the epididymis descends into the vaginal process while the testis remains in the abdomen.[16,17]

At 8½ months of gestation, the gubernaculum begins to shorten. Simultaneously, the testis regresses in size, mainly because of loss of interstitial cells, until during the last month of gestation when it becomes one tenth its former size.[16] The epididymis and subperitoneal gubernaculum expand in diameter, thus dilating the vaginal ring and inguinal canal. This, along with an increase in intra-abdominal pressure, allows the testis to pass into the inguinal canal at between 270 and 300 days of gestation.[7,15-17] The mass of extra-abdominal gubernaculum prevents the testis from moving directly into the scrotum so that, at birth, most testes lie in the inguinal canal. This extra-abdominal portion of gubernaculum can be quite large at birth and easily mistaken for a testis. The vaginal rings contract to approximately 1 cm in diameter during the first few weeks of neonatal life and become so fibrous that the testis cannot be forced through from either direction.[16]

Spermatic Cord

The spermatic cord consists of structures carried ventrad by the testis in its migration through the inguinal canal from the abdomen to the scrotum.[1,10] The spermatic cord begins at the deep inguinal ring, where its constituent parts converge. It extends obliquely and ventrad through the inguinal canal, passes beside the penis, and terminates at the dorsal border of the testis.[1] The structures that make up the cord are the tunica vaginalis; the blood vessels, nerves, and lymphatics of the testis; and the ductus deferens.[1,10,11] Technically, the cremaster muscle is not included as a component of the cord because it lies external to the parietal tunic.[4,14] The neurovascular components of the cord are enclosed in the mesorchium, a fold of serous membrane formed by invagination of the parietal tunic along its caudal wall (Figure 59-4). The mesorchium extends from the origin of the testicular vessels to the testis. The mesoductus deferens is a caudomedially located fold of the parietal tunic containing the ductus deferens and the deferential vessels.[1,3,4] The mesoductus deferens is continuous with the mesorchium. The mesofuniculum is the thin part of the mesorchium between the mesoductus deferens and the parietal tunic.

The *ductus deferens* is a direct continuation of the epididymal duct.[1] It stores spermatozoa, and during ejaculation, it propels spermatozoa from the epididymis to the pelvic urethra.[5] The ductus deferens is somewhat convoluted near the epididymis but straightens as it continues dorsad.[1,2] At the vaginal ring, it separates from other constituents of the spermatic cord and turns caudomedially into the pelvic cavity, where it lies in the genital fold, a horizontal sheet of peritoneum lying between the bladder and the rectum. Dorsal to the bladder, the ductus deferens increases in diameter and forms the ampulla, which in the stallion, is 15 to 20 cm long and about 1 to 2 cm in diameter. The ampulla is not nearly as pronounced in geldings. Beyond the ampulla, the ductus deferens narrows and opens with the ducts of the seminal vesicles at the ejaculatory orifices

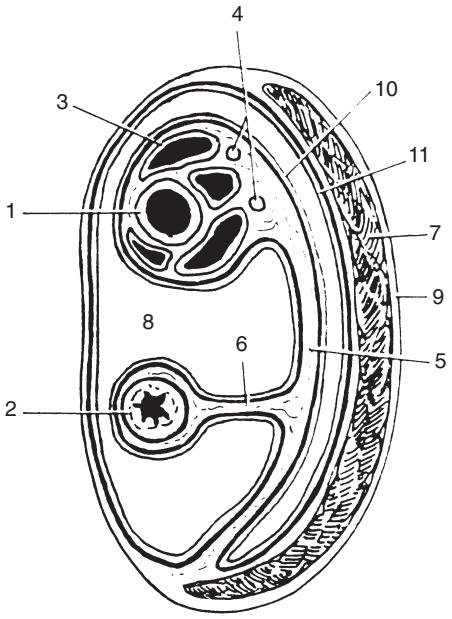

Figure 59-4. Transverse section of the spermatic cord. *1,* Testicular artery; *2,* ductus deferens; *3,* pampiniform plexus; *4,* testicular nerves and lymphatic vessels; *5,* mesorchium; *6,* mesoductus; *7,* cremaster muscle; *8,* vaginal cavity; *9,* spermatic fascia; *10,* visceral layer of vaginal tunic; *11,* parietal layer of vaginal tunic.

on the colliculus seminalis, a protuberance on the dorsal wall of the urethra, located about 5 cm caudal to the internal urethral orifice. The ductus deferens is supplied by the deferential artery, a branch of the umbilical artery.[4]

The *testicular artery*, a branch of the abdominal aorta, descends in the cranial part of the cord.[1] As the artery descends in the inguinal canal, it becomes greatly convoluted. At the cranial pole of the testis, it gives off the epididymal artery, then continues caudad with the epididymis, turns ventrad around the caudal pole of the testis, and travels craniad on the free border, sending off branches to the medial and lateral surfaces.[1,19] The testicular artery and its branch, the epididymal artery, constitute most of the vascular supply to the testis.[20] The cremasteric artery and the deferential artery also contribute to the blood supply to the testis. These three arteries are connected by numerous anastomosing vessels.

Testicular veins ascend from the dorsal border of the testis. Small veins coalesce into larger veins about 10 cm proximal to the testis.[21] These larger veins contain valves that prevent retrograde flow of blood. The veins divide and convolute to form the *pampiniform plexus,* which lies around the coiled testicular artery and forms the bulk of the spermatic cord.[1,3,4,7] The veins unite within the abdomen to become the testicular vein. The right testicular vein usually joins the caudal vena cava, and the left testicular vein usually joins the left renal vein.[1] The lymphatic vessels of the testis and epididymis ascend directly to the medial iliac and lumbar lymph nodes.[1,3,4] A plexus of autonomic and visceral sensory nerves accompany the blood vessels to supply the testis and epididymis.

The *cremaster muscle*, a slip of the internal abdominal oblique muscle, lies on the caudolateral surface of the parietal tunic and attaches to this tunic at the caudal pole of the testis.[1,10] Its blood supply derives from the cremasteric artery.[3,4] Contraction of the cremaster muscle retracts the testis.[1]

PATHOPHYSIOLOGY

Congenital Monorchidism

Horses possessing one descended testis and one that has failed to descend are sometimes described incorrectly as being monorchids.[21] The term *monorchidism* should be reserved to describe the rare situation of complete absence of one testis. Horses in possession of only one testis usually reach this state from failure of a surgeon to remove both testes at the time of castration, but the condition can also occur from unilateral testicular agenesis or when a vascular insult, presumably caused by torsion of the spermatic cord, causes an abdominally located testis to degenerate.[22-27]

Monorchidism of an apparently congenital nature is usually caused by testicular degeneration rather than by atresia,[25] and the vascular insult responsible for the degeneration may occur in the undescended testis before birth or in an abdominal testis after birth.[25,27,28] Torsion of the spermatic cord of the abdominal testis may cause the horse to show moderate to severe signs of abdominal pain,[29] but some similarly affected horses do not demonstrate any signs of pain.[28]

Congenital absence of testicular tissue or absence of testicular tissue caused by testicular degeneration is discovered when, during inguinal or abdominal exploration to locate a cryptorchid testis, the tail of the epididymis is found attached to both the ligament of the tail of the epididymis and the ductus deferens, but the testis is absent.[25,27] More rarely, the epididymis or even the ductus deferens may also be missing. Monorchidism can be confirmed with a human chorionic gonadotropin (hCG)-stimulation test after the contralateral testis is removed and the horse has recovered from surgery, but determining whether monorchidism was caused by degeneration, atresia, or surgical excision of a testis may be difficult.[25] The presence of a vaginal process suggests that monorchidism was acquired, and identifying remnants of the cremaster muscle, ductus deferens, and testicular vessels within the inguinal canal suggests that the testis was excised.

Cryptorchidism

Cryptorchidism is an anomaly of testicular position and is the most prevalent, nonlethal developmental defect of the horse.[16,30,31] Abnormal testicular location occurs when one testis or both testes fail to descend completely from the fetal position in the sublumbar area through the inguinal canal into the scrotum.[16] The term *cryptorchid* refers to the nondescended testis (by extension, a horse with this condition is also termed a cryptorchid), and removal of an undescended testis is sometimes referred to as cryptorchidectomy. Colloquial terms for the cryptorchid include *rig, ridgling,* or *original.*[32-34] If a testis and its epididymis are both abdominally retained, the horse is called a complete abdominal cryptorchid. If the epididymis, but not the testis, has descended through the vaginal ring, the horse is called a partial or incomplete abdominal cryptorchid.[33-35] An inguinal cryptorchid or "high flanker" is a horse with a testis retained within the inguinal canal.[32-35]

In boys testes that usually reside in the inguinal region, because of retraction of the cremaster muscle, but that can be manipulated into the scrotum are termed *retractile.*[36] The retractile testis resides in the inguinal region until puberty, when testicular growth causes the testis to reside permanently within the scrotum. The retractile testis of boys is considered to be a

normal variant[36] and is distinguished from a truly inguinal retained testis. Although retractile testes of horses have not been described, at least some inguinally located testes of young horses that can be retracted into the scrotum may warrant being distinguished from truly retained testes and classified as retractile.

Cryptorchidism of stallions is of considerable economic significance, because the seminiferous tubules of the cryptorchid testis are rudimentarily developed and incapable of producing sperm.[16] Unilateral cryptorchids are usually fertile but have reduced production of sperm,[37] and horses affected with bilateral testicular retention are sterile.[16] The androgen-producing cells of Leydig of a cryptorchid testis are functional and produce testosterone, although at reduced concentrations, so that the cryptorchid horse exhibits sexual behavior. Exposure of the testis to the high temperature of the abdomen or inguinal canal appears to be responsible for hypoplasia of the seminiferous tubules and results in a small, soft testis. Location of the testis at sites other than the scrotum complicates removal of the testis. Because malfunction of descent may be inherited, cryptorchid horses are generally considered genetically unsound.[30] For this reason, registration of cryptorchid stallions is disallowed by some breed associations.

Undescended testes of humans may be at risk of developing neoplasia,[38] and the same may be true of undescended testes of horses, although a direct link between equine testicular neoplasia and cryptorchidism has not been proved, perhaps because of the few reports of testicular neoplasia in stallions. Testicular neoplasia of humans may be the result of progressive degeneration of germ cells caused by the abnormal temperature to which the cryptorchid testis is subjected.[39] Factors other than position of the testis may be responsible for the increased incidence of malignancy, raising the question of whether the testis develops neoplasia because it is undescended or whether some abnormality of the testis (resulting in neoplasia) is responsible for failure of the testis to descend.

An epidemiologic survey investigating the prevalence of equine cryptorchidism found that approximately one of six (i.e., about 17%), 2- to 3-year-old colts presented to a veterinary medical teaching hospital was cryptorchid.[30] Of these cryptorchid testicles, only 14 had tumorous derangement, representing 0.3% of the total cryptorchid testes evaluated.[30] Groups most commonly represented were Percherons, American Saddlebreds, American Quarter Horses, ponies, and crossbred horses. Thoroughbreds had the lowest prevalence of cryptorchidism.

Etiology

Improper function of the gubernaculum has been postulated to cause failure of testicular descent.[15,16] Failure of the subperitoneal portion of the gubernaculum to enlarge may result in failure of the vaginal ring to expand sufficiently to allow the testis to pass. Excessive enlargement of the subperitoneal portion of the gubernaculum, followed by its failure to regress adequately, may also inhibit passage of the testis into the inguinal canal.[14]

Events that lead to cryptorchidism in horses have not been determined, but a deficiency in production of gonadotropin by the pituitary gland or the placenta has been shown to result in cryptorchidism in boys, by leading to insufficient production of androgens by the descending testes.[40] These androgens are responsible for development of the vaginal process, testicular vessels, and vas deferens and for changes in the gubernaculum, all of which are necessary for the descent of the testes into the scrotum. Rather than acting directly on the gubernaculum, androgens likely act primarily on the cell body of the genitofemoral nerve in the spinal cord.[41] The genitofemoral nerve, which innervates the gubernaculum, causes the gubernaculum to differentiate, creating a potential space into which the testis descends under the influence of abdominal pressure.[38] Androgens thus act on the gubernaculum indirectly and probably must do so at a specific time during gestation.

The testis may also fail to descend if it does not regress to a sufficiently small size to traverse the vaginal ring.[16,18] A testicular cyst or teratoma or persistence of the suspensory ligament of the testis during gestation may account for failure of testicular descent.[34,42] The complexity of the process of testicular descent suggests that the cause of failure of descent is multifactorial.

Although genetic studies of cryptorchid horses indicate that in many instances the condition is hereditary, no definitive studies have supported a plausible genetic mechanism. The effects of maternal environment and mechanical factors (e.g., dystocia) in the etiology of cryptorchidism have not been examined.[30] The conflicting observations of numerous studies of genetic transmission of equine cryptorchidism suggest that the mechanism of inheritance is complex and most likely involves several genes.[43] One of the most frequently cited investigations is a thesis published in 1943 on the genetic nature of cryptorchidism of several species of domestic animals. The Technical Development Committee of Great Britain (1954), citing this thesis, concluded that equine cryptorchidism is transmitted by a dominant factor.[44] Other researchers, citing the same thesis, reported that the investigation had determined that equine cryptorchidism is caused by a simple autosomal recessive gene.[45] A German study of 21 cryptorchid stallions postulated that at least two genetic factors are involved, one of which is located on the sex chromosomes.[45] Another German study, however, concluded that equine cryptorchidism is transmitted as an autosomal dominant gene.[45]

Researchers at Texas A&M University found that 56 of 58 colts sired by a cryptorchid American Quarter Horse stallion had both scrotal testes.[46] The other two colts were unavailable for assessment of testicular location. This stallion was unlikely to have carried a dominant autosomal gene for cryptorchidism, unless the gene had low penetrance. If equine cryptorchidism is caused by an autosomal recessive gene, the frequency of the recessive gene in the dams of these 56 colts would have to have been nearly zero.

Cryptorchidism is often a feature of intersexuality.[47,48] For example, the testes of the male pseudohermaphrodite are often located within the inguinal canals or the abdomen.[6,47,49] Cryptorchidism may also be a manifestation of an abnormal karyotype.[47,48] Horses that are cryptorchid because of an aberration of genetic sex, however, are usually easily identified by their intersexual phenotypes.[6,47] The prevalence of horses that are cryptorchid because of intersexuality appears to be quite low; in a study of 5018 cryptorchid horses, only 9 were intersexes.[30]

Incidence

Retrospective studies of large numbers of cryptorchid horses indicate that failure of right and left testicular descent occurs with nearly equal frequency.[50-52] Right-sided testicular retention

predominates in young cryptorchid ponies, but the incidence of right-to-left retention decreases as the ponies age.[51] The decrease in incidence of right testicular retention is probably the result of descent of the right testis from an inguinal location.

A study of 350 cryptorchid horses and another of 205 cryptorchid horses found abdominal testicular retention to be more common than inguinal testicular retention.[50,52] Another study of 500 cryptorchid horses, however, found inguinal cryptorchidism to be more common than abdominal cryptorchidism.[51]

The study of 350 cryptorchid horses found that 75% of left undescended testes were located within the abdomen, whereas only 42% of right undescended testes were retained abdominally.[52] The reason for this difference may be that the right testis is smaller than the left during the stage of testicular regression.[17] Consequently, if the gubernaculum fails to expand sufficiently to dilate the vaginal ring, or if the testes incompletely regress in size, the larger left testis is more likely to be incapable of traversing the vaginal ring. Because this difference in testicular size remains after birth, the smaller right testis is more apt to be inguinally retained. Increase in testicular size with the onset of puberty may cause an inguinally retained testis to descend into the scrotum.[50,51]

In one study, 14% of cryptorchid horses were bilaterally affected, and most of these (60%) had abdominal testicular retention.[52] Another study found a 9% incidence of bilateral testicular retention among cryptorchid horses, but in that study, most bilaterally retained testes (67%) were inguinally located.[50] The incidence of cryptorchid horses with one inguinally located testis and one abdominally located testis was quite low in both studies (i.e., when both testes of a horse had failed to descend into the scrotum, the testes were usually either both inguinal or both abdominal).

Inguinal Herniation and Rupture

Inguinal herniation occurs when intestine, usually a portion of the ileum or a loop of the distal portion of jejunum, protrudes through the vaginal ring into the inguinal canal. If the contents extend into the scrotum, the condition is sometimes referred to as scrotal herniation (Figure 59-5), but the terms are used interchangeably.[53,54] Inguinal hernias are sometimes called *indirect hernias,* a term borrowed from descriptions of the comparable condition in humans (for more information on hernias please review Chapter 39).[53]

A *ruptured inguinal hernia* occurs when intestine protrudes through the vaginal ring and then passes through a rent in the parietal tunic and scrotal fascia so that it lies subcutaneously in the inguinal or scrotal region[55] (Figure 59-6). The term *inguinal rupture* describes a similar condition that occurs when intestine protrudes through a rent in the peritoneum and transverse fascia adjacent to the vaginal ring, causing intestine to reside subcutaneously beside the vaginal process[53] (Figure 59-7). Inguinal ruptures have been referred to as *direct hernias,*[56] but this term is derived from descriptions of hernias in humans and inaccurately describes the condition in horses.[53] In humans, the transverse fascia adjacent to the vaginal ring becomes weakened and protrudes, forming a peritoneum-lined sac. In horses, the integrity of the tissue adjacent to the vaginal ring is disturbed, and the intestine passing through the rent is not surrounded by peritoneum. The purported direct hernias of horses are actually ruptures, not hernias.[53,57]

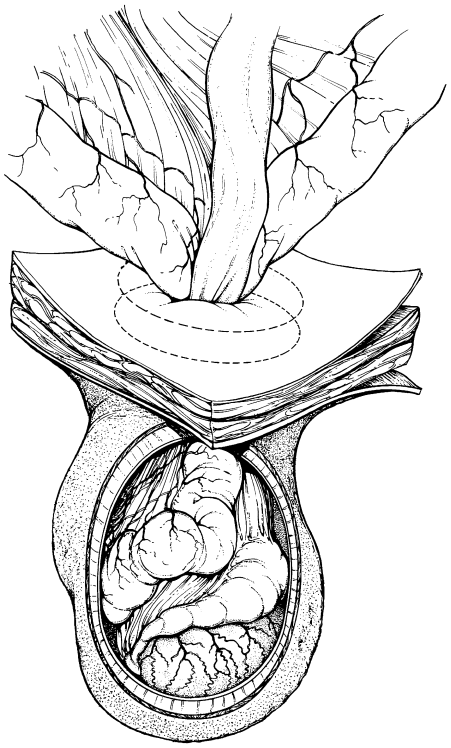

Figure 59-5. Inguinal hernia. Intestine protrudes through the vaginal ring into the inguinal canal or scrotum. The intestine lies within the vaginal cavity.

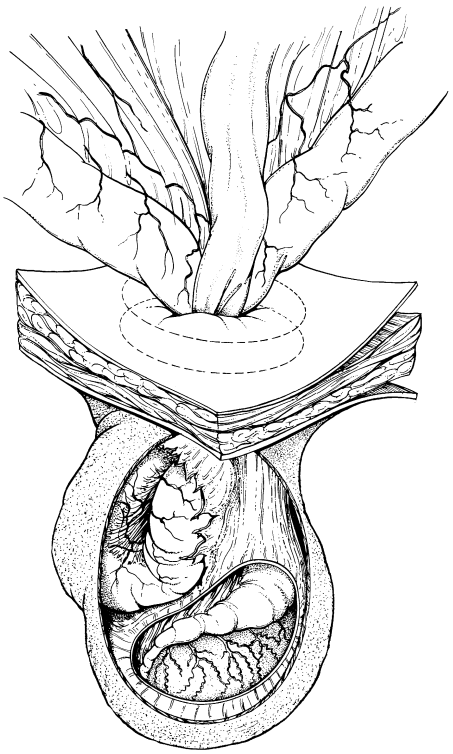

Figure 59-6. Ruptured inguinal hernia. Intestine protrudes through the vaginal ring and passes through a rent in the parietal tunic and scrotal fascia so that it lies subcutaneously in the inguinal or scrotal region.

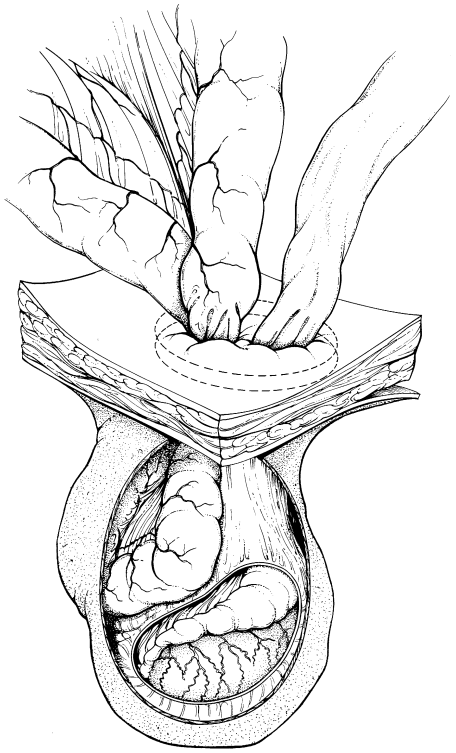

Figure 59-7. Inguinal rupture. Intestine protrudes through a rent in the peritoneum and transverse fascia outside the vaginal sac but adjacent to the vaginal ring.

Etiology

Inguinal herniation and rupture occur almost exclusively in stallions,[54,55,58] but inguinal herniation has been reported to have occurred in a few geldings[54,59,60] and in a mare.[61] Geldings rarely develop an inguinal hernia, because the vaginal rings decrease in size soon after castration.

Inguinal hernias in foals are congenital, and most are hereditary in origin.[6] The left inguinal canal is most often involved.[62] Congenital inguinal herniation may be caused by excessive outgrowth of the extra-abdominal part of the gubernaculum, which results in a vaginal process with an unusually wide neck.[7,15] Congenital inguinal hernias may occur unilaterally or bilaterally, are usually reducible, cause no strangulation, and spontaneously resolve by the time the foal is 3 to 6 months old.[53,60] Longstanding congenital inguinal hernias, however, may lead to testicular atrophy.[60]

Inguinal hernias occurring in adult horses are usually acquired and can occur during exercise or copulation, either of which may contribute to herniation by altering the anatomy of the inguinal canal and increasing abdominal pressure.[54] The incidence of acquired inguinal herniation in Standardbreds, draft breeds, and Andalusian horses is reported to be higher than that of the general equine population.[63-65] Other breeds with anecdotal evidence of increased incidence of inguinal herniation are Tennessee Walking Horses and American Saddlebreds.

An acquired inguinal hernia of an adult horse commonly results in strangulation of intestine and represents a surgical emergency.[54] Small intestine (jejunum and ileum) is the most commonly herniated viscus, but inguinal herniation of small colon, large colon, omentum, and the urinary bladder have been reported.[66-68] Clinical signs are usually referable to obstruction of the small intestine and include recovery of nasogastric reflux after nasogastric intubation and rectally palpable distended small intestine.[54,58] Strangulation of herniated intestine is caused by constriction of the intestine by the vaginal ring.[58] An acquired inguinal hernia usually causes obstruction of the vasculature of the spermatic cord, which leads to edema of the external genitalia and testicular degeneration or necrosis.

Ruptured inguinal herniation appears to occur much more commonly in foals than in adults, and those that occur in foals may be caused by abdominal compression during parturition.[55,69] Inguinal ruptures are probably traumatic in origin, but the traumatic event is not always obvious. With an inguinal rupture or a ruptured inguinal hernia, the intestine that protrudes through the rent may become strangulated or may adhere to subcutaneous tissue, and the separation of the skin from its blood supply may reduce the viability of the skin.

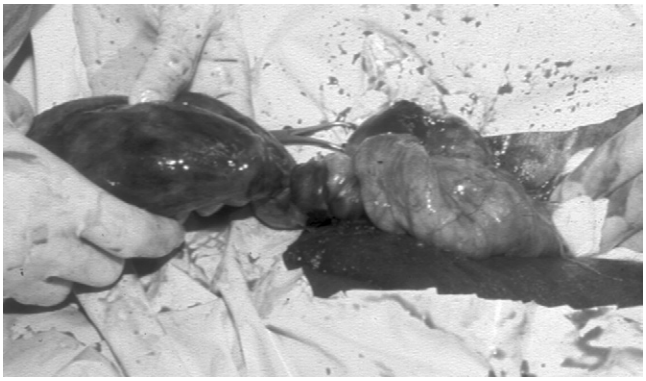

Figure 59-8. Torsion of the spermatic cord has caused necrosis of the testis and the portion of the cord distal to the torsion.

Torsion of the Spermatic Cord

Torsion of the spermatic cord occurs when the attached testis rotates on its vertical axis.[70] The condition is sometimes improperly referred to as testicular torsion. Torsion of the spermatic cord causes the testicular vessels to twist, producing venous and often arterial obstruction, which leads to testicular congestion and edema in mild cases and complete testicular infarction in severe cases.[28,70,71] (Figure 59-8). Clinically significant torsion of the spermatic cord in the horse occurs rarely, and few reports of the condition can be found.[28,72-75] This condition is reported to occur most commonly in trotting Standardbreds.[6]

Torsion of the spermatic cord of 360 degrees or more is accompanied by signs of acute pain, which may resemble signs of colic, and enlargement of the affected testis and cord.[74] The condition represents a surgical emergency that usually requires removal of the affected testis, because the testis is rarely salvageable. Torsion of the spermatic cord of an abdominally located testis may cause the affected horse to show mild signs of colic pain,[29] but acute torsion of the spermatic cord has been recognized during cryptorchidectomy of several apparently normal horses that apparently displayed no signs of colic.[28] The incidence of torsion of the spermatic cord of undescended testes of humans is probably higher than the incidence of torsion of the descended testes,[76] and the same may be true of horses. Because torsion of the spermatic cord of an abdominal testis often goes

unrecognized in horses, the condition may account for some reports of horses with congenital monorchidism.

Evidently, torsion of the spermatic cord of a scrotal testis of the horse less than 180 degrees causes no clinical abnormalities and is considered an incidental finding.[74] Evaluation of testicular blood flow of stallions using color Doppler ultrasonography, however, shows that 180-degree torsion of a spermatic cord may cause retrograde blood flow and suggests that torsion of this degree may have a detrimental effect on testicular function, even in the absence of clinical signs of substantial vascular compromise.[77]

Torsion of the spermatic cord of 180 degrees is relatively easy to diagnose because it causes the tail of the epididymis to reside in the cranial portion of the scrotum, rather than in its normal caudolateral position. Acute torsion of the spermatic cord may be difficult to determine when the spermatic cord is twisted 360 or 720 degrees, because the tail of the epididymis resides in its normal caudolateral position in the scrotum. Torsion of the spermatic cord of 360 degrees or more may be accompanied by so much scrotal, testicular, and epididymal swelling that the epididymis cannot be palpated.

Torsion of the spermatic cord of the descended testis of horses apparently occurs intravaginally (i.e., within the vaginal process).[74,75] Horses may be predisposed to testicular torsion because the testis resides horizontally, rather than vertically, in the scrotum.[75] Torsion may occur because the ligament of the tail of the epididymis (caudal ligament of the epididymis) or the proper ligament of the testis is abnormally long. The ligaments of the contralateral testis may also be abnormally long, making that spermatic cord also prone to torsion.[75] To prevent torsion of the contralateral spermatic cord, the contralateral testis can be fixed permanently in position (orchidopexy or orchiopexy) by placing a nonabsorbable suture through the tunica albuginea and the dartos tissue at the cranial and caudal poles of the testis.

The spermatic cord of an abdominal testis may be more apt to develop torsion than is the spermatic cord of a descended testis, because the proper ligament of the testis and sometimes the ligament of the tail of the epididymis of an abdominal testis are abnormally long.[23,28,37] None of 350 cryptorchid horses in one study, however, developed torsion of the spermatic cord.[52]

Hydrocele (Vaginocele) and Hematocele

A hydrocele, or vaginocele, is an abnormal collection of serous fluid between the visceral and parietal layers of the tunica vaginalis.[78,79] Hydroceles form when fluid normally secreted by the vaginal tunic is produced at a rate greater than that at which it can be absorbed by the lymphatic vessels and veins of the spermatic cord.[80] The cause of the discrepancy between the rate of production and the rate of resorption of the fluid is usually idiopathic.[78] Hydroceles may accompany testicular neoplasia or orchitis, and because the vaginal and peritoneal cavities communicate, some hydroceles could be caused when fluid present in excess in the abdominal cavity enters the vaginal cavity.[81] Migration of parasites through the vaginal cavity, 180-degree torsion of the spermatic cord, trauma, and a hot climate in conjunction with lack of exercise have been implicated as causes of hydroceles.[78,82] Hydroceles may develop acutely or insidiously. A hydrocele is generally considered to cause temperature-induced dysfunction of spermatogenesis by insulating the testis and epididymis, but one investigation found they caused no important effect on semen quality.[78] A hydrocele is occasionally a complication of castration, but only when the parietal tunic is retained (i.e., after an open castration).

Palpation of a hydrocele usually causes no apparent discomfort to the horse, and the involved testis is freely moveable and feels as though it resides within a compressible, fluid-filled bag.[81] Anechoic to semi-echoic fluid surrounding the involved testis and epididymis is seen during ultrasonographic examination of the scrotum, and a yellowish, clear fluid is obtained during aspiration of the vaginal cavity. The involved testis of a chronically affected horse may be abnormally small.

The size of a hydrocele may decrease temporarily with exercise, and some hydroceles spontaneously resolve when the affected horse is moved to a cooler environment.[78,81] Fluid usually reaccumulates quickly after aspiration. Treatment of a horse for hydrocele should be aimed at removing the inciting cause. However, the cause can rarely be identified, so the usual treatment of horses affected with persistent unilateral hydrocele is removal of the affected testis and parietal tunic. The testis and tunic should be removed before spermatogenesis of the contralateral testis becomes affected by increased scrotal temperature. Prognosis for fertility is guarded if bilateral hydroceles persist, but some stallions affected with hydrocele retain normal spermatogenesis.[78]

Sclerotherapy, using a 2.5%, 5%, or 10% solution of tetracycline or using polidocanol injected into the vaginal cavity, has been used successfully to treat men affected with a hydrocele,[83,84] but this treatment may also decrease fertility.[85] Sclerotherapy has not been evaluated as a treatment for stallions with a hydrocele.

A *hematocele* may resemble a hydrocele, but it is characterized by a collection of blood within the vaginal cavity.[86,87] Hematoceles are usually caused by testicular or scrotal trauma, but they may occur as an extension of hemoperitoneum.[37] Ultrasonographic examination of the scrotum and its contents and aseptic aspiration of the vaginal cavity may help differentiate other causes of scrotal enlargement, such as hydrocele, from hematocele.

A horse with a hematocele may be treated successfully by aspiration of blood from the vaginal cavity followed by lavage of the cavity with a balanced electrolyte solution, provided that the tunica albuginea is not ruptured.[86] This treatment may minimize the insulating effect of blood and reduce the formation of adhesions between the visceral and parietal layers of the tunica vaginalis. Ultrasonographic examination of the ipsilateral testis may help determine whether the tunica albuginea has been ruptured. If the integrity of the tunic remains uncertain, the testis should be examined surgically, which allows blood to be evacuated from the vaginal cavity. If torn, the tunica albuginea should be sutured, or the testis should be removed. A horse that has developed a hematocele may have also developed a breach in the blood–testis barrier, which may trigger an immune response to the horse's spermatozoa and, in turn, result in infertility.[88]

Varicocele

A varicocele is an abnormally distended and tortuous pampiniform plexus.[79,89] Valvular incompetence of the testicular vein, where it empties into the vena cava or renal vein, may increase hydrostatic pressure in the pampiniform plexus by causing reflux of caval or renal blood into the testicular vein.[89] Varicoceles in

men are associated with a low total sperm count, but motility and morphology of spermatozoa may remain normal. Varicoceles may disturb countercurrent exchange of heat from the arterial to the venous blood, causing temperature-induced dysfunction of spermatogenesis,[79,89] or they may affect spermatogenesis by impairing drainage of blood from the testis, which results in increased scrotal temperature, testicular hypoxia, and elevated testicular pressure.[90] Although varicoceles are known to cause infertility in men and rams, their effect on fertility of horses has not been evaluated, perhaps because varicoceles of stallions are so rarely encountered. Unilateral varicoceles have been noted in some stallions with normal ejaculates.[91] Varicoceles of stallions usually occur unilaterally and, when palpated, do not cause the horse to display signs of pain. The affected spermatic cord has the texture of a "bag of worms," and the neck of the scrotum on the affected side may be wider than usual.

Humans affected with varicocele have been treated by ligation of the venous loops of the pampiniform plexus,[92] but there is no good evidence that treating subfertile men for varicocele enhances the probability of conception.[90] Treatment of stallions with varicoceles is removal of the affected cord and testis, but treatment is not necessary if the varicocele does not affect seminal quality.

Retraction of a Testis into the Inguinal Canal

Rarely, a stallion retracts one or both scrotal testes into the inguinal area, apparently from persistent contraction of the cremaster muscle caused by pain, either from genital trauma or from musculoskeletal pain of the pelvic limbs. The position of the retracted testis inhibits spermatogenesis from that testis, which may lead to infertility. To reestablish fertility or to improve the quality of semen, the retracted testis can be returned to its normal scrotal position by transecting the cremaster muscle.

To transect the cremaster muscle, the horse is anesthetized and placed in dorsal recumbency, and the ipsilateral inguinal region is prepared for aseptic surgery. To expose the cremaster muscle, a 5- to 7-cm skin incision is created over the long axis of the cord. The incision is extended through fascia until the spermatic cord is encountered. A 3-cm section of cremaster muscle, which is attached to the outer caudolateral surface of the parietal tunic, is isolated from the cord and transected with scissors. The fascia and skin are each closed separately. Descent of the testis into the scrotum can be observed when the horse recovers from anesthesia, and results of improvement in spermatogenesis can be observed in about 3 months.

Testicular Neoplasia

The classification of testicular neoplasms of horses is far less extensive than that of humans. Primary testicular tumors are usually divided into germinal and nongerminal types. Germinal neoplasms arise from germ cells of the spermatic epithelium, and nongerminal neoplasms arise from testicular stromal cells. Reported *germinal testicular tumors* of the horse include the seminoma, teratoma, teratocarcinoma, and embryonic carcinoma. *Nongerminal testicular tumors* of the horse are the Sertoli cell and Leydig cell tumors. Secondary tumors (i.e., tumors that are the result of metastases) of the equine testis are extremely rare.[70,93,94]

The prevalence of primary testicular neoplasia in horses is unknown but is probably low, perhaps because most stallions are castrated while young, before neoplasia has had an opportunity to develop,[93,94] and perhaps because testes removed from apparently normal stallions are seldom examined closely for the presence of neoplasia.[95] Consequently, the few acknowledged characteristics of testicular neoplasms of horses have been established by collecting information gathered from a small number of case reports. Characteristics of equine testicular neoplasms, such as hormonal effects, tendency toward malignancy, and relationship to cryptorchidism, have been inferred from characteristics of testicular neoplasms of other species more commonly affected, such as humans and dogs. Characteristics of testicular neoplasms differ among species, so extrapolating characteristics of testicular neoplasms of other species to horses may result in mistaken assumptions.

Testicular enlargement is the primary presenting sign of testicular neoplasia when the neoplastic testis is located scrotally.[96] Usually, the enlargement is insidious. Signs of tenderness during palpation are uncommon but may be a feature of the neoplastic testis.[93,97] The surface of the neoplastic testis may be characterized by multiple, irregular bumps, and the consistency of the testis may be firmer than normal.[97,98] Horses with neoplastic abdominal testes may be presented for examination because of intermittent signs of colic.[96,99] Metastases may cause weight loss or dyspnea. Identifying a tumor is difficult without histologic examination of either the entire testis or tissue obtained by biopsy.[97] Metastatic spread of testicular tumors is rare, but when it occurs, metastases can sometimes be palpated as an enlargement of the sublumbar lymph nodes.[37]

Seminoma

Seminomas arise from the germinal cells of the seminiferous tubules and are the most common testicular tumors of horses.[93,96] These tumors probably do not produce hormones.[37] As in other species, seminomas of the horse seem to appear with greater frequency in cryptorchid testes and are most commonly found in old horses[96-98,100-102]

Although most equine seminomas behave benignly, they have a higher incidence of malignancy than do some of the other types of testicular neoplasms, and they metastasize more frequently.[93,94,96] The relative risk of a seminoma in a human becoming malignant is highest if the testis is abdominal,[103] but no such correlation has been made between the location of a seminoma in the horse and malignancy. All seminomas of horses should be considered potentially malignant, because clear histologic differentiation between benign and malignant seminomas is almost impossible.[104] Abdominal invasion should be suspected if an enlarged spermatic cord can be palpated externally.[93] Thoracic metastases have been reported.[94,101] The sectional surface of a seminoma is lobulated, homogeneous, and white or grayish white. The tumor is soft to moderately firm, and when squeezed, the surface may exude a milky fluid.[37,101]

Sertoli Cell Tumor

The Sertoli cell or sustentacular cell tumor of the horse arises from the nonspermatogenic cells of the seminiferous tubules.[37] Sertoli cell tumors of horses are less frequently encountered than are other types of testicular tumors.[104-106] Their biological behavior in horses is unknown because of the small number of reports, but in dogs and people, these tumors often cause hyperestrogenism and feminization.[70]

Malignant Sertoli cell tumors in dogs are rare,[70] but of the few Sertoli cell tumors of horses that have been reported, several were malignant.[107,108] A malignant Sertoli cell tumor that had metastasized to many organs was found in a descended testis of a horse,[107] and a malignant Sertoli cell tumor that also had metastasized to many organs was found in an abdominal testis of another horse believed previously to be a gelding.[108] Whether cryptorchidism is a factor predisposing to development of the Sertoli cell tumor cannot be determined from the few reports.[93] The sectional surface of a Sertoli cell tumor is firm, white or tan, and homogeneous.[106,107]

Leydig Cell Tumor

The interstitial or Leydig cell tumor is an infrequently reported equine testicular tumor.[93,104,109,110] Although these tumors arise from the androgen-producing cells of the testis, evidence of production of androgenic hormones by these tumors is lacking in horses and other animals.[37,95,104] Because the Leydig cells of the testes of horses secrete a large amount of estrogen, in addition to androgenic hormones, the hormonal effect of Leydig cell tumors in the horse could vary.[9,111] Cryptorchidism may be associated with this neoplasm on the basis of the observation that, in a study of nine stallions affected unilaterally or bilaterally with Leydig cell tumor, the tumors in all but one horse were found in one or both undescended testes.[95]

Most Leydig cell tumors of horses are benign, and metastases are rare.[37] The sectional surface of the tumor is yellow to brown, which may help grossly to differentiate this tumor from other types of testicular tumors.[93,95,112] The demarcation between the tumor and adjacent normal parenchyma is poor.[95]

Teratoma

Teratomas are tumors of multiple tissues whose embryologic origins are different from that of the tissue in which they arise,[104] and evidence indicates that they develop from pluripotential tumor cells capable of giving rise to any type of tissue found in the body.[113] Most teratomas occur in the gonads.[70] Histologic examination of equine testicular teratomas shows that they are benign, slow growing, and composed of mixed, well-differentiated tissues.[114] Derivatives from all three germinal layers, such as bone, cartilage, brain, and respiratory and glandular epithelium, may be present within the testis.[104,115] Occasionally, teeth are found.[22,104] Teratomas that consist primarily of cysts lined by squamous epithelium and contain hair have incorrectly been referred to as dermoids or dermoid cysts. The term *dermoid*, however, should not be used when referring to a teratoma, because unlike dermoids, teratomas display progressive growth.[22]

The teratoma is the second most common form of equine testicular neoplasia.[93,104] It is found in both descended and cryptorchid testes, but some investigators believe it occurs more frequently in the latter.[105,116-118] Others dispute this claim, stating the opposite.[104,119] Failure of descent of a testis, rather than being a predisposing factor in the formation of a teratoma, may more probably be a result of the teratoma itself.[105,117]

Teratocarcinoma and Embryonal Carcinoma

Teratocarcinomas are similar to teratomas but contain undifferentiated embryonic tissue interspersed among the disarranged mix of differentiated tissues.[120,121] Embryonal carcinomas resemble teratocarcinomas but are composed entirely of undifferentiated embryonic tissue.[121] The undifferentiated embryonic tissue is responsible for the malignant properties of teratocarcinomas and embryonal carcinomas. Teratocarcinomas and embryonic carcinomas of the horse are evidently quite rare but appear to be rapidly fatal.

Intersex

Because male and female reproductive organs arise from the same embryonic structures, errors of development may result in a horse that possesses sexual structures common to both sexes.[70] An individual whose sexual identity is confused because of congenital anatomic abnormalities of the genital organs is called an intersex.[6,48,70] An animal's sexual identity can be defined according to its genetic makeup, the type of its gonads, or the morphology of its accessory genitals. A normal animal is the same sex in all three categories, but the intersex differs in one of the categories.

Intersexes can be divided into three main classes: "true" hermaphrodites, female pseudohermaphrodites, and male pseudohermaphrodites. Hermaphrodites are usually defined on the basis of gonadal sex, with true hermaphrodites having both testicular and ovarian tissue.[6,48,122] Pseudohermaphrodites have gonads of only one sex and are classified as male or female, depending on whether the gonads are testes or ovaries.

The true hermaphrodite is much rarer than the pseudohermaphrodite.[6,48,122,123] One gonad of a hermaphrodite may be a testis and the other an ovary, or one or both gonads may consist of both ovarian and testicular tissue (i.e., ovotestes). The hermaphrodite's external as well as internal genitalia usually represent both sexes, but its external genitalia may tend toward either the male or female. Some cases of equine hermaphroditism have been attributed to chimerism resulting from double fertilization or fusion of blastocysts, but other equine hermaphrodites are one genetic sex.[47,123]

Gonads of the pseudohermaphrodite are of one sex, but the external genitalia resemble those of the opposite sex.[6,48,122] The male pseudohermaphrodite is by far the most common intersex of horses and typically has hypoplastic testes within the abdomen or inguinal canal and a penislike structure, which often resembles a clitoris, emerging from a rudimentary prepuce (Figure 59-9). The rudimentary "penis" and prepuce are positioned anywhere on the midline from the perineum to a scrotal or abdominal location.[6,49] Although the phenotypic appearance of a male pseudohermaphrodite is often that of a female, sexual behavior is that of a male.[122] Most equine male pseudohermaphrodites appear to be masculinized, genetic females (64, XX).[6,48,70,122,124] The contradiction between gonadal and genetic sex has not been fully explained.

DIAGNOSTIC PROCEDURES

History and Physical Examination

History pertaining to problems of the testes and related structures may include such information as infertility, history of unilateral or bilateral testicular retention, enlargement in the inguinal or scrotal areas, testicular pain, and changes in testicular size. Knowledge of events surrounding the onset of testicular pain or increase in testicular size, such as occurrence after exercise or copulation, is sometimes helpful in making a diagnosis.

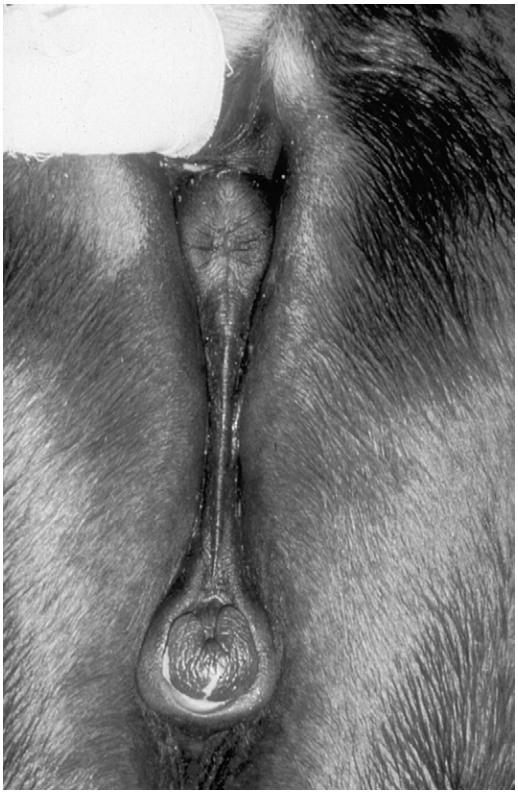

Figure 59-9. Male pseudohermaphrodite. A rudimentary penis and prepuce are positioned in the perineal region. (Courtesy E. Behrens, MV, Ocala, FL.)

Other considerations include previous urogenital surgery, illnesses, and drug therapy.

A physical examination of the testes and associated structures should include inspection and palpation. Notice should be given to the size, shape, texture, and temperature of the testes, and the horse should be observed for evidence of pain when the testes are palpated. Right and left testes should be compared. The scrotum of a normal stallion usually appears asymmetric, because the left testis has a longer spermatic cord and is, therefore, often more pendulous than the right. With cryptorchidism, the scrotum on the involved side is poorly developed. Scrotal scars should be noted, bearing in mind that a scrotal scar may mean only that an orchidectomy was attempted, not that it was accomplished. Occasionally, scrotal edema is noted. This does not usually imply disease related to the genitalia but is more likely a consequence of general retention of fluid associated with disease of other systems.

The testes should feel smooth and elastic. Irregularities in size and texture may indicate trauma, orchitis, torsion of the spermatic cord, thrombosis of the testicular artery, inguinal herniation, or neoplasia. Often, a neoplastic testis is insensitive to digital compression that would cause a normal horse to show signs of pain. Insidious increase in testicular size suggests neoplasia, whereas acute increase in size may suggest torsion of the spermatic cord, inguinal herniation or rupture, or orchitis. As a testicular neoplasm grows, it may seem to replace the entire organ. The neoplastic testis characteristically feels heavier than the normal testis. Because neoplasia may be associated with inguinal or pelvic lymphadenopathy, palpation *per rectum* of internal lymph nodes should, if possible, accompany an examination for testicular enlargement.

The testes of the prepubescent stallion are often quite small and retractile and therefore often difficult to palpate. Before declaring the stallion a cryptorchid, a tranquilizer or sedative should be administered to relax the cremaster muscles and thus facilitate palpation. The medial crus of the superficial inguinal ring is easily palpated if the palm of the hand is turned toward the abdomen.[3] If the palm is turned toward the thigh, the fingers may pass into the inguinal canal without encountering the ring, because the lateral border of the ring is not as readily palpated. Because the average-sized stallion's canal is about 15 cm in depth,[32] only the most ventral part of the inguinal canal can be palpated. An inguinal testis lies with its long axis oriented vertically and is preceded in its descent by the tail of the epididymis. A partial abdominal cryptorchid can be easily mistaken for an inguinal cryptorchid if the epididymis within an everted vaginal process is, by chance, palpated and mistakenly identified as a small testis. The vaginal rings can be palpated only by examination *per rectum*.

The epididymis, located on the dorsolateral surface of the descended testis, should be easily identifiable. The tail of the epididymis should be easily palpated on the caudal pole of the testis. If the tail of the epididymis is located craniad in the scrotum, the spermatic cord has rotated 180 degrees. As mentioned before, torsions of 180 degrees or less probably cause no clinical problems.[74] When palpating the scrotum of a foal to ascertain if testicular descent is complete, the epididymis should be identified so that the remnant of the gubernaculum is not mistaken for a descended testis.

A bulge in the spermatic cord or scrotum may suggest the presence of an inguinal hernia, hydrocele, or hematocele. Because the hernial contents of most acquired inguinal hernias are strangulated, palpation of the scrotum and examination of the vaginal rings *per rectum* should be part of the physical examination of every adult stallion with signs of intestinal obstruction. Unapparent, congenital inguinal hernias may become apparent after castration. Examining the inguinal area of foals before castration for the presence of a congenital inguinal hernia is especially important to avoid evisceration. If a scrotal hernia is present, palpation may elicit a sensation of crepitus. Occasionally, peristalsis may be noted by movement of the skin overlying the bulge.[58] A ruptured inguinal hernia or an inguinal rupture should be suspected if the skin over the inguinal or scrotal swelling is cold, edematous, or macerated. A strangulated inguinal hernia or rupture should be suspected when a stallion develops signs of colic after copulation or exercise, especially if a testis and its spermatic cord are swollen and tender. A varicocele should be suspected if the spermatic cord appears larger than normal and has the texture of a "bag of worms." A hydrocele should be suspected if the scrotum appears to be smooth, nontender, and fluid filled, and the testis is smaller than normal. The presence of a fluid-filled, nontender scrotum weeks after castration also indicates a hydrocele.

Examination *per Rectum*

Per rectum examination of the vaginal rings and structures that traverse them may be helpful in the diagnosis and evaluation of some genital abnormalities, such as cryptorchidism, inguinal or scrotal herniation, scirrhous cord, and testicular neoplasia. The vaginal rings and associated structures are located about

6 or 8 cm cranial to the iliopectineal eminence and 10 to 12 cm abaxial to the linea alba in the average-sized horse.[1] The vaginal ring is palpable in geldings as a slight depression, but in stallions it is large enough to insert a finger.[125]

The risk of rectal injury should be weighed against the value of the diagnostic information to be gained before performing an examination *per rectum*. For example, examination *per rectum* is probably not necessary before removing a cryptorchid testis by an inguinal approach, provided that the owner is confident that orchidectomy has not been attempted previously. If, during surgery, the testis is not encountered in the inguinal canal, it can usually be extracted from the abdomen through the vaginal ring or through a parainguinal incision. Even though mares are more commonly subjected to examination *per rectum*, the incidence of rectal injury in geldings and stallions is higher, perhaps because males are less accustomed to the procedure and resist it more forcibly.[126] Most cryptorchid stallions presented for examination are young, and the small size and fractious nature of young horses predispose them to rectal injury during examination *per rectum*.

To palpate the vaginal rings, the examiner introduces a hand into the rectum until the lateral aspect of the wrist rests on the pubic brim at the pelvic symphysis.[127] The examiner may find it advantageous to palpate the right vaginal ring with the left hand and the left vaginal ring with the right hand. The fingertips are flexed and pressed against the lateral aspect of the abdominal wall, and fingers are extended in a downward and forward direction against the abdominal wall until a finger enters the vaginal ring. If the search is made with a backward flexing motion of the finger, the medial border of the deep inguinal ring tends to close, causing the finger to slide over the vaginal ring. When the vaginal ring is located, the components of the spermatic cord can be felt as a cordlike structure entering the canal. The ductus deferens is more readily palpated at the ring than are the testicular vessels,[3] and it can be palpated on the caudomedial aspect of the ring, if the testis or epididymis has descended.[22]

The vaginal rings should always be examined for evidence of intestinal incarceration when examining a colicky stallion. Palpating distended loops of intestine and a loop descending through the vaginal ring indicates that intestine has become inguinally incarcerated. Traction on the loop usually causes a painful reaction from the horse. For horses with septic funiculitis, thickening of the spermatic cord at the vaginal ring is evidence of ascension of infection into the abdominal cavity. For horses with testicular neoplasia, thickening of the spermatic cord at the vaginal ring suggests that the neoplasm has metastasized. The lymph nodes surrounding the terminal portion of the aorta and its branches should also be palpated for evidence of metastases.

For a horse with an unknown history of castration that displays masculine behavior, or for a stallion known to be cryptorchid, examination *per rectum* may be useful in determining whether a testis has traversed the vaginal ring.[127] Determining the location of a retained testis before orchidectomy is especially important if a flank or paramedian approach is to be used, because an inguinally retained testis is difficult to remove using any approach other than the inguinal one. Palpating an abdominal testis *per rectum* is difficult, because the testis is small and flaccid, and because the proper ligament of the testis is usually elongated, allowing the testis a wide range of movement. Palpation of an abdominal testis *per rectum* by an inexperienced examiner should be regarded as a fortuitous occurrence, and failure to palpate an abdominal testis should not be relied on diagnostically. The vaginal ring cannot be palpated in horses with complete abdominal testicular retention, but palpation of the vaginal ring is evidence that the testis or at least its epididymis has descended through the ring into the inguinal canal. Unfortunately, a partial abdominal cryptorchid cannot be distinguished from a horse whose testes have descended through the vaginal ring by examining the vaginal rings *per rectum*.

Testicular Biopsy

Testicular biopsy is indicated when less-invasive diagnostic methods have failed to provide an etiologic or pathologic diagnosis of testicular disease and when a diagnosis is essential to determine treatment and prognosis.[128] An obviously neoplastic testis should be removed rather than biopsied, because biopsy may disseminate neoplastic cells. Testicular biopsy has been used to a limited extent in the horse, perhaps because of fear of the complications reported after incisional (especially) or needle biopsy of the testes in other species.[129,130] Because horses are usually able to maintain breeding soundness after removal of one testis, obviously diseased testes have often been removed rather than biopsied, even though the testicular disease was not definitively identified.

Biopsy provides the only method for directly assessing stages of spermatogenesis and rates of sperm production and for identifying space-occupying lesions.[113] A biopsy may be helpful in differentiating among causes of testicular enlargement, such as septic orchitis, neoplasia, or trauma. Despite complications reported to occur in other species, aspiration and needle biopsies have been performed successfully in a small number of horses.[130-132]

Aspiration Biopsy

Aspiration biopsy presents little risk to the horse because decreased spermatogenesis is not a likely complication.[128] Although an aspiration biopsy is less damaging to the testis than a needle biopsy, an aspiration biopsy usually does not offer useful information about spermatogenesis.[129] Its main use is to help determine the cause of testicular enlargement. Aspiration biopsy can be performed by inserting a 23- or 25-gauge needle into the testicular parenchyma.[128] Local anesthesia of scrotal skin is unnecessary. A 12-mL syringe is attached to the needle, and the plunger is withdrawn. The needle is retracted without exiting the parenchyma and is reinserted into several areas. The plunger is released, and the needle, still attached to the syringe, is removed from the testis. Although hemorrhage after biopsy is uncommon, digital pressure maintained over the puncture site for several minutes ensures hemostasis. Aspirated material is expelled onto a glass slide, smeared, dried, and stained. Aspirated material should be smeared gently, because cells collected from the testis by aspiration are extremely fragile. The slides should be examined by an experienced cytologist, because normally developing spermatocytes have cytologic characteristics that could lead to a mistaken diagnosis of malignancy.

Needle Biopsy

The needle biopsy provides the most useful information about spermatogenesis, and the technique is unlikely to have

deleterious effects on the testis.[128,130,131] Needle biopsy of a testis can be performed using a 12- or 14-gauge Vim-Silverman needle[130] or a 12- or 14-gauge automated biopsy needle (Monopty Biopsy Instrument).[131] Biopsy can be performed with the horse standing and sedated. The scrotum is prepared as if for aseptic surgery, and a small volume of local anesthetic solution is injected subcutaneously at the proposed site of biopsy using a 25-gauge needle. Unless there is a particular area of interest, the site of biopsy should be the craniolateral quarter of the testis, where the vasculature is the least prominent, and away from the head of the epididymis. The biopsy needle can be guided ultrasonographically to sample a discrete lesion. The testis is held tightly against the scrotal skin, and a small stab incision is created on the lateral surface of the scrotum, down to and through the parietal tunic. The Vim-Silverman needle or automated biopsy needle is inserted through the visceral tunic and tunica albuginea into the testicular parenchyma. Two or three samples are collected through the same skin incision but at slightly different angles. Pressure is maintained over the incision for several minutes, and the cutaneous incision is closed with a single suture. Samples are fixed in Bouin solution for 6 to 12 hours, washed, and stored for about 12 hours in 70% ethanol. They are then shipped to a laboratory in 50% ethanol. The biopsy should be examined by a pathologist trained in reproductive pathology.

Complications associated with needle biopsy of the testis in other species include transient scrotal edema; intratesticular hemorrhage resulting in pressure necrosis; immune reaction to spermatozoa caused by disruption of the blood–testis barrier; dissemination of neoplastic cells if a tumor is biopsied; formation of a hematoma between the testis and the parietal tunic or between the parietal tunic and scrotum, which can result in insulation-induced damage to the seminiferous epithelium; and transient decrease in semen quality.[128] Formation of antisperm antibodies and decrease in semen quality reported to occur in other species after testicular needle biopsy have not been reported to occur in the horse.[128,131]

Hormonal Assays

Occasionally, physical examination is inadequate to determine whether a horse possesses a retained testis, in which case a hormonal assay may be necessary to distinguish between psychic and hormonal causes of persistent masculine behavior. Concentration of testosterone or estrogen in the plasma or serum can be used to distinguish between geldings and horses with extrascrotal testicular tissue.[133-138] Stallions with descended testes (sometimes referred to as "entire stallions") and cryptorchid stallions have significantly higher concentrations of androgens and estrogens in the serum or plasma than do geldings. Concentration of testosterone and estrogen in serum decreases rapidly after castration and stabilizes within about 6 hours.[139]

In several studies, basal concentration of testosterone in castrated horses was generally less than 40 pg/mL of serum, and that of entire stallions was greater than 100 pg/mL.[134,135] The concentration of testosterone of entire stallions was often 1000 to 2000 pg/mL, although the concentration during winter was often as low as 200 pg/mL.[135] In one study, geldings were found to have a testosterone concentration in serum of less than 240 pg/mL, and horses with testicular tissue had a concentration of greater than 440 pg/mL.[133] Another investigation found the concentration of testosterone in the serum of ponies with testicular tissue to be 440 to 1550 pg/mL; 6 weeks after castration, the concentration had decreased to an average of 40 pg/mL.[136] These studies found the concentration of testosterone in the serum of stallions to depend on the age of the horse and the season, with the concentration being lowest in horses younger than 3 years and during the winter.[133-135]

Some investigators noted that cryptorchid stallions generally had a slightly lower concentration of testosterone than did entire stallions.[135-137] In contrast, other investigators observed that mature, bilateral, cryptorchid stallions and hemi-castrated stallions (i.e., unilateral cryptorchid horses whose scrotal testis had been removed) had a similar or higher concentration of testosterone in the serum than did entire stallions.[133] These authors suggested that retained abdominal testes may produce as much testosterone or more than do scrotal testes.

Wide variations in basal concentration of testosterone occasionally lead to confusion.[134-138] Because the concentration of testosterone in some entire stallions is exceptionally low and that in some geldings is exceptionally high, values may overlap, causing erroneous conclusions to be drawn as to whether a horse possesses testicular tissue. One investigator reported 14% error using basal concentration of testosterone to differentiate geldings from horses with testicular tissue.[117] These wide variations in concentration of testosterone were not observed in another study, which found basal concentrations of testosterone to be accurate in predicting the presence of testicular tissue.[133] That study found an error of only 5%.

A rise in the concentration of testosterone in response to stimulation by administration of hCG increases the accuracy of detecting cryptorchidism.[134-138] Investigators reported increased concentration of testosterone in cryptorchid stallions at any time between 30 and 120 minutes after intravenous administration of 6000 or 12,000 units of hCG.[134,135] Horses were classified as cryptorchid if the concentration of testosterone both before and after administration of hCG was greater than 100 pg/mL, or as geldings if the concentration in both samples was less than 40 pg/mL. The hCG-stimulation test was 94.6% accurate in predicting the presence of testicular tissue. Response to hCG was poor in horses less than 18 months old and during the winter.[134] Other investigators found that the response of bilateral cryptorchid stallions and hemi-castrates to hCG was minimal, but that best results were achieved if poststimulation samples were obtained at 24 hours rather than at 1 hour.[133] Other investigators found that administration of hCG to entire stallions induced an increase in concentration of testosterone that lasted about 10 days, and they suggested that for detection of a retained testis, a poststimulation sample should be taken at 2 to 3 days.[138]

In one study, quantification of total free (i.e., unconjugated) estrogen alone was superior to quantification of total androgens, with or without hCG stimulation, for detecting retained testicular tissue.[137] Although other investigators were unable to confirm superiority of free estrogens for detecting testicular tissue, they found a high correlation between the presence of testicular tissue and the concentration of conjugated estrogen (e.g., estrone sulfate) if cryptorchid horses younger than 3 years and cryptorchid donkeys of any age were excluded from the study.[134,135] The investigators found that young cryptorchid horses and cryptorchid donkeys did not consistently produce enough conjugated estrogens to yield reliable results. Quantification of conjugated estrogen was 96% accurate in predicting

the presence of testicular tissue when these animals were excluded from the study. Although cryptorchid horses produced less estrogen than did entire stallions, the lower threshold of conjugated estrogen for a cryptorchid horse was higher than that of testosterone. Horses with a concentration of estrone sulfate less than 50 pg/mL in plasma or serum were determined to be geldings. A concentration in excess of 400 pg/mL indicated cryptorchidism. Another investigation also revealed a high correlation between the concentration of conjugated estrogens and the presence of testicular tissue in horses older than 3 years.[133] In that investigation, geldings had a concentration of estrone sulfate less than 120 pg/mL, and cryptorchid stallions had a concentration greater than 1000 pg/mL.

Knowing the laboratory's standards for normal hormonal concentrations of geldings and horses with testicular tissue is important when evaluating results of hormonal assays. Comparing test results with values from a known gelding may be necessary, if the laboratory cannot furnish standards. False-positive results from hormonal assays to determine the presence of testicular tissue have not been reported.[140] The clinician can be confident that a horse has testicular tissue if the result of a hormonal assay indicates that testicular tissue is present.

Other Diagnostic Tests

Diagnostic tests that can be used to identify or characterize disease of the testes include ultrasonography, semen evaluation, cytological examination of peritoneal fluid, thermography, karyotyping, and possibly measurement of serum biomarkers. Ultrasonographic examination may delineate abnormalities within the testis and associated structures or assist in determining the location of a cryptorchid testis.

To ultrasonographically image an undescended testis, the inguinal area is scanned with a longitudinally placed 3.5-MHz convex probe or a 5-MHz transrectal transducer.[141,142] The testis is recognized as a round structure with a homogeneous granular texture with an anechoic line inside, representing the central vein of the testis. The echotexture of the cryptorchid testis is less dense than that of a normal descended testis.[141] If the testis is not located, the probe is placed more axially to examine the caudal ventral aspect of the abdomen. Most abdominal testes can be visualized lying on the ventral abdominal wall adjacent to the urinary bladder. If the testis is still not detected, the lateral aspect of the flank is scanned.

If the testis cannot be located during transabdominal ultrasonographic examination, transrectal ultrasonography can be performed. To image an abdominal testis, the transducer is inserted rectally, cranial to the pelvic brim, and the abdomen is scanned in a to-and-fro pattern as the transducer is advanced craniad. Transrectal ultrasonography is ineffective in locating an inguinal testis.[141] The sensitivity of transabdominal ultrasonography in detecting an undescended testis is about 98%.[142] Withholding feed from the horse for 24 to 36 hours before examination enhances visualization of an abdominal testis.[142]

Serum concentrations of biochemical markers, such as α-fetoprotein and hCG, are measurable in minute quantities using radioimmunoassay and have been used to monitor the response of humans to treatment for testicular neoplasia.[103] Apparently, no such markers have been used to detect testicular neoplasia of horses or to monitor the presence of metastatic neoplasms after a neoplastic testis has been removed.

Cytologic examination of peritoneal fluid may be valuable in diagnosing certain diseases of the testes and associated structures because the vaginal and peritoneal cavities communicate, so that changes in vaginal cavity fluid can be reflected in the peritoneal fluid. Semen evaluation may be valuable in diagnosing orchitis, epididymitis, or seminal vesiculitis. Thermographic examination of the scrotum may detect a variation in temperature between the testes. A horse with morphologic abnormalities suggestive of intersexuality, such as cryptorchidism accompanied by ambiguous genitalia, can be karyotyped to determine its genetic sexual identity.

SURGICAL PROCEDURES

Castration

Indications

Synonyms for castration include orchidectomy, orchiectomy, emasculation, gelding, and cutting. Castration is one of the most common equine surgical procedures and is usually performed to sterilize horses unsuitable for contributing to the genetic pool and to eliminate masculine behavior. By removing the primary source of androgens, castration renders the horse more docile and manageable.

Although stallions can be safely castrated at any age, managerial convenience usually governs the age at which a horse is castrated. Typically, a horse is castrated simply because facilities are insufficient to safely contain a stallion. Most horses are castrated when they are 1 to 2 years old, when masculine behavior becomes intolerable to the owner. Sometimes castration is delayed until a masculine feature, such as a crest on the neck, has developed or until it becomes apparent that the horse is unsuitable for breeding. Castration may be performed to alter conformation. Bulls castrated before puberty grow to a greater height because castration delays closure of the growth plates of their long bones,[143] and the same may be true of stallions castrated before puberty. The age when colts reach puberty varies greatly between and within breeds. One study found that the mean age at which American Quarter Horse colts reached puberty, defined as the age at which an ejaculate containing a minimum of 100×10^6 sperm with at least 10% progressive motility was first collected, was 68.68 ± 12.7 weeks and ranged between 55 and 101 weeks.[144]

Orchitis, epididymitis, testicular neoplasia, hydrocele, varicocele, testicular damage caused by trauma, torsion of the spermatic cord, or inguinal herniation may necessitate unilateral or sometimes bilateral orchidectomy. Impotent or infertile breeding stallions may be salvaged for other uses by castration.

Preoperative Considerations

A general physical examination of the horse should precede castration, and the scrotum, especially that of very young horses, should be inspected for inguinal herniation and for the presence of both testes. Discovery of inguinal herniation or cryptorchidism may affect the choice of anesthesia and the surgical approach. Preoperative sedation of a fractious horse usually permits safe palpation of the scrotal and inguinal areas and occasionally facilitates palpation of an inguinal testis by causing the cremaster muscles to relax.

Standing Castration

CASE SELECTION

Castration performed with the horse standing can be difficult and dangerous to the surgeon if candidates for the procedure are not selected prudently. Standing castration of horses with poorly developed testes and of ponies is mechanically difficult. Donkeys and mules can be dangerous to castrate while they are standing because of their athletic agility. Stallions that elicit a hostile or evasive response to genital palpation are best castrated while they are anesthetized. Docile stallions with well-developed testes whose genitalia can be palpated without being sedated are usually the safest candidates for standing castration.

PREPARATION OF THE HORSE

Sedating the horse to be castrated while standing is optional but advised. Drugs commonly used, either alone or in combination, are xylazine HCl, detomidine HCl, pentazocine, and butorphanol tartrate. Acetylpromazine, although commonly administered to tranquilize stallions before castration, can result, on rare occasion, in priapism or penile paralysis, and so its use in stallions should be avoided. Additional information about standing restraint of the horse can be found in Chapter 22.

The scrotum must be anesthetized on each side of the scrotal raphe from the cranial to the caudal pole of the testis along the proposed lines of incision. The spermatic cords can be anesthetized by injecting local anesthetic solution, usually 15 to 30 mL, through a 22- to 20-gauge needle directly into each cord. This anesthetic technique ensures good anesthesia of the cord but occasionally causes a hematoma that interferes with application of the emasculator. Alternatively, about 25 mL of local anesthetic solution (without epinephrine) can be injected directly into the parenchyma of each testis through an 18-gauge, 1½-inch needle. The anesthetic solution diffuses proximally into each spermatic cord. The horse's tail should be bandaged to prevent it from contaminating the surgical field, and the scrotum should be scrubbed before and after administering the local anesthetic solution.

RESTRAINT

The horse should be restrained by a competent handler, and to ensure adequate immobilization, application of a lip twitch may be necessary. The standing castration should be performed with both the surgeon and the handler positioned on the same side of the horse, usually the left side for the right-handed surgeon. The surgeon should be positioned at the horse's shoulder well out of kicking range with his or her head and shoulder pressed firmly into the horse's flank.

Standing castration can be performed safely and efficiently if candidates are selected prudently, the horse is adequately sedated, the spermatic cord and scrotum are properly desensitized with a local anesthetic agent, and the surgeon is technically proficient. A standing castration requires less expense and assistance and is often less time-consuming because the surgeon need not wait for the horse to recover from general anesthesia. Risks to the horse that attend general anesthesia are avoided. Because the spermatic cords and scrotum are locally desensitized, measures to rectify immediate postoperative complications, such as hemorrhage, can be accomplished without anesthetizing the horse.

Recumbent Castration

ANESTHESIA

To castrate a horse in the recumbent position, a clean, safe area in which to anesthetize and recover the horse is a prerequisite. A variety of intravenous anesthetics, alone or in combination, can be administered to provide safe and predictable anesthesia of sufficient duration. A thiobarbiturate administered as a bolus produces rapid anesthesia characterized by moderate analgesia and muscular relaxation, particularly if the horse has been sedated with xylazine.[145] Recovery is usually satisfactory if repeated administration of the thiobarbiturate is not necessary. Ketamine, administered after sedating the horse with xylazine, provides 10 to 15 minutes of surgical anesthesia. Muscular relaxation and analgesia are only moderate but can be enhanced if butorphanol tartrate or diazepam is added to the preanesthetic regimen.[145-147] If necessary, anesthesia can be extended by readministering half the dosage of xylazine and ketamine combined in one syringe. Guaifenesin (5% to 10%), in combination with ketamine or a thiobarbiturate, provides smooth induction and recovery and good analgesia with excellent muscular relaxation, but guaifenesin must be administered in large volumes.[148] Succinylcholine, a muscle relaxant, has been widely employed as a chemical restraint for recumbent castration, but because it provides no analgesia, its use alone for castration is inhumane. Additional information on anesthesia can be found in Chapters 18 and 19.

POSITIONING

The horse is anesthetized and positioned in lateral recumbency with its upper pelvic limb pulled forward and secured with a rope. The right-handed surgeon generally finds the recumbent castration most easily performed with the horse positioned in left lateral recumbency, and vice versa.

Approach

SCROTAL INCISION

For the standing castration and most techniques of recumbent castration, the testes are removed through a scrotal incision. If the horse is anesthetized, and if the testes are small and difficult to grasp, as is often the case with prepubescent stallions, the scrotum can be safely incised by pulling the cranial end of the prepuce craniad and upward to tense the scrotal raphe (Figure 59-10). Another method of incising the scrotum is to make two parallel 8- to 10-cm incisions 2 cm distant from the raphe on either side while compressing the testes against the bottom of the scrotum. The incisions should be sufficiently long to provide adequate postoperative drainage. To ensure adequate drainage, many surgeons prefer to partially ablate the scrotum, which can be accomplished by connecting the two parallel incisions craniad and caudad and removing the portion of scrotum between the incisions.

Another method of removing the bottom of the scrotum is to grasp the scrotal raphe between the thumb and forefinger, and while applying traction to the bottom of the scrotum, to excise a portion of the tented tissue with a scalpel (Figure 59-11). When surgery is performed with the horse standing, stab wounds to the horse or surgeon that could result from sudden movement by the horse are prevented by incising the scrotum with a scalpel blade held between the fingers rather than

attached to a handle. A large portion of the scrotum should be excised to ensure adequate drainage. To avoid cutting large vessels, the dissection should remain close to the scrotal skin. After removing a portion of the scrotum, the testes are isolated using digital dissection.

Figure 59-10. Incising the scrotum for castration. If the testes are difficult to grasp, the cranial end of the prepuce can be pulled craniad and upward to tense the scrotal raphae.

INGUINAL INCISION

For the inguinal approach, the horse is anesthetized and positioned in dorsal recumbency. The superficial inguinal ring is exposed through an 8- to 15-cm skin incision (depending on the horse's size) made directly over the superficial inguinal ring.

EMASCULATORS

The emasculator models most commonly used are the improved White's, the Reimer, and the Serra emasculators (Figure 59-12).[149] The Reimer emasculator crushes the cord, and a blade operated by a separate handle severs the cord distally. Because the cord is severed with a separate handle, there is no danger of cutting the cord before it is satisfactorily crushed. The extra handle on the Reimer emasculator makes the instrument somewhat unwieldy for standing castration. The Sand's emasculator is similar to the Reimer emasculator but has no cutting component and only crushes the cord (Figure 59-13). The cord must be severed distal to the crushed segment with scissors or a scalpel blade. This emasculator is used more commonly in Europe than in North America. The jaws of the Serra emasculator are curved, so that the cord is evenly crushed, and the grooves on the crushing blades are oriented parallel to the cord, decreasing the chance of accidentally transecting the cord with the crushing portion of the jaws (Figure 59-14).[21]

The Henderson Equine Castrating Instrument is another tool designed for castration (Figure 59-15). One handle of this pliers-like instrument is attached to a 12 W, or greater, variable-speed drill (slippage is likely to occur with a less-powerful drill) with a $\frac{3}{8}$-inch or larger chuck. With one hand holding the testis, the instrument is clamped across the entire cord, just proximal to the testis. With slight tension on the drill and with the instrument held parallel to the cord, the testis is rotated slowly for about five turns. The speed of the rotations is gradually increased while keeping slight tension on the cord. After 20 to 25 rotations, the cord separates about 8 to 10 cm proximal to the instrument. The twisting of the cord effectively seals the severed

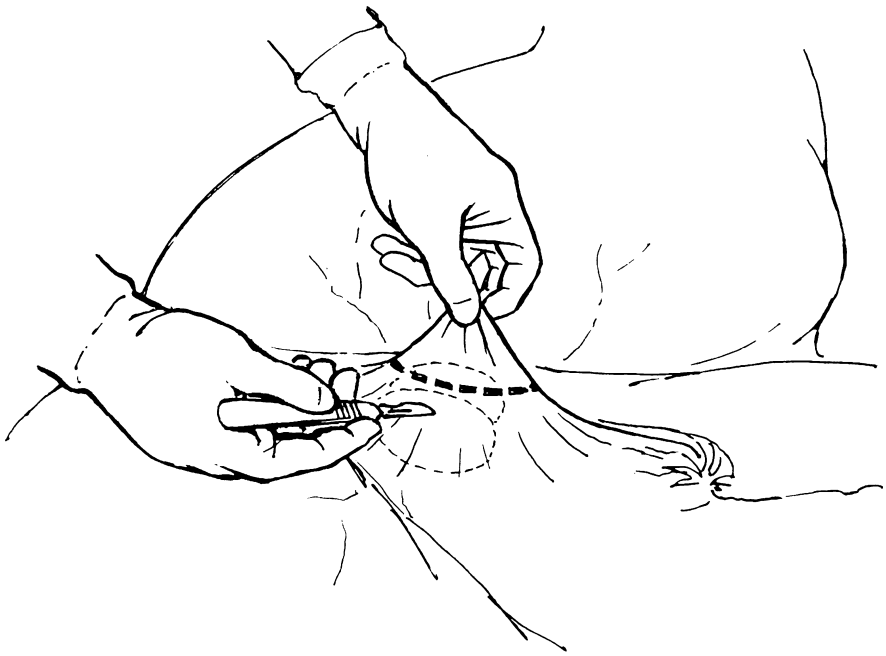

Figure 59-11. The bottom of the scrotum is removed by placing traction on the scrotal raphe with a thumb and forefinger and excising a portion of the tented tissue with a scalpel.

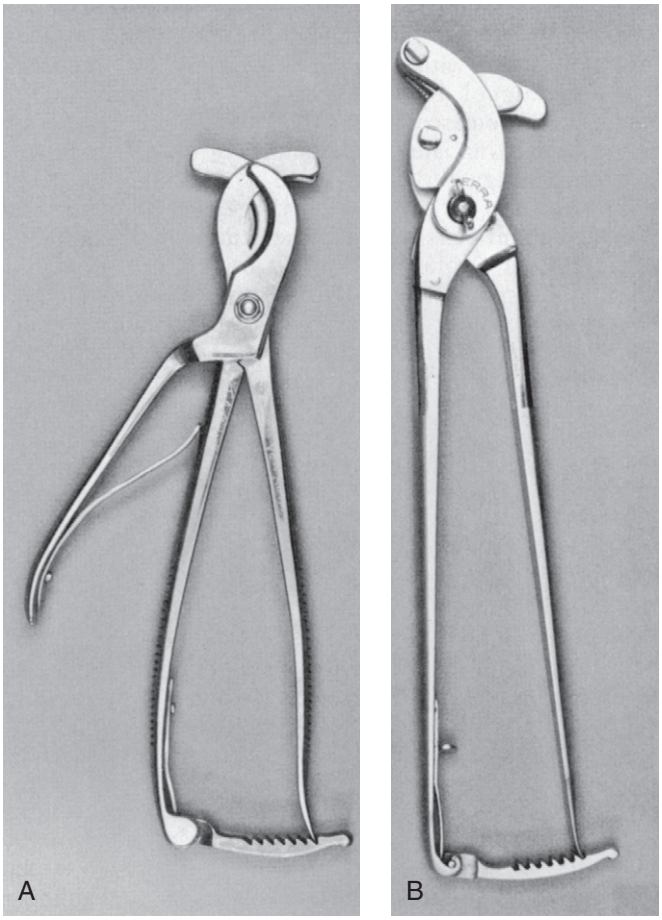

Figure 59-12. Reimer **(A)** and Serra **(B)** emasculators. The Reimer emasculator severs the cord with a blade on a separate handle so that the cord is not accidentally cut before it is satisfactorily crushed.

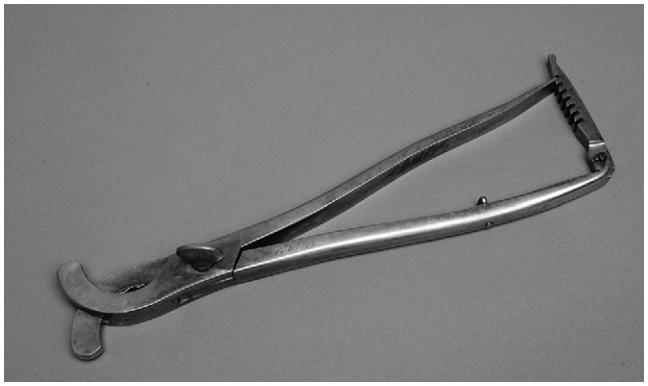

Figure 59-13. The Sand's emasculator is similar to the Reimer emasculator but has no cutting blade. The spermatic cord must be severed distal to the emasculator with a scissors or scalpel blade.

vessels. Because the parietal tunic is sealed using this device, herniation is theoretically less likely than when castration is performed using other instruments. Although castration using this instrument is usually performed with the horse anesthetized, horses can also be castrated with this instrument while they are standing.

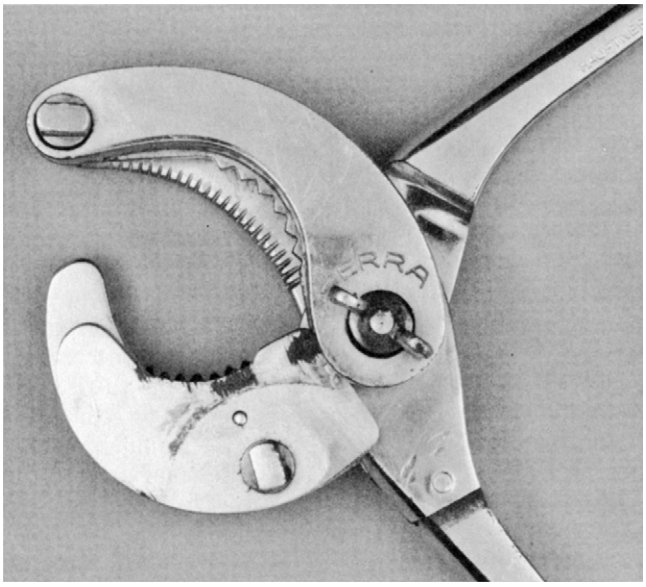

Figure 59-14. Serra emasculator. The grooves of the crushing blade are oriented vertically to prevent the blade from accidentally cutting the cord.

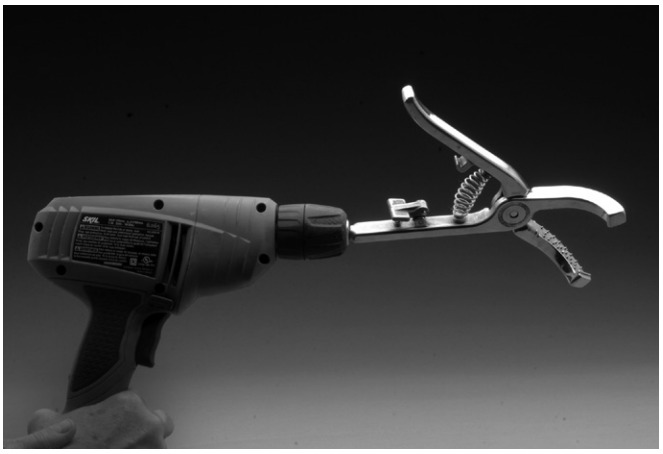

Figure 59-15. A Henderson Equine Castrating Instrument. One handle of this instrument is attached to a variable-speed drill. The instrument is clamped across the spermatic cord and rotated slowly for about five turns before the speed of rotations is increased. The cord is rotated until it separates proximal to the instrument. The twisting of the cord seals the severed vessels.

Surgical Techniques

Techniques of orchidectomy are the open, closed, and half-closed techniques, regardless of whether the horse is castrated while standing or recumbent, or whether the approach is inguinal or scrotal. With the open technique of castration, the parietal (or common vaginal) tunic is retained. With the closed and the half-closed techniques, the portion of the parietal tunic that surrounds the testis and distal portion of the spermatic cord is removed. Regardless of the technique, the scrotal skin is most commonly left unsutured to heal by second intention. When the skin is left unsutured, the castration is sometimes referred to as an open castration, adding confusion to the terminology associated with castration. When the scrotal or inguinal skin is

sutured, the castration is sometimes referred to as a closed castration. To avoid confusion, the terms *open* and *closed* should be used to describe whether the parietal tunic of each testis was removed and should not be used to describe whether the scrotal or inguinal wound was sutured.

OPEN TECHNIQUE

When performing the open technique of castration, the parietal tunic of testis is incised. The ligament of the tail of the epididymis (caudal ligament of the epididymis), which attaches the parietal tunic to the epididymis, is severed or bluntly transected. By transecting the fold of the mesorchium and mesofuniculum, the testis, epididymis, and distal portion of the spermatic cord are completely freed from the parietal tunic and removed using an emasculator. The open technique of castration is probably the most commonly used technique.[149]

CLOSED TECHNIQUE THROUGH A SCROTAL APPROACH

With the closed technique, the parietal tunic is not incised, so it also is removed along with the testis and a portion of the cord. Using digital dissection, the parietal tunic surrounding the testis is freed from the scrotal ligament and scrotal fascia. By placing mild traction on the testis with one hand, the parietal tunic surrounding the cord is then separated from fascia surrounding the spermatic cord with the other hand. After the parietal tunic is separated from the surrounding fascia, it and its contents are removed using an emasculator. Care should be taken, when separating the fascia from the spermatic cord, to include the large pudendal vessels that lie within the fascia, so that these vessels are not included in the jaws of the emasculator.

HALF-CLOSED TECHNIQUE

The closed technique just described can be converted to a half-closed technique by making a 2- to 3-cm vertical incision through the exposed parietal tunic at the cranial end of the testis or the distal end of the spermatic cord. A thumb (the left thumb if the operator is right-handed) is inserted through the incision into the vaginal cavity. The testis and a portion of the spermatic vasculature are prolapsed through the incision by applying downward traction on the tunic with the thumb while simultaneously using the fingers of the same hand to push the testis through the incision. The fundus of the parietal tunic inverts and follows the testis through the incision because of its attachment to the testis by the ligament of the tail of the epididymis. Traction is applied to the parietal tunic with the index and middle fingers, which are placed into the sac formed by the inverted fundus. Traction can also be applied to the parietal tunic and the testis by applying a large Carmalt or Allis forceps to the parietal tunic before prolapsing the testis from the vaginal cavity. The half-closed technique should be considered a closed technique because the parietal tunic is removed along with the testis and the distal portion of the spermatic cord.

CONSIDERATIONS CONCERNING ALL TECHNIQUES

For each technique, the emasculator is applied at a right angle to the spermatic cord, loosely closed to avoid incorporating scrotal skin, and slid farther proximally. The emasculator is applied so that the crushing component is proximal to the cutting blade. When correctly applied, the wing-nut of the emasculator is oriented distad toward the testis, and the emasculator is said to be positioned "nut to nut." This positioning is not critical when using an emasculator that does not have a cutting blade. The scrotal skin is pushed toward the abdomen with one hand (with the spermatic cord positioned between the index and middle fingers) toward the horse's abdomen, and the jaws of the emasculator are inspected to ensure that they do not contain scrotal skin. The tension on the cord is relieved, and the handles of the emasculator are compressed completely to crush and, depending on the emasculator used, to sever the cord. The time that should elapse before the emasculator is removed from the spermatic cord depends on the size of the cord being severed and the dependability of the emasculator used, but applying the emasculator for about 2 minutes is usually sufficient to achieve hemostasis. If the cord is exceptionally large, the emasculator can be applied for a longer time, or the parietal tunic and cremaster muscle can be crushed and severed separately from the testicular vessels and the ductus deferens. The cutting blade of the emasculator should not be so sharp that the cord is completely severed but rather should be slightly dull so that the cord must be torn, although with only slight effort, from the blade. If a noncutting emasculator is used, the cord is severed with a scalpel blade or scissors 2 to 3 cm distal to the emasculator. The emasculator should be directed toward the horse's inguinal area before the cord is released, so that the testicular vessels do not recoil. It is customary, and perhaps prudent, when performing a standing castration to leave the scrotal wound unsutured. Loose scrotal fascia protruding from the scrotal opening is trimmed with scissors.

A technique (i.e., the Zurich technique) frequently used in Europe to ensure adequate scrotal drainage involves suturing a 30-cm-long gauze drain to the stumps of the cords with heavy catgut suture.[150] The drain that exits the scrotal wound is removed 2 days after castration by rupturing the catgut suture with a sharp tug on the drain.

SELECTION OF TECHNIQUE

An advantage of the closed and half-closed techniques of castration is that removal of the parietal tunic decreases the incidence of postoperative complications, such as septic funiculitis and hydrocele[32,35] (see "Postoperative Complications," later). The half-closed technique permits inspection of the components of the cord and allows a greater portion of the ductus deferens and testicular vasculature to be exteriorized. For horses at risk of having an unapparent inguinal hernia, such as Standardbreds, evisceration can be avoided by using a closed technique and placing a ligature proximal to the proposed site of transection.

The closed technique of castration has no advantage over the open technique in preventing evisceration if a ligature is not applied to the cord proximal to the site of transection. The closed and half-closed techniques are indicated for disease conditions that may involve the parietal tunic, such as neoplasia and orchitis. The closed and half-closed techniques require more dissection than does the open method of castration, and this may be a disadvantage when performing a standing castration on a fractious stallion.

Primary Closure of the Incision

By convention, scrotal incisions are usually left unsutured to heal by secondary intention. Primary closure, however, speeds healing and recovery, decreases the possibility of infection, and decreases edema, pain, and muscular stiffness.[151-154] Primary closure may be particularly useful if vigorous exercise cannot be enforced postoperatively.

In one study, 6% of horses castrated under aseptic conditions, while anesthetized, and whose scrotal wound was sutured, suffered from one or more complications, including diarrhea and fracture of limb during recovery from anesthesia, whereas 22% of horses castrated while standing and whose scrotal wound was left open to heal by second intention experienced a complication, which for most was infection at the scrotal wound.[155] The cost of performing a castration with the horse anesthetized and under aseptic conditions was about three times greater than the cost incurred when the horse was castrated while standing. Even considering the cost associated with treatment for complications incurred when castration was performed with the horse standing, however, the overall cost of performing surgery with horses standing was still less than that of performing surgery with horses anesthetized and under aseptic conditions.[155]

Primary closure decreases the time of convalescence but is time-consuming and requires meticulous hemostasis and strict adherence to aseptic technique. In one study in which the scrotal wound was sutured, no complications were encountered when the spermatic cords were simply transected with an emasculator.[151] Hemorrhage from a cord, however, even a small amount, into the sutured scrotum, increases the risk of hematoma formation. Therefore ligation of the cord proximal to the point of division with the emasculator ensures good hemostasis and should be considered an important part of the procedure.

One investigator, pointing out the importance of eliminating dead space, closed the subcutaneous tissue with multiple rows of absorbable suture.[151] Another investigator, however, found multiple-layer closure unnecessary and reported minimal postoperative complications when only the scrotal skin was sutured.[152] The cutaneous incision is best closed with an absorbable 2-0 monofilament suture using a simple-continuous intradermal suture pattern. Because the cutaneous incision is sutured intradermally, removal of cutaneous sutures is not necessary.

The testes can be removed *per primam*, with the horse anesthetized, through an inguinal incision created over each spermatic cord.[156,157] This technique is frequently used in Europe when castrating horses 2 years old or older to avoid evisceration, because the vaginal rings of horses more than 2 years old are thought by some surgeons to be wider than those of younger horses. Using this technique, the testis is pushed craniad from the scrotum so that it lies close to the superficial inguinal ring, and a 5- to 7-cm cutaneous incision is created over the superficial inguinal ring. The inguinal fascia overlying the testis is incised to expose the parietal tunic of the testis. Care is taken to avoid lacerating large branches of the external pudendal blood vessels. The parietal tunic is incised longitudinally for 5 cm in an area not covered by the cremaster muscle.

The ligament of the tail of the epididymis is located with an index finger, and by applying traction on this structure, the testis is pulled from the vaginal cavity. The ligament of the tail of the epididymis, which attaches the testis to the parietal tunic, is transected. Bleeding vessels are cauterized to prevent hemorrhage into the vaginal cavity. Two transfixing ligatures of absorbable heavy monofilament suture are applied 1 cm apart, as far proximad as possible, to the testicular vasculature and ductus deferens. The vasculature and ductus deferens are severed 2 cm distal to the distal ligature, and the stump is replaced into the vaginal cavity. If this technique of *per primam* castration is performed with the expectation of preventing incarceration of intestine into the inguinal canal, a ligature should be applied to each spermatic cord proximal to the site of transection.[64]

The incision in the parietal tunic and the subcutaneous tissue are each sutured with an absorbable 2-0 monofilament suture using a simple-continuous pattern. The cutaneous incision is closed with the same suture using a simple-continuous intradermal suture pattern.

Aftercare

All horses not previously immunized with tetanus toxoid should receive both tetanus antitoxin and toxoid. The previously immunized horse should receive a booster vaccination if more than a year has passed since its last vaccination.[158] The horse's activity should be restricted for the first 24 hours after castration to prevent hemorrhage from the severed testicular and scrotal vessels. After this period, the horse should be exercised to the degree necessary to prevent excessive preputial and scrotal edema. A large, grass-covered field is an ideal environment for postoperative recuperation, but the owner should be cautioned that turning a horse into a field does not ensure that it will receive adequate exercise.

Antimicrobial treatment is probably unnecessary if clean surroundings are provided, but a survey of practitioners, undertaken to determine the type and frequency of complications that occur after castration found that horses may be less likely to develop infection at the castration site if they receive perioperative antimicrobial treatment.[149] The operative site can be hosed vigorously, if the horse permits it, to keep the scrotal wound clean, open, and draining; but this same survey found that horses that receive hydrotherapy after castration may be more prone to develop excessive swelling and infection of the scrotum.

Protecting the wound against flies is usually unnecessary, even during fly season, if the horse's tail-hairs are long enough to reach the scrotal area. The horse should be isolated from mares for at least 2 days after castration. Ejaculates are highly unlikely to contain sufficient spermatozoa to cause impregnation after 2 days.[159] The castration wound should be nearly healed by 3 weeks.

Laparoscopic Castration

Ligating and transecting the blood supply and ductus deferens of scrotal testes laparoscopically with the horse standing or anesthetized results in avascular necrosis of the testicular parenchyma with the testes *in situ*.[160,161]

The standing approach is preferred by most surgeons when the horse is castrated laparoscopically. To ensure safe insertion of a laparoscopic sleeve and cannula into the abdomen, the abdomen can be insufflated through a Verres needle, IV catheter, teat cannula, chest drain, or metal uterine catheter introduced into the abdomen through a stab incision created slightly dorsal to the crus of the internal abdominal oblique muscle, midway between the last rib and the tuber coxae. The abdomen can also be insufflated, using the same devices, through a stab incision created on the linea alba. The abdomen is insufflated using a gas, such as carbon dioxide, nitrous oxide, or helium, until the intra-abdominal pressure rises to 8 to 10 mm Hg. Care should be taken to avoid insufflating the retroperitoneal space. After the abdomen is inflated, the insufflation device is removed, and a laparoscopic sleeve with a trocar is inserted through an incision in the flank. The laparoscopic sleeve and trocar can also be

inserted safely, without insufflating the abdomen, by allowing air to enter the abdomen through a blunt cannula, such as a chest drain, inserted into the abdomen through the flank. Air entering the abdomen causes the viscera to fall away from the body wall, allowing safe introduction of the laparoscopic sleeve and trocar. The trocar is removed, and the laparoscope, which is attached to a fiberoptic light source and a video camera and viewing monitor, is introduced into the abdomen. The laparoscope is directed caudad to view the inguinal area. A 10-mm-diameter instrument portal is created 8 to 10 cm cranioventral to the laparoscopic portal, and another is created 8 to 10 cm caudoventral to the laparoscopic portal. A third 5-mm-diameter instrument portal is created 8 to 10 cm caudodorsal to the laparoscopic portal.

The testicular vessels and ductus deferens are identified in the mesorchium as they course toward the vaginal ring. A ligating loop is placed through the 5-mm instrument portal, and a right-angle dissecting forceps is inserted through the cranioventral portal and the ligating loop. The ductus deferens and testicular vessels are grasped with the forceps. Using a bipolar cautery forceps placed through the caudoventral instrument portal, the ductus deferens and testicular vessels are coagulated distal to the forceps. The cautery instrument is removed and replaced with a laparoscopic scissors, which is used to transect the ductus deferens and spermatic vessel immediately distad to the site of coagulation. The ligating loop is now slid over the right-angle forceps onto the coagulated stump of the ductus deferens and testicular vessels, tightened, and tied, and the ends of the ligature are cut. After releasing the forceps, the stump is inspected for hemorrhage. By elevating the small colon manually *per rectum*, the contralateral testis can be removed using the same portals and technique. Removing the contralateral testis using portals created on the contralateral side, however, may be faster and easier.

The testes, deprived of their blood supply, swell during the first week and then begin to decrease in size. The atrophied testes can be palpated in the scrotum for at least several months,[161] but by 5 months the remnants are no longer palpable.[160] Within 7 days after the testicular vessels are ligated, the concentration of testosterone falls to that expected of a horse with no functional testicular tissue. The body and tail and sometimes the head of the epididymis remain viable, but because the epididymis has no contribution to masculine behavior, the horse behaves as a gelding.[20] Swelling and discomfort observed after laparoscopic castration seem to be less severe than that seen after routine castration.[161]

In one study that evaluated the results of laparoscopic castration, 5.6% of inguinally retained testes and 3.4% of normally descended testes failed to become completely necrotic, as a result of an alternative blood supply from the cremasteric or external pudendal artery, or both, resulting in preservation of stallionlike behavior.[20,162] The owner should be warned of this uncommon complication.

Vasectomy

A stallion used for detecting estrous (i.e., a teaser stallion) can be vasectomized to render it incapable of ejaculating spermatozoa and thus from accidentally impregnating mares. A stallion can be vasectomized through an incision created over each spermatic cord or through a single incision created over one testis.[163] To transect a portion of the ductus deferens through a single incision in the scrotal area, the horse is anesthetized and positioned in dorsal or lateral recumbency, and the scrotum is prepared for surgery. A 2-cm, longitudinal, cutaneous incision is made on the medial aspect of one testis, and the incision is extended through the dartos and parietal tunic. The ductus deferens, which is identified as a white, 2- to 3-mm-diameter, cordlike structure, is exteriorized and separated for a length of several centimeters from its mesorchium, using a curved hemostatic forceps. Two ligatures of 2-0 absorbable or nonabsorbable suture are placed around the most proximal aspect of the exposed portion of the ductus deferens, and a third ligature is placed around the most distal aspect of the exposed portion of the ductus deferens. The segment of ductus deferens between the two proximal ligatures and the distal ligature is removed. Double-ligating the proximal end minimizes the likelihood of spontaneous reanastomosis and formation of a sperm granuloma.

The incision in the parietal tunic is sutured with an absorbable 2-0 suture using a simple-continuous pattern. The ductus deferens on the medial aspect of the other testis is subsequently palpated through the cutaneous incision and exposed by incising the scrotal septum and overlying parietal tunic. A segment of the ductus deferens is exteriorized, ligated, and transected as described. The incision in the parietal tunic and the subcutaneous tissue are each sutured with an absorbable 2-0 suture using a simple-continuous pattern. The cutaneous incision is closed with the same suture using a simple-continuous intradermal suture pattern.

Immunologic Castration

Immunization against luteinizing hormone–releasing hormone (LHRH), a neuropeptide produced by the hypothalamus, was used in a cryptorchid stallion to decrease serum concentration of testosterone,[164] and immunization against gonadotrophin-releasing hormone (GnRH) was used experimentally to suppress testicular function of entire stallions.[165,166] Repeated immunization was necessary to maintain a sufficient binding titer for complete neutralization of LHRH or GnRH and inhibition of the reproductive endocrine axis. Immunization against GnRH caused decreased concentrations of testosterone and estrogen in the serum, diminished sexual behavior, and decreased testicular size, and it had a negative effect on semen quality. Stallions varied in response to immunization, and in one study, libido was not totally suppressed.[165]

If a vaccine against LHRH or GnRH becomes commercially available, unwanted male sexual behavior by cryptorchid or entire stallions can be prevented temporarily. A vaccine against GnRH may enable a stallion to perform at its peak ability at athletic competitions, by decreasing undesirable sexual behavior, until the stallion's genetic potential can be determined, while allowing the stallion to retain its ability to produce progeny. The time required for recovery of libido and semen quality needs to be determined before a vaccine against LHRH or GnRH is used clinically for temporary diminution of male sexual behavior.

Cryptorchid Castration

A retained testis can be removed through an inguinal, parainguinal, suprapubic paramedian, or flank approach. For each of these approaches, except the flank approach, the horse must be anesthetized. The approach is termed noninvasive if the testis

can be removed by introducing only one or two fingers into the abdominal cavity, and an approach that requires insertion of a hand into the abdomen is considered invasive. Only the inguinal and parainguinal approaches allow noninvasive removal of a cryptorchid testis.

Inguinal Approach

For the inguinal approach, the horse is anesthetized and positioned in dorsal recumbency. The superficial inguinal ring is exposed through an elliptical, scrotal incision or through an 8- to 15-cm skin incision (depending on the horse's size) made directly over the superficial inguinal ring. A cryptorchid testis and the contralateral scrotal testis (or two cryptorchid testes) can be removed from one incision if the incision is created over the scrotum rather than over the superficial inguinal ring.

The inguinal fascia is separated digitally to expose the superficial inguinal ring. An inguinal testis is readily encountered when the superficial inguinal ring is exposed. The vaginal sac should always be opened and its contents examined to avoid mistaking the descended tail of the epididymis of a partial abdominal cryptorchid for a small, inguinal testis. If the testis has already been removed, the stump of the spermatic cord is encountered as it exits the canal. Finding a stump of a severed ductus deferens, the remnant of spermatic vessels, and a well-developed cremaster muscle indicates that the horse has been castrated.[25,134]

After the superficial inguinal ring has been exposed, an abdominal testis can be retrieved using one of several noninvasive techniques. One noninvasive technique requires locating the rudimentary common vaginal tunic, or vaginal process. This structure contains a portion the epididymis or sometimes a portion of the gubernaculum testis. The body of the epididymis can be exposed through a small incision in the vaginal process and traced to the tail of the epididymis, which is connected to the testis by the proper ligament of the testis. By placing traction on this ligament, the abdominal testis can usually be exteriorized through the vaginal ring. The key to this technique is locating the vaginal process. The vaginal process of the partial abdominal cryptorchid testis lies everted within the inguinal canal and is readily encountered during inguinal exploration. The vaginal process of the complete abdominal cryptorchid lies inverted within the abdominal cavity, along with the epididymis and testis, and difficulty may be encountered in locating and everting it into the canal.

An inverted vaginal process can be everted into the inguinal canal by exerting traction on the scrotal ligament, which is also known as the inguinal extension of the gubernaculum testis (IEGT)[12] (Figure 59-16). This ligament is a remnant of the gubernaculum testis and attaches the vaginal process to the scrotum. The IEGT is located by carefully examining the margin of the superficial inguinal ring for a fibrous band that descends into the canal. The IEGT can be found on either the medial or lateral aspect of the ring, usually at the junction of the middle

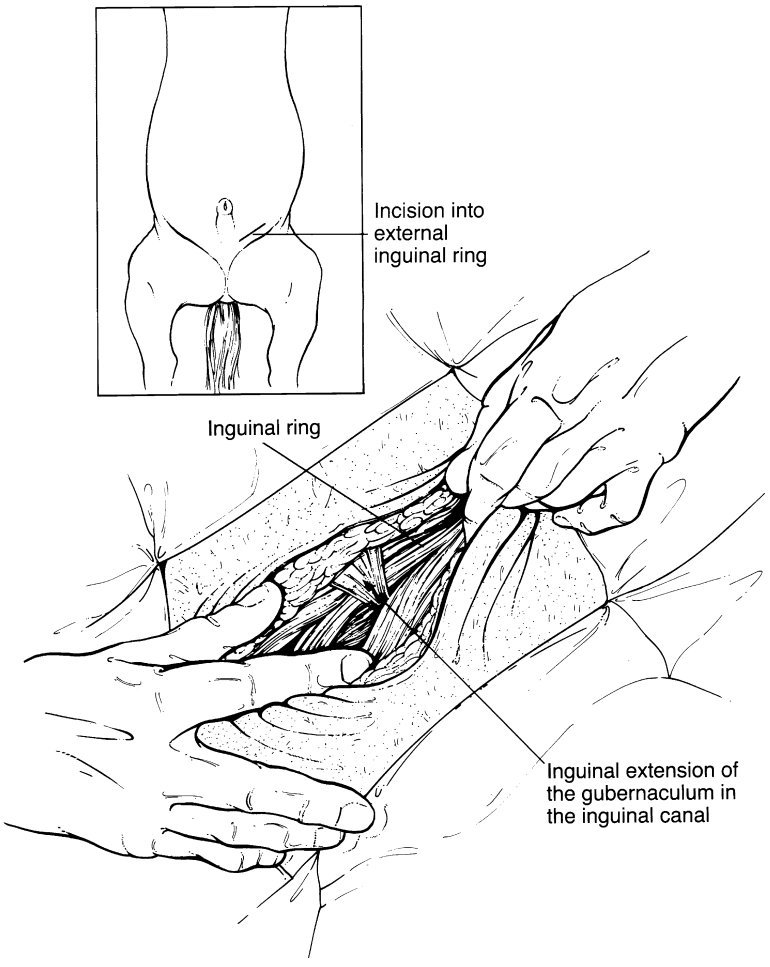

Figure 59-16. An inverted vaginal process can be everted into the inguinal canal by exerting traction on the inguinal extension of the gubernaculum testis. This ligament is a remnant of the gubernaculum and attaches the vaginal process to the scrotum.

and cranial third of the ring. The genitofemoral nerve courses through the canal and can be mistaken for the IEGT. This nerve usually lies farther caudad, in the middle or caudal third of the superficial inguinal ring. The IEGT is most easily located by grasping and retracting loose fascia at the junction of the middle and cranial thirds of the ring with the thumb and index finger of one hand and tracing it into the canal with the index finger and thumb of the other hand. The fascia tears if the IEGT is not contained within the fascia, but if the IEGT lies within this fascia, traction causes the inverted vaginal process to evert into the canal, where it and the epididymis or gubernaculum contained within can be seen and palpated. The everted process is a glistening white structure, usually about the size of a fingertip. A hypoplastic cremaster muscle can be seen attached to the lateral aspect of the vaginal process.

An inverted vaginal process can also be everted using a sponge forceps.[22] A finger is inserted through the vaginal ring into the inverted vaginal process, and a 25-cm curved sponge forceps is introduced beside the finger. The jaws of the forceps are opened and closed to grasp the apex of the vaginal process. Traction applied on the forceps everts the inverted vaginal process. The difficulty of this technique is locating the vaginal ring. The ring can usually be found beneath the third finger when four fingers are inserted into the inguinal canal.

After the vaginal process is everted and stripped of inguinal fascia, it is incised longitudinally (a No. 12 scalpel blade works

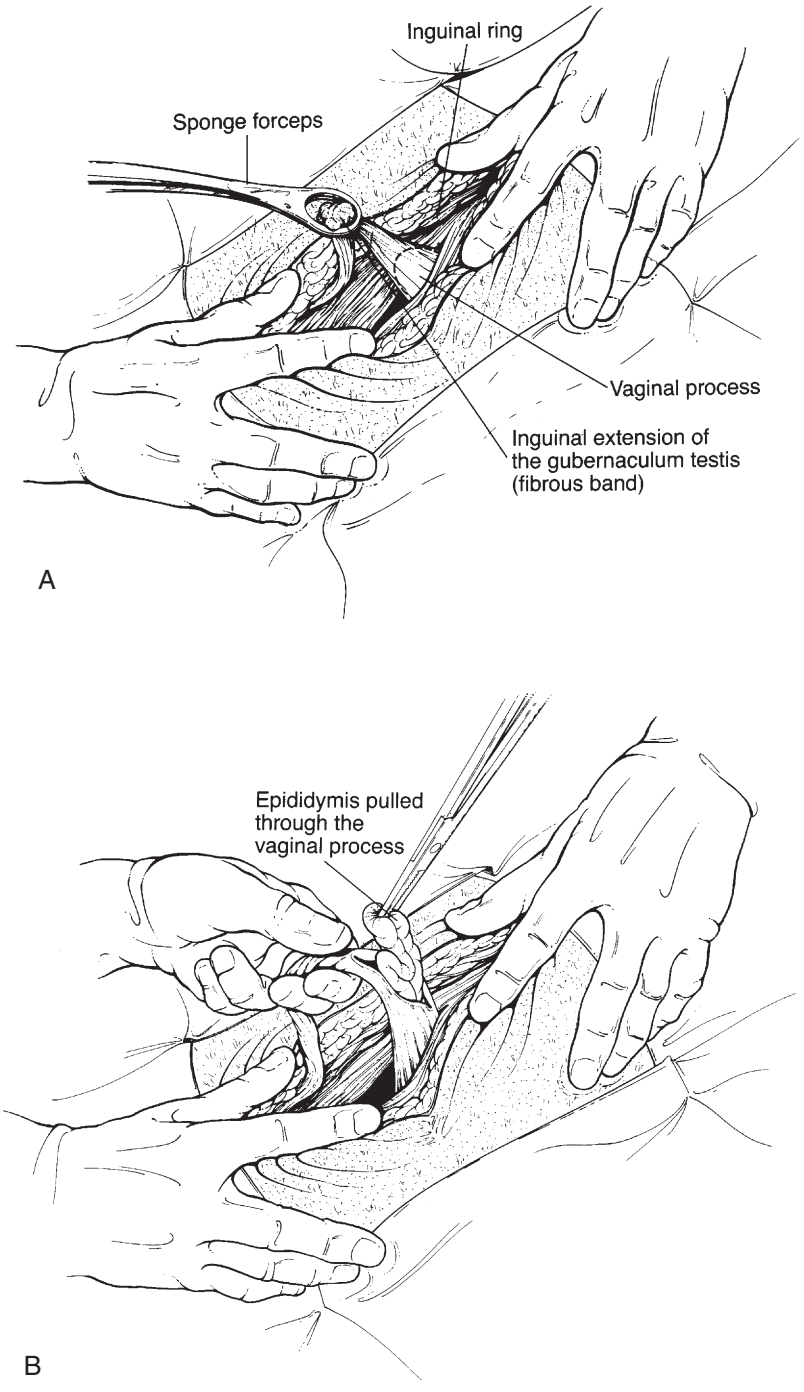

Figure 59-17. A, The everted vaginal process is stripped of inguinal fascia and longitudinally incised. **B,** The epididymis contained within is grasped with a hemostat and exteriorized.

best for this) to expose a portion of the epididymis contained within (Figure 59-17, *A*). The epididymis is grasped with a hemostat and exteriorized until the tail of the epididymis is located (see Figure 59-17, *B*). The proper ligament of the testis connects the tail of the epididymis to the caudal pole of the testis (Figure 59-18), and by applying traction to this structure, the testis can be pulled through the vaginal ring and exteriorized for removal (Figure 59-19). Stretching the vaginal ring to accommodate passage of the testis may not be necessary if the stallion is immature. Usually though, the vaginal ring must be stretched to allow passage of the testis, and this is accomplished by inserting a finger through the incision in the vaginal process and through the vaginal ring. The finger is inserted through the vaginal ring to the level of the second joint, and by flexing the finger, the ring is dilated. The vaginal ring of a mature stallion is usually much more difficult to dilate than that of an immature stallion.

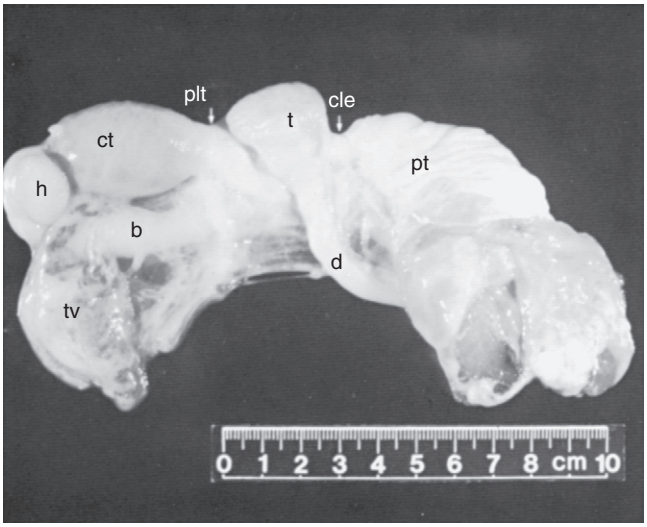

Figure 59-18. Cryptorchid testis. *b,* Body of epididymis; *cle,* caudal ligament of the epididymis; *ct,* cryptorchid testis; *d,* ductus deferens; *h,* head of epididymis; *plt,* proper ligament of the testis; *pt,* parietal tunic; *t,* tail of epididymis; *tv,* testicular vessels.

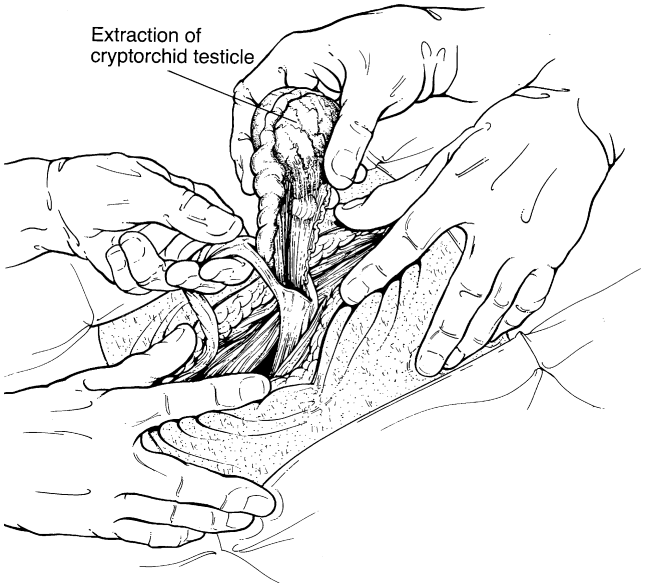

Figure 59-19. With traction on the proper ligament of the testis, the testis is pulled through the vaginal ring.

Rarely, the vascular pedicle of the testis is so short that placing an emasculator proximal to the testis and epididymis is impossible. The cord must then be crushed and transected using an écraseur or severed with scissors after occluding the testicular vasculature with one or two ligatures. The contralateral testis is then removed, and the skin incision is sutured or left to heal by secondary intention.

PREVENTING EVISCERATION

The vaginal ring should be repalpated after the abdominal testis is removed. If the ring accommodates no more than the tips of the index and middle fingers, the horse can be recovered, and unrestricted activity can be safely allowed after several days. If the ring has been dilated beyond this diameter, one of two measures must be taken to prevent evisceration. To prevent evisceration, the inguinal canal can be packed to the level of the vaginal ring with sterile gauze for 24 to 36 hours. The pack is maintained in the canal by partially suturing the skin incision. Evisceration may follow removal of the pack, especially if gauze was inadvertently inserted through the vaginal ring into the abdomen. Not only does gauze in the abdomen prevent the vaginal ring from contracting but it also becomes adhered to viscera. Evisceration can be prevented by palpating the vaginal ring *per rectum* after the pack is inserted and before it is removed to ensure that gauze did not enter the abdomen. After the pack is removed, the horse's activity should be restricted to hand-walking for several days before forced exercise is imposed. Jumping, cantering, and galloping should not be allowed for 3 weeks.

Although the deep inguinal ring is inaccessible for suturing, the superficial inguinal ring can be closed with an interrupted or continuous pattern of heavy, absorbable suture to prevent evisceration. A half-circle, hernia, or kidney needle should be used to suture the inguinal ring. These are heavy, eyed needles with blunt ends, and they are difficult to break. Using a looped suture allows the needle to be pulled through the loop after the first bite, preventing the necessity of tying a knot before proceeding with the continuous suture pattern, and allows the ring to be closed with a doubled strand of suture. Not only does suturing the superficial inguinal ring provide better security against evisceration than does packing the canal with gauze, but it also allows primary closure of the inguinal fascia and skin. Although viscera can enter the inguinal canal, incarceration of intestine by the vaginal ring has not been reported. Inguinal fascia and skin can be sutured after closure of the superficial inguinal ring or allowed to heal by secondary intention. Activity should be restricted to hand-walking for several days before forced exercise is imposed. Heavy exercise should not be allowed for 3 weeks after surgery.

Parainguinal Approach

If the vaginal process cannot be located using the previously described techniques, the testis can be removed noninvasively by converting the inguinal approach to a parainguinal approach.[167] A 4-cm incision is made in the aponeurosis of the external abdominal oblique muscle, 1 to 2 cm medial and parallel to the superficial inguinal ring (Figure 59-20). The incision is centered over the cranial aspect of the ring. The internal

Figure 59-20. Parainguinal approach to cryptorchidectomy. A 4-cm incision is made in the aponeurosis of the external abdominal oblique muscle 1 to 2 cm medial and parallel to the superficial inguinal ring. The incision is centered over the cranial aspect of the ring.

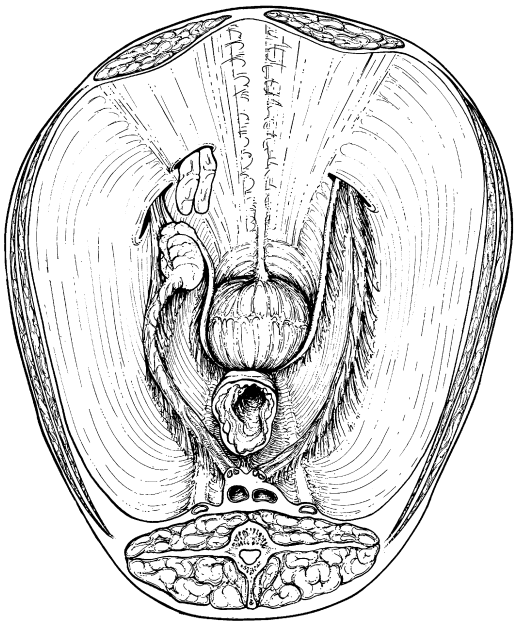

Figure 59-21. Parainguinal approach to cryptorchidectomy. The vaginal ring is palpated caudolateral to the point of entry into the abdomen. Either the epididymis, gubernaculum, or ductus deferens is located at the vaginal ring and exteriorized.

abdominal oblique muscle underlying the aponeurosis is spread in the direction of its fibers, and the peritoneum is penetrated with a sharp thrust of the index and middle fingers. The vaginal ring is palpated caudolateral to the point of entry into the abdomen (Figure 59-21). The epididymis, ductus deferens, and gubernaculum are situated near the ring, and by sweeping the region with index and middle fingers, one of these structures can be grasped between them and exteriorized. The body of the epididymis is followed to the tail. Traction on the proper ligament of testis pulls the testis through the incision.

If difficulty is encountered in locating the epididymis or associated structures, or if exteriorizing the testis is difficult, the incision can be enlarged to accommodate a hand. After excising the testis, the incision in the aponeurosis of the external abdominal oblique muscle is apposed using heavy absorbable sutures in an interrupted or continuous pattern. The subcutaneous tissue and skin can be sutured or left unapposed to heal by secondary intention. The horse can receive exercise after surgery, excluding cantering and galloping, provided that the parainguinal incision was short enough that it could accommodate only several fingers. Unrestricted activity is allowed 3 weeks after surgery.[167]

The parainguinal approach is preferred over the inguinal approach by some surgeons because the vaginal ring is not disrupted.[167] The aponeurosis of the external abdominal oblique muscle is more easily sutured than the superficial inguinal ring.

Suprapubic Paramedian Approach

For the suprapubic paramedian approach, an 8- to 15-cm, longitudinal skin incision is made 5 to 10 cm lateral to the ventral midline.[168-170] The incision begins at the level of the preputial orifice and extends caudally. The large subcutaneous vessels encountered caudally in the incision are ligated. The abdominal tunic and the closely adherent ventral sheath of the rectus abdominis muscle are incised longitudinally, and the underlying fibers of the rectus abdominis muscle are bluntly separated in the same direction. The dorsal rectus sheath, retroperitoneal fat, and peritoneum are penetrated with a finger. The perforation is bluntly enlarged, and a hand is introduced into the abdomen.

The testis is usually encountered near the vaginal ring. If the testis cannot be palpated, accessory structures at the vaginal ring can be located and followed to the testis, or the ductus deferens can be found in the genital fold of the bladder and traced to the testis. Both testes of a bilateral cryptorchid can be removed through one incision, but the contralateral testis is difficult to exteriorize, and its cord usually must be transected with an écraseur. After removing the testis, the abdominal tunic, the subcutis, and skin are each closed separately with interrupted or continuous sutures.

Flank Approach

For the flank approach, a 10- to 15-cm incision is made through the skin and subcutis in the paralumbar fossa of the affected side with the horse standing or recumbent.[171] In a standing horse, the incision site must be anesthetized before the surgery. The external abdominal oblique muscle is transected in the direction of the skin incision, and the peritoneum is exposed by splitting the internal abdominal oblique and transversus abdominis muscles in the direction of their fibers. The peritoneum and retroperitoneal fat are perforated with a finger to enter the abdomen. The testis is located and exteriorized as described for the paramedian approach. If the testis cannot be exteriorized, an écraseur is used to transect the testicular vasculature. After the abdominal testis is removed, the internal and

external abdominal oblique muscles, subcutis, and skin are each closed separately with interrupted or continuous sutures. Closing the peritoneum and transversus abdominis muscle is difficult and not necessary.

Selection of Approach

The paramedian and flank approaches allow removal of only an abdominal testis, because retraction of an inguinal testis into the abdomen can usually be accomplished only with difficulty. Abdominal testicular retention should be confirmed before either of these approaches is used, but often the testicular location cannot be determined reliably. The inguinal approach allows removal of either an abdominal or an inguinal testis. Because an inguinal testis is quickly encountered using an inguinal approach, prior determination of testicular location is not necessary. If the testis is not encountered in the inguinal canal using an inguinal approach, the testis can be removed from the abdomen noninvasively through the vaginal ring or through a small parainguinal incision in the abdominal musculature.

Because the inguinal and parainguinal approaches allow removal of an abdominal testis through a finger-sized abdominal perforation, surgery is rapid, and convalescence is short. The lengthy incision required for the suprapubic paramedian and flank approach prolongs surgery and convalescence. The paramedian approach also increases the risk of postoperative evisceration or herniation. Rarely, an invasive approach is necessary to remove a large, neoplastic, abdominal testis. An abdominal testis can be removed with the horse standing using a flank approach, when general anesthesia is not practical.

Laparoscopic Technique of Cryptorchidectomy

An abdominal testis can be removed laparoscopically with the horse standing or recumbent, but fractious stallions should be anesthetized. Food should be withheld for at least 12 hours before surgery to allow the colon to empty, to decrease the risk of penetrating a viscus when instruments are introduced and to optimize visualization of intra-abdominal structures.[172-174]

To perform laparoscopic removal of an abdominal testis with the horse standing, the horse is restrained in a stock and sedated.[172-175] The flank region is prepared for aseptic surgery, and proposed sites for inserting the laparoscope and grasping forceps are infiltrated subcutaneously and intramuscularly with a local anesthetic agent. The surgical approach is identical to the one described for laparoscopic castration (see "Laparoscopic Castration," earlier).

The testis is located by inspecting the region surrounding the vaginal ring. The vaginal ring and associated structures are easier to see when the horse is standing than when it is anesthetized and recumbent, because the abdominal contents fall away from the inguinal area. The contralateral vaginal ring can be observed by manipulating the laparoscope under the descending colon, or by passing the laparoscope through a small perforation created in the mesocolon of the descending colon, or by elevating the descending colon, either with an instrument placed through an abdominal portal or with a hand *per rectum*.[176] The mesorchium, which contains the testicular vasculature, can be seen coursing caudad from the area of the kidney to the area of the deep inguinal ring. An abdominal testis can be observed to lie anywhere between the kidney and the vaginal ring,[161] but most commonly it is located cranioventral to the ring. The testis is attached to the tail of the epididymis by the proper ligament of the testis, and the epididymis is attached to the vaginal ring and sac by the caudal ligament of the epididymis (ligament of the tail of the epididymis).

The testis and mesorchium are desensitized by injecting a local anesthetic agent into the mesorchium or the testis, using a 30-cm, 18-gauge needle introduced through the flank.[172] Infiltrating the testis with a local anesthetic agent instilled at one site is technically easier than injecting the thin mesorchium in several sites, and the analgesia provided is similar.[177] Desensitizing the testis and mesorchium may not be necessary, especially if caudal epidural anesthesia, using either a combination of 2% mepivacaine (5 mL) and xylazine (0.18 mg/kg), or xylazine (0.18 mg/kg) diluted to 10 to 15 mL with physiologic saline solution, is administered before surgery.[176] An instrument portal close to the vaginal ring is created caudal and ventral to the laparoscopic portal.[172-175] The testis is grasped and exteriorized using grasping forceps inserted through this portal. If triangulation is inadequate, a new portal for the laparoscope can be created between the last two ribs using laparoscopic control.[178] The testicular vessels and ductus deferens are ligated and cut, or crushed and transected using an emasculator, and the stump is returned to the abdomen and inspected through the laparoscope for hemorrhage. If the contralateral testis is also located abdominally, the laparoscopic procedure is repeated on the contralateral side. The abdomen is decompressed by opening the cannula. The abdominal fascia and skin are sutured.

The testicular vessels and ductus deferens of an abdominal testis can also be transected intra-abdominally, with the horse standing, before removing the testis.[176] This technique requires the use of a third portal, created close to the other portals, to introduce instruments used to occlude and transect the testicular vasculature and ductus deferens. Vessel-sealing devices, such as the LigaSure (see Figure 13-13) or SurgRx EnSeal (see Figure 13-14), are ideal for severing and sealing the spermatic cord intra-abdominally. The use of a morcellator to mince the abdominal testis prevents the necessity of enlarging an incision to exteriorize a large testis.[179]

The scrotal testis of a unilateral cryptorchid or an inguinal testis is removed through a scrotal incision, or its ductus deferens and vasculature can be ligated and severed intra-abdominally or severed with a vessel-sealing device (see earlier), which causes the testis to atrophy in the scrotum or inguinal canal.[161] Palpable but nonfunctional remnants of the testis may be detectable in the scrotum several months after the testis is destroyed by ligation of the testicular artery and vein. A scrotal testis of a juvenile stallion or an inguinal testis can be retracted into the abdomen for intra-abdominal transection of the ductus deferens and testicular vessels. The testis is retracted into the abdomen by placing traction on the mesorchium, after enlarging the vaginal ring with scissors. Retracting the testis into the abdomen inverts the vaginal tunic into the abdomen, and the exposed ligament of the tail of the epididymis, which attaches the vaginal tunic to the tail of the epididymis, is severed. The incision in the vaginal ring can be closed with staples or left open.[161,180]

To perform laparoscopic removal of an abdominal testis with the horse anesthetized, the horse is positioned in dorsal recumbency.[172-175,180] After the ventral aspect of the abdomen is prepared for aseptic surgery, a stab incision is made through the umbilicus, and through this incision, the abdomen is insufflated to 10 to 15 mm Hg, as described earlier. A laparoscopic sleeve with a trocar is inserted through the incision into the

abdominal cavity. The trocar is removed and replaced with a laparoscope. The horse is tipped into the Trendelenburg position (i.e., head down approximately 30 degrees) to displace the viscera craniad (see Figure 13-16),[172-175,180] and the laparoscope is directed caudad to view the inguinal areas. Because the hindquarters are elevated, positive-pressure ventilation is necessary. If the testis is not readily visible, it can be located by following the ductus deferens cranially over the lateral ligament of the bladder to the inguinal ring. Traction on the ductus deferens elevates the testis into view.

The testis can be removed before occluding and transecting the testicular vessels and ductus deferens,[172-175] or the testicular vessels and ductus deferens can be occluded and transected intra-abdominally before the testis is removed.[172-175,180] If the testis is to be exteriorized before transecting the testicular vessels and ductus deferens, the instrument portal is created 4 cm cranial and axial to the superficial inguinal ring, on the side of testicular retention. The testis is exteriorized using a grasping forceps introduced into the abdomen through this incision.[172-175] The exteriorized testicular vessels and ductus deferens are ligated and cut or are crushed and transected using an emasculator, and the stump is returned to the abdomen. Both testes of a horse with bilateral, abdominal, testicular retention can be viewed from one portal, but a portal must be created cranial and axial to each inguinal ring to remove each testis. The abdomen is deflated through the laparoscopic cannula, and the portals are closed by suturing the external lamina of the rectus abdominis muscle, the subcutaneous tissue, and skin.

To occlude and transect the testicular vessels and ductus deferens intra-abdominally with the horse anesthetized and positioned in dorsal recumbency, a grasping forceps for manipulating the testis is introduced through a cannula inserted 8 to 10 cm axial and cranial to the superficial inguinal ring.[180] A third instrument portal is created at the cranial, abaxial edge of the sheath to introduce instruments used to occlude and transect the testicular vasculature and ductus deferens.

The testicular vessels and ductus deferens can be occluded using an endoscopic clip or an endoscopic ligating loop and transected using an endoscopic scissor. Ligating the pedicle is generally the most economical method of providing hemostasis, but intra-abdominal ligation requires more experience than does the use of other methods to provide hemostasis. Severing the testicular vessels and ductus deferens with monopolar or bipolar electrocoagulation alone provides adequate control of hemostasis.[181] The risk of accidental thermal injury to adjacent viscera is far greater when using monopolar electrocoagulation than when using bipolar electrocoagulation. Alternatively, vessel-sealing devices, such as LigaSure (see Figure 13-13) or EnSeal SurgRx (see Figure 13-14), can be used to coagulate and sever the testicular vessels and ductus deferens (see Chapter 13). Vessels can also be occluded and transected using an endo-GIA stapler (Endo-GIA 30).[180]

After the attachments to the testis have been transected, the testis is removed from the abdomen by expanding the skin incision over the portal through which the grasping forceps was introduced. Placing the testis in a retrieval bag facilitates exteriorization of the testis and eliminates the risk of dropping it in the abdomen.[181] The testis can also be removed using a morcellator, which minces the tissue, allowing it to be removed by suction through one of the cannulas inserted during the surgery, thus avoiding the necessity of enlarging an incision to exteriorize the testis.[179]

An advantage of intra-abdominal transection over extra-abdominal transection is that with intra-abdominal transection, the severed stump of the vasculature can be inspected before the testis is removed from the abdomen. Exteriorizing a testis causes loss of insufflation, which impairs visibility. A hemorrhaging stump is more easily noted and ligated when the abdomen is still inflated. Insufflation of the abdomen can be preserved during bilateral cryptorchidectomy by occluding and transecting the ductus deferens and vessels of each testis before either testis is removed from the abdomen. In a study in which the abdominal testis of 123 cryptorchid horses was not removed after ligating the spermatic cord at two sites, the entire testis became completely necrotic and incapable of production of testosterone,[20] indicating that removal of the abdominal testis is not necessary after its blood supply has been completely interrupted.

Laparoscopic cryptorchidectomy simplifies locating the cryptorchid testis, prevents disruption of the vaginal ring, which minimizes the likelihood of evisceration, and permits early return to exercise because the incisions are small.[172-175,182] Laparoscopy may be particularly useful in evaluating a horse that displays stallionlike behavior but has the appearance of a gelding, especially when the presence or absence of testicular tissue cannot be determined conclusively by hormonal assay.[174] Laparoscopy is also useful for removing an abdominal testis when the side of the testicular retention is not known.

A disadvantage of laparoscopic cryptorchidectomy is the expense of the equipment.[172-174] A viscus, a large vessel, or the spleen can be penetrated inadvertently if the instruments are not inserted carefully into the abdomen.[183,184] Improper use of electrosurgical coagulation during the procedure may also result in perforation of a viscus.[180] If the procedure is performed with the horse anesthetized, the hindquarters must be elevated to displace the viscera craniad, making positive-pressure ventilation necessary. Familiarity with laparoscopic equipment and experience in laparoscopic techniques are required.

Repair of Inguinal Hernias and Ruptures

Nonsurgical Management

The majority of inguinal hernias are congenital, cause no distress, and spontaneously reduce by the time the foal is 3 to 6 months old.[58,185] Repeated manual repositioning of the herniated viscera may encourage spontaneous reduction, and applying a truss after manually reducing the hernia may speed resolution.[91] To apply a truss, the foal is sedated and positioned in dorsal recumbency. The hernia is reduced, and the superficial inguinal ring is packed with rolled cotton. The cotton is maintained within the ring with elastic gauze and tape wrapped over the back and over both inguinal rings in a figure-of-eight (Figure 59-22). The bandage can be left in place for up to a week. Surgical reduction of a congenital inguinal hernia is not necessary unless contents of the hernia become incarcerated or unless the hernia fails to regress.

Horses with an acquired inguinal hernia, a ruptured inguinal hernia, or an inguinal rupture usually require immediate treatment, because the intestine that has escaped through the vaginal ring or hole in the peritoneum is likely to become strangulated. Nonsurgical reduction of inguinal hernias by external manipulation or rectal traction can be attempted if herniation is diagnosed soon after the onset of signs.[185] Nonsurgical reduction should not be attempted if the viability of the incarcerated

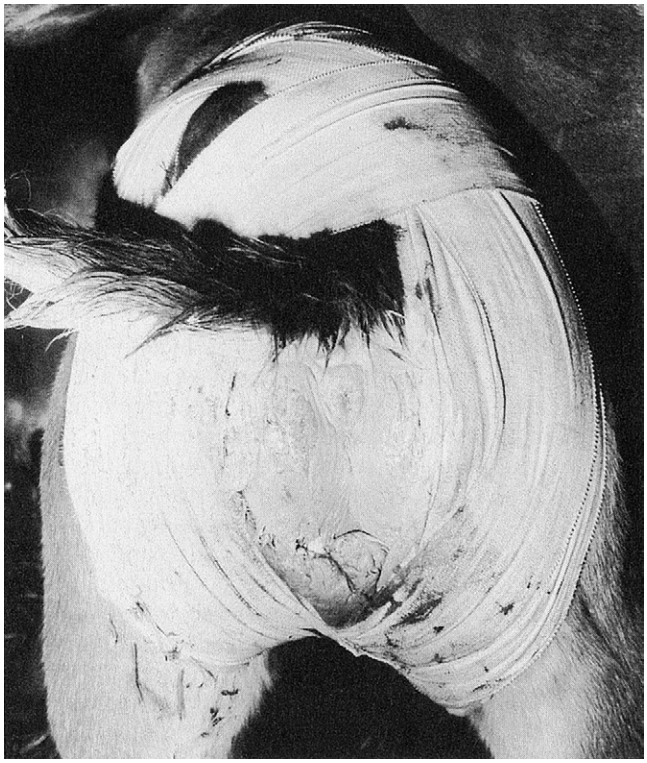

Figure 59-22. Application of a truss after manual reduction of a congenital hernia.

intestine is in doubt or if ruptured inguinal herniation or inguinal rupture is suspected.

To replace hernial contents into the abdomen by external manipulation, the horse is sedated, and the testis of the affected side is grasped and pulled downward so that the vaginal sac is tensed into a rigid, tubelike configuration.[185] With the second hand, the cord is grasped above the testis and slid proximad until herniated intestine is encountered. The second hand maintains traction on the cord and prevents herniated intestine from slipping downward. The first hand releases the testis and grasps and squeezes the cord above the second hand to force the contents of the hernia proximad. The hands are alternated in this manner until the intestine has been returned through the vaginal ring into the abdomen.

If reduction by external manipulation fails, it may be possible to retract the herniated loop of intestine into the abdomen by grasping the loop *per rectum* as it enters the vaginal ring and applying traction.[185,186] The horse should be sedated, and an epidural anesthetic should be administered to minimize the risk of rectal injury or damage to incarcerated intestine. Administering scopolamine butylbromide (Buscopan) to relax the rectum may be helpful. The horse should be observed closely after nonsurgical reduction of an inguinal hernia, because the health of the reduced intestine cannot be assessed directly. Intestinal viability should be monitored periodically by ultrasonographically imaging intestine or by evaluating the horse's peritoneal fluid.

Surgical Management

A congenital inguinal hernia of a foal should be reduced surgically if the hernia cannot be reduced, has escaped into subcutaneous tissue through a rent in the vaginal sac, fails to resolve or enlarges, or is so large that spontaneous resolution is unlikely.[187] To surgically correct inguinal herniation, the foal is anesthetized and positioned in dorsal recumbency. An incision is made directly over the superficial inguinal ring of the affected side, and the vaginal sac is isolated from surrounding fascia using blunt dissection. The scrotal ligament, which attaches the vaginal sac to the scrotum, is transected. While applying traction to the testis, the intestinal contents of the vaginal sac are milked back into the abdomen. Twisting the spermatic cord may facilitate replacement of intestine into the abdomen. The cord is ligated and resected proximal to the superficial inguinal ring. Ligating the cord prevents reherniation, but for added security, the superficial inguinal ring can be closed with absorbable suture placed in a continuous or interrupted pattern. The skin and subcutaneous tissue can be left open to heal by secondary intention or closed primarily.

Nonstrangulating congenital inguinal hernias of foals can be corrected laparoscopically.[187-189] The contents of the hernia are reduced into the abdominal cavity laparoscopically, with the foal anesthetized, and the testis of the affected side is retracted from the vaginal cavity into the abdominal cavity and removed, after ligating and transecting the testicular vessels and ductus deferens. The deep inguinal ring and the vaginal ring are closed using a laparoscopic stapling device or sutures. Closing the deep inguinal ring and the vaginal ring with sutures provides a more secure closure than with staples, because a larger volume of tissue can be incorporated into the closure.[189] The testis of the nonaffected side can also be removed in this manner. Nonstrangulating congenital inguinal hernias can be corrected using a similar laparoscopic technique without removing the testes.[187] Advantages of laparoscopic herniorrhaphy over open herniorrhaphy are the quickness of the procedure, the little postoperative swelling, and the rapid return of the foal to normal activity.

Emergency surgery is usually indicated for horses with an inguinal rupture or a ruptured inguinal hernia and for horses with an acute inguinal hernia that cannot be reduced by nonsurgical manipulation. Surgical correction of inguinal hernias is always indicated if viability of the testis or incarcerated intestine is questionable, because surgery allows these structures to be assessed visually.

Fluids should be administered intravenously in volumes sufficient to combat shock. The horse should be positioned in dorsal recumbency, and the ventral aspect of the abdomen and inguinal area should be prepared for aseptic surgery. The vaginal sac and its contents are exposed and isolated by blunt dissection through an incision created over the superficial inguinal ring. The vaginal sac is opened to expose its hernial contents and the testis. Devitalized intestine may be resected at the inguinal wound, but resection and anastomosis are usually more easily accomplished through a celiotomy. Dilating the vaginal ring and applying traction to the intestine through a ventral midline, paramedian, or parainguinal celiotomy may assist reduction of the herniated intestine. The affected testis should be removed if it appears nonviable or even if its viability is questionable.

If the testis is removed, reherniation through the inguinal canal can be prevented by ligating the spermatic cord and the vaginal tunic as proximally as possible with absorbable suture. For additional security, the inguinal canal can be packed with gauze for 24 to 48 hours, or the superficial inguinal ring can be

closed with heavy absorbable suture. Suturing the superficial inguinal ring gives greater security against reherniation than does packing the inguinal canal. The inguinal fascia and skin can be closed primarily or left unsutured to heal by secondary intention. The celiotomy is closed in routine fashion.

Reherniation through the inguinal canal can be prevented, while at the same time trying to preserve a viable testis, by partially suturing the superficial inguinal ring around the spermatic cord.[190] Suturing starts at the cranial aspect of the ring and ends near the middle of the ring to give a snug, but not tight, closure around the spermatic cord. Difficulty arises in closing the ring adequately to prevent reherniation while still maintaining blood supply to the testis. A more certain method of salvaging the testis is laparoscopic inguinal herniorrhaphy. One method of laparoscopic inguinal herniorrhaphy entails implanting a polypropylene mesh beneath the peritoneum over the deep inguinal ring and the ductus deferens and testicular vessels that enter it, with the horse anesthetized and in the Trendelenburg position.[186] Another laparoscopic technique used to prevent viscera from entering the vaginal ring of a stallion is to cover the vaginal ring with a peritoneal flap using a technique referred to as peritoneal flap hernioplasty.[191] With the horse anesthetized, peritoneum ventrolateral to the vaginal ring is transected on three sides, separated from underlying muscle, inverted, transposed over the vaginal ring, and attached dorsomedially and laterally to the abdominal wall using sutures or staples. A less-sophisticated technique of laparoscopic inguinal herniorrhaphy involves inserting a coiled polypropylene mesh through the enlarged vaginal ring into the inguinal canal.[192] The coiled mesh is pushed into the inguinal canal until the proximal edge of the mesh is distal to the vaginal ring and then allowed to uncoil. The mesh is fixed to the inguinal canal with endoscopic staples. Fibrous reaction to the mesh obliterates the canal, preventing intestine from entering it. The procedure can be performed with the horse standing. Infertility of men, sometimes accompanied by atrophy of the testis, has been reported after inguinal hernioplasty.[193] Infertility is presumably caused by ischemic orchitis because of impaired blood supply, or it may be due to an immunological response.

SPECIAL CONSIDERATIONS

Hemicastration

Fertility is maintained after hemicastration of normal horses. Testes undergo compensatory hypertrophy after hemicastration in response to increased secretion of interstitial cell–stimulating hormone from the hypophysis.[194] A hydrocele, neoplasia, or orchitis of one testis can cause temperature-induced dysfunction of spermatogenesis of the other testis, but removing the diseased testis may allow the remaining testis to regain normal spermatogenesis.[87] Postponing removal of the diseased testis can result in permanent dysfunction of the nondiseased testis.

The descended testis of a cryptorchid should never be removed without first removing the nondescended testis. Failure to find and remove the nondescended testis after the descended testis has been removed enables an unscrupulous owner to fraudulently represent the horse as a gelding and may complicate subsequent surgery, especially if there is no written record of which testis was removed. This significantly increases the cost of cryptorchidectomy.[195] In one study, 8 of 16 unilaterally castrated horses were described as geldings when they were purchased by the owner.[195] Compensatory hypertrophy of the nondescended testis occurs after hemicastration and may complicate removal of an abdominal testis.[18] Removing the descended testis has been advocated to promote descent of the cryptorchid testis,[196] but descent of an abdominal testis becomes impossible within several weeks after birth.[16,197] There is no proof that hemicastration actually results in descent of an inguinal testis. An inguinally retained testis is located easily during inguinal exploration and should be removed along with the descended testis.

Hormonally Induced Testicular Descent

Undescended inguinal testes of boys have been fixed in scrotal position (orchidopexy or orchiopexy) using sutures,[198] but because cryptorchidism of horses may be hereditary, bringing about descent of an inguinal testis by surgical means is considered by most practitioners to be disreputable. There is good evidence that an impaired hypothalamic-pituitary-gonadal axis, which leads to insufficient production of testosterone and dihydrotestosterone, is the main cause of cryptorchidism in boys, and so boys with one or more cryptorchid testes have been successfully treated using GnRH, hCG or LHRH.[40,199] Because testicular descent is hormonally controlled, treatment of a horse for cryptorchidism with hormones would seem to be an attractive mode of therapy, but factors responsible for testicular descent in horses are still obscure, and no specific therapy has been developed to correct the hormonal defect responsible for testicular retention. GnRH and hCG have been administered separately and in combination to horses to bring about testicular descent, but conclusive evidence of efficacy is lacking.[200] Treatment of eight colts, each of which had an inguinally located testis, with 2500 mg hCG twice weekly for 4 weeks, however, was thought to facilitate descent of the inguinal testis of four of the eight colts.[201]

If a cryptorchid testis of a human descends into the scrotum or is surgically placed in the scrotum, degenerative changes within the testis can reverse so that the testis becomes capable of producing sperm; but for degenerative changes to reverse, the cryptorchid testis must enter the scrotum while the child is very young.[202] The optimal time to effect descent of a cryptorchid testis in cryptorchid boys is within 2 years after birth, because after this time, the abnormally warm environment of the undescended testis may irreparably damage the seminiferous tubules.[203] Biopsy of nondescended testes of cryptorchid boys, performed at the time of orchiopexy showed that the concentration of germ cells decreases overtime, beginning as early as 1 year of age.[204] In horses, the age at which a cryptorchid testis must descend into the scrotum to contribute to fertility has not been determined.

After the first few weeks of neonatal life, the vaginal rings contract, reducing the likelihood of descent of an abdominal testis.[16] Hormonal therapy, therefore, is likely to have no effect on descent of an abdominal testis. Testicular growth, brought about by onset of puberty, may bring about descent of an inguinal testis,[18] and this supports the rationale for administration of GnRH or hCG. The effect of exogenous hormones on descent of inguinal testes, however, is unclear. Because genetic studies of equine cryptorchidism indicate that testicular retention is probably hereditary, hormonal therapy to bring about testicular descent is no more ethical than surgically placing the testis in the scrotum.

POSTOPERATIVE COMPLICATIONS

Complications associated with castration are a common cause of malpractice claims against North American equine practitioners.[205] To avoid complications, the practitioner should have a good understanding of male reproductive anatomy and of the various techniques of orchidectomy. The practitioner also should be able to recognize and resolve complications of castration.

Hemorrhage

Excessive hemorrhage is usually the result of an emasculator that is improperly applied or is in imperfect working order. Reversing the emasculator (i.e., placing the cutting edge toward the abdomen) usually results in severe hemorrhage, because the cord is crushed distal to the site of transection. The emasculator should be applied perpendicular to the cord, because transection of the cord other than at a right angle increases the diameter of the severed ends of the testicular vessels. The blade of the emasculator should not be so sharp that the testicular vessels are severed before they are crushed properly. A blade that is too sharp can be dulled by using it several times to cut rope.[190]

The testicular vessels may be insufficiently crushed if scrotal skin is inadvertently included in the emasculator's jaws. The thick cords of a mature stallion may require double application of the emasculator to sufficiently crush the vessels. Using this technique, the parietal tunic and cremaster muscle are crushed and transected separately from the testicular vessels and ductus deferens.

A ligature placed around the entire spermatic cord or around the testicular vessels alone can be used alone, or in conjunction with an emasculator, to prevent hemorrhage. Although a ligature, with or without an emasculator, may be more effective than the emasculator alone in preventing hemorrhage, the use of a ligature may increase the incidence of infection at the surgery site.[149] The increase in risk of infection associated with the use of a ligature is likely the result of reduced resistance of tissue contaminated with bacteria to infection in the presence of foreign material, especially if a nonabsorbable suture is used.

Dripping of blood from the wound for several minutes after emasculation is expected and should cause no concern. Unabated streaming of blood for 15 to 30 minutes is a signal for alarm. The testicular artery is the usual source of severe hemorrhage.[21] Because testicular veins are valved, hemorrhage from these vessels is usually mild. Hemorrhage from scrotal vessels is usually not serious and soon ceases spontaneously. If, after allowing the horse to stand quietly for 15 to 30 minutes, hemorrhage does not diminish, the end of the cord can be grasped, using fingers, and stretched to allow application of a crushing forceps or an emasculator. A crushing forceps with curved jaws, such as a kidney clamp, is easier to apply and maintain in position than a straight forceps. If the horse was castrated while standing, the end of the cord is likely to still be desensitized, and the forceps or emasculator can usually be applied without causing serious discomfort to the horse. The forceps is removed the next day. If the horse was castrated while recumbent, the cord is not desensitized (unless the testes were infiltrated with local anesthetic solution before they were removed), so to safely grasp and crush the end of the cord, the horse may need to be reanesthetized.

If the end of the cord is inaccessible through the scrotal incision, hemorrhage can be stopped by ligating the testicular vessels intra-abdominally using the procedure described for laparoscopic castration.[206,207] Laparoscopic surgery to stop hemorrhage after castration can be performed with the horse standing or anesthetized and positioned in dorsal recumbency. The testicular artery can be coagulated using electrocoagulation or occluded with a laparoscopic suture loop or a vascular clip.

If the end of the cord is inaccessible, and if intra-abdominal ligation of the testicular vessels using laparoscopy is not an option, sterile gauze can be packed tightly into the inguinal canal and scrotum, and the scrotum can be closed with sutures or towel clamps. The pack is removed the next day.

Ten percent formalin (i.e., a 4% aqueous solution of formaldehyde gas created by diluting a 37% to 40% solution of formaldehyde gas with 9 parts of water) has been used with questionable success to stop hemorrhage. In one study, 8 to 16 mL of 4% to 12% formalin administered intravenously to average-sized horses decreased the time of coagulation by 67% for 24 hours.[208] Another study, however, found that 0.37% formaldehyde solution administered intravenously did not appear to enhance primary or secondary hemostasis.[209]

Formaldehyde solution is pyrogenic and accelerates pulse and respiration.[208] Other side effects include restlessness, lacrimation, salivation, elevation of the tail, nasal discharge, increased peristalsis with frequent defecation, sweating, quivering of muscles, signs of severe abdominal pain, and tenesmus. Physical reaction is minimal when 10 mL of 4% formaldehyde solution (i.e., 10% formalin), diluted in a liter or more of isotonic saline solution, is administered intravenously. I have observed dramatic reduction of hemorrhage, with no clinically apparent side effects, within minutes after intravenously administering this amount, but convincing scientific evidence of the safety and efficacy of a formaldehyde solution in reducing hemorrhage is lacking.

Evisceration

An uncommon but potentially fatal complication of castration is evisceration through the vaginal ring and open scrotal incision. A survey of practitioners (performed to determine the type and frequency of complications that occur after castration) reported the incidence of evisceration after castration to be about 0.2%.[149] Horses that eviscerate after castration may have a preexisting, unapparent, congenital, inguinal hernia.[62] Most unapparent inguinal hernias resolve by the time the horse is 3 to 6 months old,[187] so horses castrated before they are 6 months old may be at greater risk of eviscerating after castration than are horses castrated after they are 6 months old. Standardbreds and draft horses may be more frequently affected by postoperative evisceration, because they have a higher incidence of congenital inguinal herniation.[64,149] Based on anecdotal evidence and unpublished data, Tennessee Walking Horses and American Saddlebreds may also have a higher incidence of congenital inguinal herniation and so may be predisposed to evisceration after castration.[210]

To decrease the likelihood of evisceration after castration, the horse's inguinal area should be palpated for the presence of an inguinal hernia before the horse is castrated, and the owner should be questioned as to whether the horse suffered from a congenital inguinal hernia as a foal. If the horse has or has had an inguinal hernia, is less than 6 months old, or is a member

of a breed that has an increased incidence of inguinal herniation, the surgeon should consider taking measures to prevent evisceration. Some clinicians examine the vaginal rings *per rectum* before castrating a horse deemed to be at risk for evisceration because of its breed.[211] The vaginal ring of stallions is normally large enough to accommodate the tip of one finger. According to some clinicians, precautions to prevent evisceration should be taken if a vaginal ring is larger than two fingers. The risk of rectal injury should be weighed against the value of the diagnostic information to be gained before examining the vaginal rings *per rectum*.

For horses that have a higher than normal risk for evisceration, castration should be performed with the horse anesthetized. The testes should be removed using a closed technique, and the spermatic cord should be ligated. The cremaster muscle should be isolated from the cord before the cord is ligated, so that it is not included in the ligature. Including the cremaster muscle in the ligature could cause the ligature to loosen when the cremaster muscle contracts. The closed technique of castration does not diminish the incidence of evisceration unless a ligature is applied to the cord proximal to the site of transection.[64]

Evisceration usually occurs within 4 hours after castration and may be precipitated by attempts to rise from anesthesia.[62,212] Evisceration has occurred up to 6 days after castration,[62] and one horse was reported to have herniated into its sutured scrotum and vaginal cavity 12 days after castration.[213] After intestine enters the canal, peristalsis encourages further protrusion, and intestinal strangulation accompanied by severe signs of colic rapidly ensues. Treatment of a horse that has eviscerated through the inguinal canal is similar to emergency treatment of horses with acquired inguinal herniation.

If evisceration occurs, the protruding intestines should be collected in a clean sheet and tied over the back of the horse to prevent the horse stepping on the intestines. The horse should be transported immediately to a hospital equipped with surgical facilities. The horse should be anesthetized immediately to prevent contamination of and damage to the herniated intestine. Torn mesentery should be repaired, and the intestine should be meticulously cleaned, copiously irrigated, and replaced into the abdomen as rapidly as possible to avoid ischemic damage. Dilating the vaginal ring and applying traction to the intestine through a celiotomy may assist reduction of the intestine. Parenteral antimicrobial therapy should be initiated, and peritoneal fluid should be examined postoperatively if the horse displays signs of septic peritonitis. Factors that negatively influence survival include the amount of intestine that has protruded through the vaginal ring and whether intestinal resection and anastomosis were required.[214]

Protrusion of greater omentum through the scrotal incision after castration causes no immediate distress to the horse and need not be considered a dire emergency (Figure 59-23). The horse should be examined *per rectum* to determine the size of the vaginal ring and to confirm that intestine has not entered the inguinal canal. Exposed omentum is transected as proximally as possible using an emasculator. This can usually be accomplished with the horse standing. To prevent further protrusion of omentum, the horse should be forced to stand in a stall for 48 hours. Suturing the superficial inguinal ring or packing the inguinal canal and scrotum gives additional security against evisceration but is not always necessary, because omentum occupying the vaginal ring prevents intestine from entering the canal. If omentum continues to exit the scrotal incision, the superficial inguinal ring should be sutured.

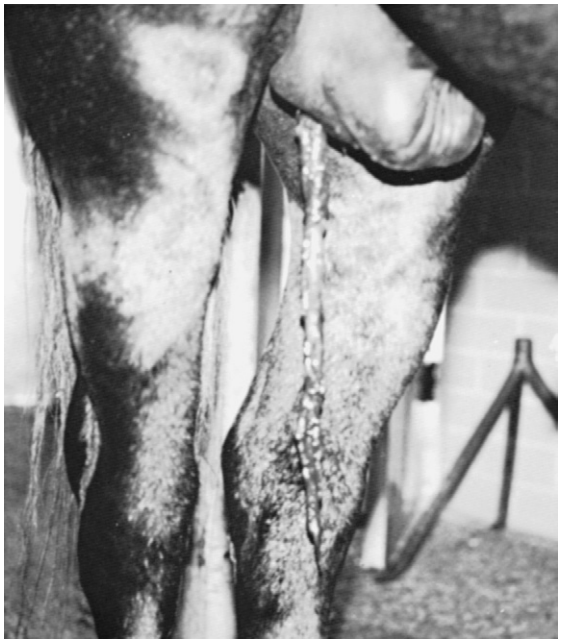

Figure 59-23. Protrusion of greater omentum through the scrotal incision after castration.

Edema

Preputial and scrotal edema develops after nearly every castration and is generally greatest around the fourth postoperative day.[21] Excessive edema, reported to be the most common complication of castration,[149] can usually be avoided by removing a generous portion of the scrotum during castration and by vigorously exercising the horse for several weeks to promote drainage from the open wound. Without vigorous exercise, the scrotal skin may seal and trap fluid containing bacteria or inflammatory products within the scrotal cavity. Excessive edema can usually be relieved by opening the sealed wound with scrotal massage or by inserting a gloved finger into the scrotal cavity and enforcing vigorous exercise. High-pressure lavage of the scrotal wound with tap water administered using a garden hose may assist in keeping the wound open and clean, but a survey of practitioners (performed to determine the type and frequency of complications that occur after castration) indicated that horses that receive hydrotherapy after castration may be more prone to develop excessive swelling and infection of the scrotum.[149]

Signs of Colic

In one study of 238 horses castrated *per primam*, 8.8% displayed transient signs of colic interpreted to be caused by postoperative pain.[157] Horses older than 10 years were significantly more likely than horses younger than 5 years to display signs of colic. A horse that displays signs of colic after castration and that fails to respond favorably to administration of an analgesic drug should be examined to ensure that signs of pain are the result of castration and are not caused by intestinal pain.

Lameness

Occasionally, a horse is presented for lameness examination with a history of having developed the lameness within several weeks after castration.[215] Palpation of the inguinal region on the side of lameness may reveal abnormally firm tissue in the region of the surgical scar, and examination of the abdomen, performed *per rectum*, may reveal swelling of the abdominal wall in the area of the vaginal ring of the affected side. Resection of the stump of the cord proximal to the abnormal portion of the cord resolves the problem.

Septic Funiculitis

A scrotal wound, like any other wound left unsutured to heal by secondary intention, becomes contaminated and may subsequently become infected. The infection remains confined to the scrotal cavity as long as the scrotal incision remains open and draining, and it resolves as the scrotum heals. Septic funiculitis, or infection of the spermatic cord, can occur from extension of the scrotal infection, especially if the scrotal cavity does not properly drain. Septic funiculitis can also be caused by a contaminated emasculator or ligature. The open method of castration, in which the vaginal tunic and cremaster muscle are not removed, may predispose the horse to septic funiculitis,[34] but the condition can occur if the horse has been castrated using the closed technique of orchidectomy. The condition is characterized by preputial and scrotal edema, pain, pyrexia, and sometimes lameness.[66] Septic funiculitis may resolve with antimicrobial therapy and reestablishment of drainage, but removal of the infected stump is likely to be required, especially if the cord has been ligated.

Champignon (French for "mushroom") is a term used to describe a type of septic funiculitis of the stump of the cord caused by infection with *Streptococcus.*[21] Champignon is characterized by purulent discharge and a mushroom-shaped mound of granulation tissue that protrudes from the scrotal incision.[216] This was a common complication of castration before the invention of the emasculator, when hemorrhage was controlled by ligatures or "clams," but now its importance is mostly historical.[21]

A stump chronically infected with pyogenic bacteria is commonly referred to as a scirrhous cord.[32,70] Scirrhous cord, caused by *Staphylococcus,* is sometimes referred to as botryomycosis.[21,70] The scrotal incision of a horse affected with septic funiculitis may eventually heal, but if the septic funiculitis does not resolve, the cord enlarges with granulation tissue and abscesses, which may eventually discharge through sinus tracts. The cord is hard, often painless, and adhered to scrotal skin. The stump may become so large that it mechanically interferes with locomotion of the rear limb. In extreme cases, infection ascends the cord into the abdomen, where a hard mass can be palpated *per rectum* at the deep inguinal ring.[34] Occasionally, the lesion does not become apparent for several years after castration.[21,217] Treatment is removal of the infected mass. The horse is anesthetized and positioned in dorsal recumbency. An incision is made over the scrotal scar or superficial inguinal ring, and the infected cord is isolated from normal tissue (Figure 59-24). The cord is transected proximal to the mass, using an emasculator or écraseur, and the wound is left unsutured to heal by secondary intention. Removing an infected cord within a few weeks after castration is relatively simple, but a chronically infected cord is difficult to remove because of fibrous adhesions to the parietal tunic and large blood vessels associated with the adhesions. The infected portion of the cord is likely to extend far into the inguinal canal if the horse was castrated using the closed technique of orchidectomy, making dissection difficult.

Figure 59-24. The exteriorized portion of the spermatic cord is thickened and hardened from infection. The demarcation between normal and abnormal portions of the cord is obvious. A thick, hard, infected cord after castration is commonly referred to as a scirrhous cord.

Clostridial Infection

Clostridial infection of the castration wound is particularly severe, because tissue necrosis and toxemia produced by clostridial organisms may lead to death within several days. Specific systemic signs of clostridial infection vary according to the clostridial species involved. *Clostridium tetani* causes general spasms and paralysis of the voluntary muscles. Horses develop a characteristic "saw-horse" stance and protrusion of the third eyelid.[218] *Clostridium botulinum* causes flaccid paralysis, and early signs include decreased tone of the eyelids and tail, weakened gait, muscular tremors, and dysphagia.[219] *Clostridium septicum, Clostridium perfringens, Clostridium chauvoei,* and *Clostridium fallax* have been identified as etiologic agents of malignant edema, a highly lethal disease characterized by fever, depression, toxemia, subcutaneous accumulation of gas, and fulminating cellulitis.[220] Treatment of horses for clostridial infection at the castration site includes administration of high doses of penicillin and nonsteroidal anti-inflammatory and analgesic drugs, supportive therapy, radical débridement of all necrotic scrotal tissue, and establishment of scrotal drainage. Horses infected with *C. botulinum* and *C. tetani* can also be treated with antitoxin.[219,221]

Septic Peritonitis

Subclinical, nonseptic peritonitis occurs in many horses after castration because the peritoneal and vaginal cavities communicate.[222] Postoperative, intra-abdominal hemorrhage may be responsible, at least in part, for nonseptic peritonitis, because free blood in contact with the peritoneum causes inflammation.[223] A concentration of nucleated cells greater than 10,000/μL in the peritoneal fluid indicates peritoneal inflammation. A concentration greater than 10,000/μL can be found routinely in

peritoneal fluid for at least 5 days after uncomplicated castration, and a concentration greater than 100,000/μL is not uncommon.[222]

Septic peritonitis occurs when peritoneal inflammation is accompanied by bacterial infection. Signs of septic peritonitis include colic, pyrexia, tachycardia, diarrhea, weight loss, and reluctance to move.[224] Septic peritonitis should not be diagnosed on the basis of the concentration of nucleated cells in the peritoneal fluid alone, because a concentration greater than 10,000/μL indicates only that the peritoneum is inflamed.[222] The presence of degenerated neutrophils or intracellular bacteria in the peritoneal fluid is more indicative of bacterial peritonitis, especially when accompanied by clinical signs.[225] Treatment of horses for septic peritonitis includes administration of antimicrobial and nonsteroidal anti-inflammatory and analgesic drugs, supportive therapy, and peritoneal lavage. Proper drainage of the scrotum must be established. The occurrence of septic peritonitis after castration is rare, perhaps because the funicular portion of the vaginal process is collapsed as it courses obliquely through the abdominal wall[226] and because mesothelial cells of the vaginal process are phagocytic.[80]

Penile Damage

The surgeon may encounter the shaft of the penis while separating scrotal fascia in search of an inguinal testis and, if inexperienced in orchidectomy, may mistake it for the testis. A portion of the shaft of the penis may be stripped of fascia, partially exteriorized, and amputated before its true identity is recognized (Figure 59-25). Even if the penile shaft is recognized before the penis is damaged, damage to the penile fascia created by dissection can result in excessive edema and even paraphimosis. Damage to the urethra during sharp dissection results in severe necrosis of tissue from extravasation of urine into surrounding tissue.[227,228] Penile damage is easily avoided if the surgeon possesses some familiarity with genital anatomy and proper techniques of castration.

Figure 59-25. Stump of penis emerging from a scrotal incision. Much of the shaft of the penis was inadvertently removed during a standing castration.

Excessive edema after castration can result in protrusion of the penis from the preputial cavity, and if the protruded penis is improperly cared for, its integument can be irreparably damaged. Prolonged penile protrusion can result in permanent penile paralysis.[190] Administering a phenothiazine-derivative tranquilizer as a preanesthetic agent can result in priapism or penile paralysis. Penile paralysis and priapism are described in detail in Chapter 60.

Hydrocele (Vaginocele)

A hydrocele, or vaginocele, is a fluid-filled, painless swelling in the scrotum that may appear months or years after castration and is the result of the accumulation of sterile, amber-colored fluid in the vaginal sac.[21] The fluid can often be reduced into the abdomen. The condition is rare and idiopathic, but open castration predisposes to the condition because the vaginal tunic is not removed. Hydroceles may occur more frequently in mules than in horses after castration.[229] Enlargement of the scrotum with fluid may give the horse the appearance of an entire stallion; or the horse may appear to have an inguinal hernia.

If the hydrocele does not increase in size, is not aesthetically displeasing to the owner, and does not inconvenience the horse, no treatment is necessary; otherwise, the hydrocele should be surgically removed. Drainage only temporarily alleviates the condition. To remove the hydrocele, the horse is anesthetized, placed in dorsal recumbency, and prepared for aseptic surgery. Skin is incised directly over the fluid-filled vaginal sac, the sac is bluntly separated from adherent fascia, and the scrotal ligament, which attaches the vaginal sac to the scrotum, is severed. The sac is transected, using a scissors or an emasculator, as proximad as possible. The wound can be sutured or left open to heal by secondary intention.

Continued Masculine Behavior

Even though the serum concentration of testosterone and estrogen decline precipitously to basal levels within 6 hours after castration, libido is normally lost gradually; in one study, mean scores of libido by horses declined gradually until they stabilized on day 56 after castration.[139] Castration is not always successful in completely eliminating objectionable masculine behavior, and some geldings display sexual behavior, such as genital investigation, erection, mounting, and even copulation. Objectionable masculine behavior of geldings is especially common in spring and early summer. Geldings with objectionable sexual libido and aggressive temperament characteristic of stallions are sometimes referred to as *false rigs.*[230]

Persistent masculine behavior of false rigs has been attributed to failure to remove all epididymal tissue during castration, and geldings that display masculine behavior as a result of retention of epididymal tissue are said to be *proud cut.*[230] Epididymal tissue is unlikely to be inadvertently left with the horse, because the epididymis is intimately attached to the normal descended testis. Regardless, androgens are neither produced

nor released by the epididymis, and therefore, the presence or absence of epididymal tissue should not influence masculine behavior.[136] Because masculine behavior of a false rig cannot be justly attributed to retention of epididymal tissue, there is no such thing as a proud cut horse.[230]

Masculine behavior of a false rig has also been attributed to production of testosterone by the adrenal cortex.[136] The circulating concentration of luteinizing hormone is increased after castration in response to the falling concentration of testosterone, and the rise in luteinizing hormone concentration may produce some adrenal hypertrophy. False rigs, however, have no higher concentration of testosterone or dihydrotestosterone in serum than do normal, quiet geldings, and the presence of significant concentration of any other androgen is unlikely.[230] Moreover, administering hCG to false rigs produces no discernable rise in concentration of testosterone in serum.

Heterotopic testicular tissue has been incriminated as a cause of continued masculine behavior after castration.[231] Failure of embryonic primordial germ cells to migrate caudad and dispersion of the cells of the embryonic germinal epithelium from trauma have been theorized as causes of transplantation of testicular tissue throughout the peritoneal cavity of pigs.[231] The occurrence of heterotopic testicular tissue in horses, however, has not been reported. Administering hCG to a horse with heterotopic testicular tissue would probably produce a rise in concentration of testosterone in the serum.

Objectionable masculine behavior after castration is most likely innate and not caused by extragonadal production of testosterone.[230,232] Masculine behavior of false rigs is probably part of the normal social interaction between horses.[230] About 20% to 30% of castrated horses can be expected to display stallionlike sexual interest in mares and aggression toward other horses, and about 5% can be expected to display stallionlike aggression toward people.[232] Because the prevalence of masculine behavior of horses castrated as juveniles is similar to that of horses castrated as adults, castration before puberty is no more effective in preventing objectionable behavior than castration after puberty. These percentages should be considered the normal prevalence of masculine behavior in geldings.

Amputating the stumps of the spermatic cords was reported to abolish objectionable masculine behavior in 75% of 18 false rigs,[233] but no satisfactory rationale was offered to account for the success of this procedure. Because spermatic cords contain no Leydig cells and are incapable of producing androgens, the efficacy of shortening cords to eliminate libido seems doubtful. Limiting social interaction with other horses or imposing stricter discipline may be more successful in eliminating or diminishing undesirable masculine behavior.[230] Administration of a progestagen (such as altrenogest, 50 to 75 mg daily) may ameliorate sexual and aggressive behavior of geldings.[234,235] Progestagens suppress sexual behavior of stallions by inhibiting production of luteinizing hormone from the anterior pituitary gland, resulting in reduced production of testosterone by Leydig cells. But they also exert a direct calming effect on the central nervous system of horses, and this function may be responsible for the effect of progestagens on geldings that display objectionable, masculine behavior.[235]

Incomplete Cryptorchid Castration

The tail of the epididymis of the partial abdominal cryptorchid lies within the inguinal canal, enclosed within a well-developed vaginal process. Excessive length of the proper ligament of the testis and body of the epididymis may allow the tail of the epididymis to descend through the inguinal canal. A surgeon unfamiliar with testicular anatomy and the mechanism of testicular descent may amputate the tail of the epididymis after mistaking it for a hypoplastic inguinal testis (Figure 59-26). The horse naturally continues to display objectionable, stallionlike behavior.

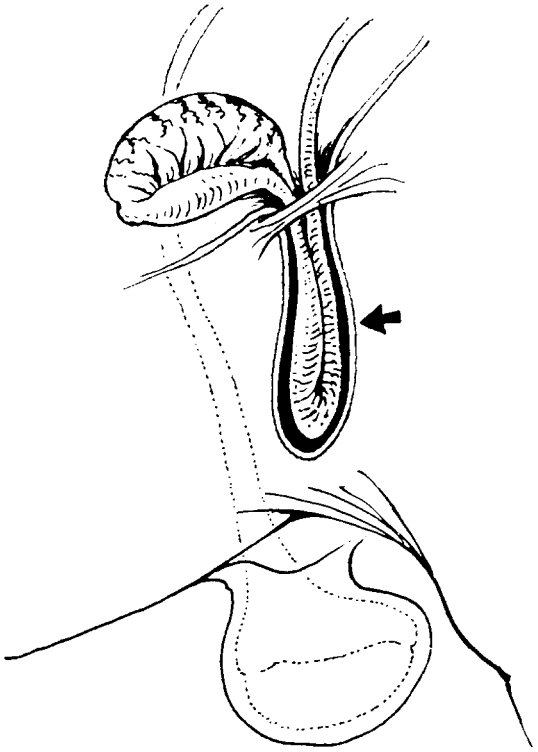

Figure 59-26. The *arrow* points to the epididymis of a partial abdominal cryptorchid. A portion of the epididymis lies within the inguinal canal, enclosed within the vaginal process. This portion of the epididymis could be mistaken for an inguinal testis by an inexperienced surgeon, and amputated.

The vaginal tunic should be opened before removing a structure presumed to be an inguinal testis to ensure that a testis, and not just the epididymis, is contained within. An hCG-stimulation test differentiates a horse with testicular tissue from a false rig. Locating both the end of the epididymis and the stump of the ductus deferens in the inguinal canal at surgery identifies a horse as a partial abdominal cryptorchid whose epididymis has been partially resected.[13]

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CHAPTER
60 Penis and Prepuce

James Schumacher

ANATOMY AND PHYSIOLOGY

Penis

The penis is the male organ of copulation and is composed of erectile tissue that encases the extrapelvic portion of the urethra (Figure 60-1).[1-4] The penis of the horse is musculocavernous and can be divided into three parts: the root, the body or shaft, and the glans penis. The penis originates caudally at the root, which is fixed to the lateral aspects of the ischial arch by two crura (leglike parts) that converge to form the shaft of the penis. The shaft constitutes the major portion of the penis and begins at the junction of the crura. It is attached caudally to the symphysis ischii of the pelvis by two short suspensory ligaments that merge with the origin of the gracilis muscles (Figure 60-2). The glans penis is the conical enlargement that caps the shaft. The portion distal to the point of attachment of the prepuce is referred to as the free part of the penis.[5] The urethra passes over the ischial arch between the crura and curves cranioventrad to become incorporated within the erectile tissue of the penis. The ventral surface of the penis is nearest the urethra, and the dorsal surface is farthest from the urethra.[5]

The mobile shaft and glans penis extend cranioventrally to the umbilical region of the abdominal wall.[4] The body is cylindrical but compressed laterally. When quiescent, the penis is soft, compressible, and about 50 cm long[3]; 15 to 20 cm lie free in the prepuce. When maximally erect, the penis is up to three times longer than when it is quiescent.[6]

Erectile Bodies

Two cavernous spaces make up the majority of the penile shaft: the dorsally located corpus cavernosum penis (CCP), which is responsible for erection, and the ventrally located corpus spongiosum penis (CSP), formerly termed the corpus cavernosum urethrae (Figure 60-3).[3,4] The CCP originates below the ischial arch at the union of the crura, which attach to the ischial arch, and makes up the bulk of the shaft.[2,3] It ends distally in one long central and two blunt ventrolateral projections (Figure 60-4).[4] Along the ventral surface of the CCP runs the urethral groove or urethral sulcus.

The CSP lies in the urethral groove of the CCP and surrounds the urethra (see Figure 60-3).[2-4] The bulb of the penis is the proximal enlargement of the CSP. At its distal termination, the CSP expands into the glans penis, which caps the central projection of the CCP (see Figure 60-4). The tunica albuginea of the glans is thinner than that of the rest of the penis, making it softer in the erect state than the CCP.[6] A long dorsal process of the glans penis extends 10 cm proximally on the dorsum of the CCP. The circular edge of the glans penis is termed the corona glandis, and the collum glandis represents the constriction behind it. The convex cranial surface of the glans contains a deep depression, the fossa glandis, into which the urethra protrudes 1.5 to 3 cm as a free tube surrounded only by thin integument (Figure 60-5).[1,3-4,7] This tubular protrusion of the urethra is termed the urethral process. A dorsal diverticulum of the fossa

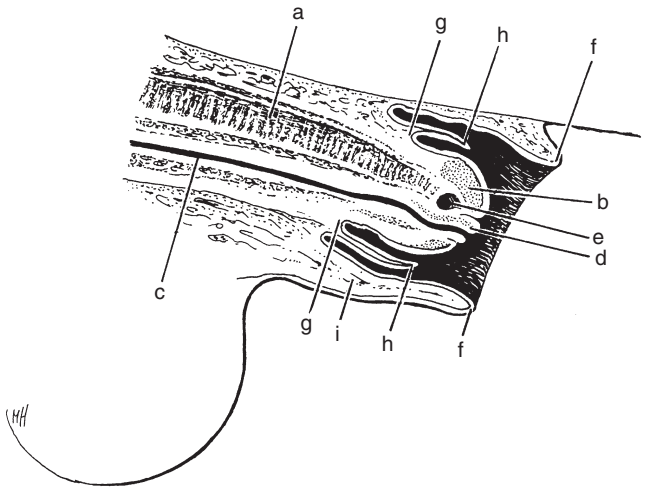

Figure 60-1. The cranial end of the penis in median section *in situ* in the horse, medial aspect. *a,* Corpus cavernosum penis; *b,* corpus spongiosum glandis; *c,* urethra; *d,* urethral process; *e,* fossa glandis; *f,* external preputial orifice; *g,* preputial cavity (internal); *h,* plica preputialis; *i,* prepuce.

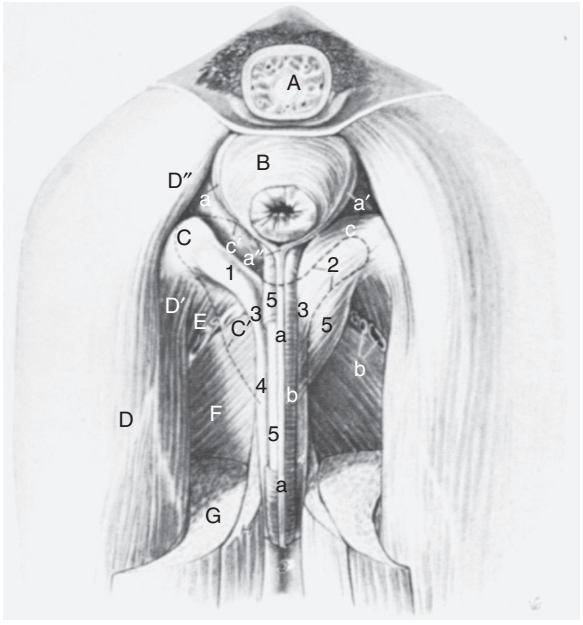

Figure 60-2. Perineum of stallion, deep dissection, caudal aspect. *A,* Cross section through root of tail; *B,* external anal sphincter; *C,* tuber ischiadicum; *D,* semitendinosus; *D′,* short head from tuber ischiadicum; *D″,* vertebral head; *E,* obturator externus; *F,* adductor; *G,* ventral stump of semimembranosus (the dorsal part of the muscle has been removed); *H,* gracilis; *J,* caudal wall of scrotum; *a,* penile part of retractor penis; *a′, a″,* rectal part of retractor penis; *b,* bulbospongiosus, partly removed on the left side to expose the urethra; *c,* right ischiocavernosus, covering right crus penis *(broken line); c′,* outline of left ischiocavernosus, which has been removed to expose left crus penis; *1,* left crus penis; *2,* outline of right crus penis under cover of ischiocavernosus; *3,* union of crura penis; *4,* corpus cavernosum penis; *5,* urethra, surrounded by corpus spongiosum; *6,* muscular branches of obturator vessels. (From Nickel R, Schummer A, Seiferle E, Sack WO: The Viscera of the Domestic Mammals, Paul Parey Verlag, Berlin, 1973.)

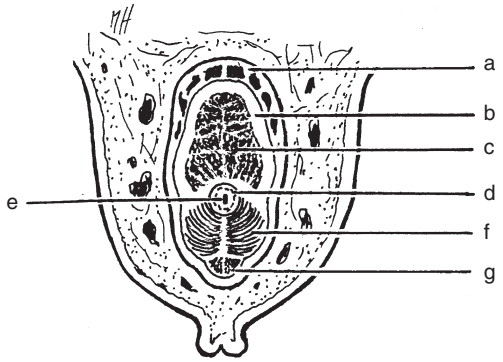

Figure 60-3. Cross section of the penis. *a,* Dorsal veins of penis; *b,* tunica albuginea; *c,* corpus cavernosum penis with dividing trabeculae; *d,* corpus spongiosum; *e,* urethra; *f,* bulbospongiosus; *g,* retractor penis muscle.

glandis, the urethral sinus, is often filled with smegma, a caseous mass of sebaceous matter and epithelial debris. A collection of hardened smegma in the urethral sinus is termed a bean. Large beans have been purported to interfere with urination. Two ventrolateral recesses also project from the fossa glandis.[3] The fossa glandis and its recesses can harbor bacteria capable of producing venereal disease.[8]

The erectile bodies are surrounded by the thick, fibroelastic tunica albuginea, which sends fibrous trabeculae inward to form the supporting framework of the cavernous spaces.[1-2,7] The CSP has thinner trabeculae with larger, veinlike cavernous spaces than seen in the CCP. The tunica albuginea of the CSP is thin and elastic and merges distally with the integument of the glans.[3] The cavernous spaces are lined with endothelial cells and longitudinally oriented bundles of smooth muscle. Tonus of these muscular trabeculae and the retractor penis muscles maintains the nonerect penis within the prepuce. Decrease in tonus, as during micturition, causes the nonerect penis to protrude from the prepuce.

Mechanism of Erection

Penile erection is a neurovascular phenomenon, and the primary hemodynamic event leading to erection is increased arterial flow to the cavernous spaces.[3] Sexual excitement stimulates parasympathetic outflow from the sacral portion of the spinal cord and results in dilation and straightening of the helicine arteries (coiled branches of the deep artery of the penis) and relaxation of the sinusoidal smooth muscles, enabling blood to pass into the sinusoidal spaces.[3,9] Engorgement of the cavernous spaces, which is controlled and finally stopped by the unyielding tunica albuginea and trabeculae, lengthens and stiffens the penis.

Obstruction of venous return from the CCP appears to be important during erection in the stallion.[10,11] Circulation to and from the CCP passes between the ischium and the ischiocavernosus muscles. Contraction of the ischiocavernosus muscles from stimulation of the pudendal nerves occludes arterial and venous flow against the ischium, making the CCP a closed system during peak erection. Compression of the crura by the ischiocavernosus muscles forces blood into the CCP to produce high pressures. In one study, the mean pressure within the quiescent CCP measured 13 mm Hg, but it rose to 107 mm Hg

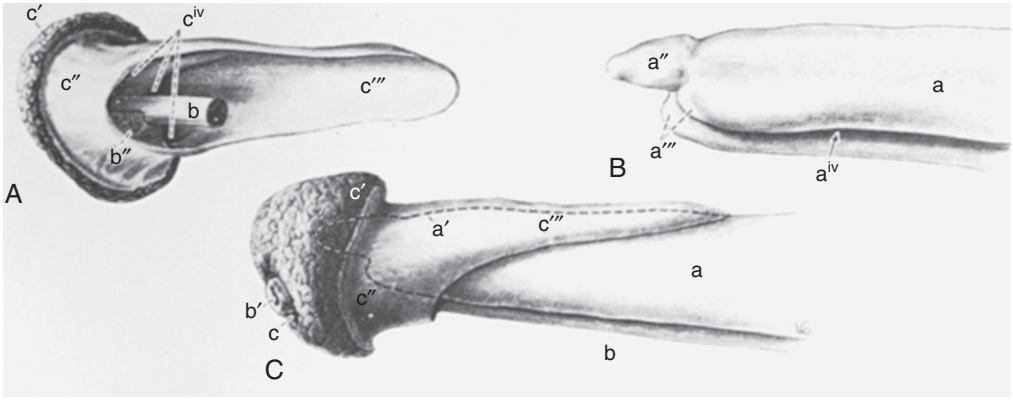

Figure 60-4. Distal end of the penis of the horse. *A,* Caudoventral aspect of the glans, and of the terminal part of the urethra with corpus spongiosum; *B,* ventrolateral aspect of corpus cavernosum; *C,* lateral aspect of tip of the penis (the skin of the penis has been removed proximal to the corona glandis); *a, a′,* corpus cavernosum; *a″,* dorsomedian process of corpus cavernosum; *a‴,* ventrolateral processes of corpus cavernosum; *a^iv,* urethral groove; *b,* urethra, surrounded by corpus spongiosum; *b′,* urethral process and external urethral orifice; *b″,* stump of bulbospongiosus; *c,* fossa glandis; *c′,* corona glandis; *c″,* collum glandis; *c‴,* dorsal process of glans; *c^iv,* recesses on the interior of the glans for the three processes (*a″, a‴*) of the corpus cavernosum. (From Nickel R, Schummer A, Seiferle E, Sack WO: The Viscera of the Domestic Mammals, Paul Parey Verlag, Berlin, 1973.)

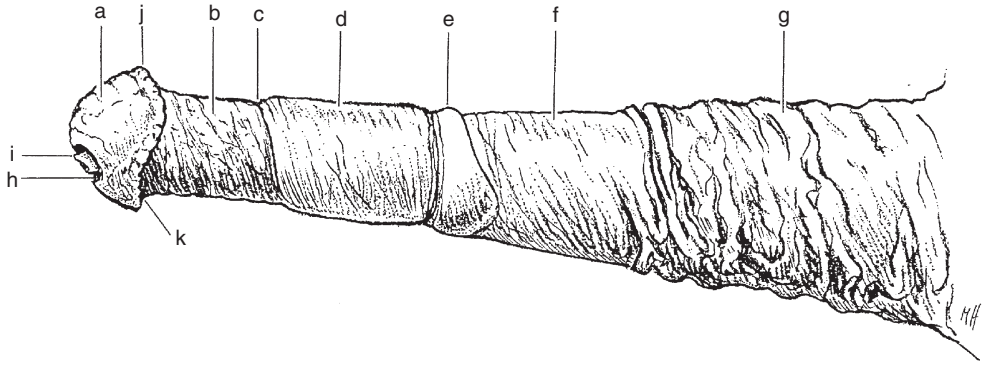

Figure 60-5. Extended penis of a stallion (protruded from the prepuce), left lateral aspect. *a,* Glans penis; *b,* free part of the penis; *c,* attachment of the inner lamina of the preputial fold to penis; *d,* inner lamina of the preputial fold; *e,* preputial ring; *f,* outer lamina of the preputial fold; *g,* internal lamina of the external fold of the prepuce; *h,* fossa glandis; *i,* urethral process; *j,* corona glandis; *k,* collum glandis.

during sexual arousal, and finally to 6530 mm Hg during coitus.[11] Anesthesia of the ischiocavernosus muscles caused diminution of erection by reducing the peak pressure in the CCP to a value close to that of the systemic blood pressure.

During coitus, increased arterial blood flow into the CSP and contraction of the bulbospongiosus muscle lead to increased pressure within the CSP and considerable distention of the glans penis.[12] The glans penis is greatly distensible and may become so large before coitus that the stallion is unable to accomplish intromission. In one study, mean pressure within the CSP was 17 mm Hg during the quiescent state, 76 mm Hg on arousal, and finally, 994 mm Hg during coitus.[12] Contractions of the bulbospongiosus muscles were likely responsible for the high pressure, because anesthesia of this muscle greatly reduced pressure in the CSP during coitus. The CSP remains an open system during erection because vessels entering the bulb of the penis do not pass between an osseous structure and the bulbospongiosus muscles.[12] Blood passes down the CSP to the glans penis and out through the dorsal veins.

Detumescence occurs after ejaculation because parasympathetic impulses diminish and because sympathetic impulses that facilitate emission of semen also cause the helicine arteries to return to their coiled state, thus restricting inflow of arterial blood. Sympathetic impulses also decrease venous compression and allow emissary veins to open, thereby increasing venous outflow.[3,9,13,14]

Muscles

The short, paired, ischiocavernosus muscles that arise from the tuber ischii and the adjacent part of the sacrotuberous ligament attach to the crura and adjacent parts of the body of the penis (see Figure 60-2).[3,4] Contraction of these muscles elevates the erect penis, bringing it into position for intromission. By compressing the penis against the ischium, the ischiocavernosus muscles assist in producing and maintaining erection by impeding venous return from the CCP.

The urethralis muscle surrounds the pelvic urethra and the bulbourethral glands and, by its contractions, forces release of seminal fluid during ejaculation as well as emptying of the last vestiges of urine during urination.[4] The bulbospongiosus muscle, formerly termed the bulbocavernosus muscle, covers

the CSP ventrally and extends nearly the entire length of the penis (see Figure 60-2).[3] It originates near the bulbourethral glands, where it is continuous with the urethralis muscle, and ends at the free part of the penis near the glans penis. It sends transversely directed fibers from the edges of the urethral groove to meet at a median septum. Rhythmic contractions of the bulbospongiosus muscle during ejaculation force blood from the bulb, causing a pressure wave to be sent down the CSP to forcefully expel semen from the urethra.[4]

The ischiourethral muscles extend from the ventral surface of the ischium and crura, pass around the ischial arch into the pelvic cavity, and end at the ventral layer of the urethralis muscle.[3,4] They may assist erection by compressing the dorsal veins of the penis. The paired, longitudinal retractor penis muscles arise on the ventral surface of the first few coccygeal vertebrae and pass ventrad on each side of the rectum to form a loop beneath the terminal end of the rectum and anus (see Figure 60-2).[3,4] From the loop, the muscles pass distad along the bulbospongiosus muscle and end at the glans penis. They retract the penis into the prepuce after erection or protrusion.

Blood Vessels, Nerves, and Lymphatics

Arteries supplying the penis include the terminal branches of the internal pudendal (or internal pudic), obturator, and external pudendal (or external pudic) arteries.[1,2,4] The external pudendal artery supplies the cranial (or dorsal), artery of the penis, a major source of blood for erectile tissue.[2] It supplies a branch to the scrotum and continues as the caudal superficial epigastric artery, which provides branches to the prepuce.[2,3] The deep arteries of the penis originate from the obturator arteries and supply the CCP.[9] The internal pudendal artery supplies the pelvic portion of the urethra and terminates in the CSP as the artery of the bulb of the penis, which supplies the CSP.[2,3] Blood flows from the penis through a venous plexus on the dorsum and sides of the penis.[2,4] The venous plexus is emptied by the external pudendal and obturator veins; blood is carried from the root by the internal pudendal veins.[4]

Nervous supply to the penis is primarily via the pudendal nerves and the pelvic plexus of the sympathetic nervous system.[4] The pudendal nerves branch into the dorsal nerves of the penis, and the sympathetic fibers supply the smooth muscle of the vessels and the erectile tissue. The deep perineal and caudal rectal nerves supply the bulbospongiosus, ischiocavernosus, and retractor penis muscles.[3] Efferent lymphatic vessels of the penis carry lymph to the superficial and deep inguinal lymph nodes.

Accessory Genital Glands

The accessory genital glands of the horse are the paired seminal vesicles, the prostate, the paired bulbourethral glands, and the paired ampullae of the ductus deferens (Figure 60-6).[3,4,15] The accessory genital glands are fully developed in the sexually mature stallion but resume their juvenile size in the gelding.[5] The ducts of these glands empty into the pelvic urethra and provide the major portion of the ejaculate and serve to transport, nourish, and buffer spermatozoa.[9]

The distal 10 to 15 cm of each deferent duct widens to form an ampulla, the wall of which is thickened with secretory glands.[6,16] Each ampulla is evident as an enlargement near the midline of the pelvic floor. The seminal vesicles are two hollow, pear-shaped glands that lie on the dorsal surface of the neck of the bladder lateral to the ampullae.[3,4] They are often difficult to palpate *per rectum*, but identification can be enhanced if the stallion is sexually aroused before palpation.[15] Each vesicle of the stallion is 10 to 15 cm long and 3 to 6 cm wide, but those of geldings are much smaller. The seminal vesicles narrow as they converge toward the midline, and each forms a single excretory duct, which travels beneath the prostate and opens together with or beside the ipsilateral ampulla on the colliculus seminalis. The combined terminal portion of the ampulla and excretory duct of the seminal vesicle is termed the ejaculatory duct. The seminal vesicles are true glandular structures and not just reservoirs for the storage of spermatozoa as was once thought.[9] The secretions of the seminal vesicles are viscous and contribute the major portion of the volume of ejaculate.[3,4,9] Secretions from the seminal vesicles are the last to enter the urethra.[5] Seminal vesiculitis, a rare but important problem in stallions, may be associated with infertility. Surgical removal of chronically infected vesicles has been described.[17]

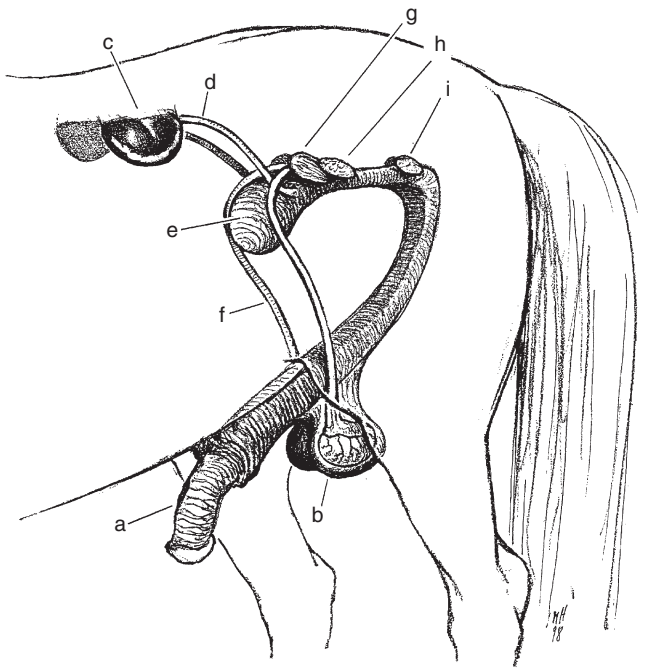

Figure 60-6. Graphic representation of the urogenital tract of the stallion. *a,* Penis; *b,* testes; *c,* kidneys; *d,* ureters; *e,* urinary bladder; *f,* ductus deferens; *g,* seminal vesicles; *h,* prostate gland; *i,* bulbourethral glands.

The prostate is a nodular, bilobed gland that lies dorsal to the neck of the bladder.[3,4] The prostate of a stallion can, with some difficulty, be palpated *per rectum*, especially if the horse is sexually aroused. Each lobe is 5 to 9 cm long, 3 to 6 cm wide, and about 1 cm thick; the lobes are connected across the midline by a 3-cm-long isthmus. Prostatic secretion from each lobe is carried through 15 to 20 prostatic ducts, which open into the urethra through small, slitlike openings located lateral to the colliculus seminalis. The prostate produces a watery, alkaline secretion that neutralizes the acidity of fluid entering the urethra from the ductus deferens.[9]

The two bulbourethral (or Cowper) glands are situated on the dorsolateral surface of the urethra at the ischial arch, 2 to 3 cm caudal to the prostate.[1-4,6] Each is covered by

a bulboglandularis muscle. The bulbourethral glands of the stallion are 4 to 5 cm long and 2.5 cm wide and are difficult to palpate *per rectum*. Six to eight excretory ducts from each gland open in two longitudinal rows of small papillae on the dorsal surface of the pelvic urethra caudal to the openings of the prostatic ducts. The bulbourethral glands produce an alkaline, mucinous secretion that clears the urethra of urine before ejaculation and lubricates the urethra for the passage of seminal fluid.[9]

Prepuce

The prepuce, or sheath, is a voluminous, folded sleeve of integument covering the mobile portion of the quiescent penis.[4] The prepuce consists of the haired external lamina, which is continuous with the skin of the abdominal wall, and an internal lamina, which is in contact with the penis (see Figure 60-5).[3,4,7] The external lamina extends craniad from the scrotum to within 5 to 8 cm of the umbilicus and is continuous with the internal lamina at the opening of the prepuce, the preputial orifice (see Figure 60-1).[3] Close to the cranial extent of the external lamina are two rudimentary teats. The preputial raphe, a cranial continuation of the scrotal raphe, divides the external lamina sagittally on its ventral midline. The prepuce is supported by an elastic suspensory ligament that lies within the external lamina and is derived from the abdominal tunic.[18] When the horse trots or canters, movement of the penis within the prepuce often creates a sucking noise.

The prepuce of the horse differs from that of other species in that it is formed by a double fold of preputial skin, one inside the other.[2-4,7] When the penis is retracted, the internal lamina doubles on itself to form a cylindrical internal fold, the plica preputialis or preputial fold. The preputial cavity is thus divided into external and internal cavities, of which the external is the more spacious.[3] The opening of the plica preputialis is termed the preputial ring. When the plica preputialis unfolds during erection, the preputial ring can be recognized as a thickened band on the extended penis.[1,2]

The penis is not free in the preputial cavity at birth, because epithelium of the internal lamina of the prepuce and epithelium of the free part of the penis are fused into a single lamina. The lamina is split into external and internal laminae by a cytolytic process that forms vesicles that coalesce to form the preputial cavity.[19,20] Separation of the internal and external laminae occurs in the first month after birth and is controlled by androgens.

DIAGNOSTIC PROCEDURES

History

Most preputial and penile abnormalities are easily diagnosed from the horse's history and during physical examination, and further studies are not required. History pertaining to problems of the penis and prepuce may include such information as copulatory performance, drug therapy, behavioral changes, conception rates, duration of disability, and previous injuries, illnesses, or urogenital surgery.

Clinical Examination

Urination

Physical examination of a horse with a penile or preputial disorder should include observation of urination. The horse can sometimes be stimulated to urinate by placing it in a freshly bedded stall; shaking the bedding while whistling may increase the horse's urge to urinate. If this technique fails, intravenously administered furosemide generally results in urination within 15 minutes. If the horse makes painful and unsuccessful attempts to urinate, urethral obstruction should be suspected, and the bladder should be palpated *per rectum*. If the bladder is distended, it should be catheterized to relieve its distention and to determine the location of urethral obstruction. A large accumulation of hardened smegma within the fossa glandis can produce stranguria by distorting the urethral process, but this accumulation is readily identified and easily removed.

Erection and Ejaculation

A breeding stallion that is experiencing difficulty with erection or ejaculation should be observed servicing a mare. Inability of a sexually excited stallion to achieve erection could be caused by a vascular shunt from the CCP to a vessel outside the tunica albuginea or by fibrosis of cavernous tissue from an unresolved episode of priapism. Shunts between the CCP and one or more dorsal veins of the penis could result from a congenital anomaly or from laceration or rupture of the tunica albuginea. Damage to the CCP caused by priapism (persistent erection without sexual excitement) can be assessed by palpating the cavernous tissue. Fibrous, noncompliant cavernous tissue indicates that the CCP has been permanently damaged. A stallion that is reluctant to ejaculate or displays pain during ejaculation may suffer from a urethral rent or seminal vesiculitis. If so, semen and urine should be examined grossly and microscopically for the presence of blood, and the urethra should be endoscopically inspected for evidence of seminal vesiculitis or a urethral abnormality.

Palpation

The penis can be palpated as it lies retracted in the prepuce by inserting a gloved and lubricated hand through the preputial orifice and preputial ring. This may be the only method of physically evaluating the penis and internal preputial lamina of a horse with phimosis (an inability to protrude the penis from the prepuce because of a stricture of the preputial orifice or preputial ring). The external and internal preputial cavities, preputial ring, and free part of the penis, including the urethral sinus and process, can be evaluated by palpation. Beans within the urethral sinus can usually be palpated by compressing the tip of the penis. Dense, brown-black, greasy smegma is normally encountered at the preputial fornix.

Visual Inspection

To ascertain the exact nature and extent of penile or preputial abnormalities, visual inspection of the horse's penis and internal preputial lamina is usually necessary. The horse's penis can be protruded by administering xylazine HCl or preferably by stimulating sexual arousal. Administration of phenothiazine-derivative tranquilizers to stallions should be avoided because of association of these tranquilizers with penile paralysis and priapism. The gelding's penis can be protruded by administering a tranquilizer or sedative or by placing a loop of gauze behind the corona glandis and with steady traction overcoming the pull of the retractor penis muscles. Producing penile

protrusion by chemical means is preferable to pulling the penis from the prepuce, because traction on the penis is resented by the horse and could damage the penis.

The penis can be desensitized and extruded by anesthetizing the pudendal nerves at the level of the ischial arch.[2,21] The point of injection is 2 cm dorsal to the ischial arch and an equal distance lateral to the anus. The needle is inserted at an angle until its point contacts the ischial arch on the midline where the pudendal nerves course around the ischium. The penis usually protrudes within 5 minutes after deposition of 3 to 5 mL of a local anesthetic agent adjacent to each nerve. Unless prolonged penile desensitization is required, a short-acting local anesthetic, such as lidocaine HCl, should be used to avoid prolonged penile protrusion. Desensitization and extrusion of the penis and internal lamina of the prepuce by anesthetizing the right and left pudendal nerves where they are embedded in the sacrotuberous ligament is described, but this pudendal nerve block is difficult.[22]

The urethral process and fossa glandis should be inspected for lesions of cutaneous habronemiasis. With the penis extended, the entire internal preputial lamina is visible and can be evaluated for wounds, scars, hematomas, neoplasia, and granulomas. Penile and preputial wounds should be closely examined to determine if they penetrate the tunica albuginea or invade the urethra. Leakage of urine from a traumatized area may be noted, especially when the horse urinates. A hematoma should be differentiated from an abscess. Physical findings of hematoma include penile swelling and ecchymosis, particularly noticeable in nonpigmented areas. Aspiration of a hematoma confirms the diagnosis. Examination of a horse with paraphimosis (an inability to retract the protruded penis into the prepuce) should include an evaluation of penile sensory innervation, because protrusion accompanied by penile paralysis may be permanent.

If preputial or penile neoplasia is suspected, the entire external genitalia should be examined meticulously for other primary lesions, and the inguinal regions should be palpated to detect enlarged lymph nodes. Superficial inguinal lymph nodes may enlarge initially from inflammation but later from malignant infiltration. Lymph nodes adhered to overlying skin or with fistulous tracts have most likely been infiltrated by malignant emboli. Metastases to internal lymph nodes may be detected by palpation *per rectum*. Recognition of carcinoma of the external genitalia should lead to examination of other structures commonly affected by carcinoma, such as the third eyelids and the perineum.

Other Diagnostic Procedures

Endoscopy

Endoscopy may be useful for identifying the source of hemorrhage noted to occur during urination or ejaculation. Endoscopy of the urethra and bladder is performed with the horse standing and sedated, using a sterile, 100-cm (or longer), flexible endoscope with a diameter no larger than 12 mm. An endoscope with an outside diameter of 9 or 10 mm is suitable for most male horses. The endoscope should be capable of distending the urethra with air, and it should not be so large that it restricts exit of air introduced proximal to it.

To endoscopically examine the urethra, the distal end of the penis is grasped behind the glans by an assistant. The urethra is occluded around the endoscope while the urethra is insufflated as the scope is inserted proximad. The urethral mucosa is normally pale pink and has longitudinal folds, but when the lumen is distended, the mucosa becomes reddened and assumes a smooth, tubular configuration. The blood-filled cavernous spaces of the corpus spongiosum penis that surround the translucent urethra should not be mistaken for an inflamed mucosa.

Two longitudinal rows of six to eight papillae that mark the openings of the excretory ducts of the two bulbourethral glands are seen on the dorsal surface of the pelvic portion of the urethra, and lateral to these papillae lies a longitudinal row of papillae that mark the urethral openings of the excretory ducts of the urethral glands. After advancing the scope beyond the papillae of the bulbourethral glands, the colliculus seminalis, which contains the opening of each of the two ductus deferentes and the opening of the duct of each of the two seminal vesicles, is identified as a round prominence on the dorsal aspect of the urethra. The small orifice of the uterus masculinus, a remnant of the ducts of Müller and the homologue of the uterus and vagina, can sometimes be seen on the center of the colliculus. The openings of the seminal vesicles are crescent-shaped. The lumen of a seminal vesicle can be examined for evidence of infection by passing a 10-mm or smaller diameter endoscope into its duct.[23] The orifices of the 15 to 20 prostatic ducts open into the urethra through small, slitlike openings located lateral and proximal to the colliculus seminalis, but these orifices are difficult to identify.

Ultrasonography

Ultrasonography can be used to assess the physical status of cavernous tissue and to identify urethral lesions, such as calculi or stenosing scars. Ultrasonographic examination of a penile hematoma may identify a rupture of the tunica albuginea. Abnormality of an accessory sex gland can sometimes be detected using transrectal ultrasonography.[15] The glands can be more readily identified if the stallion has been sexually aroused before ultrasonographic examination.

Cavernosography

Cavernosography may be useful for determining the cause of persistent impotence. Contrast medium (100 to 200 mL of iohexol, a 24% water-soluble, organic iodine radiographic contrast medium) is injected into the CCP, and serial radiographs of the penis are obtained. If shunts are present, contrast medium appears in the nutrient vessels of the penis and prepuce. If trabeculae are damaged, the sinusoidal spaces fill incompletely with contrast medium. Cavernosography also may be useful in identifying a rupture or laceration of the tunica albuginea of the CCP.

Miscellaneous Diagnostic Procedures

Urethral and ureteral catheterization may be useful for determining the source of hemorrhage observed to occur during urination or ejaculation or to obtain fluid expressed from the seminal vesicles for cytologic examination and culture.[23] Cytologic or histologic evaluation of penile and preputial lesions may be necessary to distinguish between various diseases, such as cutaneous habronemiasis and squamous cell carcinoma.

PENILE AND PREPUTIAL DISORDERS

Penile and Preputial Injuries

Etiology

Horses can lacerate their penis while jumping barriers, in attempting to breed a mare over a fence, or by falling on sharp objects. During coitus it may be caused by the mare's tail hairs or a loosely tied "breeding stitch." Stallion rings that are too small or improperly cleaned may cause erosions of the shaft.[24] Penile hematomas are usually caused by trauma to the erect penis and can occur when stallions are pastured with other horses or are permitted to breed improperly restrained mares. Severe bending of the penile shaft during coitus or semen collection may cause tearing of a corporeal body or subfascial vessels on the surface of the penis.

Penile lacerations or erosions are usually superficial, but lacerations into the cavernous tissue and urethra have been reported.[25-29] A tear in the urethral sinus leading directly into the CSP caused severe penile hemorrhage in a stallion during coitus,[25] and improper castration of two horses caused urethral damage and necrosis of tissue at the scrotum from escape of urine.[28-29]

Hematomas usually arise from rupture of the extensive vascular plexus located subfascially on the surface of the penis,[30] but occasionally a hematoma originates from a torn corporeal body.[31] Rupture of the CCP is sometimes referred to as fracture of the penis. Rupture of the bulb of the penis, presumably from a blow, eventually led to the death of a stallion by causing urethral stenosis and subsequent rupture of the bladder.[32] A hematoma confined within the intact tunica albuginea of the CCP, apparently caused by a breeding accident, resulted in deviation of a stallion's penis during erection, presumably from disruption of blood flow through the cavernous structure.[33] Aspiration of the hematoma, using ultrasonographic guidance, resulted in straightening of the penis.

Pathophysiology

Unsutured preputial lacerations inevitably become infected, and migration of infection through the loose preputial connective tissue results in cellulitis and generalized swelling. If cellulitis and swelling become severe, the penis and internal lamina of the prepuce protrude through the preputial orifice. Superficial wounds, if properly treated, heal without complication, but large unattended wounds that heal by cicatrization may restrict action of the prepuce. An unsutured wound into cavernous tissue may lead to impotence caused by creation of a shunt between the cavernous tissue and the superficial penile vasculature. Although longitudinal urethral lacerations generally heal without stricture when left to heal by secondary intention, unsutured, transecting urethral lacerations usually heal with obstructing stenosis. An improperly attended urethral injury could result in a cutaneous-urethral or cavernosourethral fistula.

Rupture of superficial penile vessels or corporeal bodies causes extravasation of blood into the loose preputial fascia. Extreme preputial swelling may occur within minutes of injury and may prevent the horse from retracting its penis into the preputial cavity. The penis may rapidly enlarge to several times its normal size. The hematoma may interfere with venous and lymphatic drainage by impinging on undamaged veins and lymphatic vessels, thus exacerbating the swelling,[24] or the hematoma may interfere with urination by impinging on the urethra.[32]

Treatment

OPEN WOUNDS

To avoid infection, fresh penile and preputial wounds should be débrided and sutured. Sutures can be either absorbable or nonabsorbable but should be soft and nonirritating. Infected wounds should be cleansed with a mild antiseptic solution several times daily and covered with an antimicrobial ointment. If the urethra is completely disrupted, it should be reapposed with absorbable sutures; stenting the urethral lumen with a male urinary catheter during surgery simplifies the anastomosis. Stenosis of the urethra caused by cicatrix formation after injury can sometimes be relieved by transendoscopic laser ablation.[34] Often, penile amputation is the most expedient means of treating complete urethral disruption accompanied by severe trauma of surrounding tissue, especially if the injured horse is a gelding.[27]

HEMATOMAS

Treatment of a horse with a penile hematoma should be instituted immediately after injury and aimed at decreasing hemorrhage. Compressing the penis and internal lamina of the prepuce with a pneumatic bandage or tight wrap may relieve edema and minimize hemorrhage. To compress the penis and prepuce, the horse is anesthetized, and starting at the distal end of the penis, the penis and internal lamina of the prepuce are wrapped snugly from the glans to the preputial orifice with an elastic bandage.[35] The wrapped penis and prepuce and the preputial orifice are massaged until the wrap loosens from the decrease in size of the penis and prepuce. The process is repeated until maximal decrease in size is achieved. The penis and internal lamina of the prepuce should then be supported against the abdomen or within the preputial cavity to diminish hemorrhage and edema. Hydrotherapy with cold water may hasten vasoconstriction. If the hematoma continues to expand despite treatment, the area of the hematoma should be examined ultrasonographically for evidence of a rent in the tunica albuginea, or it should be surgically explored. Failure to repair a rupture of the tunica albuginea could result in formation of a shunt between the damaged erectile body and the dorsal veins of the penis.

Aftercare

To avoid erection and more hemorrhage, a horse with a penile or preputial injury should not be subjected to sexual stimuli. Because exercise may exacerbate hemorrhage, the horse should initially be closely confined. After 5 or 6 days, when hemostasis is ensured, the horse should be exercised lightly to decrease the edema. Hot packs applied to the penis at this time stimulate vasodilatation and thus resorption of the hematoma.

Paraphimosis

Etiology

Paraphimosis, or the inability of the horse to retract its protruded penis into the prepuce, occurs most frequently from preputial edema caused by genital trauma, such as preputial laceration, penile hematoma, or castration. Paraphimosis may be a manifestation of disease characterized by extensive edema,

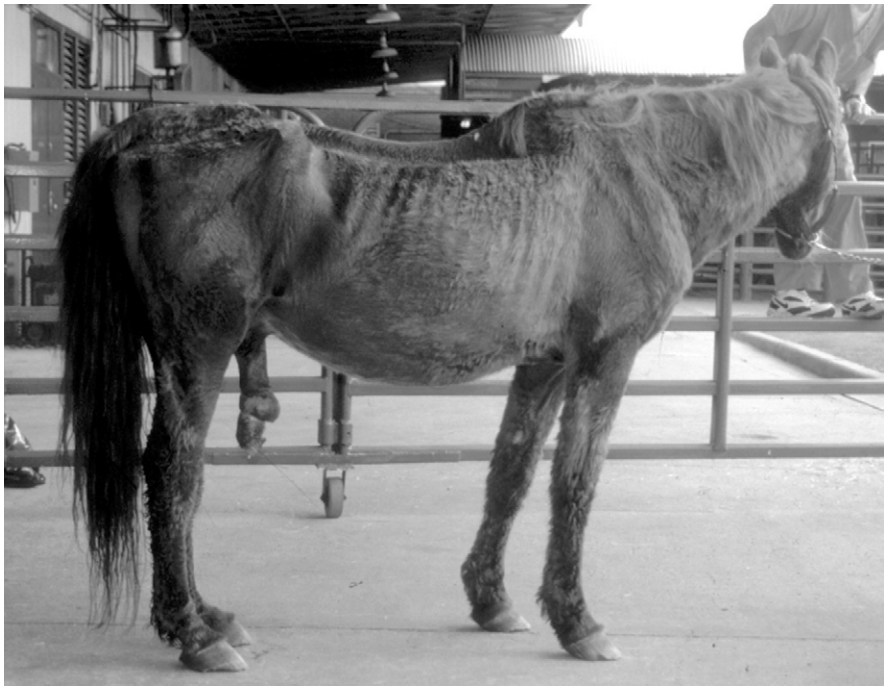

Figure 60-7. Paraphimosis caused by severe debilitation. The preputial ring has become a constricting cuff.

such as dourine and purpura hemorrhagica,[24,36] or it may be caused by damage to penile innervation. The last has been associated with spinal disease, trauma, and infectious diseases, such as equine herpesvirus 1 and rabies.[24,37] Paralysis associated with priapism, debilitation, or exhaustion has been reported (Figure 60-7).[38-40] Penile paralysis has followed administration of phenothiazine-derivative tranquilizers, most notably propiomazine (formerly termed propiopromazine).[41,42]

Pathophysiology

Tonus of the retractor penis muscles and the smooth muscle of the cavernous spaces normally maintains the penis within the prepuce.[40] With penile or preputial injury, edema develops in loose connective tissue between the penis and the internal lamina of the prepuce, and the weight of edema causes muscular fatigue, followed by protrusion of the penis and internal preputial lamina from the preputial cavity. The relationship between debilitation or exhaustion and penile paralysis is obscure, but general debilitation may cause loss of muscular tonus, allowing the penis to protrude and the pudendal nerves to become contused or stretched at the ischial arch.[40,43,44]

Penile paralysis occurring after administration of a phenothiazine-derivative tranquilizer may likewise be caused by mechanical damage to the pudendal nerves from prolonged penile protrusion and not from direct damage to penile innervation by the tranquilizer, as suggested by one investigator.[42] Motor innervation of the retractor penis muscles is probably supplied solely by α-adrenergic fibers, and phenothiazine-derivative tranquilizers block these α-adrenergic fibers.[1] The retractor penis muscles can, however, be transected without causing the penis to protrude. Tranquilization may also block sympathetic impulses to the smooth muscle of the cavernous tissue, allowing the sinusoidal spaces to fill with blood and the penis to drop from the preputial cavity.[37]

Regardless of the reason for penile protrusion, blood within the CCP pools and clots within 2 to 5 hours, making the penis somewhat rigid, rather than flaccid.[45] This rigidity may cause the clinician to conclude erroneously that the penis is protruded because the horse suffers from priapism (see "Priapism," later). Prolonged protrusion itself produces edema of the penis and prepuce by impairing venous and lymphatic drainage. As the penis and internal preputial lamina swell from edema, the preputial ring becomes a constricting cuff that compounds swelling distad. After several days, fluid begins to seep diffusely through the penile and preputial epithelium. Edema increases fragility of tissues, and because the exposed penis is subjected to trauma and effects of temperature, the penile and preputial epithelium soon becomes extensively excoriated.

Bacterial invasion of excoriated epithelium causes inflammation of the penis and prepuce, or balanoposthitis, and bacterial migration through the loose preputial connective tissue causes cellulitis. Eventual invasion of edematous and inflamed tissue by fibroblasts results in fibrosis of the penile integument and fascia, causing permanent impairment of the normal telescoping action of the prepuce.[40] The pendulous weight of the penis may eventually damage the pudendal nerves.[41,44]

The protruded penis becomes curved with the glans penis pointing caudoventrad. Urination is usually unimpeded.[41] Paralysis is usually accompanied by loss of erectile function, but ejaculatory capability is often still preserved.[24]

Treatment

Treatment of a horse affected with paraphimosis should be directed toward controlling edema and preventing further trauma. To preserve normal venous and lymphatic drainage and to protect against injury, the penis should be retained within the external preputial lamina. The penis can be temporarily retained with sutures or towel clamps placed across either the

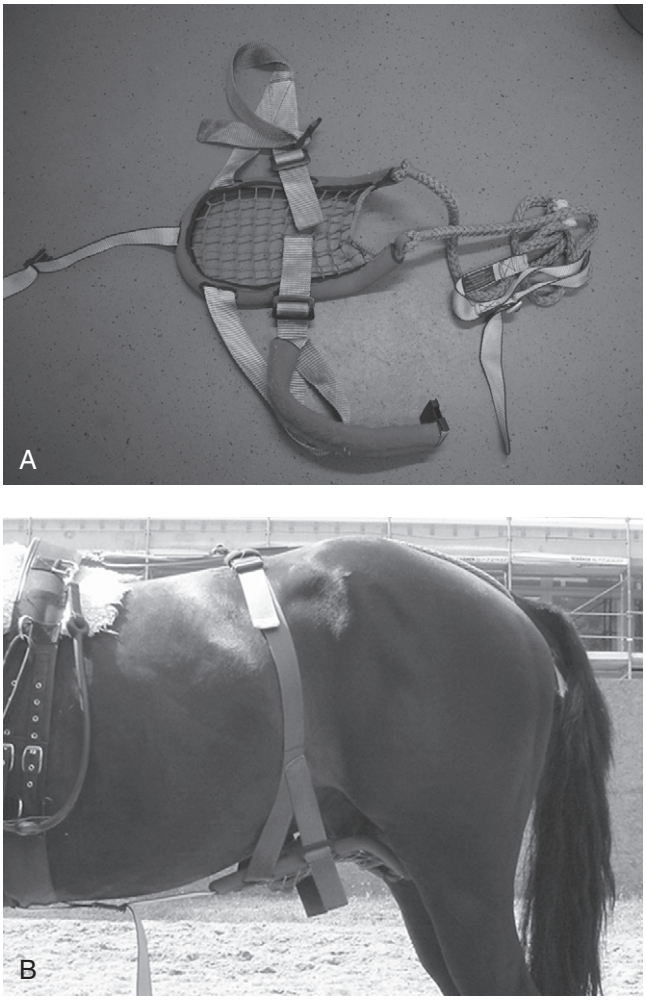

Figure 60-8. A, Suspensory device manufactured from a lightweight aluminum tube and nylon net. **B,** The device fitted to a horse. (Courtesy A. Fürst and R. Keller, University of Zurich.)

preputial orifice or preputial ring, but these devices should not be relied on for more than several days, because they damage the prepuce. Prolonged, atraumatic support can be provided by a nylon net or hosiery suspended at the preputial orifice by a crupper and surcingle made of rubber tubing (Figure 60-8).

If the protruded penis is too edematous to be replaced within the preputial cavity, it should be compressed against the abdomen with a bandage until edema subsides. A pneumatic bandage or a tight bandage applied directly to the penis may also be effective in reducing edema. Applying a nonirritating, hydrophilic agent, such as glycerin, or sulfa-urea to the penis may increase the effectiveness of the compressive bandage. Massaging the penis between bandage changes is helpful for dissipating edema. Applying an antimicrobial ointment to the penis prevents epithelial maceration and infection, and a systemically administered nonsteroidal anti-inflammatory drug reduces inflammation. Daily application of a 2% testosterone cream compounded with an equal amount of an udder cream also helps in maintaining the health of the penile and preputial epithelium.[45] The horse should be lightly exercised to reduce edema.

If the relatively inelastic preputial ring prevents penile retraction or impedes venous and lymphatic drainage, a preputiotomy can be performed. The preputial ring is severed with a longitudinal incision after administration of local, regional, or general anesthesia, and the incision is allowed to heal by secondary intention.[37]

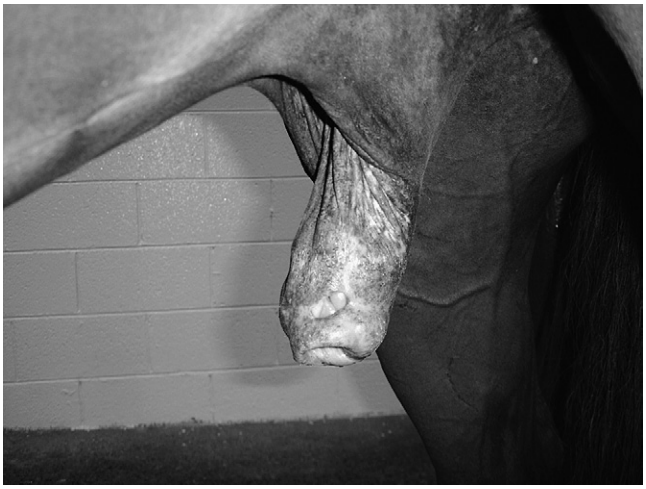

Figure 60-9. Phimosis in a horse caused by a cicatrix at the preputial ring.

With prompt treatment, paraphimosis resulting from acute trauma usually resolves within several days. Even after initial swelling and inflammation subside, preputial cicatrization may restrict normal telescoping action of the prepuce. Excision of restrictive cicatricial tissue by segmental posthetomy (reefing) may be necessary to restore normal preputial function (see "General Surgical Procedures," later). Horses with chronic paraphimosis accompanied by penile paralysis or generalized preputial fibrosis are unlikely to regain the ability to retract the penis. Stallions with penile paralysis generally retain their libido but are unable to achieve erection.[24,42] For some stallions, however, ejaculation is still possible. Penile paralysis need not necessarily end a stallion's breeding career, if the stallion can be trained to ejaculate into an artificial vagina (provided that the horse's breed registry permits artificial insemination). The horse can be salvaged for purposes other than breeding by permanently retracting its penis into the preputial cavity with sutures (i.e., the Bolz procedure) or by extensive posthetomy (i.e., the Adam's procedure) or by partial phallectomy (see "General Surgical Procedures," later, for descriptions of these procedures).

Phimosis

Etiology

Phimosis refers to the inability of the horse to protrude its penis from the prepuce because of a congenital or acquired stricture of the preputial orifice or preputial ring. Discounting the normal fusion of the internal lamina of the prepuce to the free part of the penis present during the first month after birth, congenital phimosis rarely, if ever, occurs in horses.

Acquired phimosis can result from tumors or cicatrizing lesions at the preputial orifice or preputial ring (Figure 60-9) or from impairment of the normal telescoping action of the prepuce. When the horse cannot protrude its penis, urine enters the preputial cavity and produces mucosal inflammation that may eventually lead to more cicatrization and occlusion of the

preputial orifice or preputial ring. An unusual cause of phimosis occurred when a gelding's penis became entrapped in a rent in the suspensory ligament of the prepuce. The ligament had apparently been torn when the horse was castrated.[18]

Treatment

If phimosis is caused by constriction of the preputial orifice, a wedge of external preputial lamina based toward the preputial orifice is removed.[46] The internal and external preputial laminae are joined with a row of closely spaced interrupted sutures. If phimosis is caused by constriction of the preputial ring, a similar wedge can be removed from the internal preputial fold, and after the penis is exposed, the constricting cicatrix can be removed by segmental posthetomy (reefing) (see "General Surgical Procedures," later). Phimosis caused by rupture of the suspensory ligament of the prepuce is corrected by suturing the torn ligament.[18]

Priapism

Priapism, or persistent erection without sexual excitement, occurs when the erect penis fails to detumesce.[47] The condition derives its name from the Greek god Priapus, symbol of fertility, but a frequent outcome of the condition in all species in which it occurs is infertility resulting from impotency.

Etiologic Factors

Etiologic factors in the development of priapism in men include hematologic diseases that cause vascular sludging, such as sickle cell anemia and leukemia; administration of antihypertensive or antidepressant drugs, especially when combined with alcohol; perineal trauma; spinal cord injury; and inflammatory disorders of the urogenital tract.[47] The cause of priapism in about half of all affected men is idiopathic.[48] Priapism of horses usually occurs after administration of a phenothiazine-derivative tranquilizer, usually acetylpromazine.[49] Phenothiazine-derivative tranquilizers may cause failure of detumescence by blocking α-adrenergic impulses that mediate detumescence.[50] Other, less commonly reported causes of priapism of horses include general anesthesia,[51] nematodiasis of the spinal cord,[52] and neoplasia of the pelvic canal.[53] Priapism occurs in both stallions and geldings, but stallions are more commonly affected, perhaps because of a direct influence of androgens on development of the condition, or perhaps because stallions develop erections more frequently.[53,54]

Pathophysiology

During normal erection, the rate of flow of blood into the CCP equals the rate of flow of blood from the structure. The precise mechanisms by which priapism occurs are unknown, but basically, priapism is a result of a disturbance of either the arterial inflow or the venous outflow to the CCP, causing the erect penis to fail to detumesce.[47] Priapism in men is classified as being either high flow or low flow. *High-flow priapism* occurs when arterial blood flow to the cavernosal tissue is increased, usually as a result of a traumatically induced arteriocavernosal shunt, and venous drainage cannot compensate for this increase.[55] High-flow priapism of men almost always results from trauma to the perineum or penis.[55] *Low-flow, or veno-occlusive, priapism* occurs when the neural impulses that mediate detumescence are altered or when vascular or hematologic alterations mechanically interfere with venous drainage. Low-flow priapism occurs in men and horses much more commonly than does high-flow priapism, and in fact, only one stallion has been reported to suffer from high-flow priapism. Priapism of this stallion was classified as the high-flow variety based on the stallion's response to treatment and the content of oxygen and carbon dioxide in blood obtained from the CCP, but the stallion developed priapism after it was administered a phenothiazine-derivative tranquilizer,[56] the most commonly reported cause of low-flow priapism of horses.

Low-flow priapism, regardless of its etiology, is characterized by stasis of blood within the CCP (Figure 60-10). Blood aspirated from the CCP of men affected with low-flow priapism has a low pH (typically, less than 7.25), a low partial pressure of O_2 (typically, less than 30 mm Hg), and a high partial pressure of CO_2 (typically, greater than 60 mm Hg).[57] High partial pressure of CO_2, caused by vascular stasis, causes erythrocytes to sickle and causes endothelial damage to the vessels and trabeculae in the CCP. The sickled erythrocytes occlude the venous outflow from the CCP, eventually irreversibly.[58] Endothelial damage and occlusion cause trabecular edema, which eventually progresses to fibrosis, thereby decreasing the size of the sinusoidal spaces in the CCP and the capacity of the CCP to expand during erection. Arterial flow to the CCP remains patent during early stages of priapism, but ultimately, it too becomes occluded permanently by clots, edema, or fibrosis.[58] Protracted erection in the horse may also damage the pudendal nerves, presumably from tension on the nerves or from compression of the nerves against the ischium, causing paralysis of the penis.[53] The end result of unresolved priapism in the horse is impotence caused by loss of both erectile function and sensitivity of the glans and shaft of the penis.

Clinical Signs

A horse suffering from priapism may not have a full erection, causing the penis to appear to be paralyzed or merely protruded rather than erect. Turgidity of the CCP can be detected when the penis is palpated however, and the engorged penis cannot be reduced manually into the prepuce. Unless properly cared

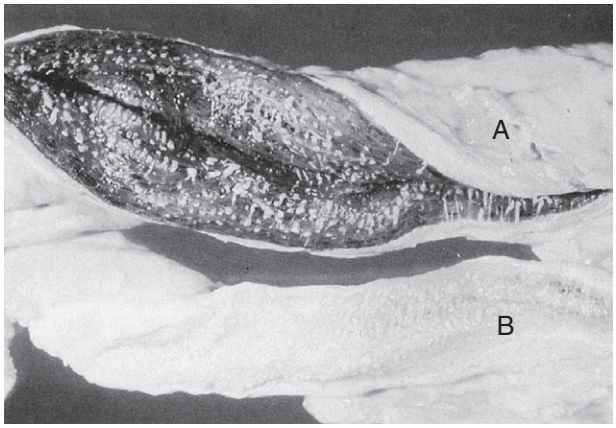

Figure 60-10. *A,* Longitudinal section of corpus cavernosum penis (CCP) of a stallion with long-standing priapism. The CCP is hemorrhagic and edematous. *B,* Longitudinal section of the CCP of a normal gelding.

for, the penis and the internal lamina of the prepuce become edematous soon after the onset of priapism.[54] Although uncommon, dysuria may be a feature of the condition. The CCP of a horse chronically affected by priapism feels fibrous, and during ultrasonographic examination, it appears to be densely echogenic. A chronically affected horse may not respond when a noxious stimulus is applied to the distal portion of the penis or internal lamina of the prepuce.[53]

Treatment

MEDICAL

Horses with priapism have been treated by massaging the penis, by applying an emollient dressing to the penis, and by compressing the penis against the body wall with a sling.[54] These treatments, although important in preventing preputial edema and damage to the penile and preputial integument, have no effect on reestablishing normal circulation in the erectile tissue of the CCP. Affected horses have been treated by administration of benztropine mesylate to reestablish normal venous drainage impaired by drugs that cause α-adrenergic blockade, such as acetylpromazine.[47,51,59-61] Benztropine mesylate, commonly used to treat people for Parkinson disease, is a synthetic drug created by combining the active portions of atropine and diphenhydramine.[51] This drug is most successful in resolving priapism if treatment is initiated soon after the onset of priapism.[51,60] The effects of the drug on horses with priapism are attributed to its anticholinergic effects.[51] The usual dose for a horse of average size is 8 mg, administered by slow intravenous injection. Side effects seen at higher dosages include paralytic ileus, impaction, dysuria, and muscular weakness. Terbutaline, a β_2-adrenergic receptor agonist, has been used successfully to treat men suffering from priapism and may be effective in treating affected horses.[62] Clenbuterol, a β_2-adrenergic receptor agonist commonly used in horses to cause bronchodilation, might also be effective.

α-Adrenergic agents, such as ephedrine, adrenaline, and phenylephrine, are often injected into the CCP of men in the early stages of priapism to achieve detumescence by promoting contractility of cavernous and arterial smooth muscle.[13,47,57,61] Instillation of 10 mg of phenylephrine diluted in 10 mL physiologic saline solution directly into the erect CCP is sometimes effective in resolving priapism of horses, provided that treatment is initiated soon after onset of priapism.[63] Phenylephrine can be injected safely into the cavernous tissue of men every 15 minutes until detumescence occurs,[13] and the same is probably true for horses. Horses chronically affected by priapism experience only temporary detumescence after this treatment.

IRRIGATION OF THE CORPUS CAVERNOSUM PENIS

A horse that does not respond within a few hours to cholinergic blockage or to three or more intracavernosal injections of an α-adrenergic agent, such as phenylephrine, should be treated by irrigation of its CCP with heparinized physiologic saline solution (PSS).[39] Irrigation of the CCP not only removes sickled erythrocytes, it also improves the acidotic environment within the CCP. The CCP can be irrigated with the horse standing, but the procedure is most easily accomplished with the horse anesthetized and in dorsal recumbency. After preparing the penis and perineal area for aseptic surgery, PSS containing 10 IU heparin/mL is introduced into the CCP under pressure through a large-bore needle (e.g., a 12-gauge needle) inserted into the turgid CCP just proximal to the glans penis. The PSS, along with the stagnant blood, is exited 10 to 15 cm caudal to the scrotum, either through one or two large-bore needles inserted into the CCP or through a stab incision into the CCP. The CCP is irrigated until fresh blood appears in the efflux. Phenylephrine (10 mg of 1% solution) instilled into the CCP at the end of irrigation may be useful in evacuating fluid from the CCP. Failure of arterial blood to appear in the efflux after stagnant blood has been evacuated indicates that the arteriolar supply to the CCP is permanently damaged and that the horse is likely to be impotent. The stab incision in the tunica albuginea of the CCP is sutured after irrigation is complete.

CREATION OF A SHUNT

Erection recurs after irrigation if venous outflow remains occluded, provided that arteriolar inflow vessels remain patent. If erection fails to resolve after several irrigations of the CCP, blood trapped in the CCP should be removed by creating a shunt between the CCP and the CSP. The CSP offers a convenient exit for blood trapped in the CCP, because in contrast to the CCP, the CSP does not act as a closed system during erection.[47,48] Shunting should be performed before the cavernous tissue or the pudendal nerves become irreversibly damaged, but the time at which damage to the cavernosal tissue becomes irreversible after the onset of priapism has not been determined for horses.

The shunt is probably best performed in the perineal region of the horse. It could be created farther distally, but creating it in the perineal region allows a more thorough evacuation of the stagnant blood by irrigation and provides drainage of blood from a greater length of the CCP. Because the CSP is thickest in the perineal region, the urethra is less likely to be perforated during the procedure.

To create the shunt, the horse is anesthetized and positioned in dorsal recumbency. The penis and inguinal and perineal regions are prepared for aseptic surgery, and a stallion catheter is inserted into the urethra. A 15-cm incision is created along the perineal raphe, about 4 to 8 cm caudal to the base of the scrotum to expose the penis, and the retractor penis muscle is retracted to expose the bulbospongiosus muscle, which covers the ventral aspect of the CSP. The right or left edge of the bulbospongiosus muscle is elevated from the edge of the urethral groove to expose 4 to 5 cm of the underlying tunica albuginea of the CSP. A 3-cm longitudinal incision is made through the tunica albuginea of the CCP adjacent to the CSP to expose the sinusoidal spaces of the CCP, and stagnant blood is evacuated from the cavernous spaces of the CCP through this incision, using irrigation as described earlier (Figure 60-11).

A matching 3-cm longitudinal incision through the tunica albuginea of the CSP is created adjacent to the 3-cm incision into the CCP. The urethral catheter compresses the sinusoidal spaces of the encircling CSP, so the CSP must be incised carefully to avoid extending this incision into the lumen of the urethra. Bright red blood exits from the incised CSP, and at this point of the procedure, suction is usually required to maintain visibility. The medial edge of the incision in the tunica albuginea of the CCP is sutured to the lateral edge of the incision in the tunica albuginea of the CSP with 3-0 or 2-0 absorbable suture material using a simple-continuous suture pattern. To complete the shunt, the lateral edge of the incision into the CCP is sutured to the medial edge of the incision into the CSP using a simple-continuous suture pattern. The bulbospongiosus

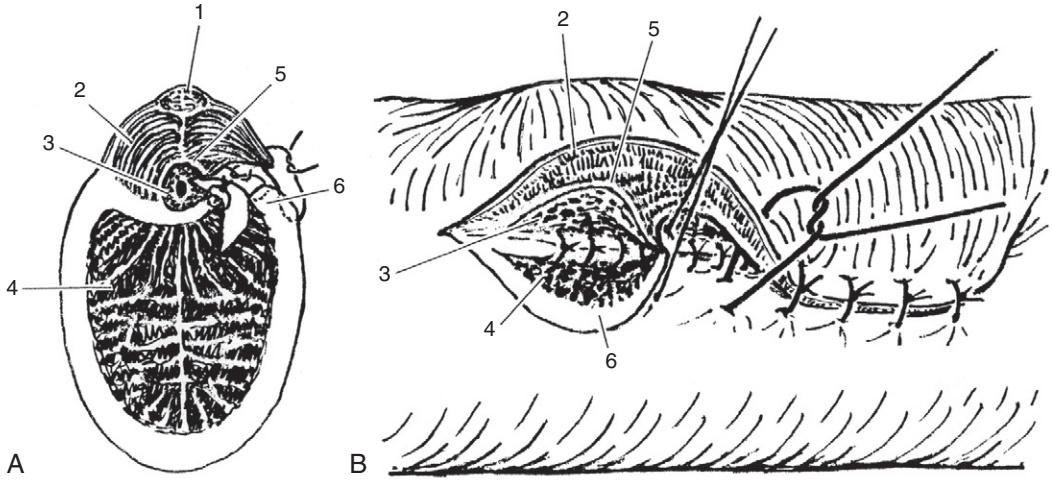

Figure 60-11. Cross-sectional view **(A)** and three-dimensional view **(B)** showing creation of a vascular shunt between the corpus cavernosum penis (CCP) and corpus spongiosum penis (CSP). *1*, Retractor penis muscle (not shown in **B**); *2*, bulbospongiosus muscle; *3*, CSP surrounding the urethra; *4*, CCP; *5*, tunica albuginea of CSP; *6*, tunica albuginea of CCP. The medial portion of the incision through the tunica albuginea of the CCP is sutured to the lateral portion of the incision through the tunica albuginea of the CSP. The medial portion of the incision through the tunica albuginea of the CSP is sutured to the lateral portion of the incision through the tunica albuginea of the CCP. The bulbospongiosus muscle is sutured to the tunica albuginea of the CCP.

muscle is sutured to its origin on the tunica albuginea of the CCP at the edge of the urethral groove, and the subcutaneous tissue and skin are apposed. Horses appear to suffer no discomfort after the surgery, and swelling is minimal.[59] The stallion should receive no sexual stimulation for at least a month after surgery.

Complications of the cavernosal shunt in men include urethrocavernous or urethrocutaneous fistulas, penile gangrene, infection, and a painful CSP during erection.[48] Men receiving a shunt for treatment for priapism may become impotent from failure to achieve or maintain pressure in the CCP required for intromission,[47,48] and this failure can be the result of damaged cavernous tissue or from the shunt itself.[48] The shunt may close as normal blood outflow in the CCP resumes, but whether closure is essential for return of potency is not known. Bulls may become impotent after developing a trauma-induced shunt between the CCP and CSP,[37,64] but a shunt created surgically between the CCP and the CSP of normal stallions does not seem to interfere with subsequent erection and ejaculation, even if the shunt does not close.[59] In one report, a stallion that developed penile paralysis after suffering from priapism for several days regained normal erectile and ejaculatory function within 1 year after resolution of priapism, even though the horse had received two shunts between the CCP and the CSP.[56] Failure of an affected stallion to develop a normal erection after creation of a shunt between the CCP and the CSP is most likely the result of damage to erectile tissue caused by protracted priapism, rather than the result of the shunt.

Erectile function in men with cavernosal tissue damaged by protracted priapism has been enhanced by injecting a vasoactive drug, such as papaverine, phenoxybenzamine, or phentolamine, into the cavernosal tissue.[47,65] Administering a vasoactive drug into the cavernous tissue may likewise be useful for enhancing erectile function of horses with damaged erectile tissue. Some stallions with damaged erectile tissue may regain the ability to achieve intromission if they are assisted in placing their penis into the vagina of the mare, and some may be trained to ejaculate into an artificial vagina. If a stallion has decreased penile sensitivity resulting from damage to the pudendal nerves caused by priapism, an antidepressive drug, imipramine, can be administered before breeding to lower the stallion's ejaculatory threshold.[66]

PARTIAL PHALLECTOMY

Partial phallectomy may be necessary if all other treatments to relieve priapism fail (see "General Surgical Procedures," later).[54,67] Hemorrhage during or after phallectomy may be no worse than that expected during and after amputation of the penis of a horse not affected by priapism, but the remaining portion of the penis may remain erect until the CCP fibroses.

Hypospadias

Hypospadias is a congenital anomaly in which the urethral meatus (opening) is situated on the ventral aspect of the penis proximal to its normal location on the glans penis (Figure 60-12). The condition is sometimes accompanied by chordee, or an abnormal ventral curvature of the penis (Figure 60-13). It can also be accompanied by an incomplete prepuce and by stenosis of the urethral meatus [68] The position of the urethral meatus in affected humans is usually glandular, coronal, or subcoronal, but it may be penile, penoscrotal, or perineal.[69,70] Associated urogenital anomalies that sometimes accompany hypospadias in affected humans include cryptorchidism, inguinal herniation, and hydrocele. Anomalies of other systems include cleft lip and palate, imperforate anus, and cardiac irregularities.[69] The more proximal the urethral defect, the more likely the affected human is to have one or more additional anomalies.

Hypospadias results from arrested development of the penis before the penis and urethra are completely formed[71] and is probably a manifestation of a wide assortment of endocrinopathies. The condition may result from an inadequate production of androgens by the fetal testes, defective conversion of

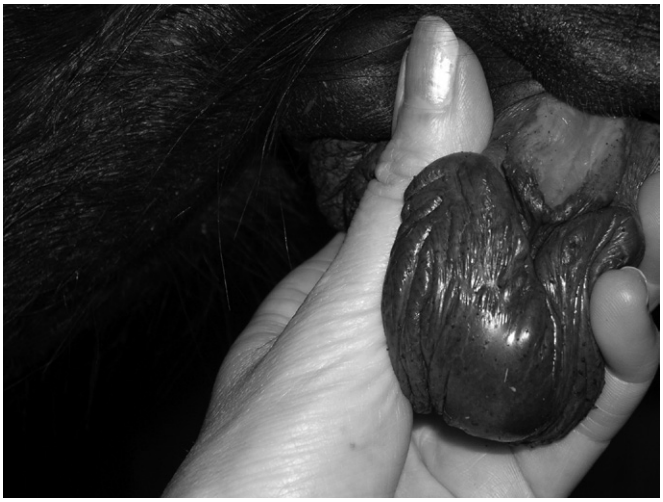

Figure 60-12. Hypospadias of a stallion. The urethral meatus is located subcoronally and the internal lamina of the prepuce is complete. The stallion also had chordee (see Figure 60-13).

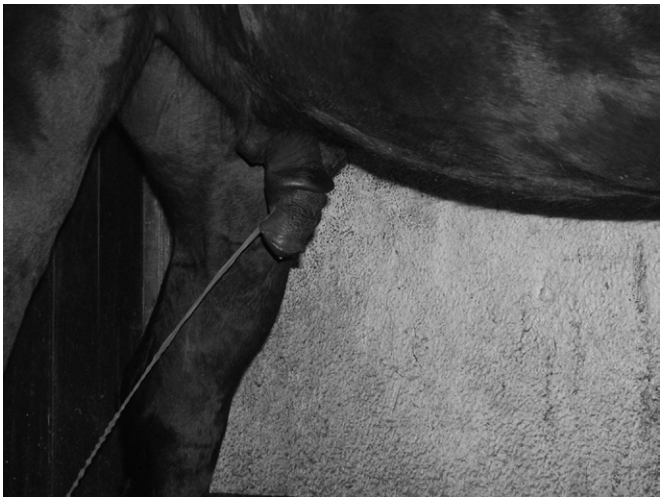

Figure 60-13. Hypospadias of the stallion in Figure 60-12 was accompanied by chordee, or an abnormal ventral curvature of the penis. Chordee caused this stallion to develop urine-induced dermatitis and discomfort during urination.

testosterone to dihydrotestosterone, or poor sensitivity of the target tissues of the developing external genitalia to androgens.[72-74] Hypospadias of men has a familial tendency, but no such tendency for the horse has been reported.[69]

Hypospadias is the most common congenital urogenital anomaly of men,[75,76] but the anomaly is encountered rarely in horses. A Friesian stallion in one report had a 4-cm-long glanular coronal and subcoronal urethral defect,[77] and a horse in another report had a urethral defect that extended from the region of the ischium to the glans penis.[78]

A horse with the anomaly may require no treatment unless the anomaly causes urine-induced dermatitis and discomfort during urination (see Figure 60-13). The most expedient method of resolving the problem may be to amputate the deformed portion of the penis.[79] Correcting hypospadias or chordee of a stallion to permit coitus should be discouraged because the condition may be genetic.

Intersex

Clinical Features

Anomalous development of the external genitalia of intersexes confuses their sexual identification. The most common intersex, the male pseudohermaphrodite, usually has a rudimentary penis, which resembles an enlarged clitoris, and prepuce, which resembles a vulva, situated on the midline somewhere between the ischial arch and the normal ventral abdominal location of the preputial orifice (see Figure 59-10).[46,80-82] Its testes are usually located within the abdomen or inguinal canals or hidden subcutaneously beneath a moderately developed udder (see Chapter 59). Despite its deceptive feminine appearance, the intact male pseudohermaphrodite displays masculine behavior and is even capable of achieving an erection of the rudimentary penis.

Treatment

If the genitalia are located close to the ischium, the pseudohermaphrodite's appearance can be altered to more closely resemble that of a female by amputating the rudimentary penis and constructing a vulva using the prepuce.[46] Alternatively, if the genitalia are less than 15 cm caudal to the inguinal area, the male pseudohermaphrodite's appearance can be altered to more closely resemble that of a male by cranially rotating the genitalia to a more normal position caudal to the umbilicus.[64] The horse should be castrated 4 to 6 weeks before surgery.

Neoplasia

Incidence and Etiology

The incidence of neoplasia of the external genitalia is second only to that of the skin.[83] Neoplasms of the penis and prepuce include squamous papillomas, squamous cell carcinomas, sarcoids, melanomas, mastocytomas, and hemangiomas.[84-87] Melanomas are occasionally found on the prepuce of old gray horses,[24] and squamous papillomas, the benign counterparts of squamous cell carcinoma, are often found on the external genitalia of young horses[88] or adjacent to penile or preputial carcinomas of old horses.[89] Squamous cell carcinoma is by far the most common penile and preputial neoplasm.[84,89] Genital squamous cell carcinoma may arise *de novo* or from malignant transformation of a squamous papilloma.[88,89] Any papillomatous lesion present on the penis or prepuce of a horse should be considered to be premalignant.

Squamous cell carcinoma is usually found on old horses, especially those of breeds with nonpigmented genitalia, such as Appaloosas and American Paint horses.[37,85,90] Lack of preputial or penile pigmentation seems to predispose to carcinoma, even though the external genitalia are unexposed to direct sunlight.[91] One study performed on mice showed that squamous cell carcinoma may be caused by unidentified carcinogenic agents in smegma,[92] but another study, also performed on mice, which used human smegma, was not able to substantiate this finding.[93] Regardless of whether smegma contains a carcinogenic agent, it may stimulate neoplastic changes in penile and preputial integument by causing chronic irritation. Geldings and old horses, therefore, may be predisposed to development of genital squamous cell carcinoma, because they produce a greater amount of smegma than do young horses and stallions.[91,92]

Squamous cell carcinoma occurs most commonly on the glans and internal lamina of the prepuce.[89,90] Penile and

preputial squamous cell carcinomas are locally invasive but have a low grade of malignancy and grow surprisingly slowly for carcinomas.[85,89,90] Metastasis to the superficial and deep inguinal lymph nodes occurs late in the disease. In one study, 12% of 48 horses affected with penile or preputial squamous cell carcinoma had metastatic involvement of the inguinal lymph nodes.[89]

Diagnosis

Most affected horses are presented for examination when the owner observes the lesion, but some may be presented because of a malodorous, purulent, blood-stained preputial discharge.[89,90] The duration of disease is usually unknown but lengthy, because most owners inspect their horse's penis infrequently.[90] Precancerous lesions may appear as a small, heavily keratinized plaque, and cancerous lesions may first appear as a shallow, flat ulceration with an indurated base.[85,90,94] Long-standing carcinomas may attain a cauliflower-like excrescence, containing areas of necrosis, ulceration, and hemorrhage that can interfere with coitus or normal protrusion and retraction of the penis (Figure 60-14).[95] The tumor can cause dysuria by impinging on the urethra. Most affected horses are also affected by balanoposthitis.[87,90]

Metastatic spread of penile and preputial squamous cell carcinoma can sometimes be palpated as an enlargement of the inguinal lymph nodes, but mild, metastatic enlargement of the inguinal lymph nodes may be difficult to differentiate from lymphadenopathy secondary to balanoposthitis.[89] Penile or preputial squamous cell carcinoma can metastasize to internal organs without causing gross enlargement of the inguinal lymph nodes.[96]

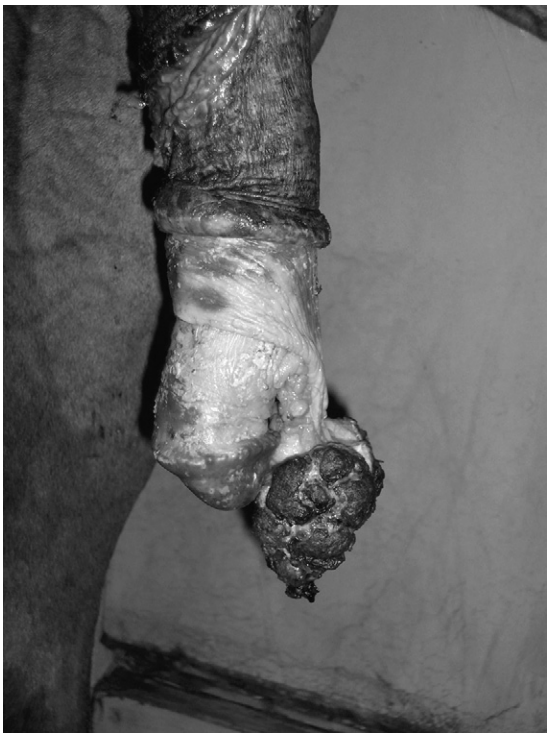

Figure 60-14. Squamous cell carcinoma of the inner lamina of the preputial fold.

Treatment

A variety of treatments for horses affected with squamous cell carcinoma of the penis or prepuce have been described, including surgical excision, cryosurgery, chemotherapy, and hyperthermia.

SURGICAL EXCISION

A small lesion on the prepuce can be excised and the wound sutured. Using a carbon dioxide laser to locally excise squamous cell carcinoma from the external genitalia may decrease the incidence of recurrence of the neoplasm.[97] Using a laser to excise a neoplasm not only decreases postoperative swelling by sealing lymphatic vessels but also has a thermal killing effect on marginal tumor cells.[98] Horses with extensive neoplastic lesions of the external genitalia require preputial reefing or partial phallectomy (see "General Surgical Procedures," later). Occasionally, neoplasms become so extensive that prescrotal urethrostomy combined with *en bloc* resection of the penis, prepuce, and inguinal lymph nodes becomes necessary.[99]

CRYOTHERAPY

Cryotherapy is a useful treatment of horses with early squamous cell carcinoma lesions. Cryotherapy can be performed using liquid nitrogen administered as a spray or through a cryoprobe, or using CO_2 administered through a cryoprobe. Thermocouple needles in a pyrometer (i.e., tissue temperature indicator) are used to monitor the depth and degree of freezing. A double, fast freeze–slow thaw cycle gives the best results.[100] More information on cryosurgery is found in Chapter 14.

CHEMOTHERAPY

Horses with small genital lesions of squamous cell carcinoma have been treated successfully by applying 5% 5-fluorouracil to the lesions at 14-day intervals.[101] Lesions need not be debulked, provided that the lesion is raised no more than to 2 to 3 mm above the surrounding integument. Up to seven treatments may be required to effect resolution of lesions. The drug is also effective in causing regression of preneoplastic lesions to which it is applied.

Horses with squamous cell carcinoma of the external genitalia have been treated by intratumoral injection of cisplatin in sesame oil[102,103] or by intratumoral implantation of cisplatin beads.[104] The drug is administered at a dosage of 1 mg/cm^3 tissue every 2 weeks, usually until the tumor has been injected four times. Intratumoral chemotherapy with cisplatin can be used alone for treatment of horses with small tumors or in combination with surgery for treatment of horses with large tumors. Debulking the tumor increases the efficacy of the cisplatin because antineoplastic drugs are most effective when the tumor burden is low.[105] Tumor cells still present after the tumor has been excised or debulked are stimulated into active proliferation and hence are more susceptible to cisplatin. Administration of cisplatin treatments intraoperatively and at 2-week intervals appears to result in a better outcome, at least for horses with tumors that have a high proliferation index, than does delaying chemotherapy until the wound has healed.[103] Injecting the tumor bed and a margin of normal tissue with cisplatin at the time of surgery and during all phases of wound healing does not have clinically apparent detrimental effects on healing of wounds closed primarily or wounds left open to heal by secondary intention.[102,103] (For more information on skin tumor treatments, and chemotherapy in general, see Chapter 29.) Topical

or intratumoral chemotherapy is impractical for treatment of horses affected with squamous cell carcinoma that has metastasized to internal organs. Horses affected with metastatic squamous cell carcinoma can be treated with a systemically administered chemotherapeutic agent, such as doxorubicin or piroxicam,[106] but little information is available on the efficacy of systemically administered chemotherapy to treat horses for neoplasia.

HYPERTHERMIA

Radiofrequency-induced hyperthermia has been used to treat horses with sarcoids and cattle and horses suffering from ocular neoplasia.[107,108] Although its use to treat horses with genital neoplasia has not been reported, radiofrequency-induced hyperthermia could be useful for treatment of horses with genital neoplasia. Intratumoral hyperthermia is induced by a radiofrequency current of 2 MHz. Using this treatment, the tumor is heated to approximately 50° C for 30 seconds. Large tumors are heated in sections. Multiple treatments are required, but the length of the interval between treatments is determined subjectively.

Prognosis

In one study of 48 male horses with genital carcinoma, 64.5% of horses were alive 18 months after surgical therapy.[89] In another study, about 81% of 45 affected horses survived at least 1 year after surgical therapy with no evidence of recurrence of the disease.[90] Both studies found that prognosis for survival was poor if squamous cell carcinoma had metastasized to the inguinal lymph nodes.[89] Invasion of the cavernous tissue by squamous cell carcinoma is a pejorative prognostic factor for survival in men[109] and is likely to be so in horses as well, because neoplastic invasion into a corporeal body may be more likely to result in hematogenous spread of the neoplasm. In one study, three of four horses that had metastases of penile carcinoma to the abdomen had gross or histologic evidence that the neoplasm had invaded the cavernous tissue.[90] Horses that have corporeal invasion by a carcinoma seem to have a high likelihood of abdominal metastases, and therefore laparoscopic examination of the abdomen of a horse that has neoplastic invasion of a corporeal body may be prudent. Invasion of the CSP by a carcinoma may result in stricture of the urethra.[110] Because lesions of squamous cell carcinoma sometimes recur, horses should be monitored periodically for recurrence of disease after apparently successful treatment.

Habronemiasis

Etiology

Cutaneous habronemiasis, also known as "summer sore" or granular dermatitis, is a granulomatous, mildly pruritic disease caused by cutaneous migration and encystment of the larvae of the equine stomach worm *Habronema*.[94,111] Larvae passed in the feces are ingested by fly maggots, and after the maggot pupates, the larvae are deposited on wounds from the feeding fly. The disease appears in spring and summer, when flies are prevalent, and usually disappears with onset of cold weather. The penis and prepuce are common sites of infestation by these larvae because moisture on these structures attracts flies. Horses that tend to protrude their penis while resting and horses that receive anthelmintic drugs infrequently may be more prone to developing genital habronemiasis.[111]

Pathophysiology

Infestation stimulates an acute granulomatous reaction characterized by exuberant granulation tissue that contains numerous small, yellow, hard, caseous granules composed of eosinophils, nuclear remnants, and larvae. The larvae may excrete a substance lytic to the host's tissue,[94] but a local hypersensitivity reaction to the larvae resulting from repeated reinfestation is probably responsible for the extreme granulomatous response.[111] The presence of mature *Habronema* in the stomach may induce a state of general hypersensitivity, because horses affected by cutaneous habronemiasis are almost always heavily parasitized by adult worms. Some horses appear to be more susceptible and are plagued by yearly recurrence of lesions.

Clinical Signs

The preputial ring and urethral process are the genital sites of predilection. Preputial lesions may appear as ulcerated, red areas demarcated by edges of depigmentation.[94] Lesions may be granulomatous and extensive (Figure 60-15). The infested urethral process may be enlarged from periurethral fibrosis, and hyperemic prolapsed mucous membrane may protrude from the urethral orifice.[112] Preputial lesions may mechanically impede the telescoping action of the preputial laminae, and lesions of the urethral process may partially obstruct the flow of urine.[112] A horse with a distorted urethral process may spray itself or show signs of discomfort during urination. Erosions into the CSP may result in hemorrhage at the end of urination or ejaculation.[113] Horses with a genital lesion of cutaneous habronemiasis may have lesions of cutaneous habronemiasis elsewhere on the body.

Diagnosis

Cutaneous habronemiasis of the external genitalia is usually diagnosed by its typical appearance, but lesions can be confused with squamous cell carcinoma, exuberant granulation tissue, or

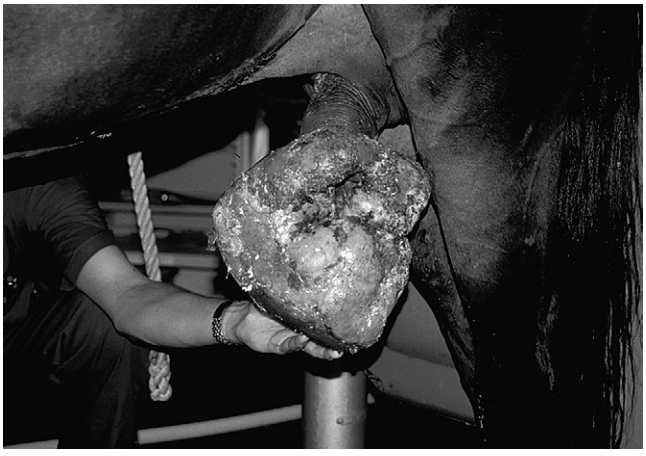

Figure 60-15. Massive granuloma on the internal preputial lamina caused by cutaneous habronemiasis. This mass was removed by segmental posthetomy.

phycomycosis.[111] A nonhealing, granulating wound accompanied by marked circulating eosinophilia is suggestive of the disease. Squeezing the lesion may cause granules to extrude, and occasionally a larva is found if exudate is squeezed onto a slide and examined microscopically. Eosinophils, multinucleated giant cells, granules, and, sometimes, cross sections of larvae can be seen by examining affected tissue histologically.[112,114]

Treatment

NONSURGICAL TREATMENT

Lesions are resolved by eliminating the larvae or by reducing the horse's hypersensitivity to them.[111] Ivermectin, administered orally at 200 µg/kg, or organophosphates administered topically, orally, or intravenously have been effective in destroying the larvae.[115] Prednisolone, administered orally at 1.5 mg/kg once a day for 10 to 14 days, or diethylcarbamazine, administered orally at 1.5 mg/kg twice a day for 7 to 14 days, has brought about resolution of lesions by diminishing the body's response to the larvae. Daily topical application of a cream containing an organophosphate, such as trichlorfon, and a corticosteroid, such as dexamethasone, may bring about resolution of small granulomatous lesions caused by cutaneous habronemiasis.

SURGICAL TREATMENT

Elliptical or circumferential resection of fibrotic areas of the internal lamina of the prepuce caused by chronic infestation may be required to restore normal preputial function, and amputation of an affected urethral process may be required to restore normal urination or to prevent hemospermia[112] (see "General Surgical Procedures," later).

Hemospermia

Etiology

Hemospermia, or blood in the ejaculate, is an important cause of infertility of stallions and has been attributed to bacterial urethritis occurring usually at the area of the ejaculatory ducts; habronemiasis or neoplasia of the urethral process; improperly fitted stallion rings; seminal vesiculitis; and trauma to the urethral process or glans penis.[26,116,117] Viral urethritis has been suspected, but not proved, to cause hemospermia.[118] Hemospermia has been reported to occur from urethral rents, the etiology of which is unknown.[119] The source of voluminous hemorrhage in the ejaculate is usually the CSP. Hemorrhage from the CSP typically occurs at the end of ejaculation when contraction of the bulbospongiosus muscle causes pressure within the CSP to increase from 17 to nearly 1000 mm Hg.[12]

Red blood cells in the ejaculate are associated with reduced fertility, even though seminal quality appears otherwise unaffected.[117] Red blood cells affect the integrity of the cellular membrane and the motility of spermatic cells.[120] Seminal quality is proportional to the degree of contamination. The condition may be more common in Quarter Horses,[121] and frequently bred stallions are more often affected.[117]

Diagnosis

Stallions affected by hemospermia may require several mounts to ejaculate and sometimes exhibit pain during erection or ejaculation.[121] Blood in the semen is most easily identified by collecting the stallion's ejaculate with an artificial vagina. Semen of affected horses is usually pink to red, but because blood in amounts too minute to be detected grossly can cause infertility, microscopic examination of the semen may be necessary to detect the condition.[23] Microscopic examination of semen may also reveal a large number of white blood cells if septic seminal vesiculitis is the cause of hemospermia.

Septic seminal vesiculitis, an occasional cause of hemospermia, may be detected by identifying numerous clumps of purulent material in the semen and by finding blood in the gel fraction of the ejaculate.[122] A thickened vesicle filled with echogenic fluid may be seen during transrectal ultrasonographic examination of a septic seminal vesicle. The causative organism of septic seminal vesiculitis can be cultured from fluid obtained directly from the infected seminal vesicle.[122,123]

Rents in the urethra, a common cause of hemospermia, can be detected by examining the urethra with a sterilized flexible endoscope that is at least 100 cm long. A urethral tear into the CSP appears endoscopically as a 5- to 10-mm-long, linear defect on the convex surface of the urethra, distal to the openings of the bulbourethral glands, near the level of the ischial arch.[119,121] The shaft of a hypodermic needle can be introduced percutaneously into the lumen of the urethra at the level of the ischial arch during endoscopic examination to confirm the location of the defect (Figure 60-16). By endoscopically examining the urethra of a horse affected with hemospermia immediately after ejaculation, the examiner may be able to observe blood emanating from an otherwise undetectable rent.[23]

Urethrography to diagnose urethral lesions has been described.[117,118] The penis is radiographed after injecting 180 mL of barium suspension into the urethra. The barium is allowed to drain, 180 mL of air is injected to provide double contrast, and the penis is again radiographed. Bacterial and viral cultures and biopsy and histologic examination of urethral lesions may establish the cause of urethritis.

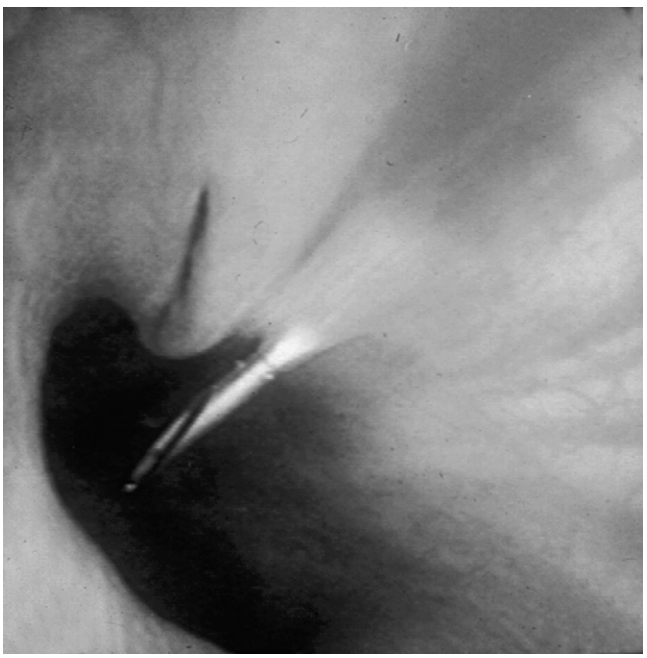

Figure 60-16. Endoscopic view of a urethral rent. The shaft of a hypodermic needle has been inserted percutaneously into the lumen of the urethra at the level of the ischium to pinpoint the location of the defect.

Treatment

NONSURGICAL TREATMENT

Sexual abstinence seems to be important in the treatment of stallions affected with hemospermia, regardless of the origin, because erection and contractions of the bulbospongiosus muscle during ejaculation dilate and further traumatize the urethra.[117,121] Medical treatment of affected horses has included intravenous administration of formaldehyde solution, oral administration of methenamine, and systemic administration of antimicrobial drugs.[118]

Horses affected with hemospermia caused by septic seminal vesiculitis should receive antimicrobial therapy that is effective against the causative organism. Systemic administration of antimicrobial drugs to stallions affected with septic seminal vesiculitis is often ineffective because antimicrobial drugs diffuse poorly into the gland. Infusing the appropriate antimicrobial drug directly into the seminal vesicles after the vesicles have been lavaged may be a more effective treatment.[122] Horses with low-grade hemospermia have been managed by adding an extender to the semen to dilute the effect of the red blood cells on the spermatozoa,[116,124] but adding semen extender is probably ineffective because the deleterious effects occur as soon as blood contacts the spermatozoa.[120]

SURGICAL TREATMENT

Temporary perineal urethrostomy combined with sexual rest has been effective in eliminating hemospermia caused by a urethral lesion.[118,119,121] Eleven of 15 affected horses were successfully treated by temporary subischial urethrostomy and daily installation of suppositories of nitrofurazone and hydrocortisone into the urethra, although two stallions developed a urethral fistula.[121] These investigators offered no sound rationale for the relatively high incidence of success of temporary urethrostomy, but other investigators implied that topical application of antimicrobial drugs to lesions of bacterial urethritis is responsible for resolution of hemospermia.[117] More likely, success of temporary subischial urethrostomy in eliminating hemospermia should be attributed to decreased pressure in the CSP and diversion of blood flow from the urethral lesion. When the bladder has been emptied, the bulbospongiosus muscle contracts to expel urine that remains in the urethra. Incising the CSP at the ischium decreases the pressure gradient between the urethral lumen and the CSP at the end of urination and diverts blood flow from the urethral lesion to the temporary urethrostomy, thus permitting healing of the urethral mucosa and underlying tunica albuginea of the CSP (see "Hematuria," later). Simply incising the convex surface of the tunica albuginea of the CSP at the ischium, without exposing the lumen of the urethra, may be as effective as temporary urethrostomy for eliminating hemospermia, and the risk of the stallion developing a urethral fistula is eliminated. This theory, however, has not been clinically evaluated.

The urethral rent of one stallion, which failed to heal despite subjecting the stallion to prolonged periods of enforced sexual inactivity, healed after it was covered with buccal mucosal graft inserted through a temporary perineal urethrostomy, resolving the hemospermia.[125] The ventral portion of the perineal urethrostomy, below the graft was left unsutured. Whether the graft was accepted at the recipient site on the urethra was not reported.

A simpler method of effecting healing of a urethral rent is to suture the rent.[126] The urethral rent can be sutured through a perineal incision if perineal incision into the CSP or temporary perineal urethrostomy is unsuccessful in allowing the rent to heal by second intention. To prepare for primary closure of a urethral rent, an endoscope is inserted into the urethra so that the rent can be observed. Two 3.81-cm, 20-gauge needles are inserted through the skin of the perineum and advanced so that the shafts of the needles emerge in the urethral lumen at the proximal and distal ends of the rent. The perineum is incised as if a perineal urethrostomy was to be performed, but the incision extends only into the CSP and not through the urethral mucosa. The rent in the urethral mucosa is identified between the shafts of the needle and is closed, using endoscopic guidance, with 3-0 absorbable suture material using a simple-continuous suture pattern. The sutures should incorporate the tunica albuginea of the CSP. The perineal incision can be left open to heal by second intention, or it can be sutured in layers. Sutured layers should include the incisions into the tunica albuginea of the CSP, the bulbospongiosus muscle, subcutaneous tissue, and skin.

Hematuria

Etiology

Hematuria can originate from the kidney, ureter, bladder, urethra, or reproductive organs.[113,116] Causes of hematuria include renal, ureteral, vesicular, or urethral calculi; renal, and vesicular neoplasia; and pyelonephritis. Terminal hematuria (i.e., hematuria that occurs at the end of urination) is associated with a lesion located at the proximal portion of the urethra and the trigone of the bladder. Hematuria associated with a rent of unknown cause at the proximal portion of the urethra has been observed in geldings.[119] The urethral rent appears to be identical to that often seen in stallions with hemospermia (see "Hemospermia," earlier).

The cause of urethral rents is idiopathic, but the reason the rent occurs invariably at the level of the ischial arch may be explained by the anatomy of the urethra. The diameter of the urethral lumen is approximately 1 to 1.5 cm at the origin of the urethra.[4] The lumen dilates to 3.5 to 5 cm in the pelvic portion of the urethra (i.e., at the pars pelvina) and decreases dramatically in diameter to 1 to 1.5 cm where the urethra bends sharply as it crosses the ischial arch. The sharp turn and the narrowing of the urethral lumen in the area of the ischial arch may expose the convex surface of the urethra at the level of the ischial arch to hydrodynamic forces not encountered by other portions of the urethra.

Pathophysiology

Because urethral rents communicate with the CSP, hemorrhage through the rent into the urethral lumen was thought to occur when pressure within this cavernosal space increases at the end of urination when the bulbospongiosus muscle contracts to expel the last vestige of urine.[100] Research, however, showed that the rise in pressure within the CSP associated with contraction of the bulbospongiosus muscle is slight. Investigators theorized that the most likely explanation for terminal hemorrhage in horses with a urethral rent is that the intraluminal urethral pressure suddenly decreases at the end of urination while the pressure in the CSP remains high.[127]

Even though the lesion in stallions is identical in appearance to that responsible for hematuria of geldings, the lesion in

stallions rarely causes macroscopic hematuria.[113,119] The reason for the difference in clinical signs between stallions and geldings is probably that during urination, pressure within the CSP of geldings is nearly double that of stallions.[127] The CSP of geldings is not as well developed as that of stallions, and the difference in volume of the cavernosal space between geldings and stallions results in different pressures in the CSP at the end of urination.

Diagnosis

Blood in urine that results from a urethral rent is characteristically discharged at the end of urination (i.e., terminal hematuria).[119] Occasionally, a horse with a urethral rent shows signs of dysuria, such as tenesmus at the end of urination. Endoscopic examination of the urethra reveals a 5- to 10-mm linear urethral defect on the convex surface of the urethra, distal to the openings of the bulbourethral glands, near the level of the ischial arch. Gross evidence of inflammation around the defect is not observed.

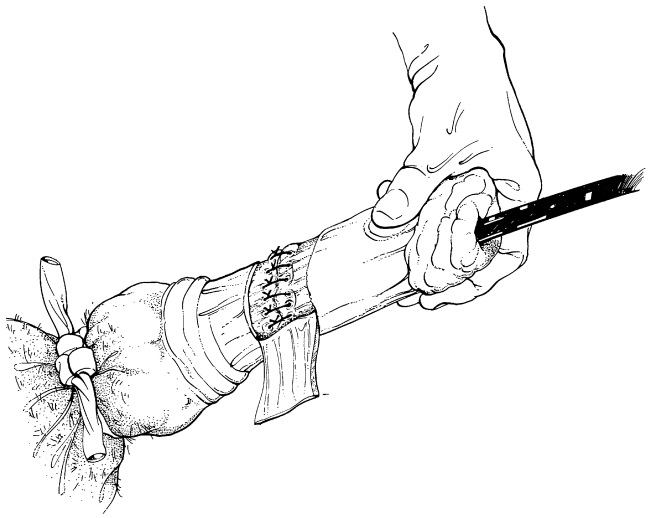

Figure 60-17. Segmental posthetomy. A cuff of epithelium is removed from the shaft of the penis.

Treatment

Some urethral rents heal spontaneously,[128] but horses with hematuria caused by a urethral rent can be treated successfully by temporary perineal urethrostomy (see "Temporary Perineal Urethrostomy," later, for a description of the surgical technique).[113,119,121] Surgery eliminates hematuria, presumably by reducing vascular pressure in the CSP, which prevents escape of blood through a rent at the end of urination, thereby allowing the rent to heal. A perineal incision that extends into the CSP but does not penetrate the urethra seems to be as effective as temporary urethrostomy in eliminating hematuria and may reduce the risk of complications associated with temporary urethrostomy, such as development of a urethral fistula[121] or stricture.[129] Although horses may bleed substantially from the perineal wound, especially at the end of urination, macroscopic hemorrhage from the urethral orifice and evidence of pain during urination are not observed after surgery.

GENERAL SURGICAL PROCEDURES

Segmental Posthetomy

Indications

Segmental posthetomy, or resection of a circumferential segment of the internal preputial lamina, is indicated for removal of preputial neoplasms, granulomas, or scars so extensive that simple excision of the lesion is impossible. Other terms for the procedure include *posthioplasty, circumcision,* and *reefing.*[130] Provided that the preputial lesions do not involve the underlying tunica albuginea, penile amputation can be avoided by segmental posthetomy. By removing most of the internal lamina of the prepuce, a paralyzed penis can be maintained permanently within the preputial cavity.[38]

Surgical Technique

Segmental posthetomy can be performed with the horse standing after anesthetizing the pudendal nerves (see "Diagnostic Procedures," earlier), but the procedure is most easily and safely accomplished with the horse anesthetized and positioned in dorsal or lateral recumbency. The urethra is catheterized, and the penis is extended by traction on gauze looped around the collum glandis. A tourniquet placed proximal to the surgical site may facilitate surgery. Parallel circumferential incisions through the preputial epithelium are created distal and proximal to the lesion, and these incisions are connected by a longitudinal incision (Figure 60-17). Care must be taken to avoid severing the large longitudinal subcutaneous branches of the external pudendal arteries and veins that lie superficial to the tunica albuginea.

When segmental posthetomy is performed to maintain a paralyzed penis within the external lamina of the prepuce (i.e., Adam's procedure), the distal circumferential incision should be made through the penile epithelium at the level where the internal preputial lamina inserts on the free body of the penis. The proximal circumferential incision should be made close to the preputial orifice.[38,131]

The cuff of integument between the incisions is dissected from the penis with scissors, taking care to avoid the large vessels. Normal alignment of tissue is maintained by placing four sutures at equidistant points around the circumference of the penis before the cuff of tissue is removed. The tourniquet is released, and all bleeding vessels are identified and ligated with absorbable sutures or cauterized. Loose adventitia is apposed with interrupted 2-0 absorbable sutures. The epithelium is apposed with interrupted 0 or 2-0 absorbable or nonabsorbable sutures.

When nearly all of the internal lamina is excised to permanently retain a paralyzed penis within the preputial cavity, the surgeon is faced with the difficult task of suturing a small distal circumferential incision to a much larger proximal circumferential incision.[38] To accomplish this, the length of the proximal circumferential incision can be decreased by removing two triangles of epithelium from the internal lamina proximal to the posthetomy, on each side of the penis. The triangles are about 3 cm wide and 4 cm long, and the base of each triangle is the circumferential incision. Suturing the sides of the triangles decreases the circumference of the proximal incision.

Aftercare

Stallions should wear a stallion ring for at least 2 weeks and must be isolated from mares for 2 to 4 weeks.[132] Regular exercise reduces postoperative edema. Nonabsorbable sutures should be removed 10 to 12 days postoperatively.

Bolz Technique of Phallopexy

Indication

The Bolz procedure is a technique used to permanently retract a paralyzed penis into the preputial cavity and is performed to avoid partial phallectomy.[41] This method of permanent retraction cannot be used if the penis or internal lamina of the prepuce is badly damaged or if the horse is still capable of attaining an erection. Damaged sections of prepuce, however, can be removed by segmental posthetomy during the same procedure.

Surgical Technique

The horse is anesthetized and positioned in dorsal recumbency. The urethra should be catheterized for easy identification. A 10-cm longitudinal incision is made on the perineal raphe just caudal to the scrotum (Figure 60-18, *A*), and the penis is bluntly separated from surrounding fascia, taking care to avoid damaging the surrounding large pudendal vessels (see Figure 60-18, *B*). The penis is retracted until the annular ring of the reflection of the internal preputial lamina onto the free body of the penis is visible at the cranial extent of the incision (see Figure 60-18, *C*). The penis is anchored in this position with two heavy, nonabsorbable percutaneous sutures through the annular ring on each side of the penis.

The anchoring sutures should penetrate the skin 2 to 4 cm from the incision at about the level of the middle of the incision. The sutures are inserted through the annular ring on the lateral surface of the penis, taking care to avoid entering the preputial cavity, the urethra, or cavernous tissue. An assistant should palpate the fornix of the preputial cavity during placement of the sutures through the annular ring to ensure that the sutures do not penetrate the preputial epithelium. The sutures exit the skin 2 to 3 cm from their entry points. They are tightened until the glans penis is flush with the preputial orifice and tied over rolls of gauze or large buttons to prevent the suture from cutting through the skin (see Figure 60-18, *D*). The subcutaneous tissue and skin are each closed separately.

The percutaneous anchoring sutures are removed after 10 to 12 days, at which time adhesions of sufficient strength to maintain the penis in its retracted position have formed. Necrosis of skin beneath the rolls of gauze is inevitable, but the technique allows adjustment of tension on the percutaneous sutures and repositioning of the penis. Precise positioning of the penis in the prepuce is important because, if the penis is inadequately retracted, the glans penis may protrude excessively through the preputial orifice, or if the penis is excessively retracted, the horse may develop urine scald from urinating in the preputial cavity. Two heavy absorbable sutures, substituted for the nonabsorbable percutaneous sutures, can be used to anchor the annular ring to the subcutaneous fascia.[133] Necrosis of skin is avoided, but the sutures cannot be adjusted after surgery.

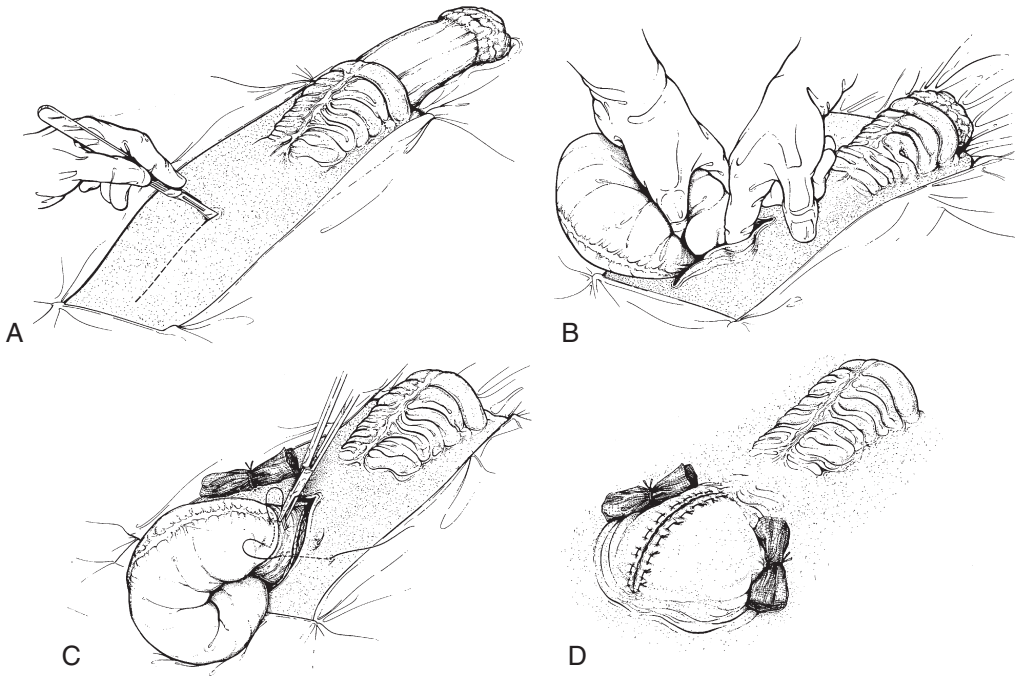

Figure 60-18. A, Bolz technique of phallopexy. A 10-cm incision is made on the perineal raphe just caudal to the scrotum. **B,** The penis is bluntly separated from surrounding fascia, taking care to avoid damaging the surrounding large pudendal vessels. **C,** The penis is retracted until the annular ring of the reflection of the internal preputial lamina onto the free body of the penis is visible at the cranial extent of the incision. The penis is fixed in this position with two heavy nonabsorbable percutaneous sutures through the annular ring on each side of the penis. **D,** The sutures are tightened until the glans penis is flush with the preputial orifice and are tied over rolls of gauze or large buttons to prevent the suture from cutting through the skin. The subcutaneous tissue and skin are closed separately.

Aftercare

The horse should be walked daily to minimize swelling, and heavy exercise can be resumed 2 to 3 weeks after the skin sutures have been removed. Retraction distorts the penis into a sigmoid curvature with acute bends, but penile blood supply and urination remain unaffected. The horse can be castrated during the same procedure, using either an inguinal or a scrotal approach, but the incision should be sutured because an open inguinal or scrotal wound may interfere with healing around the anchoring sutures. If the horse is castrated before the procedure and the scrotal wound is left unsutured to heal by secondary intention, the surgeon is confronted with the tedious task of caring for the protruded penis for several weeks while the scrotal incision heals.

Amputation of the Urethral Process

Indications

The urethral process is most commonly excised to remove a granuloma caused by cutaneous habronemiasis, when the affected horse fails to respond to medical therapy.[112] The urethral process is sometimes excised to remove a neoplastic lesion.[116]

Patient Preparation

The urethral process can be amputated with the horse standing and sedated after infiltrating the base of the urethral process with a local anesthetic agent, but the procedure is most easily and safely accomplished with the horse anesthetized and in dorsal recumbency.[112] The penis is prepared for aseptic surgery, and a urinary catheter is passed into the urethra. After placing traction on the urethral process with one or two Allis tissue forceps, two small-gauge needles (e.g., 23 or 25 gauge) are placed through the urethral process and the catheter at right angles to each other, proximal to the diseased portion of the urethral process (Figure 60-19). These needles anchor the urethral process to the catheter, making the incised margin of the process more stable and accessible for suturing.

Surgical Technique

A circumferential incision extending through the skin, CSP, and urethral mucosa is made around the base of the urethral process proximal to the affected tissue and distal to the anchoring hypodermic needles. The urethral mucosa is apposed to the epithelium of the remaining stump of the process with simple-interrupted or simple-continuous sutures of 4-0 or 5-0 absorbable suture.[116] The sutures should be closely spaced to compress the erectile tissue of the CSP. A simple-continuous suture pattern is probably more effective than a simple-interrupted one in compressing the erectile tissue of the CSP. The entire length of the process can be removed.

Aftercare

Stallions and recently castrated geldings should be isolated from mares for at least 3 weeks. Hemorrhage from the stump of the process, especially at the end of urination, should be expected for at least several days after the urethral process has been amputated. Fibrosis may occur at the site of amputation, and although this fibrous tissue may tear during copulation, it usually heals without complication.

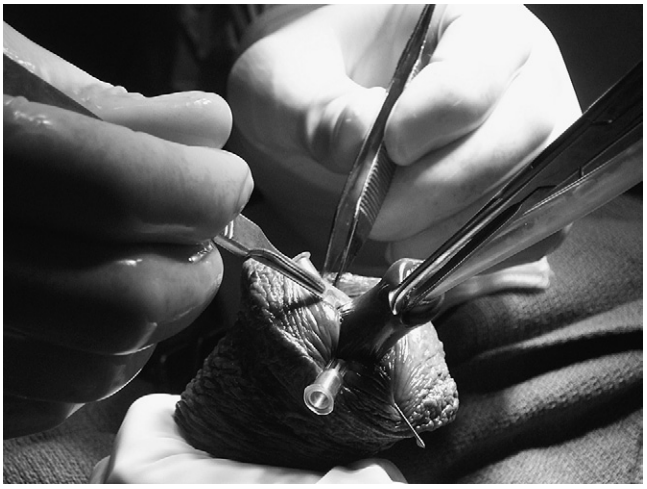

Figure 60-19. This urethral process was amputated to eliminate hemospermia caused by carcinoma of the urethral mucosa. The urethral process was stretched with tissue forceps, and two small-gauge needles were placed through the urethral process and the catheter at right angles to each other, proximal to the diseased portion of the urethral process, to anchor the urethral process to the catheter.

Partial Phallectomy

Indications

Partial phallectomy is indicated when permanent penile paralysis is accompanied by irreparable penile damage, and more commonly, when neoplasia has invaded the tunica albuginea or is so extensive that more conservative treatment by cryosurgery, hyperthermia, local excision, or segmental posthetomy is impossible. For geldings, partial phallectomy may be the most expedient means of treating urethral stenosis distal to the preputial orifice. Partial phallectomy of stallions is generally performed to salvage the horse for purposes other than breeding, but amputation of just the glans penis may not interfere with copulation.[134]

Patient Preparation

If possible, a stallion should be castrated 3 to 4 weeks before partial phallectomy, to avoid postoperative erection, which leads to hemorrhage and suture dehiscence. The procedure can sometimes be performed with the horse standing and sedated after anesthetizing the pudendal nerves or performing a ring block proximal to the site of amputation (see "Diagnostic Procedures," earlier),[27,135] but the procedure is most easily performed with the horse anesthetized and positioned in lateral or, preferably, dorsal recumbency. The urethra is catheterized with an equine male urinary catheter, and the penis is extended with gauze looped around the collum glandis. A tourniquet placed proximal to the proposed site of transection facilitates surgery.

Surgical Techniques

VINSOT TECHNIQUE OF PARTIAL PHALLECTOMY

One of the simplest techniques of phallectomy is the Vinsot procedure.[46,136] A triangular section of tissue that includes

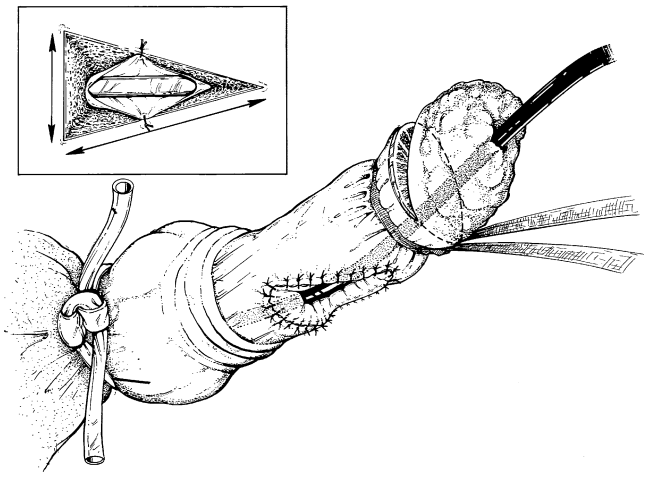

Figure 60-20. Vinsot technique of phallectomy. A triangular section of tissue is removed from the ventrum of the penis proximal to the proposed site of transection, taking care not to enter the urethral lumen. The exposed urethra is incised on its midline from the base to the apex of the triangle, and the incised edges of the urethra and the triangle's epithelial border are apposed with absorbable sutures in a simple-interrupted pattern. A nonabsorbable ligature is placed around the penis distal to the apex of the triangle, and the penis is severed distal to the ligature.

Figure 60-21. A bander castration device (Callicrate Bander) with a latex loop (ES-10) can be applied to the penis slightly proximal to the site of transection to prevent hemorrhage from the penile stump after partial phallectomy.

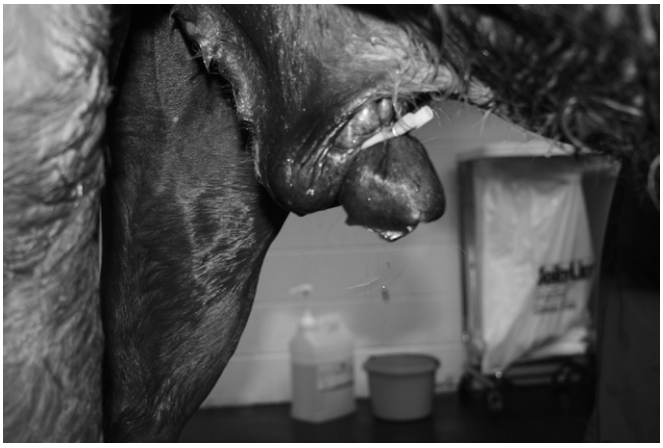

Figure 60-22. When performing a partial phallectomy using the Vinsot technique, the surgeon can leave the transected end of the penis unsutured to heal by secondary intention. To prevent hemorrhage from the corporeal bodies, a tightly fixed nonabsorbable ligature is placed around the penis 2 to 3 cm distal to the newly created stoma, before the penis is transversely severed 1 to 2 cm distal to the ligature. The ligature used to compress the stump of the penis of this horse was a latex loop applied with a bander castration device, both of which are shown in Figure 60-21.

epithelium, fascia, bulbospongiosus muscle, and CSP is removed from the ventrum of the penis proximal to the proposed site of transection, taking care not to enter the urethral lumen (Figure 60-20). The triangle has a 2.5-cm base and 4-cm sides. Its apex points distad and is located about 4 or 5 cm proximal to the proposed site of transection. The exposed urethra is incised on its midline from the base to the apex of the triangle, and the incised edges of the urethra and the triangle's epithelial border are apposed with simple-interrupted or simple-continuous absorbable sutures. The sutures should include the tunica albuginea of the CSP, and they should be closely spaced to compress the erectile tissue of the CSP. A simple-continuous suture pattern is probably more effective than a simple-interrupted one in compressing the erectile tissue of the CSP. The diseased portion of the penis is removed 4 to 5 cm distal to the urethrostomy using a wedge-shaped incision. Large vessels on the dorsal and lateral aspects of the tunica albuginea are ligated with absorbable suture material, and the corporeal bodies are compressed with absorbable sutures placed through the tunica albuginea in an everting or appositional pattern. The penile or preputial integument is sutured with absorbable or nonabsorbable sutures placed in an everting or appositional pattern.

Instead of suturing the end of the stump, the surgeon can leave it unsutured to heal by secondary intention. To prevent hemorrhage from the corporeal bodies, a tightly fixed, nonabsorbable ligature is placed around the penis 2 to 3 cm distal to the apex of the triangle, before the penis is transversely severed 1 to 2 cm distal to the ligature. A bander castration device (Callicrate Bander) with a latex loop (ES-10) is effective in maintaining continuous, maximal pressure on the stump of the penis to prevent hemorrhage from the corporeal tissue and the vasculature (Figures 60-21 and 60-22).[135]

Rather than removing a triangle of tissue overlying the urethra, the technique can be simplified by making a 4- to 5-cm longitudinal incision into the urethral lumen.[135,137] The incised edges of the urethra and the integument are apposed with simple-interrupted or simple-continuous absorbable sutures. These sutures incorporate and compress the cavernous tissue of the CSP. The Vinsot technique, especially its modification,[135,137] can often be performed with the horse standing. Primary disadvantages of the technique, or its modification, are the tendency of the urethra to stricture and the tendency for some horses to develop urine-induced contact dermatitis.[46,135]

WILLIAMS TECHNIQUE OF PARTIAL PHALLECTOMY

The likelihood of urethral stricture and urine-induced contact dermatitis is decreased when the Williams technique of partial phallectomy is employed.[134] With this technique, a triangle of tissue with similar dimensions to those described in the Vinsot technique is removed from the ventrum of the penis (Figure 60-23, *A*). The triangle's apex is directed proximad, rather than distad, and the base of the triangle is the site of penile transection. The urethra is split on its midline from the base to the apex of the triangle, and the incised edge of the urethra and the triangle's epithelial edge are apposed with 3-0 simple-interrupted or simple-continuous absorbable sutures. These sutures incorporate and compress the cavernous tissue of the CSP. A simple-continuous suture pattern is probably more

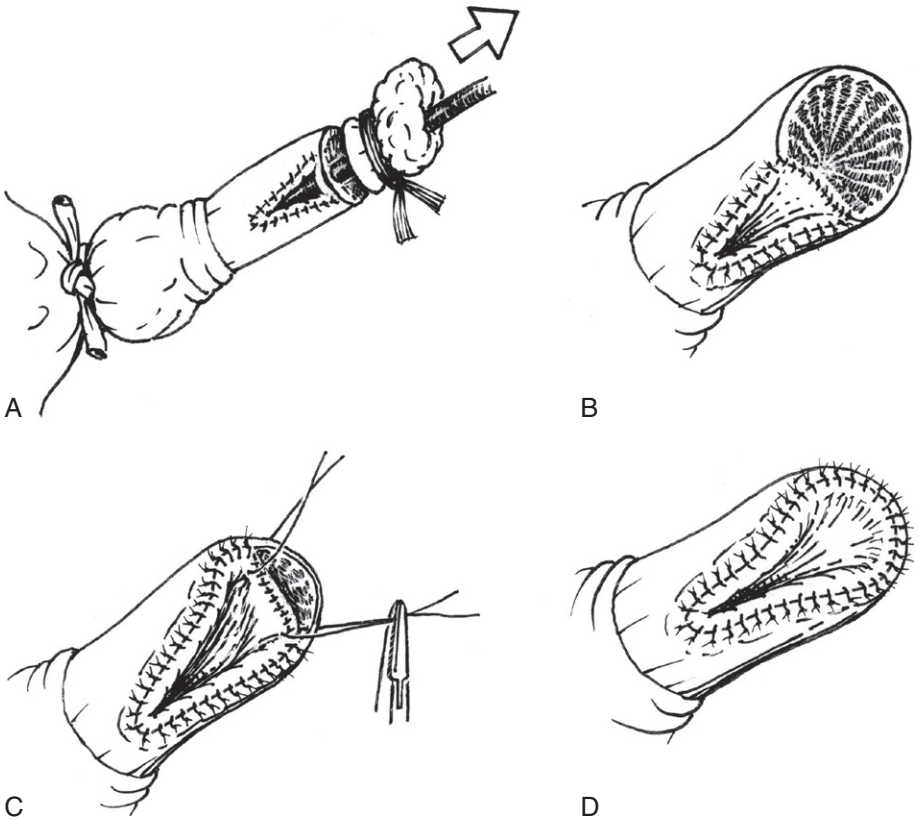

Figure 60-23. A, Williams technique of phallectomy. A triangle is removed from the ventrum of the penis. The triangle's apex is directed proximad. The urethra is split on its midline from the base to the apex of the triangle, and the edges of the urethra and the triangle's epithelial edges are apposed with simple-interrupted absorbable sutures. **B,** Before closing the stump, the transected edge of the CSP at the base of the triangle can be compressed with a simple-continuous absorbable suture line through the urethral mucosa and tunica albuginea. **C,** The stump is closed with interrupted sutures that pass through the urethra, the tunica albuginea of the urethral groove, and the tunica albuginea of the dorsum of the corpus cavernosum penis (CCP) and the penile or preputial epithelium. The sutures should be preplaced at equidistant intervals for an even closure. **D,** The sutures are tightened and tied, compressing the cavernous spaces; and the epithelium is apposed to the urethral mucosa.

effective than a simple-interrupted one in providing compression. The urethral catheter is removed, and the penis is obliquely transected at the base of the triangular urethrostomy in a craniodorsal direction, so that the dorsum of the penile stump is longer than the ventrum (see Figure 60-23, *B*). Large branches of the external pudendal vessels that reside in loose fascia on the dorsal and lateral aspects of the tunica albuginea are ligated with absorbable suture. The transected edge of the CSP at the base of the triangle can be compressed by placing a simple-interrupted or simple-continuous pattern of 3-0 absorbable sutures through the urethral mucosa and tunica albuginea at the base of the triangle.

The rest of the stump, including the CCP, is closed with interrupted absorbable or nonabsorbable sutures that pass through the urethra, the tunica albuginea of the urethral groove, and the tunica albuginea of the dorsum of the CCP and the penile or preputial epithelium (see Figure 60-23, *D*). The sutures should be preplaced at equidistant intervals for an even closure. When these sutures are tightened and tied, the erectile bodies are compressed, and the epithelium is apposed to the urethral mucosa (see Figure 60-23, *E*). Alternatively, after compressing the CSP at the base of the triangle, the stump can be closed by first suturing the urethral mucosa and tunica albuginea of the urethral groove to the tunica albuginea of the dorsum of the CCP with interrupted absorbable sutures placed at bisecting intervals. Then the penile or preputial integument is sutured to the urethral mucosa with interrupted absorbable or nonabsorbable sutures. Suturing the stump in this manner necessitates placing three rows of sutures through the urethral mucosa and CSP but ensures good compression of corporeal tissue.

SCOTT TECHNIQUE OF PARTIAL PHALLECTOMY

With this technique, a circumferential transverse incision through the epithelium of the body of the penis or prepuce is made at the intended site of transection, and branches of the external pudendal vessels are ligated.[95] Dissection is continued through the CCP to the urethral groove. The CSP is circumferentially incised to the urethra, which is easily identified by the urinary catheter in its lumen, and a 4- to 5-cm segment of urethra is dissected free from the amputated section of penis (Figure 60-24, *A*).

The stump of the CCP is closed by apposing the outer perimeter of its tunica albuginea to the tunica albuginea of the urethral groove with interrupted absorbable sutures preplaced at equidistant intervals (see Figure 60-24, *B*). The sinusoidal spaces of the CSP are closed by suturing the tunica albuginea surrounding the CSP to the submucosa of the urethra with interrupted or continuous absorbable sutures (see Figure 60-24, *C*). The urethral stump is divided into three equal triangular segments, with the apex of each triangle pointing distad. These

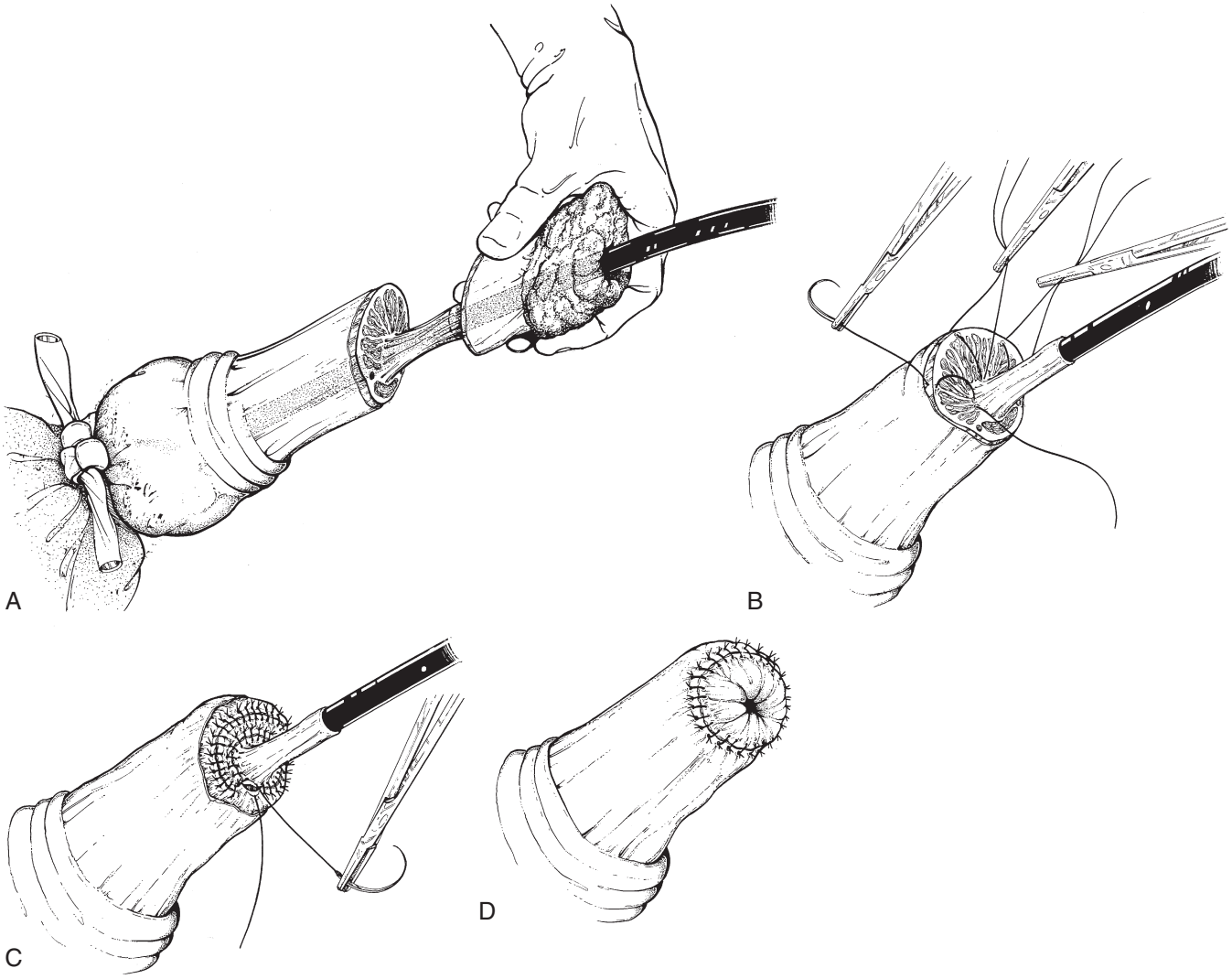

Figure 60-24. Scott technique of phallectomy. **A,** A circumferential transverse incision through the epithelium of the body of the penis or prepuce is made at the intended site of transection. Dissection is continued through the corpus cavernosum penis (CCP) to the urethral groove. The corpus spongiosum penis (CSP) is circumferentially incised to the urethra, and a 4- to 5-cm segment of urethra is dissected free from the amputated section of penis. **B,** The stump of the CCP is closed by apposing the outer perimeter of its tunica albuginea to the tunica albuginea of the urethral groove with interrupted absorbable sutures preplaced at equidistant intervals. **C,** The sinusoidal spaces of the CSP are closed by suturing the tunica albuginea surrounding the CSP to the submucosa of the urethra with interrupted or continuous absorbable sutures. **D,** The urethral stump is stretched and folded back over the end of the penis, where it is sutured to the penile or preputial epithelium and underlying tunica albuginea.

segments are intermeshed with similarly prepared segments of penile or preputial integument and are apposed with simple-interrupted absorbable or nonabsorbable sutures. Sutures should include underlying tunica albuginea. Instead of dividing the urethral stump into three triangles, the urethral stump can be stretched and folded back over the end of the penis, where it is sutured to the penile or preputial epithelium and underlying tunica albuginea (see Figure 60-24, *D*).

PARTIAL PHALLECTOMY BY *EN BLOC* RESECTION WITH PENILE RETROVERSION

Removal of the free portion of the penis, the internal lamina and external lamina of the prepuce, and regional lymph nodes may be indicated when these structures are extensively affected with neoplasia.[99] With this technique of partial phallectomy, a fusiform incision is created around the preputial orifice. The incision begins 6 cm cranial to the orifice and ends 10 cm caudal to it. The incision is carried to the deep fascia of the abdominal tunic, and if neoplasia has metastasized to the superficial lymph nodes, dissection is extended through this plane to both superficial inguinal rings, and the superficial inguinal lymph nodes are removed. The penis is amputated approximately 6 to 8 cm caudal to the fornix of the prepuce, and the amputated portion of the penis and the prepuce are removed *en bloc*. The penile shaft is amputated using a method similar to that described by Scott, so that 4 cm of urethra is left protruding from the penile stump[95] (Figure 60-25, *A*). The technique can be modified by amputating the penis using the Williams or Vinsot technique of partial phallectomy.[138]

By bluntly separating penile fascia, the stump of the penis is retroverted through a 6-cm subischial incision created approximately 20 cm ventral to the anus, so that its distal end points

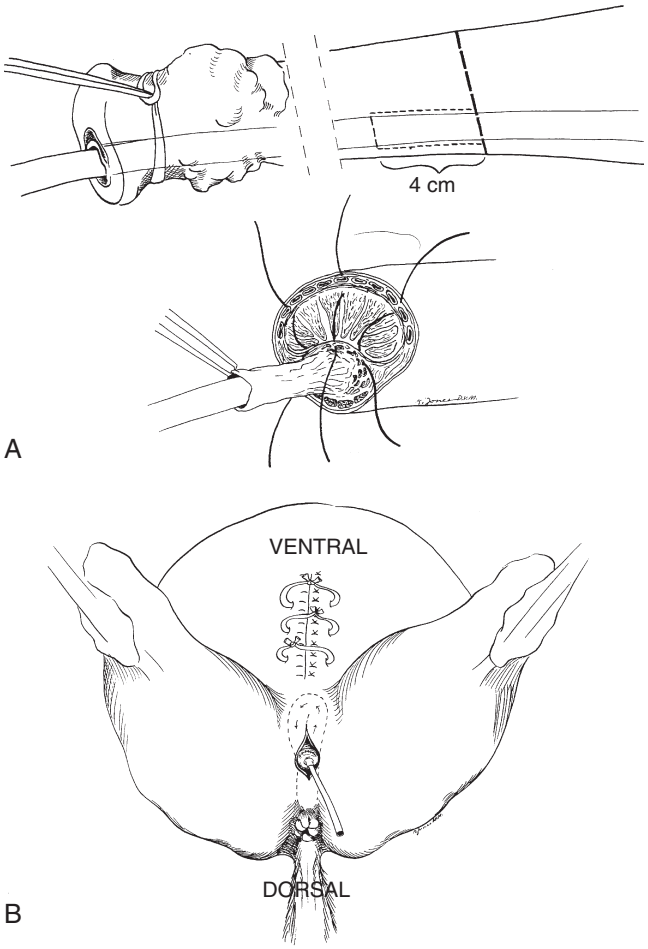

Figure 60-25. *En bloc* resection of the penis. **A,** Four centimeters of urethra is left beyond the penile stump. **B,** The position of the penile stump when retroverted is demonstrated. (From Markel MD, Wheat JD, Jones K: Genital neoplasms treated by *en bloc* resection and penile retroversion in horses: 10 cases 1977-1986. J Am Vet Med Assoc 192:396, 1988)

caudad and extends just beyond the subischial incision (see Figure 60-25, *B*). The tunica albuginea of the CCP and the fascia of the penis are sutured to the subcutaneous tissue of the subischial incision. The ventral aspect of the urethra is incised longitudinally over its 4-cm length, and the edges of the urethra are sutured to the surrounding edges of the incised subischial skin. Penrose drains are placed deeply at the cranial incision, and the subcutaneous tissue and skin are each closed separately.

PARTIAL PHALLECTOMY BY *EN BLOC* RESECTION WITHOUT PENILE RETROVERSION

This technique of phallectomy is similar in many respects to phallectomy by *en bloc* resection with penile retroversion, but with this technique, the stump of the penis is not retroverted but is maintained in its normal ventral position.[139] With this technique, a fusiform incision is created on the midline beginning at the umbilicus. The incision extends caudad on each side of the preputial orifice and continues on the midline to a point 10 cm caudal to the preputial orifice. The caudal portion of the incision is extended and deepened to expose and remove the inguinal lymph nodes, if neoplasia has metastasized to these structures.

Blunt dissection is continued into the loose areolar tissue of the prepuce, ligating large vessels as they are encountered. After the shaft of the penis is exposed, dissection is redirected along the shaft of the penis in a plane superficial to the loose subcutaneous tissue overlying the vasculature of the penis. At least 10 cm of the shaft should be exposed. A tourniquet is applied around the shaft of the penis proximal to the site of amputation. The dorsal arteries and veins of the penis are ligated and transfixed to the tunica albuginea. The penis is transected caudal to the fornix of the prepuce, using the method described by Williams.[134] After the tourniquet is removed, the stump is fixed to the body wall on the midline with heavy absorbable interrupted sutures. The subcutaneous tissue cranial to the penile stump surrounding the exposed penile shaft is apposed. Skin is sutured to the tunica albuginea and the urethral mucosa of the new urethral orifice. The skin cranial and caudal to the urethral orifice is sutured.

This technique of *en bloc* resection requires a smaller incision and results in less alteration to the appearance of the horse than does the retroversion technique, while still allowing the surgeon to remove extensive portions of the penis and extirpate the regional lymph nodes.[139]

Aftercare

Because the procedure is generally performed to salvage the horse for purposes other than breeding, stallions should be castrated several weeks in advance of partial phallectomy. Stallions and recently castrated geldings should be isolated from mares for 2 to 3 weeks and should wear a stallion ring on the penile stump during this time.

Complications

Hemorrhage from the penile stump, especially at the end of urination, should be expected for at least several days after partial phallectomy. Hemorrhage usually emanates from the CSP. Partial phallectomy of geldings is attended by less hemorrhage than partial phallectomy of stallions. Excessive hemorrhage may be caused by minor dehiscence, which is usually of no consequence. Dehiscence of sutured erectile tissue may lead to the formation of a large hematoma. Other complications of partial phallectomy in the immediate postoperative period include pain (which can be severe with *en bloc* resection), infection of the surgical wound, edema of the prepuce, and acute urinary obstruction caused by edema of the urethra.[89,90,99,139] Long-term complications include chronic recurrent cystitis, urine-induced dermatitis, dysuria caused by urethral stricture, recurrence of neoplasia at the site of amputation, and neoplastic metastases to inguinal lymph nodes and internal organs.

Temporary Perineal Urethrostomy

Indications

Temporary urethrostomy at the ischial arch is performed to provide access to small cystic calculi, to treat horses affected with hemospermia or hematuria, and to divert the flow of urine from the penile urethra for such conditions as urethral laceration or urethral urolithiasis.

Surgical Technique

Temporary perineal urethrostomy is best performed with the horse standing and sedated after administering epidural anesthesia or infiltrating the tissue at the proposed site of incision with a local anesthetic agent. A 6- to 8-cm vertical incision is created on the perineal raphe about 2 to 3 cm below the anus. The incision is extended through the skin, the retractor penis and bulbospongiosus muscles, the CSP, and the urethral mucosa (see Figure 60-2).

Preoperative insertion of a large-bore urethral catheter facilitates identification of the urethra. The perineal incision should "funnel" to a short urethral incision as it deepens to avoid postoperative pocketing of urine in the tissues. If the incision strays from the midline, profuse hemorrhage can result from laceration of branches of the external pudendal artery.[36] The urethrostomy is generally allowed to heal by secondary intention; development of clinically apparent urethral stenosis after this procedure is rare. The urethrostomy normally heals within 2 weeks.[127]

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CHAPTER
61 Vulva, Vestibule, Vagina, and Cervix

J. Brett Woodie

The caudal aspect of the reproductive tract is composed of the vulva, vestibule, vagina, and cervix. These structures are susceptible to a variety of injuries during breeding and foaling. Conformational abnormalities of the caudal reproductive tract may predispose the mare to pneumovagina, pooling of urine, and other problems. Ultimately problems associated with the caudal reproductive tract can lead to infertility.

ANATOMY

The external genitalia of the mare are the perineum and the vulva. The perineum is defined as the region bound dorsally by the base of the tail, laterally by the semimembranosus muscles and sacrosciatic ligaments, and ventrally by the ventral commissure of the vulva (Figure 61-1).[1] The vulva includes the two labia and the clitoris. The external orifice of the vulva is typically 12 to 15 cm long.[2] The labia of the vulva in their normal position should be vertical and meet dorsally to form the dorsal commissure, which is located just ventral to the anus. The labia should meet ventrally to form the ventral commissure, which should be located caudal and ventral to the ischial arch. Approximately two thirds of the vulvar cleft should be ventral to the ischial arch.[3] Normally, the labia of the vulva are muscular and resist manual separation. The constrictor vulvae muscle lies deep to the skin of the labia. The internal pudendal vessels provide vascular supply to the vulva, its labia, and the clitoris. The pudendal and caudal rectal nerves provide motor innervation to the muscles of the vestibule and vulva.[4] These nerves also supply sensory fibers to the mucous membrane of the vulva and the skin of the labia.[4]

The fibromuscular perineal body lies between the anus and the vulva. The perineal body is formed by the fibers of the external anal sphincter and the constrictor vulvae muscles (Figure 61-2).

The clitoris is the homologue of the penis and is located at the ventral commissure of the vulvar labia. The clitoral glans is approximately 2.5 cm in diameter and contains erectile tissue similar to that of the corpus cavernosum penis. The clitoral fossa that surrounds the glans is located laterally and ventrally. The

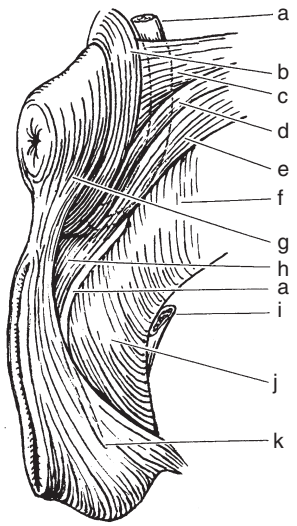

Figure 61-1. Muscles of the perineal region. *a,* Retractor clitoridis; *b,* external anal sphincter (cranial superficial part); *c,* levator ani; *d,* subanal loop of levator ani; *e,* ventral part of levator ani; *f,* urethralis; *g,* external anal sphincter (caudal superficial part); *h,* perineal septum; *i,* crus clitoridis (cut); *j,* constrictor vestibuli; *k,* constrictor vulvae.

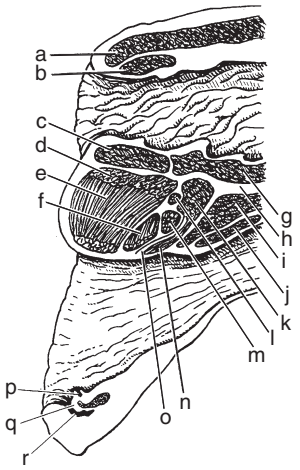

Figure 61-2. Drawing of a sagittal section of the muscles that are part of the vulvar and vestibular sphincters and the perineal body. *a,* External anal sphincter; *b,* internal anal sphincter; *c,* internal anal sphincter; *d,* external anal sphincter; *e,* muscular fibers from external anal sphincter to constrictor vulvae; *f,* cranial superficial part and deep part of external anal sphincter; *g,* rectal musculature; *h,* rectovaginal septum; *i,* vaginal musculature; *j,* vaginal musculature; *k,* rectal part of retractor clitoridis; *l,* clitoral part of retractor clitoridis; *m,* subanal loop of levator ani; *n,* decussation of retractor clitoridis; *o,* perineal septum; *p,* clitoral sinus; *q,* glans clitoris; *r,* clitoral fossa.

body of the clitoris is approximately 5 cm in length and is attached to the ischial arch by two crura.[2]

The vestibule is the terminal part of the genital tract. It is a tubular structure approximately 12 to 15 cm long that connects the vulva to the vagina.[2] The normal configuration of the vestibule is a ventrodorsal slope oriented rostrad.[5] The cranial extent of the vestibule ends at the level of the transverse fold, which is located dorsal to the external urethral orifice. The lateral and ventral surface of the vestibule is covered by the constrictor vestibuli muscle.[1] Dorsally this muscle is incomplete. The constrictor vestibuli muscle blends caudally with the constrictor vulvae muscle. The constrictor vestibuli muscles, the pillars of the hymen, and the floor of the pelvis meet to form the vestibular sphincter.[6]

The vagina is a tubular structure extending craniad from the transverse fold of the external urethral orifice to the vaginal fornix around the cervix. The vagina is related to the rectum dorsally, the bladder and urethra ventrally, and the pelvic wall laterally. The majority of the vagina is located retroperitoneally but a small cranial portion of the ventral aspect and larger portions of the dorsal aspect are always covered with peritoneum.[2] The extent that the cranial part of the vagina is covered with peritoneum is related inversely to the fullness of the rectum and bladder.[4] The vascular supply of the vagina is derived from the internal pudendal vessels. Since there is no skeletal muscle in the vagina, there is no motor innervation. There are multiple sympathetic ganglia in the vaginal wall.[7]

The cervix is an extension of the uterine body with the caudal portion positioned in the cranial aspect (fornix) of the vagina. The caudal portion of the cervix is referred to as the *external cervical os,* which is covered by vaginal mucosa. The cervix is a tubular muscular structure lined with mucosa that forms many longitudinal folds. It functions as a sphincter separating the caudal reproductive tract from the uterus.

There are essentially three protective barriers in the caudal reproductive tract. The constrictor vulvae muscles of the labia form the first barrier, the second is the vestibular sphincter, and the cervix is the third. When any of these barriers becomes incompetent, contamination of the reproductive tract may occur and result in infertility.

PATHOPHYSIOLOGY

Failure of the protective barriers in the caudal reproductive tract can be caused by numerous factors. Conformational abnormalities such as a flat croup, sunken anus, and underdeveloped vulvar labia predispose the mare to pneumovagina.[8] Poor perineal conformation is often found in older multiparous mares that have a thin body condition.

A large percentage of the injuries to the caudal reproductive tract occur secondary to trauma from breeding or foaling. Some of the injuries are readily apparent immediately after delivery, whereas others can go unnoticed until it is time to rebreed the mare. Injuries that occur during foaling include cervical lacerations, cervical contusions, perineal lacerations, rectovestibular fistulas, urinary bladder eversion or prolapse, urinary bladder rupture, vaginal contusions, vaginal lacerations, vestibular lacerations, and uterine prolapse or rupture.[9-11] These injuries range from minor to severe, and not all require intervention. However, extensive surgical repair may be necessary to restore normal anatomic function. Without repair of the injured tissue, infertility is often the result. A thorough examination is necessary to ensure that more than one anatomic structure does not require surgical intervention.

Injuries during breeding do not occur frequently but may require medical and/or surgical treatment.[12] Perforation of the vagina usually occurs during intromission and is typically located at the cranial aspect of the vagina adjacent to the cervix. Predisposing factors for this type of injury have been reported to represent an overly vigorous stallion or a large stallion breeding a small mare.[12] Depending on the location and depth of penetration, the peritoneal cavity may or may not be entered. Semen is not sterile, and peritonitis may develop secondary to ejaculation into the peritoneal cavity.[13,14] Eventration can occur through a tear in the vaginal wall.[15] The development of an abscess in the pelvic cavity may result from an injury that does not involve the peritoneal cavity.[16] Inadvertent entry of the stallion's penis into the mare's rectum during breeding can result in rectal injury or perforation. This type of accident is not common and has been associated with poor vulvar conformation, small vulvar opening after a Caslick procedure, and relaxed anal sphincter following palpation *per rectum.*[12]

DIAGNOSTIC PROCEDURES

Examination of the reproductive tract of the mare should progress from an external to an internal evaluation. A logical starting point is to visually evaluate the conformation of the perineum and vulva and examine the latter for evidence of discharge. The external genitalia should be examined for intersex conditions and diseases such as neoplasia. The conformation of the vulva is very important because this anatomic structure is the first line of defense in protecting the reproductive tract from contamination. The labia should be evaluated for symmetry, position, angle, and tone. Pascoe's Caslick Index is an objective scoring system that can be used to determine

the risk for ascending infection to the reproductive tract.[5] The length of the vulva (in centimeters) is multiplied by the angle of declination of the vulvar lips. Higher pregnancy rates have been reported in mares that had a Caslick Index of less than 150.[3]

The vestibule, vagina, and cervix should be examined digitally as well as visually using a speculum. Samples for endometrial biopsy, culture, and cytology should be taken. The reproductive tract should be assessed by palpation and ultrasonography *per rectum*.

Chemical Restraint

The majority of diagnostic and surgical procedures that are performed on the caudal reproductive tract of the mare are accomplished with chemical restraint of the standing mare. Chemical restraint is usually supplemented with local or regional anesthesia, or both, for surgical procedures. The drugs used for chemical restraint provide variable degrees of sedation and analgesia. A number of sedative-hypnotic, tranquilizing, and opioid agents are available for use in the horse (see Chapter 22). Selection is based on personal preference and the type of surgical procedure that is being performed. The use of drug combinations often optimizes restraint and analgesia as compared to the use of an individual drug.

Acetylpromazine, a phenothiazine tranquilizer, produces sedation without analgesia. The peak onset of sedation following intravenous administration (0.04 mg/kg) is at approximately 30 minutes and may last for 1 to 3 hours depending on dose and route of administration.[17] Because the phenothiazine tranquilizers do not produce analgesia, they should be used in combination with an analgesic agent if a surgical procedure is performed.

Xylazine and detomidine are the most commonly used sedative-hypnotic drugs for standing chemical restraint. These drugs produce varying degrees of sedation, muscle relaxation, analgesia, and ataxia depending on the dose administered. Both drugs induce diuresis, which may warrant placement of a urinary catheter.[18] Detomidine has a longer duration of action and greater potency compared to xylazine.[19] Each of these drugs can be used in combination with an opiate such as butorphanol. This typically produces more profound sedation and analgesia. Reduced doses for chemical restraint should be considered when working on draft breeds because the desired effect can be achieved at lower doses than those used in light breeds such as Thoroughbreds.[20]

Epidural Anesthesia

Epidural anesthesia is achieved by injecting the local anesthetic solution between the dura mater and the periosteum of the spinal canal, which blocks conduction in the caudal nerve roots. Caudal epidural anesthesia implies that sensory innervation is lost without affecting the motor control of the hind limbs. The tail should be tied overhead to support the horse if ataxia develops. The sacrocaudal or first intercaudal vertebral space is selected as the injection site for epidural anesthesia.[21] This location is found by grasping the tail and moving it up and down. The first articulation caudal to the sacrum is the first intercoccygeal space. The site should be clipped and aseptically prepared. The epidural anesthetic agent should be administered using aseptic technique. Various injection techniques and drug combinations can be used. The mare should be sedated and restrained in stocks during administration of epidural anesthesia. A small skin bleb of local anesthetic can be deposited at the proposed injection site to facilitate placement of the spinal needle. A 20-gauge, 7.5-cm (3-inch) spinal needle should be positioned just cranial to the dorsal spinous process of the second coccygeal vertebra. The needle is inserted through the skin at a 30-degree angle relative to the tail and inserted craniad. If bone is encountered, the needle should be redirected. Once the needle is placed, the stylet should be withdrawn and the hub of the needle is filled with the local anesthetic to be injected. If the needle is positioned in the epidural space, the fluid will be aspirated (hanging drop technique). Minimal resistance is encountered during epidural injection. Mares that have had previous epidural injections may develop fibrous scar tissue over the intercoccygeal space, making needle placement more difficult.[21] Following injection, the needle should be removed. Caution should be exercised during placement of the spinal needle because some mares kick during the procedure. An epidural catheter can be placed to facilitate readministration of anesthetic agents if needed during the surgery.[20] Loss of anal sphincter tone is common when epidural anesthesia is achieved.

The type of blockade (motor and/or sensory) and the duration of effect depend on the type of drug(s) and the volume that is administered. Local anesthetics should produce motor and sensory blockade, whereas only sensory innervation is lost with other drugs. A dosage of 5 to 7 mL of 2% lidocaine hydrochloride per 500 kg body weight should produce analgesia within 5 to 15 minutes, and the duration of analgesia should be 60 to 90 minutes.[21] The addition of 2% mepivacaine hydrochloride to the same dosage will produce analgesia in 10 to 30 minutes and last for 90 to 120 minutes.[21] However, ataxia is a complication when using local anesthetics.

Epidural administration of α_2-adrenergic receptor agonists such as xylazine will provide profound analgesia without the complication of ataxia.[22,23] The recommended dosage of xylazine is 0.17 mg/kg. The onset of action is 10 to 30 minutes and the duration of analgesia is 2.5 to 4 hours.[21] The xylazine should be diluted in saline to a total volume of 6 to 10 mL. Epidural administration of detomidine (30 to 60 µg/kg) provides analgesia for 2 to 3 hours but produces sedation and ataxia.[21,22] The combination of lidocaine (0.22 mg/kg) and xylazine (0.17 mg/kg) produces significantly longer analgesia (approximately 5 hours) with only mild ataxia when compared to either agent used alone.[23]

Ataxia and the potential for recumbency must be considered when determining which drug or combination of drugs and the dosage that is to administer.

Preparation of the Surgical Site and the Mare

The rectum should be evacuated before starting any surgical procedure on the caudal reproductive tract. The tail should be wrapped and secured so that it does not interfere during surgery; then the perineum and buttocks should be scrubbed with a nonirritating soap. A dilute antibacterial solution of 1% povidone-iodine (Betadine) can be used to cleanse the vestibule and vagina. All fluid should be removed from the vestibule and vagina before beginning surgery.

Tetanus prophylaxis should be administered if necessary. The use of perioperative antibiotics and anti-inflammatory drugs is

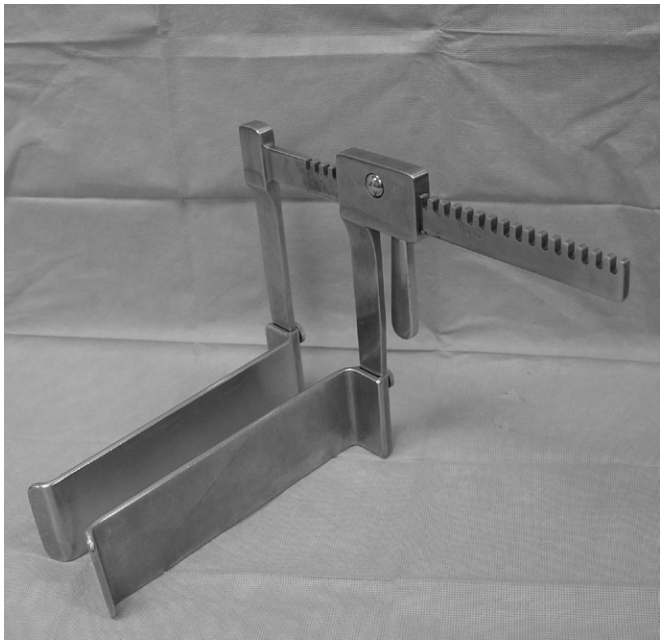

Figure 61-3. Modified Finochietto retractor with long blades. This retractor is very useful for certain surgical procedures of the caudal reproductive tract, such as repair of a cervical laceration.

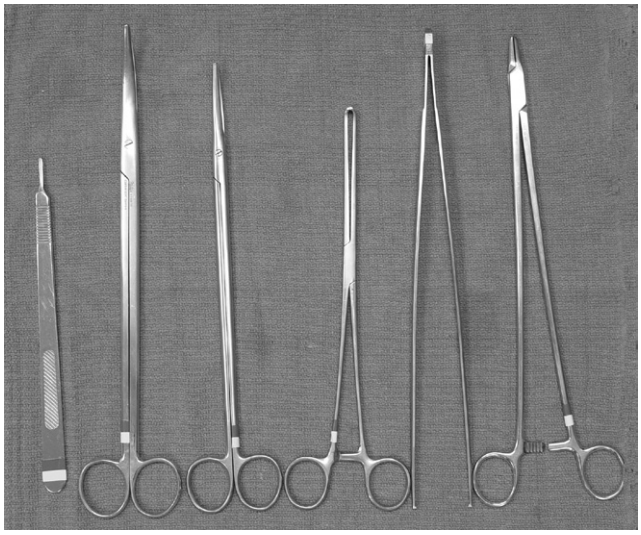

Figure 61-4. Long-handled surgical instruments.

at the discretion of the surgeon. The mare should be restrained in stocks, if available, but some procedures can be performed with the mare backed out of the stall door.

Instrumentation

Special instruments are needed to perform certain surgical procedures on the caudal reproductive tract. Illumination of the surgical field is crucial. Options include a light source that can be attached to the self-retaining retractor, a headlamp, or overhead surgical lights that can be adjusted. Retractors are needed to expose the surgical site. A modified Finochietto retractor with long blades (Figure 61-3) is very helpful in repairing cervical lacerations. Balfour retractors are useful for surgical procedures caudal to the vestibulovaginal junction. Positioning the retractor with the retaining mechanism dorsal to the anus and secured to the tail head using umbilical tape keeps the retractor in place and out of the surgical field. Handheld instruments need to be longer than conventional instruments (Figure 61-4). Scalpel handles, scissors, thumb forceps, Allis tissue forceps, and needle holders should be 25 to 30 cm in length.[24]

DISORDERS REQUIRING SURGERY

Pneumovagina

Pneumovagina leads to chronic inflammation and infection of the vagina and uterus. This is a cause of infertility in the mare. The most common cause of aspiration of air into the vagina is poor perineal conformation. Pneumovagina may develop secondary to foaling trauma with scar tissue formation, excessive stretching of the vulvar tissues from foaling, or poor body condition.[8] Sinking of the anus into the pelvic canal causes the dorsal commissure of the vulva to tip forward so that it is oriented horizontally rather than vertically. This can disrupt the vulvar seal and lead to pneumovagina as well as fecal contamination of the caudal reproductive tract. When manually separating the labia, an inrushing of air indicates a predisposition to pneumovagina. In some mares, pneumovagina only occurs during estrus when perineal tissues are more relaxed.[25] Urovagina can result from the same causes as pneumovagina, and the mare should be evaluated to determine if more than one surgical procedure is necessary.

Episioplasty

CASLICK PROCEDURE

The most common surgical procedure for correction of pneumovagina is the Caslick procedure.[6,16] The intent of the surgical procedure is to form a seal to prevent aspiration of air and fecal material. The original procedure was described by Caslick in 1937.[6] Dr. Caslick felt that introduction of air into the vagina during treatment of uterine infection exacerbated the problem. He observed a dramatic clinical improvement in uterine infections when local treatment was stopped and the dorsal half of the labia was temporarily closed. The procedure subsequently gained acceptance as treatment for pneumovagina.

Pneumovagina can be prevented in most cases by suturing the labia together to the level of the ventral border of the ischial arch. The ventral limit is determined by palpating the ischial arch just lateral to the opening of the vulva.[25] The mare should be sedated and restrained so that the clinician is protected from a kick injury. The perineum is prepared and local anesthetic is infiltrated into the labial margins extending from the ischial arch to the dorsal commissure. A 4- to 8-mm strip of mucosa is excised with scissors along the mucocutaneous junction on each labium (Figure 61-5). It is important to include the dorsal commissure of the vulva in the excision so that a seal at the dorsal aspect is achieved. A scalpel can be used to incise the labia at the mucocutaneous junction rather than excise any tissue. The incision must be of sufficient depth so that a tissue gap of 4 to 8 mm is created.

Closure is achieved using 2-0 absorbable or nonabsorbable suture material in a continuous pattern.[26] Stainless steel skin

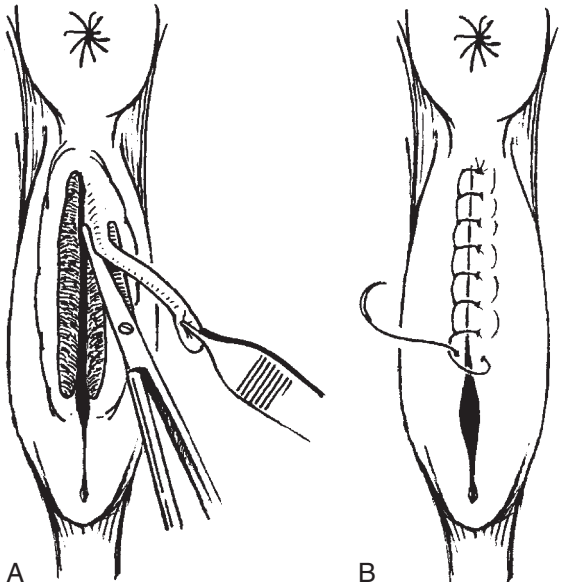

Figure 61-5. Caslick procedure. **A,** Removal of narrow strip of tissue from mucocutaneous junction. **B,** Closure using Ford interlocking pattern.

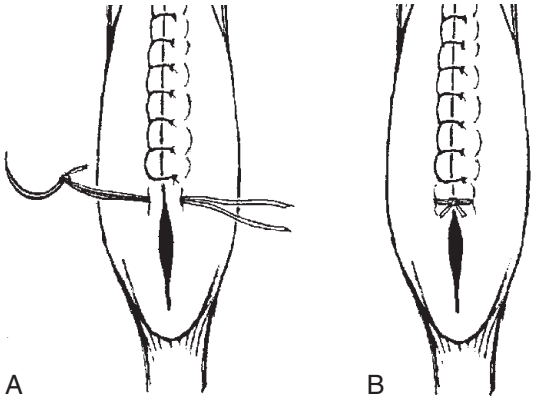

Figure 61-6. Breeding stitch placed ventral to a Caslick suture. **A,** Large suture material or umbilical tape is placed in a simple-interrupted pattern. **B,** The suture is tied and the ends are cut short so that they do not contact the stallion's penis during breeding.

staples can be used for the closure instead of sutures.[27] The sutures or staples should be removed 10 to 12 days later and the surgery site should be evaluated for fistula formation. Fistula formation can lead to aspiration of air and fecal material and negate the effects of the procedure.[28]

A mare requiring a Caslick procedure will likely need one for the rest of her broodmare life, so care should be taken to remove only as much tissue as necessary to achieve a complete seal between the labia.[25] Excessive removal of tissue will make subsequent Caslick procedures more difficult because of fibrosis and loss of tissue. Older mares that have had numerous Caslick procedures occasionally develop enough fibrous tissue or lose enough skin to make closure difficult as a result of tension. This may lead to dehiscence of a routine Caslick procedure. In such cases, a three-layer closure can be used. The inner mucosa of the two labia is apposed using 2-0 absorbable suture material in a continuous horizontal mattress pattern. The constrictor vulvae muscles are apposed using 2-0 absorbable suture material in a similar pattern. Finally the cutaneous layer is apposed using 2-0 or 0 absorbable or nonabsorbable suture material in a continuous pattern.

A breeding stitch is often placed at the ventral limits of the suture line to protect the Caslick procedure at subsequent breeding.[28] This consists of an interrupted suture of small-diameter, umbilical tape, or large-diameter suture material that is placed 2 to 3 cm lateral to both sides of the mucocutaneous border of the labia (Figure 61-6).[26] The suture must be positioned such that it does not interfere with the stallion's penis during intromission. Natural cover of the mare can be achieved with the suture in place by manually elevating the vulvar opening and guiding the stallion's penis. The breeding stitch is removed once the mare is confirmed to be pregnant.

Urovagina may result from excessive closure of the vulvar cleft.[29] Excessive closure is considered if a tube speculum cannot be readily passed.[28] Some mares have such poor perineal conformation that a Caslick procedure predisposes them to urovagina. In such cases, an alternative surgical procedure to correct the pneumovagina or a procedure to correct urovagina is necessary (see later).

In mares having undergone a Caslick procedure, an episiotomy should be performed 2 weeks before foaling to prevent damage to the vulva and perineum during the foaling process.

PERINEAL BODY RECONSTRUCTION

Reconstruction of the perineal body is useful when the vulvar and vestibular constrictor muscles have become ineffective.[25] Damage to the perineal body occurs from repeated stretching of these muscles in older multiparous mares, or from foaling trauma (second-degree rectovestibular injury). The goal of this surgery is restoration of the integrity of the dorsal aspect of the vestibule and vestibular sphincter function. Names of this surgical procedure include episioplasty, the Gadd technique, and perineal body reconstruction.[30] The surgery is performed with the mare sedated and restrained in stocks. Epidural anesthesia or local infiltration is used to desensitize the surgery site. The labia are retracted to the side using towel clamps or stay sutures. An incision is made along the mucocutaneous junction of the labia in a dorsoventral direction and is extended craniad along the dorsal commissure of the vestibule to the level of the vestibulovaginal sphincter. Dissection is continued submucosally from the dorsum and dorsolateral aspects of the vestibule. The triangular tissue flaps of mucosa are dissected so that they approximate the shape of the perineal body. Using caudal dorsal retraction on the stay sutures, the desired position for closure of the tissues is chosen (Figure 61-7). It is important that the procedure results in the vulva being oriented more vertically. Closure of the tissue layers is as follows. The vestibular mucosa is closed using 2-0 or 0 absorbable suture material in a horizontal mattress pattern, inverting the mucosa into the vestibule. The submucosal tissue is closed beginning at the cranial aspect of the vestibule using size 0 or 1 absorbable suture material in an interrupted pattern. The labial skin is apposed as for the Caslick procedure. Four weeks of sexual rest is recommended after surgery. Because the diameter of the vestibule has been decreased by this procedure, some mares require an episiotomy at the time of foaling.[31]

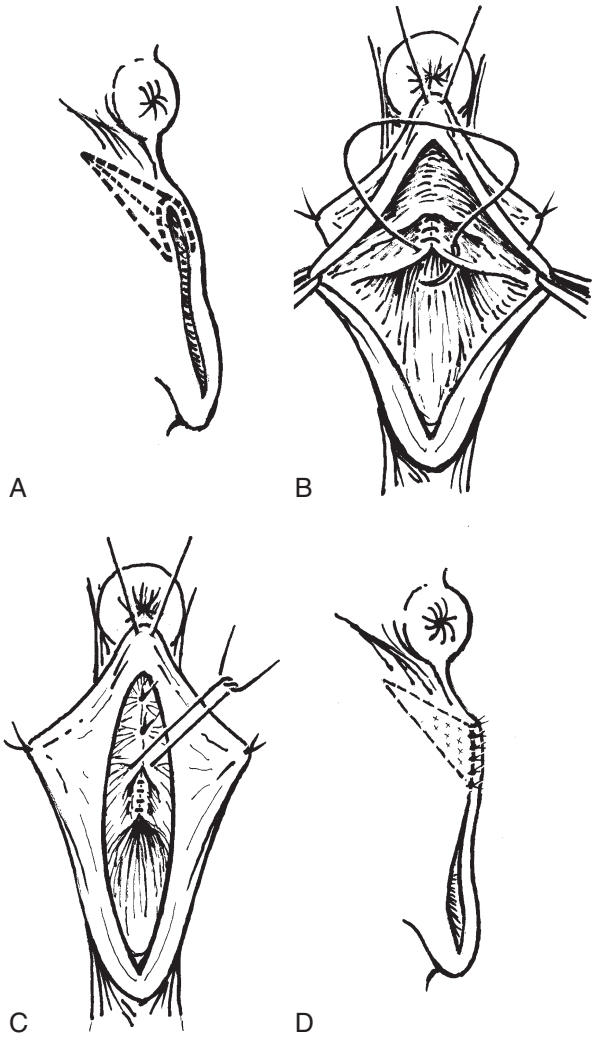

Figure 61-7. Perineal body reconstruction. **A,** The triangular piece of mucosa to be excised from the dorsal vestibule is outlined. **B,** The dorsal portion of the vulva and vestibule are retracted caudad and the vestibular mucosa is closed. **C,** Submucosal tissues are apposed with a series of interrupted absorbable sutures. **D,** The labial skin is closed with interrupted sutures and the vulva is now oriented in a more vertical position.

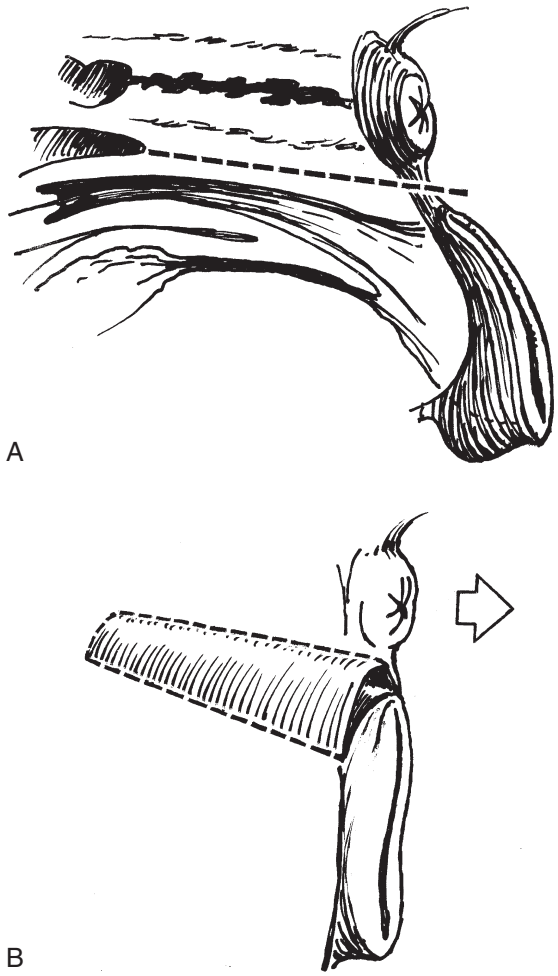

Figure 61-8. Perineal body transection. **A,** The *dotted line* shows the plane of dissection between the rectum and the caudal reproductive tract. **B,** Dissection is performed craniad using a combination of blunt and sharp separation of tissues until the vulva assumes a more vertical orientation.

PERINEAL BODY TRANSECTION

This technique was developed by Pouret and used to treat both pneumovagina and urovagina by the separation of muscular and ligamentous attachments between the rectum and the caudal reproductive tract.[32] The mare is sedated and restrained in stocks and epidural anesthesia or local infiltration of the perineal body is performed. A 4- to 6-cm horizontal skin incision is made midway between the ventral aspect of the anus and the dorsal commissure of the vulva. This incision is continued ventrad for 3 to 4 cm on both sides of the vulva. A combination of blunt and sharp dissection is used to extend the dissection in a cranial direction through the muscles of the perineal body. The dissection is continued craniad for 8 to 14 cm until the connections between the rectum and caudal reproductive tract have been severed.[30] Placing a hand in the vestibule to help guide the dissection is useful to prevent inadvertent entry into the rectum or peritoneal cavity. The dissection should be continued until the vulva has attained a normal vertical position without applying traction (Figure 61-8). No attempt is made to close the resulting dead space between the rectum and reproductive tract. Closure of the skin, either transversely or in a T-shaped configuration, has been suggested.[32,33] An alternative is to allow the wound to heal by second intention, which should occur within 3 weeks.[25] Natural cover should be delayed until the surgery site has healed properly (3 weeks). Mares may be bred by artificial insemination immediately.

Urovagina (Vesicovaginal Reflux of Urine, Urine Pooling)

Vesicovaginal reflux refers to the accumulation or pooling of urine in the vaginal fornix of the mare.[29] Urovagina and urine pooling are terms that have also been used to describe this condition. This abnormality is most often seen in thin, multiparous mares in which the cranial vagina slopes ventrally. Stretching and relaxation of the supporting ligaments of the urogenital tract leads to the excessive cranioventral sloping of the vagina. Mares often have a sunken appearance to their anus and dorsal vulva and frequently have had a Caslick procedure performed.[34] Excessive closure of the dorsal vulva during a Caslick procedure

may contribute to urine reflux by causing urine to splash back into the vagina.[35] In young fillies that are experiencing urovagina, an ectopic ureter may be present and must be ruled out.[29]

The chronic presence of urine in the cranial vagina leads to vaginitis, cervicitis, and endometritis. These inflammatory conditions can interfere with the ability of the mare to conceive and carry a foal to term.[36]

Urovagina is diagnosed by finding an accumulation of urine in the fornix of the vagina during vaginoscopy. It is best to perform a speculum examination of the vagina during estrus because in some mares urine only pools when the reproductive tract is under the influence of higher circulating levels of estrogen.[29] Examination of the mare on several occasions and finding urine in the cranial aspect of the vagina confirms the diagnosis. Differential diagnosis of urine pooling would be uterine infection with accumulation of exudate in the cranial vagina. Differentiation of the accumulated fluid using laboratory tests and cytology is often necessary. A cytologic evaluation of the fluid involves checking for bacteria, white blood cells, or calcium carbonate crystals. Creatinine and urea nitrogen testing can be performed on the fluid. Creatinine levels in accumulated urine will be at least two to three times serum creatinine levels.[29]

Surgical intervention is usually required for treatment of urine pooling.[35,37-39] Mares with poor body condition and the resulting abnormal perineal conformation can benefit from weight gain. Manual evacuation of urine from the cranial aspect of the vagina before breeding may improve conception rates, but this does not address the long-term negative side effects of urovagina. Definitive surgical treatment for vesicovaginal reflux involves modification of the external urethral orifice.

Caudal Relocation of the Transverse Fold

The Monin technique was first described in 1972 and involves caudal translocation of the transverse urethral fold.[39] This procedure has been found to be beneficial only if the reflux and abnormal perineal conformation is minimal.[29] It will not resolve severe vesicovaginal reflux and can make subsequent surgical procedures such as a urethral extension more difficult.

A Balfour retractor or stay sutures are used to access the transverse fold of the external urethral orifice (Figure 61-9). The center of the fold is grasped with Allis tissue forceps and retracted caudad toward the surgeon. The transverse fold is incised horizontally, splitting the fold into dorsal and ventral shelves. Thumb forceps are used to position the transverse fold along the ventrolateral walls of the vestibule. Mucosal incisions are then made in the walls of the vestibule at the proposed site of attachment. The transverse urethral fold is sutured to the vestibular floor in the retracted position, creating the extension. A one- or two-layer closure may be performed using 2-0 absorbable suture material.[39] This technique creates a urethral orifice that opens 2.5 to 5 cm more caudally after completion of the procedure. It is important to position the transverse fold so that it is not under excessive tension. This technique is simple to perform but it has the disadvantage of not being able to extend the urethral opening as far caudad as other urethral extension procedures (see later).[40]

Caudal Urethral Extension

Three urethral extension procedures have been described.[35,37,38] All of these procedures have the advantage of being able to extend the urethral opening as far caudad as necessary, unlike the Monin technique. A common pitfall of urethral extension procedures is fistula formation along the suture line.[40] In most instances, fistulas must be repaired. The surgeon should wait until tissue inflammation has subsided before attempting another repair (Figure 61-10).

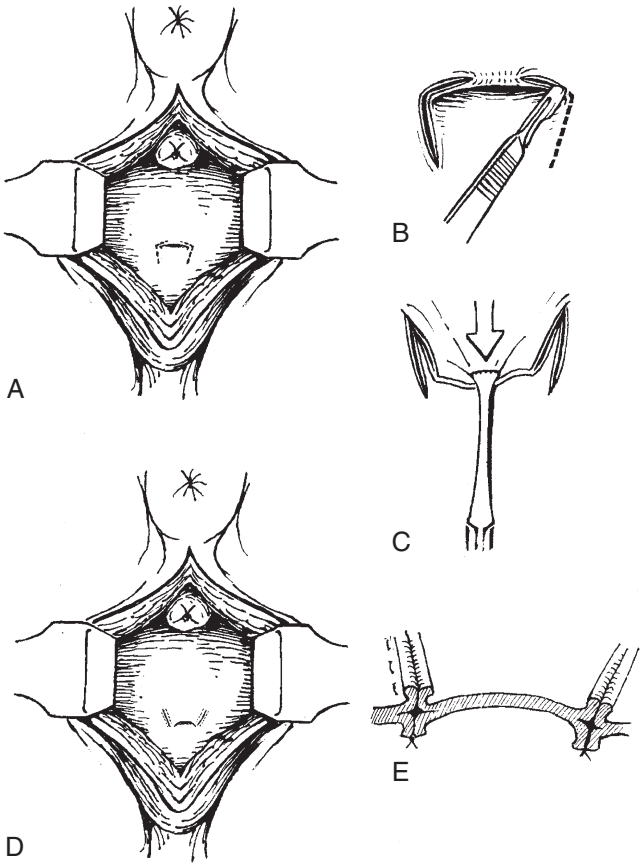

Figure 61-9. Monin urethroplasty. **A,** Incision of the transverse fold of the urethra. **B,** Mucosal incisions in walls of the vestibule. **C,** Caudal retraction of the transverse fold in preparation for suturing. **D,** Completed Monin urethroplasty. **E,** Two-layer closure using horizontal mattress pattern.

The mare should be sedated and restrained in stocks and should have epidural anesthesia to desensitize the perineum. Balfour retractors or stay sutures can be used to provide access to the surgical site. When self-retaining retractors are used, excessive lateral retraction should be avoided because this makes apposition of the vestibular mucosa more difficult. Insertion of a 30-French Foley catheter into the urethra will help ensure an adequate lumen diameter of the urethral extension and helps prevent urine contamination of the surgery site during the procedure.

BROWN TECHNIQUE

The original urethral extension procedure was described by Brown in 1978.[35] This technique involves creating tissue flaps beginning at the level of the transverse urethral fold and continuing to just inside the labia (Figure 61-11).[35] The free edge of the transverse urethral fold is incised horizontally with a scalpel, creating dorsal and ventral tissue flaps of equal thickness. It is important not to create holes in the flaps during the dissection.

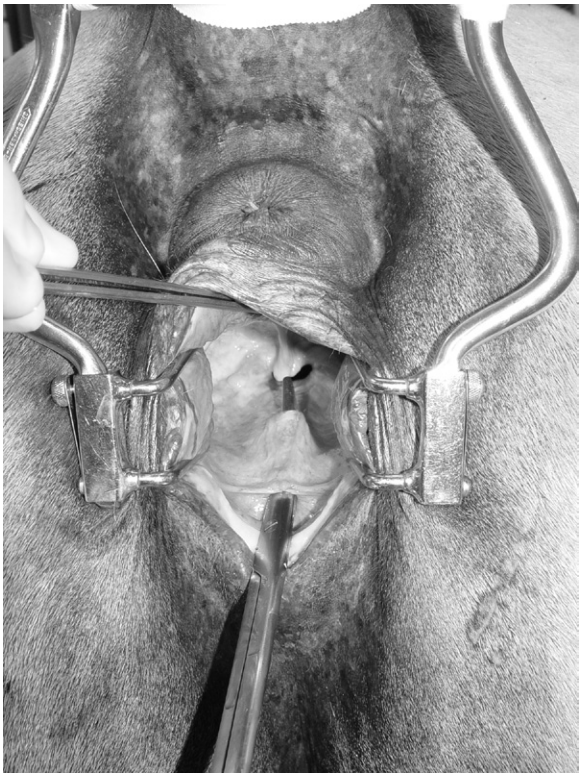

Figure 61-10. One complication of the caudal urethral extension procedures is fistula formation. Tips of scissors are sticking through the fistula.

The transverse incision is continued caudad along the vestibular wall to create a dorsal and ventral shelf of vestibular mucosa and submucosa. Dissection dorsad and ventrad allows the flaps to be apposed on the midline without any tension. It is critical that the dissection generates tissue flaps large enough to result in a urethral tunnel of adequate diameter. The ventral shelves of tissue from opposing sites are sutured using 2-0 absorbable material in continuous horizontal mattress pattern that inverts the mucosa of the ventral shelf into the new urethral lumen. The submucosa is closed using 2-0 absorbable suture material in a continuous pattern. The dorsal shelves are sutured using 2-0 absorbable suture material in a continuous horizontal mattress pattern that everts the mucosa into the vestibule. In the original description, this technique of resolving urine pooling was successful in 16 of 18 mares.[35] Following surgery, 11 mares were bred and 7 of them conceived.[35]

SHIRES TECHNIQUE

The Shires technique creates a urethral tunnel surrounded by loose mucosa from the floor of the vestibule around a Foley catheter placed in the bladder.[38] A 30-French Foley catheter is inserted through the urethral orifice into the bladder, and the balloon is inflated to secure the catheter (Figure 61-12). Interrupted horizontal mattress sutures using 2-0 or 0 absorbable suture material are placed in the ventral vestibular mucosa and tied so that a tunnel is formed over the catheter. The sutures must be positioned so that there is minimal tension on the mucosa over the catheter. The tunnel must begin cranial to the urethral orifice and extend caudad to a point approximately 2 to 3 cm cranial to the vulva. The everted ridges of mucosa dorsal to the horizontal mattress sutures are excised using scissors after

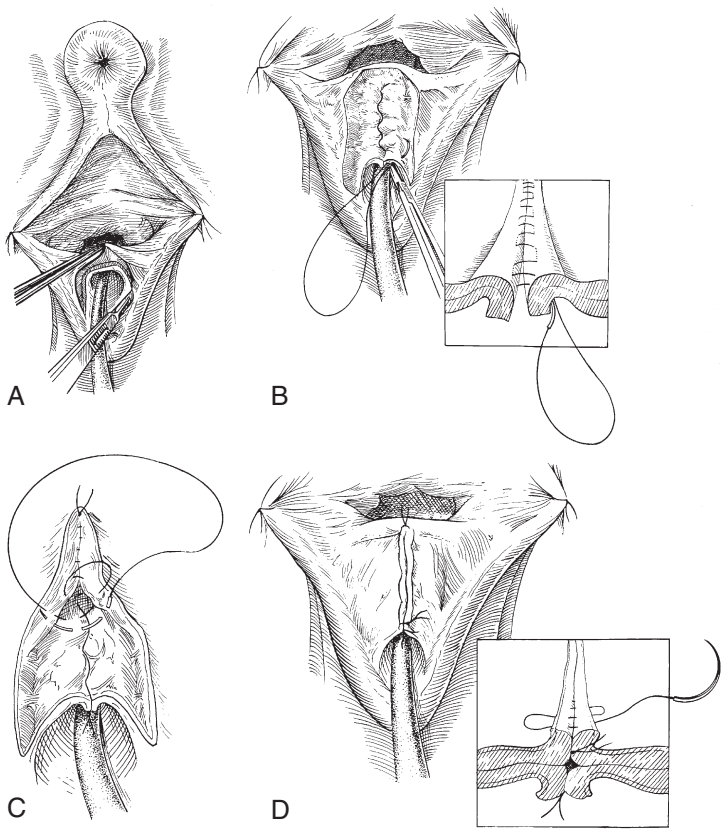

Figure 61-11. Brown technique of caudal urethral extension. **A,** The transverse fold is split into dorsal and ventral shelves, and the incisions are continued caudad along the ventrolateral walls of the vestibule. **B,** The first suture line inverts the vestibular mucosa into the new urethral lumen. **C,** The second suture line apposes the submucosal tissues. **D,** The third suture line everts the vestibular mucosa into the vestibule.

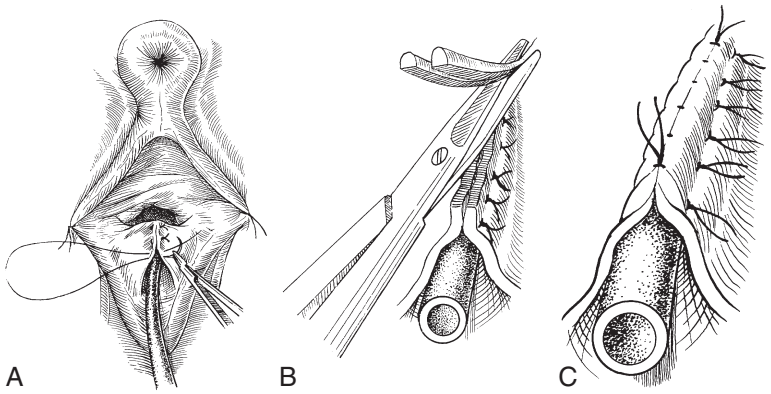

Figure 61-12. Shires technique of caudal urethral extension. **A,** Vestibular mucosa is apposed over a Foley catheter using horizontal mattress sutures. **B,** The everted mucosal ridge is excised. **C,** The exposed submucosa is apposed using a continuous suture pattern.

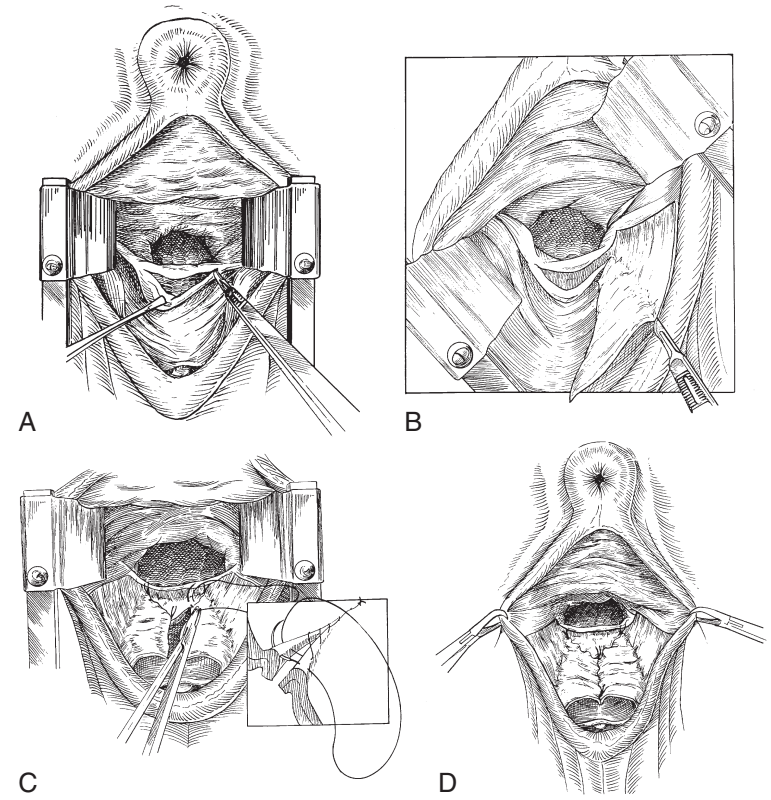

Figure 61-13. McKinnon technique of caudal urethral extension. **A,** Horizontal incision is made in the mucosa of the transverse fold. **B,** Dissection is continued caudad high along the vestibular wall to create large tissue flaps. **C,** Apposition of the tissue flaps is accomplished using a continuous horizontal mattress pattern. **D,** Completed urethral extension.

the completion of the tunnel. The cut edges of the mucosa are apposed using 2-0 suture absorbable suture material in a continuous pattern. This procedure is relatively easy to perform and was reported to be successful after a single surgery in 12 of 15 mares.[38]

MCKINNON TECHNIQUE

The McKinnon technique, described in 1988, creates a urethral tunnel that is larger and stronger than the one produced with the Brown and Shires techniques.[37] A horizontal incision is made in the mucosa of the transverse fold of the urethra 2 cm cranial to the caudal free edge (Figure 61-13). Incisions are made in the lateral walls of the vestibule approximately one half the distance from the floor of the vestibule. Dissection of the tissue flaps continues ventrad until the flaps can be apposed on the midline without tension. The tissue flaps are closed using a one-layer technique with 2-0 absorbable suture material in a continuous horizontal mattress pattern, inverting the mucosa into the lumen of the urethral tunnel. The initial dissection over the transverse urethral fold results in the cranial aspect of the closure assuming a Y pattern before the two suture lines meet on midline. Correction of urovagina using this technique was achieved in 32 of 34 mares, with fistula formation occurring in 5 of 34 mares.[37] Fistula formation is most common at the junction of the Y suture pattern. The exposed submucosal tissue heals by second intention. Mares may be bred 1 month postoperatively.

COMBINED BROWN AND MCKINNON TECHNIQUE

Combining the McKinnon and Brown techniques for urethral extension has been reported.[36] The initial dissection is similar to the Brown technique. The caudal free edge of the transverse

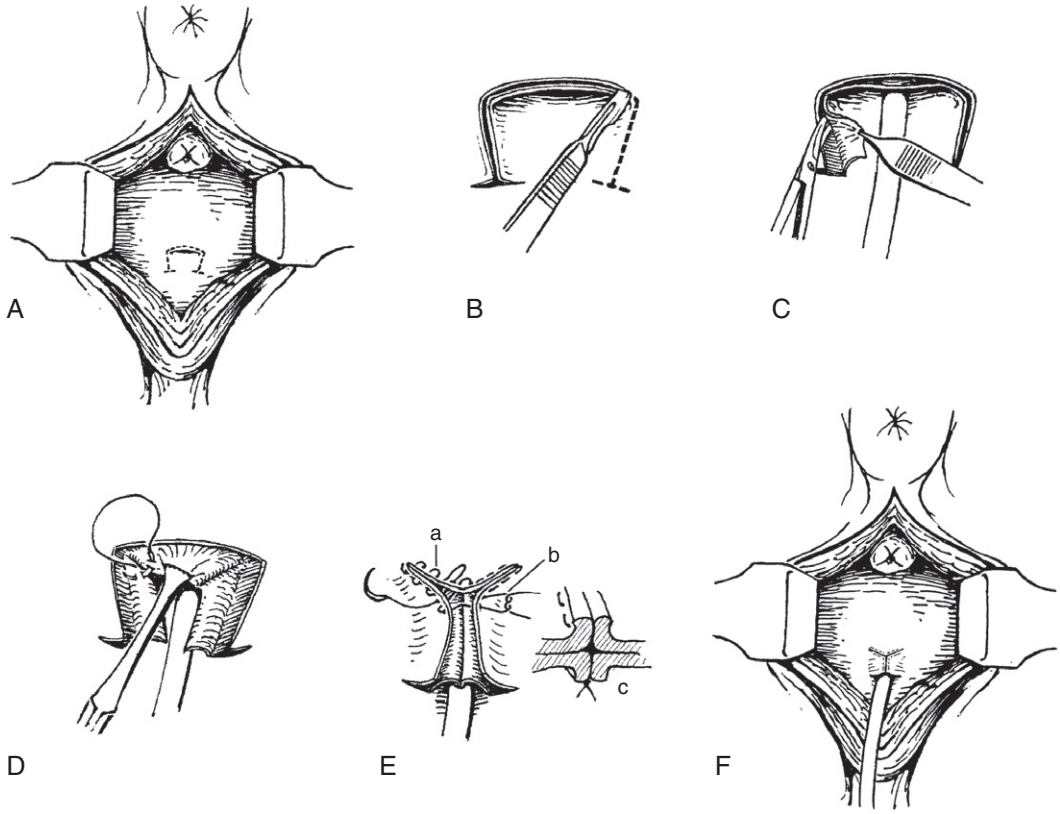

Figure 61-14. Combined Brown and McKinnon technique of caudal urethral extension. **A,** The scalpel is used to split the free edge of the transverse fold into dorsal and ventral shelves. **B** and **C,** Dorsal and ventral mucosal shelves are created by undermining the vestibular mucosa. The dissection should allow the shelves to meet on the midline without tension. **D,** The midpoint of the horizontal shelf is retracted caudad, and the ventral shelf is closed using a continuous horizontal mattress pattern to invert the tissue into the newly created urethral tunnel in a Y pattern. **E,** The dorsal shelves are sutured using a continuous horizontal mattress pattern to evert the tissue into the vestibule *(a)*. An additional continuous everting suture is placed around the three portions of the Y and tied at the end to provide further support to this very vulnerable location *(b)*. Close-up view of the new urethral shelf with the two everting patterns *(c)*. **F,** The completed urethral extension.

fold of the urethral orifice is incised into dorsal and ventral shelves of equal thickness (Figure 61-14). It is important that no holes are created in these tissue layers during dissection. The incisions are continued caudad along the ventrolateral walls of the vestibule to a point approximately 2 cm cranial to the labia. Submucosal dissection is performed to create dorsal and ventral vestibular tissue flaps that can be apposed on midline and create a urethral lumen of adequate diameter. A 30-French Foley catheter can be inserted into the bladder to help determine the location of the incisions and prevent urine contamination of the surgical field. The dissection of the vestibular walls does not reach as far dorsal as described for the McKinnon technique. Closure of the tissue flaps is as follows. The midpoint of the caudal free edge of the transverse urethral fold is grasped with Allis tissue forceps and retracted caudad. Suturing begins at the junction of the right ventral flap of the transverse fold and the right ventral flap of the vestibular wall. A continuous horizontal mattress pattern using 2-0 absorbable suture material is used. The mucosa of the ventral flaps should be inverted into the lumen of the urethral extension. During closure it is important to retract the transverse fold caudad. This suture pattern is continued caudad to the midpoint of the transverse fold, and the suture is tied. The right dorsal flap of the transverse fold and the right dorsal flap of the vestibular wall are sutured next, using a continuous horizontal mattress pattern of 2-0 absorbable suture material that everts the mucosa into the vestibule. The procedure is repeated for the left side. The remainder of the roof of the urethral tunnel is created by first suturing the right and left ventral vestibular tissue flaps, followed by suturing the dorsal flaps. The ventral flaps are apposed using 2-0 absorbable suture material in a continuous horizontal mattress pattern, which inverts the mucosa into the urethral lumen. The dorsal flaps are apposed using 2-0 absorbable suture material in a continuous horizontal mattress pattern, everting the mucosa into the vestibular lumen. The most difficult part of the repair is the junction of the Y of the three tissue layers—the transverse urethral fold and right and left vestibular tissue flaps. This location is most prone to dehiscence and fistula formation. A third layer using 2-0 absorbable sutures placed in a simple continuous pattern can be used starting just cranial to the Y junction and proceeding caudad. Maintaining a urinary catheter postoperatively for some time is based on the surgeon's preference (Figure 61-15).

Foaling Injuries

Numerous types of injuries are associated with parturition in the mare. Injuries caused by foaling and the resulting complications make up a large percentage of injuries to the perineum, rectum, and reproductive tract. Most injuries are obvious after

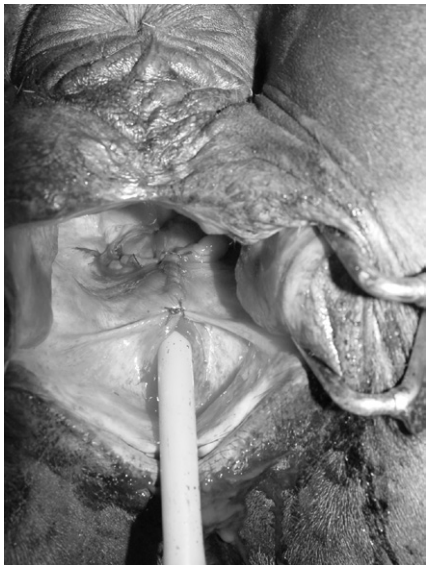

Figure. 61-15. Completed urethral extension described in Figure 61-15 with a urinary catheter in place.

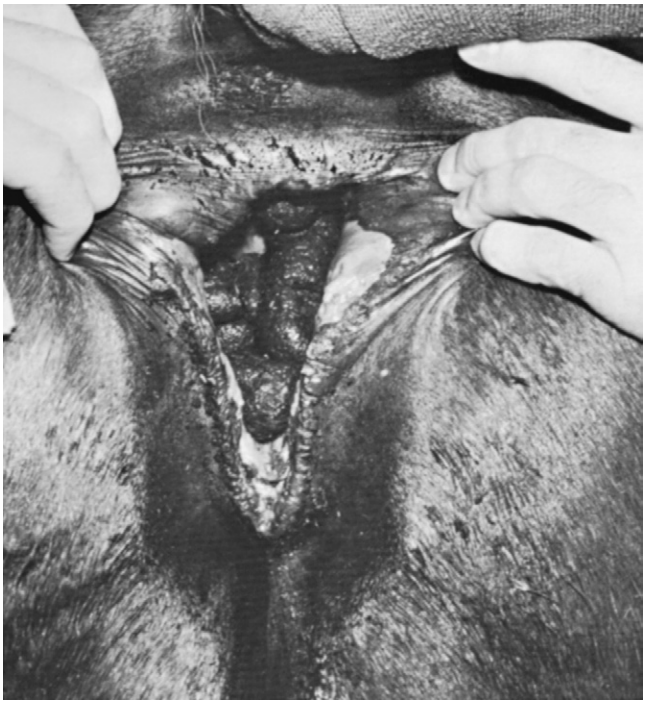

Figure 61-16. Third-degree perineal laceration with fecal contamination of the vestibule. The vagina and uterus will be contaminated if the vestibulovaginal sphincter is dysfunctional.

foaling, but some are not apparent until it is time to rebreed the mare.

Perineal Lacerations

Perineal lacerations typically occur during unassisted foaling, most commonly in primiparous mares. These types of injuries are most likely caused by the prominence of the vestibulovaginal sphincter and hymen remnants in mares foaling for the first time.[41] It is hypothesized that the forefoot of the foal catches on the dorsal transverse fold of the vestibulovaginal junction, and the mare's abdominal press forces the foal's foot through the roof of the vestibule and into the mare's rectum. A rectovestibular fistula results if a foaling attendant is present to replace the foal's foot back into the vestibule or if the foal retracts its foot. However, if the foot remains in the rectum during foaling, a third-degree perineal laceration can result (see later).

Perineal lacerations have been classified into first, second, and third degree, based on the extent and severity of the injury.[11,42] *First-degree perineal lacerations* involve the mucosa of the vestibule and the skin of the dorsal commissure of the vulva. *Second-degree perineal lacerations* involve vestibular mucosa and submucosa and continue into the muscles of the perineal body, including the constrictor vulvae muscle. These injuries do not involve the anal sphincter or rectum. Second-degree lacerations compromise the closure of the labia, predisposing the mare to pneumovagina. *Third degree perineal lacerations* are complete disruptions of the rectovestibular shelf, penetrating the rectum, perineal body, and anal sphincter. These injuries result in a common opening between the rectum and vestibule (Figure 61-16) and occur in primiparous mares that often have an excitable temperament. Fetal malposition, large fetal size, or aggressive assistance during delivery may play a role in the development of a third-degree perineal laceration.

First-degree injuries typically do not require surgical intervention. If needed, a Caslick procedure can be performed. Repair of second-degree injuries requires a Caslick procedure and reconstruction of the perineal body. The mare will develop a sunken perineum and be predisposed to pneumovagina and urine pooling if the perineal body is not reconstructed.[43] All third-degree perineal lacerations require surgical repair. The management is divided into two categories: immediate treatment and surgical repair. Repair of a third-degree perineal laceration in the acute stage should not be attempted. The tissue is very edematous and contaminated with feces, and some tissues may not be viable. Repair should be delayed at least 3 to 4 weeks or longer if possible to allow healing of the injured tissues (Figure 61-17). Initial therapy involves wound care and cleaning of the contaminated tissues. Third-degree perineal lacerations result in bacterial contamination of the vagina and uterus. Inflammatory uterine changes are reversible after surgical repair. A uterine biopsy is not needed because there are no studies correlating preoperative uterine biopsy grades with conception data after surgery.

Dietary changes may be necessary so that the mare has soft feces without excessive water content. Many methods can be used to soften the feces. Pasturing the mare on lush green grass, administering laxatives such as mineral oil or magnesium sulfate via a nasogastric tube, and feeding wet bran mashes are just a few examples. Dietary changes should be instituted well before surgery so the fecal consistency is soft by the date of surgery. If the mare has firm formed feces, the surgery should be postponed, because dehiscence of the suture repair is likely. Often the surgery is delayed until the foal is weaned so that it does not have to enter the hospital environment; but the timing of the surgery may be dictated by an urgency to repair the injury and rebreed the mare.

Surgery is performed with the mare sedated and restrained in stocks. The use of epidural anesthesia is necessary. Following

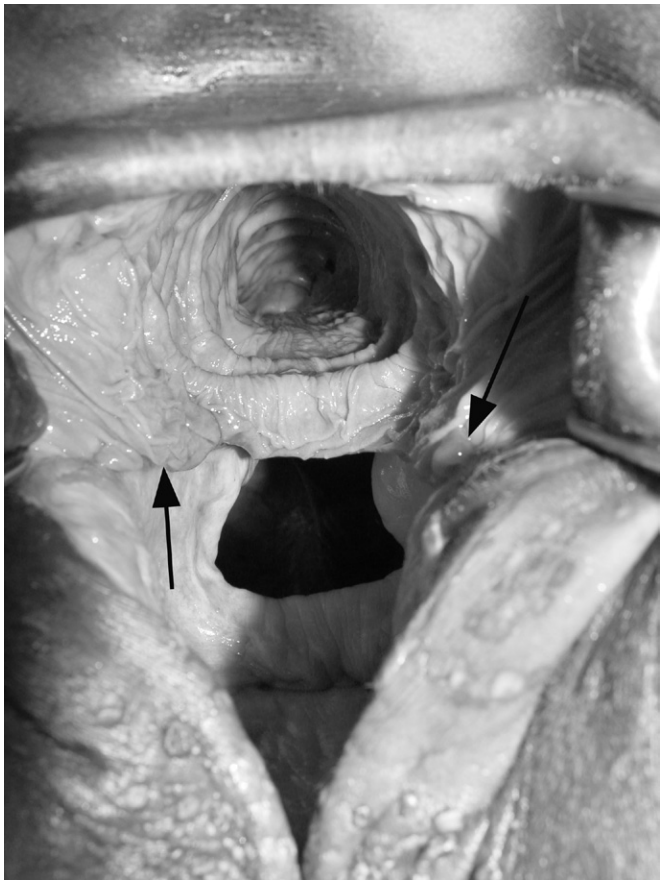

Figure 61-17. Appearance of a third-degree perineal laceration that is ready for repair. The rectal mucosa overhangs the intact shelf at the cranial extent of the laceration. *Arrows* point to the junction of the rectal mucosa and vestibular mucosa.

surgery the mare will need to maintain soft feces for at least 2 to 3 weeks.

TWO-STAGE REPAIR

The Aanes technique or a two-stage repair is designed to minimize obstipation, which can lead to failure of the repair.[42] In the first stage of the repair, the rectovestibular shelf is reconstructed without repair of the perineal body, and 3 to 4 weeks later the second stage or perineal body repair is performed.

Balfour retractors or stay sutures can be used to provide access to the surgical site. Initial dissection begins craniad in a frontal plane at the level of the rectovestibular shelf. Thumb forceps can be used to place tension on the rectovestibular shelf to facilitate dissection. A combination of sharp and blunt dissection is used to divide the tissue into rectal and vestibular shelves (Figure 61-18). The rectal shelf should comprise two thirds of the thickness and the vestibular shelf one third. The plane of dissection is continued craniad for 3 to 5 cm. The cranial dissection is important for relieving tension at the tissue edges. The incisions are continued laterad and caudad along the scar tissue junction of the rectal mucosa and vestibular mucosa. The dissection is continued laterad until the tissue shelves can be apposed on the midline without tension. Hemostasis using electrocautery or ligatures can be performed if necessary. Once sufficient dissection has been achieved, reconstruction of the tissue shelves can commence.

The first suture line apposes the vestibular shelves. A continuous horizontal mattress pattern of 1-0 or 2-0 absorbable suture material is used to invert the vestibular mucosa into the vestibule. This pattern should be interrupted when approximately one quarter to one half of the vestibular defect is closed. This allows easier access for placement of the second set of sutures. The next row of sutures is placed dorsal to the first in an interrupted fashion in the perirectal and perivestibular tissues. Then one or two absorbable sutures are placed in a four-bite purse-string fashion.[41] It is crucial that the rectal mucosa is not penetrated. The four-bite purse-string is placed by taking the first bite in the subrectal mucosal layer on the right; the second, in the subvestibular mucosal tissue on the right; the third, in the subvestibular tissue on the left; and the fourth, in the subrectal mucosal tissue on the left, followed by tying the knot. These two suture patterns are alternated until the level of the dorsal commissure of the vulva is reached. This technique does not repair the anal sphincter or perineal body. The second stage of the repair is performed 3 to 4 weeks later. It consists of removing the epithelium from the triangular surface of the perineal body and apposing these tissues on the midline (Figure 61-19), as described in the section on perineal body reconstruction. The function of the anal sphincter is gained by suturing the tissues of the perineal body. No attempt is made to isolate and suture the muscle fibers of the anal sphincter. A Caslick procedure is performed if necessary.

SINGLE-STAGE REPAIR

The initial dissection is the same in the single-stage and two-stage repair procedures. The Goetze modification of the single-stage repair uses a six-bite suture pattern (Figure 61-20). The suture is typically size 2-0 absorbable material and is placed in an interrupted pattern. The first suture is placed at the cranial edge of the dissected shelf and follows the following sequence. The first bite is deep in the left vestibular flap in a ventral to dorsal direction. The second bite is in the left rectal submucosa, taking care not to penetrate the rectal mucosa. The third bite is in the right rectal submucosa. The fourth bite is through the right vestibular flap in a dorsal to ventral direction. The fifth bite reenters the right vestibular shelf axial to the fourth bite in a ventral to dorsal direction. The sixth bite is in the left vestibular flap from dorsal to ventral and is positioned axial to the first bite. When the suture is tied the rectal edges should be apposed and the vestibular edges should be everted into the lumen of the vestibule. The sutures should be placed approximately 1.5 cm apart. Any sutures that are loose or placed too far apart should be replaced, because failure to do so will compromise the repair. Closure of the rectal mucosa has been proposed but is not necessary.[43] This repair is continued to a point approximately 4 to 6 cm cranial to the cutaneous perineum. At this point, the perineal body is repaired as previously described. A Caslick operation is performed if deemed necessary. The single-stage repair can be performed using the Aanes reconstruction technique as described for the two-stage repair. Another modification of the single-stage repair has been reported by Stickle; it involves a three-layer closure that includes a continuous horizontal mattress suture in the vestibular submucosa and rectal submucosa, inverting the mucosa into the respective lumen.[44] Simple-interrupted sutures are placed in the connective tissue shelf between the rectum and vestibule. All three sutures are started cranially and continue in an alternating fashion toward the vulva.

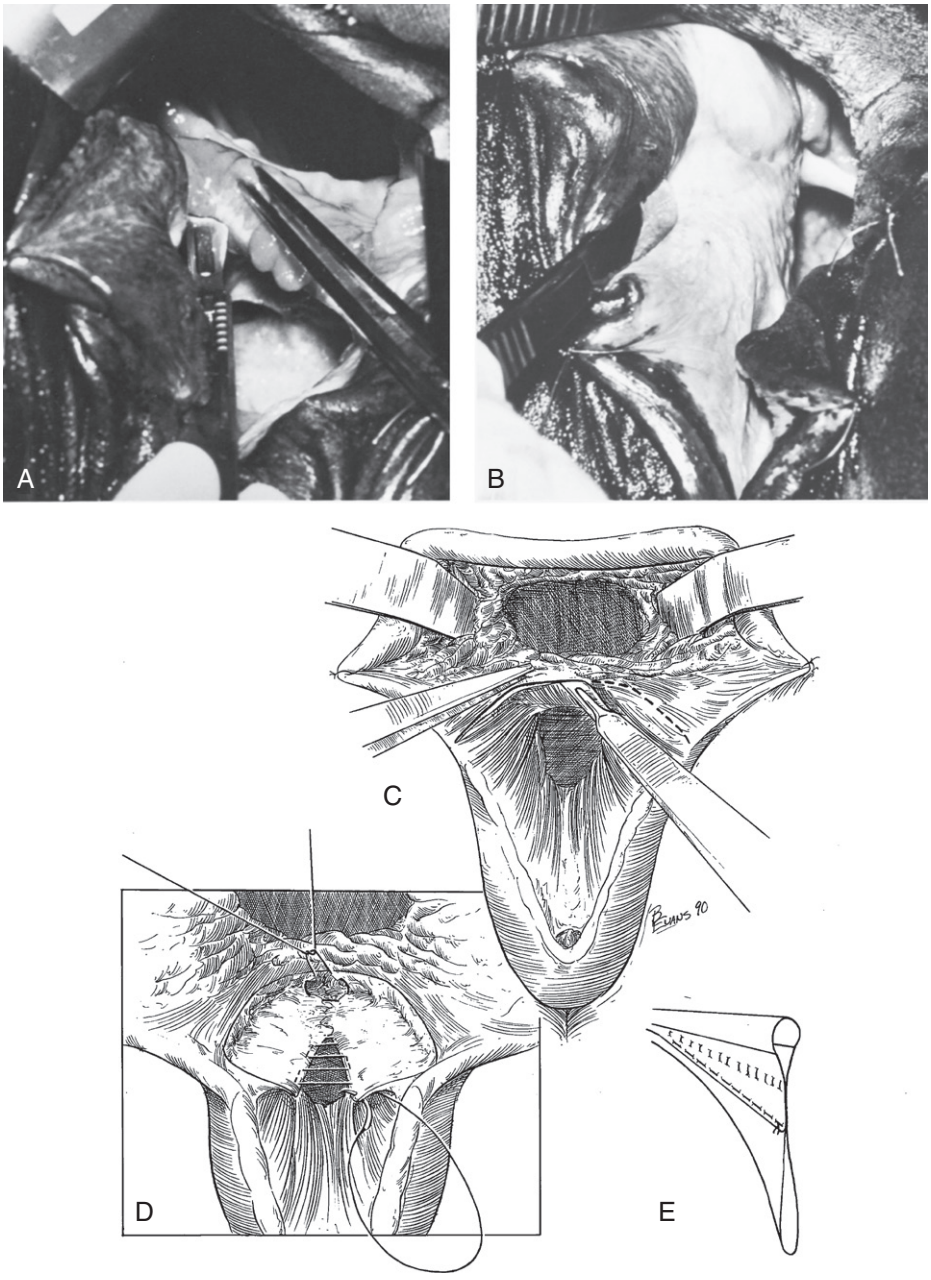

Figure 61-18. First stage of the two-stage repair of a third-degree perineal laceration. **A,** The cranial-most extent of the rectovestibular shelf is incised in a horizontal plane. **B,** The junction between the rectal mucosa and vestibular mucosa is delineated by a thin line of scar tissue. **C,** Vestibular and rectal tissue flaps are created by dissecting along the line of scar tissue. **D,** The vestibular mucosa is inverted into the vestibule using a continuous horizontal mattress pattern. The submucosal tissues are apposed using an interrupted pattern. **E,** Completed first-stage repair.

Rectovestibular Fistula

Rectovestibular fistulas result from a perineal laceration from the dorsum of the vestibule into the rectum, without disruption of the anal sphincter (Figure 61-21). They can form secondary to an unsuccessful repair of a third-degree rectovestibular laceration. Small fistulas sometimes close with conservative therapy, but larger ones require surgical repair.[40] Fistulas are most commonly 3 to 5 cm in diameter and located cranial to the perineal body.[43] Surgical approaches involve converting the fistula into a third degree perineal laceration either by a horizontal approach through the perineal body or a direct suturing technique.

When using the horizontal approach through the perineal body, a horizontal skin incision is made midway between the ventral aspect of the anus and the dorsal commissure of the vulva. A combination of blunt and sharp dissection is used to separate the perineal body. This plane of dissection is continued through the fistula for a distance of 3 cm (Figure 61-22, *A*). Stay sutures or Allis tissue forceps can be used to help retract the tissue during dissection. It is important not to penetrate the rectum or vestibule before reaching the fistula. The dissection should be such that the rectal shelf of tissue is thicker (two thirds of the thickness) than the vestibular shelf (one third of the thickness). The fistula in the rectal tissue is closed transversely using an interrupted Lembert pattern of 0 absorbable suture material (Figure 61-22, *B*). The fistula in the vestibular shelf is closed longitudinally in a continuous horizontal mattress pattern. This results in the suture lines that are at right angles to one another (Figure 61-22, *C* and *D*). The dead space created by the approach is closed using interrupted purse-string sutures. The skin is closed in continuous or interrupted pattern. An alternative is to allow the dead space and skin to heal by second intention.[11]

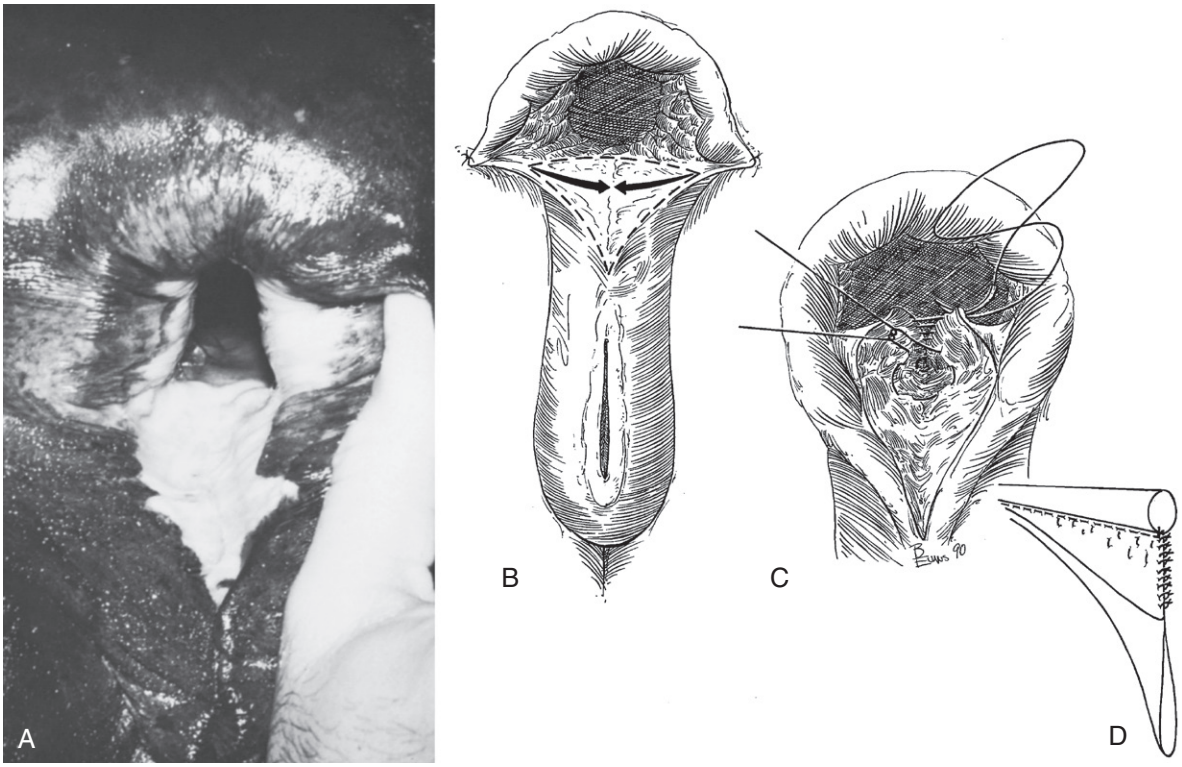

Figure 61-19. Second stage of the two-stage repair of a third-degree perineal laceration. **A,** The first-stage repair has healed and the mare is ready for the second stage of the repair. **B,** The area of epithelialized tissue that is to be excised is outlined. **C,** The submucosal tissues are apposed using interrupted purse-string sutures. This reconstructs the perineal body. **D,** Completed second-stage repair.

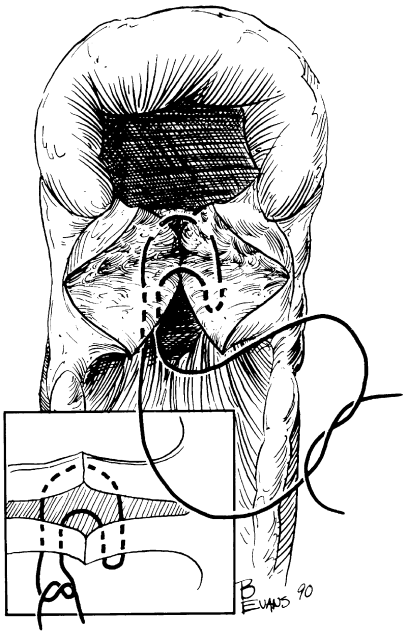

Figure 61-20. Single-stage repair of a third-degree perineal laceration. Drawings show the use of a six-bite suture pattern to appose tissues.

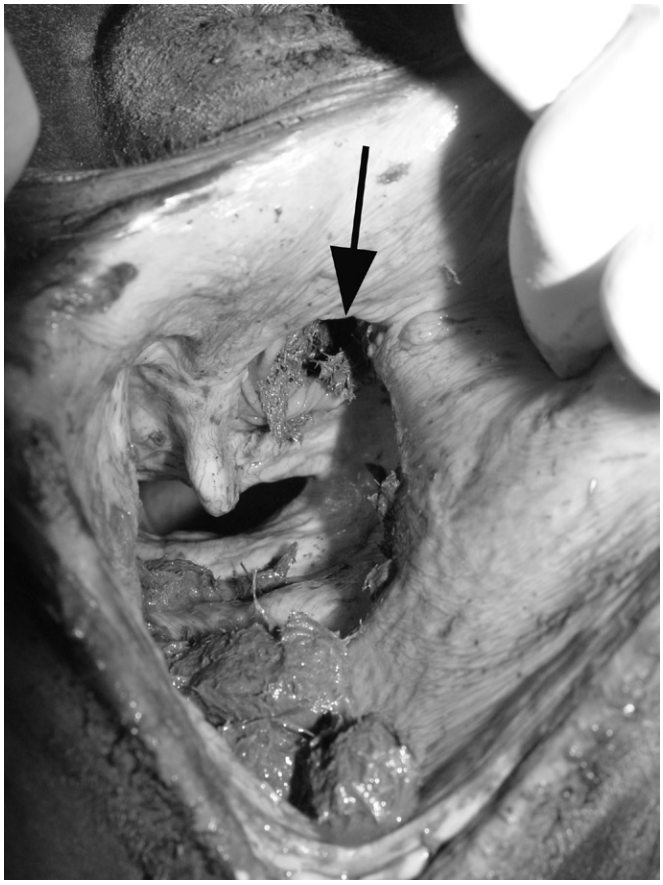

Figure 61-21. Rectovestibular fistula with manure contaminating the vestibule. *Arrow* points to the fistula.

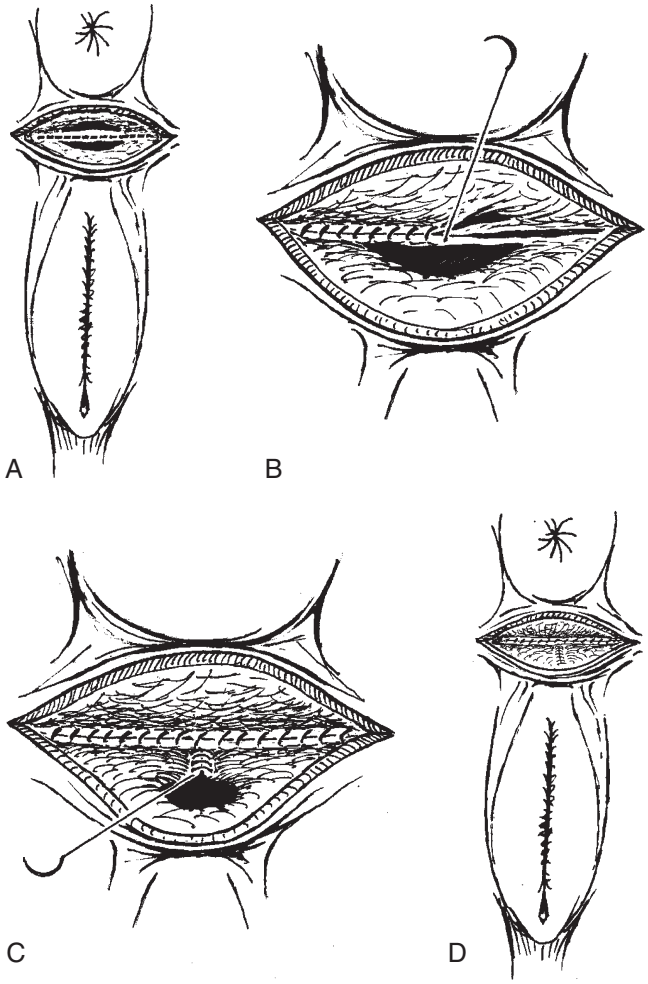

Figure 61-22. Drawing showing dissection and repair of a rectovestibular fistula using the horizontal approach. **A,** Completed dissection for repair of rectovestibular fistula. The fistula is divided into rectal and vestibular shelves. **B,** The rectal shelf is sutured transversely. **C,** The vestibular shelf is sutured longitudinally. **D,** The rectovestibular fistula is repaired. The dead space created by the approach can now be closed.

Direct suturing techniques have been described for successful repair of rectovestibular fistulas.[45,46] A mucosal pedicle flap technique for repair of rectovestibular fistulas in the standing horse has been described.[47] The edges of the fistula are débrided by full-thickness excision of 2 mm of the fistula margin. The fistula dimensions are assessed and a dorsally based U-shaped mucosal and submucosal pedicle flap is dissected from the vestibular wall. The flap is rotated 90 degrees so that the vestibular mucosa is continuous with the rectal mucosa covering the fistula. The flap is held in place by absorbable suture material placed in an interrupted pattern. Two of the three horses treated with this technique healed by first intention. The third horse required additional sutures to repair a partial dehiscence.[47]

Cervical Injuries

Injury to the cervix may lead to significant adverse effects on subsequent fertility. Cervical lacerations, adhesions, and incompetence are the most common disorders encountered.

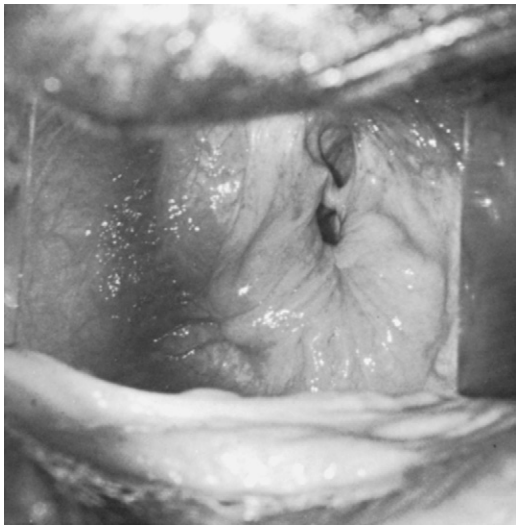

Figure 61-23. Appearance of laceration at the dorsal aspect of the cervix during a speculum examination.

Lacerations

Lacerations of the cervix usually occur as a result of excessive stretching during foaling. Cervical injury is more likely to develop during dystocia or in association with a fetotomy.[48] Cervical lacerations have been reported to occur during normal parturition or during the abortion of a relatively small fetus. Any injury that disrupts the normal function of the cervix may lead to infertility. The incidence of cervical lacerations is reported to be higher when parturition is induced.[42] This may be the result of the cervix not relaxing adequately before foaling.

The most common clinical signs associated with cervical lacerations are failure to conceive, endometritis, early fetal abortion, and persistent infertility. All mares that have undergone dystocia should have their cervix examined approximately 21 days postpartum (Figure 61-23).[49] Palpation of the cervix is crucial to making a diagnosis of a cervical laceration. Lacerations can be easily missed on visual examination alone. This is especially true if the mare is near estrus with a relaxed cervix. Diestrus is the optimal time to evaluate the cervix. During this stage of her reproductive cycle the cervix should be constricted and allow an accurate determination of cervical competency. The cervix is evaluated in the following manner. The perineum should be washed and prepared for examination. The examiner should wear a sterile glove or sleeve and apply an ample amount of lubricating jelly. The hand is inserted into the vagina of the mare and the cervix is identified at the most cranial aspect of the vaginal fornix. When using the right hand to examine the cervix, the examiner places the thumb in the lumen of the cervix and uses the index finger to palpate the cervix from the most dorsal aspect to the most ventral aspect of the right side of the cervix. To examine the left side of the cervix, the examiner inserts the index finger into the lumen and uses the thumb to palpate the cervix from the most dorsal to the most ventral aspect of the left side of the cervix. If a cervical laceration is present, the severity and extent should be determined. Not all cervical lacerations require surgical repair. It has been reported that surgery is unnecessary if 50% or less of the vaginal cervix

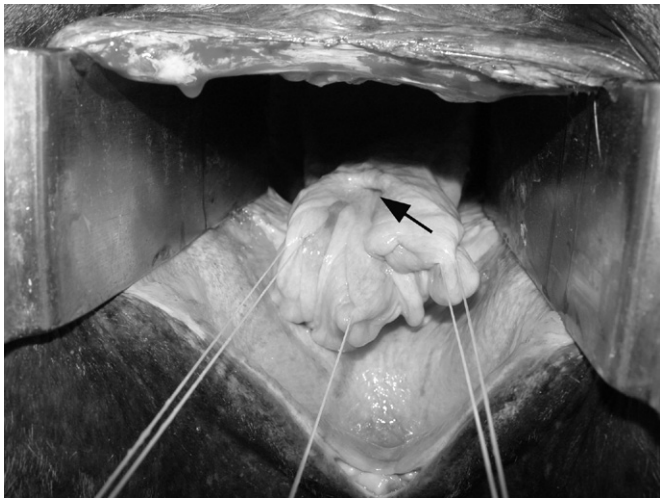

Figure 61-24. Photograph showing stay sutures placed to retract the cervix into the vestibule to facilitate repair. The sutures are placed to accentuate the defect in the cervix. *Arrow* points to the defect.

is involved.[50] However, the economic impact of fetal loss must be considered, and if it is possible to improve the chances that a mare will conceive and carry the foal to term, surgical intervention is warranted.

Surgery should be performed during diestrus and at least 3 weeks postpartum. Alternatively, the repair can be performed during estrus immediately after breeding. The mare is sedated and restrained in stocks, and epidural anesthesia is applied. A modified Finochietto retractor with long blades is very helpful in repairing cervical lacerations. Surgical interventions on the cervix should be performed in a caudally retracted position to bring it closer to the surgeon. Methods for retracting the cervix include Knowles cervical forceps and stay sutures (Figure 61-24). The Knowles cervical forceps are more traumatic than stay sutures and are not necessary. Three stay sutures using large-diameter suture material (No. 2) are placed in the external cervical os by hand or with the aid of long needle holders. The stay sutures must be positioned so they accentuate the cervical defect and do not interfere with the dissection and repair of the defect. The long ends of the stay sutures should be tagged with a hemostat and the needle removed. An assistant should apply gentle, steady caudal traction on the stay sutures so the surgeon has access to the cervix. The cervix can usually be retracted to the level of the vestibulovaginal junction. Allis tissue forceps are used to grasp the caudalmost scar tissue on each side of the cervical laceration. The scar tissue is excised using a scalpel blade or scissors. Following excision of the scar tissue, the three layers of cervical tissue should be evident. Repair of the defect is accomplished in three layers. The first layer, the inner cervical mucosa, is the most difficult to close and probably the most important. This layer is closed using 0 or 1 absorbable suture material in a continuous horizontal mattress pattern to invert the mucosa into the cervical lumen beginning at the most cranial aspect of the defect and working caudad. Following each bite, the surgeon should check whether the cervical lumen was penetrated and ensure that it is still patent. The second layer in the cervical muscle is apposed using 1 absorbable suture material placed in a simple-continuous pattern. The third layer, the outer cervical mucosa, is closed using 1 absorbable suture material placed in a simple-continuous pattern. In a report of 53 mares, a 75% pregnancy rate was achieved following surgical repair of cervical lacerations.[51] The mare should not be bred for at least 30 to 45 days following surgery. It is important to assess the uterus for signs of infection after surgery and before breeding. Uterine infection after surgery is not uncommon and must be addressed if present.

Adhesions

Transluminal adhesions interfere with the normal opening and closing of the cervix.[49] Less-severe adhesions may lead to infertility. More-severe adhesions can result in mucometra or pyometra.[16] Adhesions may form secondary to cervical trauma or cervical laceration repair. The prognosis depends on the severity of the adhesions. Transluminal adhesions can be broken down manually or surgically. Topical application of steroid-based ointment to the cervical lumen has been recommended as a means of preventing the adhesions from reforming.[16] Adhesions from the vaginal wall to the cervical opening can be relieved by sharp and blunt dissection. The surgeon must be careful not to perforate the vaginal wall and enter the peritoneal cavity when working in the cranial aspect of the vagina. Daily topical application of steroid-based ointment can be used as a means of preventing the adhesions from reforming.

Incompetence

Incompetence of the cervix can result from a tear that cannot be effectively repaired, muscle atony from repeated stretching, or a congenital anomaly.[16,52,53] Mares with an incompetent cervix may fail to conceive or experience early fetal abortion. Correction of these cervical problems has been associated with varied success.[16] The use of a buried retention suture or a nonabsorbable suture used in purse-string fashion around the base of the external opening has been described.[54] We recommended that the suture be placed during the first 48 hours following breeding and ovulation, and it must be removed before foaling. The success of this type of procedure is not known.

Clitoral Disorders

Abnormal conditions of the clitoris are rare. Squamous cell carcinoma has been reported to affect the clitoris.[55] Smegma can become impacted in the clitoral sinuses.[56] The sinuses of the clitoris can harbor *Taylorella equigenitalis,* the causative agent of contagious equine metritis.[57] Extirpation of the clitoral sinuses has been recommended as a treatment of affected mares.

Sinusectomy is completed as a standing procedure using local anesthesia. The glans clitoridis is not excised. The frenulum clitoridis, a fold of tissue overlying the clitoral sinuses, is retracted dorsad, and submucosal dissection is used to remove all clitoral sinus mucosa.[57] The resulting surgical wounds are left open to heal by second intention.

Congenital Anomalies

Congenital anomalies do not occur very often. Persistent hymen is the most frequently observed developmental anomaly of the mare's tubular genital system (Figure 61-25).[58] The hymen may be imperforate or may be present in varying

Figure 61-25. Appearance of a persistent hymen after it is exteriorized by an examiner.

degrees because of failure of the caudal sections of the Müllerian duct to fuse with the urogenital sinus. Failure of proper fusion of the Müllerian ducts may result in a dorsovental band in the cranial aspect of the vagina coursing across the external opening of the cervix.[59] Surgical removal easily solves both of these problems.

Developmental anomalies of the cranial vagina, cervix, and uterus are the result of partial or complete inhibition of Müllerian ducts. These anomalies are rare but occur more frequently in the cervix than in the vagina or uterus. Reports of congenital cervical anomalies include cervical aplasia, double cervix, and congenitally incompetent cervix.[52,53] Segmental aplasia of portions of the reproductive tract are rarely seen and may be associated with mucometra.[16] Ovariohysterectomy is recommended as a salvage procedure.

Incomplete separation of the urorectal septum leads to the formation of a rectovestibular fistula.[60] This is often seen in conjunction with atresia ani. In addition to urogenital abnormalities, other anomalies may be present. Atresia ani may be a heritable condition in the horse; therefore salvage of affected foals for breeding purposes is not advised.

Vaginal Varicosities

Vaginal varicosities are dilated veins that are most commonly located around the vestibulovaginal fold.[61] They can be solitary or form in clusters. The walls of the varicose veins are very thin and can rupture, causing recurrent or continued hemorrhage. The volume of blood loss is not life threatening but is concerning to owners and managers. The etiology of vaginal varicosities is unknown. Possible factors include elevated estrogen levels, changes in venous return, and perineal conformation during pregnancy.[62] Treatment options include ligation, cauterization, and laser photocoagulation. A vessel-sealing device such as the LigaSure (see Figures 13-16 and 13-17) can be used. Spontaneous postpartum regression of the varicosities has been reported.[63]

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CHAPTER 62

Uterus and Ovaries

Rolf M. Embertson

ANATOMY

The ovaries in the mare are approximately 70 to 80 mm (3 inches) long and 40 to 60 mm (2 inches) wide, but the size varies depending on the season and stage of the estrous cycle.[1] The ovaries are generally kidney shaped with a palpable indentation, the ovulation fossa, present along the ventrally directed free border. The cranial aspect of the broad ligament, the mesovarium, attaches to the dorsal border of the ovary. The mesovarium contains vessels, nerves, and smooth muscle fibers that extend to the ovary. The ovarian branch of the ovarian artery supplies blood to the ovary.[1]

The funnel-shaped infundibulum of the oviduct is loosely attached to the ventral aspect of the ovary around the ovulation fossa. The oviduct continues as the tortuous ampulla and then as the more straight and narrow isthmus, prior to entry into the tip of the uterine horn at the tubal papilla.

The uterine horns extend caudad to the uterine body. The majority of the uterine body is positioned within the peritoneal cavity, with the caudal aspect located retroperitoneally. The uterine body ends caudally as the thick-walled muscular cervix, which projects into the cranial vagina. The uterus is suspended in the caudal abdomen and pelvis by the broad ligament, also known as the mesometrium. The mesometrium contains

vessels, nerves, lymphatics, fat, connective tissue, and smooth muscle. The uterine blood supply, which courses through the mesometrium, originates from the uterine branch of the vaginal artery, the uterine artery, and the uterine branch of the ovarian artery.

DIAGNOSTIC PROCEDURES

A complete history and a thorough physical examination are essential to a comprehensive diagnostic workup. Palpation *per rectum* and *per vagina*, ultrasonography, and hysteroscopy provide additional information important to determining an accurate diagnosis and subsequent appropriate treatment. The results of blood work, hormone assays, culture and sensitivity, cytology, and histology are commonly used diagnostic aids. In addition, the laparoscope has become a frequently used tool for diagnosis of reproductive tract pathology.

PREPARATION FOR SURGERY

Preoperative Concerns

Decreasing the volume of ingesta within the intestinal tract generally facilitates most elective procedures involving the ovaries or uterus. Withholding food for 12 to 36 hours prior to laparotomy or laparoscopy is recommended. However, disruption of a horse's diet, feeding schedule, and environment may increase the risk of developing intestinal abnormalities (i.e., colic).

Perioperative antibiotics and anti-inflammatory drugs are usually indicated for surgery of the cranial reproductive tract. Many surgical interventions on the uterus have to be performed on an emergency basis, requiring well-trained personnel to assist in the management of these conditions.

Analgesia and Anesthesia

Standing surgery of the cranial reproductive tract requires profound sedation, visceral analgesia, and local anesthesia. Detomidine is the most commonly used drug for sedation and analgesia for these procedures. It is commonly used in combination with butorphanol and xylazine. For long procedures, sedation can be effectively maintained with a continuous-rate infusion (CRI) of detomidine. Detomidine also has been administered as an epidural injection for prolonged sedation, although it has not been shown to be more effective than the more easily administered CRI.[2] Epidural anesthesia may help facilitate some procedures.

Many of the cranial reproductive tract procedures require general anesthesia. There is concern about the effects of inhalation anesthetic agents on the fetus in mares with dystocia and those undergoing a cesarean section (C-section). Total IV anesthesia (guaifenesin, ketamine, and detomidine) has been recommended for these mares, at least until the foal is delivered.[3] However, most hospitals use inhalation anesthesia immediately following induction.

For mares encountering dystocia, where resolution requires general anesthesia, I have had consistently good results using xylazine, diazepam, and ketamine for induction and isoflurane in oxygen for maintenance of anesthesia. Anesthesia should be induced promptly and maintained at a light surgical plane, allowing the procedure to proceed as quickly as possible. Rapid delivery of the live foal combined with immediate neonatal care provides the best opportunity for the foal's survival from a difficult dystocia.

Surgery

General surgical concepts are as follows. For standing surgery, positioning the mare in stocks, tying the tail suspended upward, skin preparation, and surgical draping are essentially the same for a standing flank laparotomy or laparoscopy. The site(s) of local anesthetic infiltration for the incision(s) differ.

A modified grid approach is standard procedure used for a flank laparotomy in the mare. Generally, local anesthesia is placed as a 20-cm vertical line block between the last rib and the distal border of the tuber coxae, extending distad from just dorsal to the dorsal margin of the internal abdominal oblique muscle. A vertical incision is made through the skin and subcutaneous tissue, then extended deeper through the external abdominal oblique muscle and aponeurosis. The internal abdominal oblique and transverse abdominal muscles are bluntly separated parallel to the muscle fibers. The peritoneal lining is bluntly penetrated to gain access to the abdomen. This approach is also used by some to assist a laparoscopic ovariectomy.[4]

The ventral approach to the abdomen in the mare, under general anesthesia, is much more commonly used for laparotomy than the flank approach. In part because of staff familiarity with colic surgery, a ventral laparotomy to approach the ovaries or uterus can be performed quickly and efficiently in most hospitals. There are few indications for a ventral laparoscopic approach to the ovaries or uterus.

OVARIES

Neoplastic Conditions

Equine ovarian tumors are classified based on tissue type: surface germinal epithelium, sex cord-stromal tissue, and germ cell origin.[5] In addition, mesenchymal tissue tumors may develop in the ovary and others may gain access through metastases. The sex cord-stromal tumors, generally referred to as *granulosa cell tumors (GCTs)*, usually consist of granulosa cells but may contain granulosa and thecal cells.[5]

The GCT is the most common equine ovarian tumor, comprising approximately 85% of all equine reproductive tract tumors.[4-8] The average age of affected mares is about 11 years.[4,6,8] Affected mares may show anestrus, intermittent or continuous estrus (nymphomania), or stallion-like behavior.[5] Generally, the affected ovary is enlarged, sometimes quite massively, and the contralateral ovary is usually small and inactive. A GCT most commonly develops unilaterally but can occur bilaterally; it is usually benign but may exhibit metastases. Ultrasonographically, GCTs are usually multicystic structures, although the appearance can vary. In one study of mares with confirmed GCT, 54% had elevated testosterone levels and 87% had elevated inhibin levels.[5,9] Therefore, testing for both testosterone and inhibin may be most beneficial. Repeated palpation and ultrasonographic examination of the ovary may be useful to help rule out other causes of enlargement. Juvenile GCT has been reported in a few foals.[5,7,9] Foals with juvenile GCT, although quite rare, usual present with a hemoabdomen.[7,10] Ovariectomy is the treatment of choice to resolve this condition.

Other neoplastic conditions of the ovary are rare. They include teratoma, cystadenoma, adenocarcinoma, lymphosarcoma, melanoma, dysgerminoma, and arrhenoblastoma.[5]

Non-Neoplastic Conditions

An enlarged ovary may have non-neoplastic causes.[9,11] Occasionally, hematomas and cysts become quite large and adversely affect ovarian architecture. Abscessation can cause an enlarged, painful ovary. Ovariectomy is usually needed to resolve these conditions.

Abnormal behavior is occasionally encountered in show mares during estrus. If hormone therapy is unsuccessful, bilateral ovariectomy can be used to correct this behavior.

Ovariectomy

Colpotomy

Ovariectomy via a colpotomy has been used primarily for bilateral ovariectomy, either to improve a mare's behavior or for research applications. The procedure has been used for years and has changed little, except for improvements in sedatives and analgesics.[11-13]

The mare is sedated and placed in stocks, and the tail is wrapped. Epidural anesthesia is generally recommended. The rectum is evacuated, and the bladder is emptied if it is distended. The tail is tied upward and the perineal region is prepared for surgery. A pointed bistoury or guarded scalpel is used to make a small incision through the cranial vaginal wall, 4 to 5 cm caudolateral to the cervix. The incision is made at the 2 or 4 o'clock position if the surgeon is right handed and at the 8 or 10 o'clock position if the surgeon is left handed. These sites avoid inadvertent penetration of the bladder, rectum, and uterine branch of the urogenital artery laterally. The peritoneum is penetrated with a thrust from closed, blunt-pointed scissors. This small incision is enlarged digitally until the entire hand can pass into the peritoneal cavity. Lidocaine-soaked gauze sponges, tethered with a long strand of umbilical tape, are held around the ovarian pedicle for 1 minute. The chain loop of the écraseur is placed around an ovary, with care taken to exclude other tissues, and is slowly (over 3 to 4 minutes) tightened until the ovary falls into the surgeon's hand. The ovary is removed from the abdomen and the procedure is repeated to remove the contralateral ovary.

The vaginal incision is allowed to heal as an open wound and the mare is kept standing for 2 to 3 days to prevent the rare case of eventration. Loose apposition of the wound edges with large absorbable sutures may lessen the anxiety level of the surgeon postoperatively. Broad-spectrum antibiotics and anti-inflammatory drugs are administered for 5 days postoperatively, and the mare is walked daily for 2 weeks before resuming previous activities.

Possible complications include excessive (possibly fatal) bleeding from the ovarian pedicle, eventration through the vaginal surgery site, abscessation or hematoma formation at the incision sites, and adhesions of abdominal contents to the incision sites.[11-13]

Laparotomy

Ovariectomy via a laparotomy allows the ovary to be exteriorized under visual control, which facilitates direct hemostasis. The ovarian pedicle can be short enough that exteriorizing the ovary for resection may be difficult. A flank approach can be used in the standing mare if there is concern about general anesthesia and the ovary is less than 10 cm in diameter. A flank approach may also be used with the mare under general anesthesia. A modified grid approach is used to access the abdomen. The most common complication that occurs at the flank surgery site is development of a postoperative seroma or formation of an abscess.

A ventral approach to the abdomen can be via a midline, paramedian, or diagonal paramedian incision (see Chapter 34). The ventral midline incision may be most ideal for a very large ovarian tumor with a stretched ovarian pedicle. The diagonal paramedian laparotomy approach is the most useful approach for the majority of normal or enlarged ovaries.[11,13-15] Because the incision is positioned directly over the ovary, less tension is applied to the ovarian pedicle during vessel ligation than with the other approaches.

The mare is anesthetized and placed in dorsal recumbency, and the surgery site is prepared for aseptic surgery. A 12- to 15-cm (5- to 6-inches) long incision is made starting approximately 5 cm cranial to the mammary gland and nearly bisecting the angle formed by the ventral midline and the inguinal depression. The site of the incision can be adjusted to avoid large subcutaneous vessels. The incision is continued through the external rectus sheath, and the rectus abdominis muscle fibers are bluntly separated. Ligation and transection of 1 to 2 large neurovascular bundles found coursing perpendicular to the muscle fibers may be required. The internal rectus sheath is opened carefully with scissors. A hand is introduced into the peritoneal cavity, and the ovary is identified and pulled up to the incision. Stay sutures (No. 2 polyglactin 910) are placed through the ovary in a cruciate pattern. These sutures are very helpful for manipulating and removing the ovary. Distended follicles are aspirated to reduce the size of the ovary and to facilitate passage of the enlarged ovary through the incision. Ligatures (No. 1 or 2 absorbable suture) are placed around the cranial and caudal margins of the ovarian pedicle and used as additional stay sutures. A 90-mm thoracoabdominal stapling device (TA 90) is placed across the ovarian pedicle and discharged. The ovarian pedicle is transected between the rows of staples and the ovary. The ovary is then removed from the surgery site (Figure 62-1).

After removal of the stapling device, tension is relieved on the end of the ovarian pedicle while continuing to hold the stay sutures, and the transection site is checked for bleeding. Occassionally, minor bleeding is encountered, requiring additional ligatures. The ovarian pedicle is placed back into the abdomen and the abdomen is closed. The rectus abdominis muscle fibers are reapposed with No. 2 absorbable suture in an interrupted cruciate pattern. The external rectus fascia is closed with No. 2 absorbable suture in a continuous pattern, the subcutaneous tissue with No. 1 absorbable suture in a continuous pattern, and the skin with No. 0 absorbable suture in a continuous pattern. An experienced surgeon is able to perform this procedure bilaterally, if needed, relatively swiftly.[16]

Most mares are hospitalized overnight and discharged the following day. Postoperatively, mares should be hand-walked for 1 week, followed by 2 weeks of small-paddock turn-out prior to returning to routine care. Postoperative complications are rarely encountered but include hemorrhage from the ovarian pedicle, traumatic anesthetic recovery, postoperative abdominal pain, incisional infection, and adhesion of the uterine horn to the surgery site.

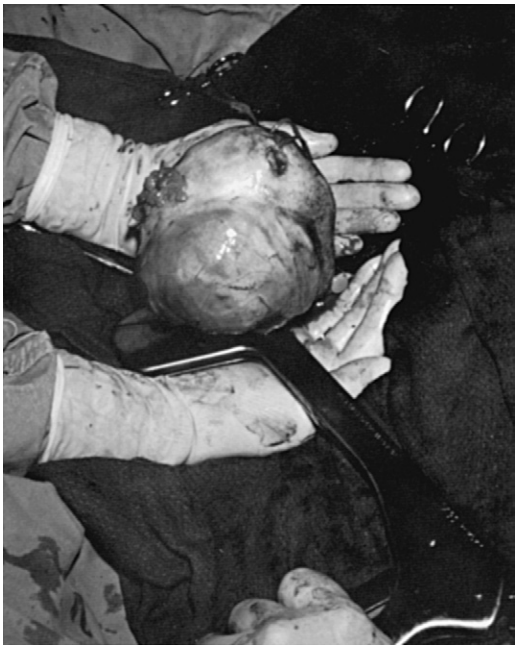

Figure 62-1. Ovariectomy, using a commercial stapling unit for hemostasis prior to ovary resection.

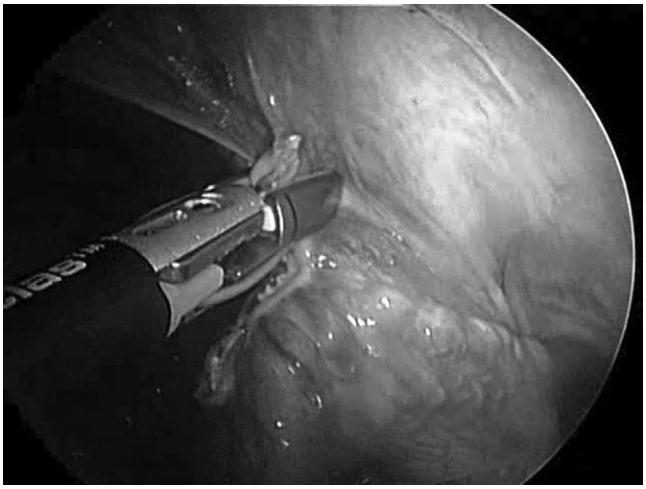

Figure 62-2. Laparoscopic ovariectomy, using the LigaSure Atlas, for hemostasis and transection of the ovarian pedicle.

Laparoscopy

Laparoscopic ovariectomy is generally performed as a standing procedure using an approach made through the flank.[4,6,17,18] The primary advantages of the flank laparoscopy compared to the diagonal paramedian laparotomy for ovariectomy are the enhanced visualization of the surgical site with minimal tension on the ovarian pedicle, the ability to quickly and more confidently address potential hemorrhage, and avoidance of general anesthesia. Complications, although different, can occur with both techniques, with surgical site drainage being more common following the flank approach. Client costs are a function of how equipment, materials, personnel, and facilities are charged. Currently the surgery time may be less with the laparotomy, and the postoperative convalescent period is not dissimilar.

Equipment needed for laparoscopic ovariectomy includes a videoendoscope camera, monitor, light source and cable, insufflator and tubing (not always used), a 0-degree or 30-degree 57-cm (22-inches) rigid endoscope, at least three 10-mm (4-inches) diameter 15- to 20-cm (6- to 8-inches) long cannulas with trocars, laparoscopic forceps, scissors, injection needle, and ligation instrumentation.[17] Ligation of the ovarian pedicle can be done with ligating loop sutures, a stapling instrument, or a bipolar vessel-sealing device. For more information on laparoscopy, please review Chapter 13.

The exact sites of the incisions for the laparoscopic portals vary between surgeons. The hand-assisted laparoscopic ovariectomy technique commences with a modified grid flank approach to the abdomen.[4] The cannulas for the endoscope and instruments then are placed carefully to avoid trauma to bowel or the spleen. The ovary is visualized and the mesovarium is desensitized with lidocaine or mepivicaine. Placing local anesthetic into the mesovarium, rather than the ovary, has been shown to be more effective in reducing pain responses during surgery.[19] The bipolar vessel-sealing device (LigaSure Atlas) (see Chapter 13) has been used effectively in our hospital, and others, for hemostasis and transection of the mesovarium (Figure 62-2).[20] Semm claw laparoscopic forceps provide a relatively secure grasp of the ovary for manipulation and extraction. However, No. 2 polygalactin 910 sutures placed in a cruciate pattern through the ovary provide a more confident purchase of the ovary than the forceps. If the hand-assisted approach was not performed, one of the portals needs to be lengthened to remove an enlarged ovary.

To avoid creating a large flank incision to extract the ovary, an enlarged ovary can be reduced in size prior to removal. Aspirating fluid from some of the enlarged follicles usually results in significant size reduction. Additionally, the ovary can be reduced into smaller pieces by direct sharp dissection or with the use of a morcellator while it is held against the body wall incision within a specimen retrieval bag.[4,6,21] Ovaries up to 30 to 40 cm in diameter have been removed laparoscopically using these techniques. Closure consists of apposing the muscles, followed by subcutaneous tissue and fascia, and finally the skin.

Most mares return to the farm the next day with the same discharge instructions as for diagonal paramedian laparotomy ovariectomy. Complications following laparoscopic surgery through the flank vary.[4,6,17,18] Complications related to sedation and analgesia include inadvertent recumbency and patient noncompliance. Cannula insertion complications include retroperitoneal insufflation, splenic or bowel puncture, and hemorrhage from laceration of the circumflex iliac artery. Loss of the ovary within the abdomen is undesirable. The most common postoperative complications are subcutaneous emphysema, mild colic, and incisional drainage.

UTERUS

Uterine Cysts

Uterine cysts are most commonly found in older mares.[22,23] They can be found on rectal exam but are usually diagnosed ultrasonographically. The presence of multiple uterine cysts may have an adverse affect on fertility. If the cysts are suspected to be the cause of infertility, they should be removed. Methods of cyst removal have included mechanical curettage, cyst rupture manually or with endometrial biopsy forceps, uterine lavage with a hypertonic saline or magnesium sulfate solution, electrocoagulation of the cyst, and laser ablation.[22,23] In our practice,

laser ablation has been the most successful method of cyst resolution and is currently the most accepted technique.

The mare is sedated and placed in stocks, and the perineal region is prepared for hysteroscopy. A 1-m videoendoscope that has been cold-sterilized and rinsed with sterile water is passed through the cervix into the uterus. The uterus is distended with air to allow visualization of the uterine body and both horns. The diode laser fiber is passed through the biopsy channel of the endoscope into the uterus. The uterine cysts are first punctured and drained with the diode laser, set on a power setting of 15 W. Following release of the fluid, lasing is continued until the remaining cystic tissue and its base are shriveled and charred. Smoke and fluid often need to be evacuated from the uterus a few times during the procedure. Immediately after the procedure, the uterus is lavaged or infused with antibiotics. The uterus should be lavaged daily for 2 or 3 days postoperatively to remove the retained cyst fluid and debris created by photoablation. An ultrasonographic examination of the uterus should be performed approximately 30 days postoperatively and prior to breeding. One report of the use of the Nd:YAG laser to ablate endometrial cysts in 55 barren mares demonstrated improved fertility.[23] In my experience, ablation of the lining and base of the cyst should prevent recurrence of the cyst. However, additional cysts may form in other locations.

Pendulous Uterus

Delayed uterine clearance has been recognized as a contributing factor to infertility in the mare.[24] The conformation of the reproductive tract commonly found in older mares, where the cranial aspect is positioned ventral to the caudal aspect, plays a role in delayed uterine clearance. In a mare with poor conformation, a recent report showed that elevating the uterus and fixing it in a more horizontal position would improve her prognosis for conception and carrying a fetus to term.[25]

The surgical technique to elevate the uterus involves creating three laparoscopic portals in the left flank. The mesometrium is infiltrated with local anesthetic solution. The left side of the uterine body and horn are sutured to the mesometrium. The suture line starts at the caudal aspect of the uterus where the mesometrium attaches to the uterus. A suture is passed through the seromuscular layers of the uterus at that level, through the mesometrium approximately 3 cm dorsal to its uterine attachment, and then tied. A continuous suture line is continued in this fashion to near the cranial aspect of the uterine horn. This suture line essentially imbricates the mesometrium. Following placement of this suture, the exact same procedure is performed to elevate the right side of the uterus from the right flank. The original report describes placing the suture line using laparoscopic needle holders.[25] The Endostitch 10-mm Suturing Device has also been used successfully for the uteropexy procedure.[26]

In one report, five mares had been barren for a mean of 3.8 years prior to surgery.[25] Three of these mares became pregnant postoperatively, with one mare having foaled by the time of publication submission. Although the procedure is initially promising, it is too early to determine an accurate success rate and any potential complications that may be encountered.

Uterine Neoplasia and Chronic Pyometra

Uterine neoplasia and chronic pyometra are uncommon conditions.[27-30] Chronic pyometra is generally the result of an obstruction of complete uterine drainage because of severe cervical trauma. If medical therapy is unsuccessful, an ovariohysterectomy is indicated. Drainage of the uterine contents prior to surgery is advisable.

The most commonly encountered uterine tumors are leiomyoma and leiomyosarcoma. Rhabdomyosarcoma, adenocarcinoma, and other types have also been reported.[27,30] Most uterine tumors can be resolved by tumor removal or partial hysterectomy. However, infrequently ovariohysterectomy is indicated.

Total ovariohysterectomy is best approached through a caudal ventral midline incision with the mare under general anesthesia and in dorsal recumbency. The ovarian pedicles are ligated, and dissection continues through the broad ligament, ligating large vessels in the process. The body of the uterus is transected as far caudad as possible, with care taken to avoid contaminating the peritoneal cavity. The uterine stump is closed with a double-inverting suture pattern. The abdomen is closed in routine fashion.[13,27] A recent report describes the laparoscopic dissection and hemostasis of ovarian and uterine structures followed by inversion of the uterus through the cervix and into the vagina, where the uterus was then resected.[30]

Solitary masses, even if quite large, can be successfully removed from the uterine wall with the mare remaining fertile.[31] The affected part of the uterus is approached through a caudal ventral midline or paramedian incision followed by exteriorization and dissection of the mass from the uterine wall. Partial ovariohysterectomy has been used to remove a focal uterine tumor and ovarian masses with adhesions to the uterine horn.[28,32-35] Surgical approaches have included a caudal ventral midline and paramedian laparotomy and a standing flank laparoscopy. Live foals have been produced by mares after partial ovariohysterectomy where various amounts of the uterine horn were removed. It is difficult to determine exactly how much uterine horn is needed to maintain a pregnancy, and this likely varies between horses. Based on a review of several papers, it appears that most mares can maintain a pregnancy with up to 50% of a uterine horn removed, but fertility decreases proportionally to the amount of horn removed beyond that.[36,37]

Uterine Torsion

Mares with uterine torsion generally present with signs of colic. Affected mares usually exhibit mild to moderate, intermittent abdominal pain. The diagnosis is usually made by rectal examination. In most cases, a taut broad ligament is palpable, coursing dorsal to the caudal aspect of the uterus in the direction of the torsion. An ultrasonographic examination may provide useful information about fetal viability, status of the uterus, and other abdominal abnormalities.

Uterine torsion can affect mares of all ages, usually during the last 2 months of gestation, although this varies.[38-40] In the most recent study of 63 mares, 90% were younger than 16 years, 60% had the torsion directed clockwise, and 80% had torsions 180 degrees or less.[40] Overall mare survival was 84%, with a survival rate of 97% if gestation was less than 320 days and 65% if gestation was 320 days or longer. Overall foal survival was 54%, with a survival rate of 72% in foals less than 320 days of gestation and 32% in foals 320 days or more of gestation.[40] These numbers compare favorably to another study involving 26 mares, where 73% of the mares and 46% of the foals survived.[39] Uterine torsion is usually diagnosed

in its acute stage, although chronic uterine torsion has been reported.[41,42]

Surgical and nonsurgical management of uterine torsions have been used successfully. The standing flank approach has been more popular than the ventral midline approach for surgical repair.[38,39] However, in the recent report of 63 mares, 30 were treated through a ventral midline approach, 23 through a flank approach, and 10 by rolling.[40] In this study, there was no statistically significant difference for mare survival regarding method of resolution.

Nonsurgical management usually requires general anesthesia and rolling of the mare 360 degrees in the direction of the torsion. A long wooden plank, firmly placed across the mare's flank, is helpful in keeping the gravid uterus in the same position while the mare is rolled around it. This method of management has been successful. However, uterine rupture in a mare at term has been reported.[43] The few mares that are at term may be successfully corrected while standing by passing a hand into the uterus, grasping the fetus, and gradually rocking it, together with the uterus, in larger and larger arcs until it untwists.[38]

For the standing flank approach, the mare is sedated and placed in stocks. Local anesthesia is administered and routine surgical preparations are made. The flank incision is made on the side toward which the uterus is twisted. A modified grid approach through the body wall is used. An arm is introduced and a hand is placed under the gravid uterine horn. The uterus is gradually rocked back and forth, eventually allowing the uterus to flip back into its normal position. The status of the fetus and of the uterus should be evaluated. Closure is routine.

A ventral midline approach should be used if uterine rupture, tearing, or devitalization is suspected, if the foal is known to be dead and the mare is preterm, and if attempts at standing correction are unsuccessful.[13,39,40] If the mare is in a surgical facility where many abdominal surgeries are performed, the ventral midline approach should be considered instead of the flank approach, since it is more versatile.

Uterine Prolapse

Uterine prolapse occurs rarely in mares.[13,44,45] It tends to occur more frequently after dystocia and when fetal membranes are retained. Tension placed on the placenta and the use of disproportionate amounts of oxytocin have also been implicated with uterine prolapse. Although rare, the condition may be complicated by bladder prolapse, uterine tear, intestinal herniation, or uterine vessel rupture.

Uterine prolapse can be quite painful for the mare, and prompt sedation is indicated. Tocolytic agents, if available, may help decrease straining and discomfort. Prompt treatment is important. The uterus should be cleaned with saline and any placenta not directly attached to the uterus should be resected. Keeping the uterus elevated and protected in a plastic bag until definitive treatment is started reduces swelling, contamination, trauma, and straining.

The uterus can often be replaced in the standing mare if she is heavily sedated. Copious amounts of lubricant are used, and the uterus is gradually pushed back into its normal position. The use of fingertips should be avoided. Epidural anesthesia can be helpful in reducing straining. However, I do not routinely use epidural anesthesia, because it may not eliminate all straining, and its use is contraindicated if general anesthesia is performed. If standing uterine replacement is unsuccessful, the mare is anesthetized and her hind limbs are hoisted. The lack of straining and aid of gravity allow the uterus to be replaced relatively easily into its normal position. Care is taken to ensure that the uterine horns are fully extended. The vulva is sutured closed, except for a small ventral opening, and the mare is recovered. Postoperatively, the mare is treated with antibiotics, anti-inflammatories, and IV fluids if needed. The sutures are removed from the vulva and uterine lavages commence the next day. Although post-reduction oxytocin has been recommended, I avoid its use for 24 to 36 hours, as it may cause discomfort and straining.[13,44,45]

Uterine Tear

A uterine tear (laceration or rupture) usually occurs during a dystocia or normal foaling, but it also rarely occurs with uterine torsion or hydramnios (Figure 62-3).[13,15,46,47] Tears near the tips of the uterine horns are likely the result of acute rapid thrusts of the fetal hind feet during foaling. Most of these injuries occur during a percieved normal foaling. Tears in the uterine body (and vagina) logically result from forceful penetration of a blunt appendage (e.g., foot, muzzle, etc.) through a stretched and relatively unyielding uterine wall. These injuries usually occur during dystocia. Caudal uterine or vaginal tears are infrequently associated with evisceration of bowel during foaling. Presented with this situation, the bowel should be cleansed and replaced in the abdomen prior to delivering the foal. The tear should then be identified and a plan formulated for repair. Very rarely, a fetus gains access to the abdomen through a uterine tear, necessitating abdominal surgery to deliver the foal and repair the uterus.

Tears in the uterine body are sometimes found relatively soon after foaling and prior to development of peritonitis. The cervix and caudal uterus should be palpated during the first postpartum uterine lavage. However, palpation of a tear in the caudal uterus can be difficult after delivery because of the swelling and folding of the uterine lining.

Most uterine tears are diagnosed 1 to 3 days postpartum.[13,15,46-48] The most common presenting signs are depression, fever, mild abdominal discomfort, and tachycardia. This history and clinical signs, ultrasonographically identified excessive, cellular peritoneal fluid, and an elevated peritoneal WBC count

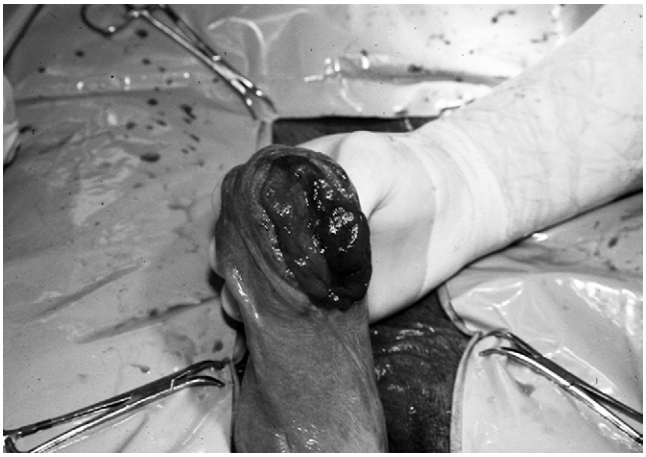

Figure 62-3. Typical appearance of a uterine tear near the apex of the uterine horn.

and total protein point strongly to a diagnosis of uterine tear. Further diagnostics, such as rectal exam, peritoneal fluid cytology, and so on, are needed to rule out intestinal tract trauma as the cause of peritonitis.

Two recent retrospective studies examining uterine tears yielded the following relatively similar results.[47,48] One study compared surgical to medical treatment of uterine tears.[47] The other examined the results of more than 70 mares with surgically treated uterine tears.[48] The most common presenting signs are listed in the previous paragraph. The median age was about 10.5 years. Tears were more common in the uterine horns (about 75%) than the body (about 25%). More tears occurred in the right uterine horn (about 75%) than the left (about 25%). Although there was a broad range, upon admission the median peritoneal fluid WBC count was about 70,000/μL and total protein value about 4.6 g/dL. The overall survival rate was about 76% in both studies.

There were some differences between the studies. In one study, there was no significant difference in survival rate between mares with tears in the uterine horns and those with tears in the uterine body.[47] In the other study, although not statistically analyzed, mares with uterine horn tears had a higher survival rate (84%) than those with uterine body tears (58%).[48] In the former study, there was no significant difference in survival rate, days of hospital stay, treatment costs, or fertility between medical and surgical management of the uterine tears.[47] Of 13 mares bred the same year as the injury, 12 concieved and carried the foal to term. In the other study, medical treatment was not evaluated.[48] All of the uterine tears in the latter study were repaired surgically.[48] All of the uterine horn and some of the uterine body tears were accessed through a caudal ventral midline celiotomy. These defects were closed using a simple-continuous pattern and oversewn with a continuous inverting pattern, both using No. 1 multifilament absorbable suture material. The abdomens were lavaged during surgery and for 2 to 3 days postoperatively. Uterine lavage started 1 day postoperatively and continued for 3 to 4 days. Perforations in the caudal body of the uterus were repaired in the standing mare *per vagina*. Closure usually consisted of a single layer of simple-interrupted or continuous No. 2 absorbable suture.

One paper referenced in this section has shown that medical treatment is a viable option for many uterine tears.[47] However, I am of the opinion that prompt surgical treatment of a uterine tear enables an accurate diagnosis, prevents further contamination of the abdominal cavity, allows the most thorough peritoneal lavage, and therefore hastens recovery.

Periparturient (Broad Ligament) Hemorrhage

Hemorrhage from one of the arteries supplying blood to the reproductive tract in the mare is the most common cause of death of the postpartum mare. A study of 98 mares that died after delivery revealed that 40 died from a rupture of one of these arteries.[49] Recognition of this condition and differentiating it from other causes of acute abdominal pain in the periparturient mare is important, as an exploratory celiotomy is generally contraindicated in these mares.

The actual site of the arterial bleeding was determined in one report of 31 mares that died from periparturient hemorrhage.[50] These sites included the uterine artery (n = 24), internal pudendal artery (n = 5), intenal iliac artery (n = 1), and caudal mesenteric artery (n = 1). The mares in this study had a mean age of 16.9 years, a mean parity of 9.5 times, and a mean time post-foaling to death of 8.5 hours. More than 90% of these mares died within 24 hours after delivery. Histological comparison of the ruptured vessels in these mares to uterine arteries collected from younger, less-multiparous mares revealed that atrophy of the smooth muscle cells with fibrosis of the arterial wall was one of the predisposing factors in aged multiparous mares.[50]

The vast majority of affected mares are aged and multiparous.[13] Often, these mares initially exhibit significant abdominal pain, especially if the blood is contained within the broad ligament. Rectal examination of persistently painful mares generally reveals a fluctuant mass in the broad ligament. If the broad ligament ruptures and the blood gains access to the abdominal cavity, the pain often dissipates and, on rectal examination, the broad ligament has a more edematous feel. On presentation, these mares generally have very pale mucous membranes, a weak pulse, and an elevated heart rate, are anxious, and are often sweating. The condition is diagnosed from the history and physical findings previously stated, identifying a hematoma or free blood on a rectal or abdominal ultrasonographic exam and the presence of blood in a peritoneal fluid sample.

Salient findings from another study of 73 mares with periparturient hemorrhage are as follows.[51] The median age was 14 years and median parity was 8 times. The hemorrhage occurred after delivery in 86% of the mares, and of these mares the median time interval from foaling to the hospital was 12 hours. Only 19% of these mares had a history of dystocia. In this study, 84% of the mares survived to hospital discharge.

Treatment varies depending on the clinician, but it should be aimed at avoiding excitement, restoring cardiovascular volume, controlling pain, enhancing hemostasis, and preventing infection.[51] Attempts to ligate the ruptured artery have met with limited success.

Cesarean Section

Elective Cesarean Section

Candidates for an elective Cesarean section (C-section) include mares that have a compromised birth canal as a result of a previous pelvic fracture or a soft tissue injury within the reproductive tract, and mares that have previously had a difficult dystocia or a severe uterine artery hemorrhage. The surgery must be well timed to yield a viable foal and have minimal adverse effects on the mare. Therefore, it is important to perform the surgery as close as possible to the natural foaling time. The mare should be hospitalized 7 to 10 days prior to her due date. Her physical status is checked frequently to determine udder development, softening of her perineal tissues, and behavior. Concentrations of electrolytes in the mammary secretions are very helpful for timing the surgery.[27] Decreasing sodium and increasing potassium and calcium levels are good indicators of impending parturition. The surgery, when performed, should proceed rapidly, with knowledgeable staff waiting to resuscitate the anesthetized foal on delivery. The fetal survival rate following elective C-section should be higher than 80%.[52-55]

Emergency Cesarean Section

The most common reason to perform a C-section in the mare is to resolve a dystocia.[27,52,54-57] This is truly an emergency situation and when the decision is made, the C-section should

proceed rapidly. The team of people involved should be well organized and prepared for this situation. Other circumstances potentially dictating the need for an emergency C-section include a near-term mare undergoing colic surgery or correction for uterine torsion. Under these circumstances, if the mare appears to have a good prognosis for survival, the fetus should remain in the mare until normal parturition. However, a C-section should be performed if the mare has a guarded prognosis. One study showed only three of eight (38%) term foals delivered during colic surgery survived to discharge.[54] It would be very unusual to encounter significant incisional complications during parturition following a recent abdominal surgery, so this should not be a factor when considering an emergency C-section.

SURGICAL TECHNIQUE

As the obstetrical manipulations for controlled vaginal delivery are being performed, the ventral midline of the mare is being prepared for a possible C-section (Figure 62-4).[56,57] When the decision is made, the surgery room is set up and the mare is positioned on the surgery table and readied for surgery. The time from the decision to perform a C-section to the delivery of the foal should be no more than 20 minutes. If the foal is known to be dead, this time is not as critical.

The most common approach to the uterus used for C-section in the mare is the caudal ventral midline.[15,27,52-58] The mare is positioned in dorsal lateral recumbency with the ventral midline tilted toward the surgeon. A 35- to 40-cm incision is made into the abdomen beginning 10 cm caudal to the umbilicus and extending craniad. The gravid uterine horn, which usually contains the hind limbs of the fetus, is located and exteriorized. One stay suture is placed in a cruciate pattern near the tip of the uterine horn, close to the position of the fetal feet, and another toward the body of the uterus, close to the position of the fetal hocks. An assistant surgeon handles the stay sutures during the procedure to minimize contamination of the abdomen with uterine fluids and to help facilitate closure. An extra impermeable drape is placed over the other drapes on the surgeon's side of the abdomen.

An incision is made through the uterine wall and chorioallantois from the level of the fetal hocks to the feet, creating a straight incision between the stay sutures (Figure 62-5). During extraction of the fetus, it is not uncommon to have the uterine wall tear slightly at the end of the uterotomy. The amniotic membrane, which has collapsed around the foal, is elevated and incised. The surgeon grasps the hind limbs and lifts the fetus up and out of the uterus. The hind limbs are handed to a third assistant, and the surgeon pulls the body up and out of the uterus. In this manner, the fetus is pulled from the mare rapidly. The umbilical cord is clamped and transected and the neonate is quickly transferred to a table set up just outside the surgery room for resuscitation and evaluation. The chorioallantois is separated from the endometrium for 3 to 4 cm along the incised edge of the uterine wall. If the placenta separates easily from the uterus, it may be entirely removed at that time. However, it is usually still well attached.

Infrequently, the hind limbs are not present in a uterine horn, making it extremely difficult to exteriorize any part of the uterus. The uterine incision is then made at the base of a horn and body of the uterus with the uterus in the abdomen. This causes significant concern about the amount of contamination occurring during surgery. After closure of the uterus, the abdomen will be lavaged with copious amounts of saline.

The incised edge of the uterine wall bleeds profusely. A continuous suture line is placed along this edge and large vessels are individually ligated to provide hemostasis. The need for the hemostatic suture line has been questioned.[59] However, I believe that it ensures close examination of the uterine edges and lessens the risk of postoperative bleeding enough to warrant the 10 minutes needed for its placement. The assistant elevates and tenses the stay sutures to facilitate closure of the uterus, which is performed in two layers with No. 1 or 2 absorbable suture material. The suture patterns used depend on the surgeon's preference. An inverting pattern is necessary in the outer layer to provide a serosa-to-serosa seal with exposed deeper tissues. This helps to prevent adhesions. After uterine closure, the uterus is lavaged and 40 units of oxytocin is administered IV. This quickly stimulates contraction of the uterus and aids expulsion of the placenta. The stay sutures are removed and the uterus is replaced into its normal position in the abdomen.

Figure 62-4. Controlled vaginal delivery. The mare's hind limbs are hoisted, the clinician is repositioning the foal, and the technicians are preparing the abdomen in case a C-section is necessary.

Figure 62-5. C-section with the uterine horn containing the hind limbs exteriorized. The amnion is partially incised near the point of the hock.

The extra impermeable drape is removed, the drape surface around the incision is lavaged, and the surgeon's gown and gloves are changed if needed. The abdomen is lavaged with 10 to 15 L of warm saline, which is removed by suction. An abdominal drain for subsequent lavage may be placed if deemed necessary. A crystalline penicillin solution is instilled into the abdomen, which is closed in routine fashion for a ventral midline celiotomy.

Terminal Cesarean Section

Significant concern for a foal in a mare with a terminal illness may require a C-section. Examples include mares recumbent as a result of neurologic abnormalities, severe laminitis, or other potentially fatal or debilitating conditions. If euthanasia is planned for the mare after the surgery, sterility is not a high priority but speed of delivery is. Most of these surgeries can be performed quickly through a low flank approach after induction of anesthesia.

Aftercare

Postoperative care for a C-section mare is very similar to that for any mare after abdominal surgery, with special attention paid to the reproductive tract. Some mares pass their placenta in the recovery stall. If the placenta has not passed, oxytocin is again administered 2 to 3 hours after delivery. The initial dose is 40 U in 1 L of lactated Ringer's solution given IV over 30 to 60 minutes. Since this often causes abdominal pain, the rate of administration is dictated by the response of the mare. In refractory cases, 80 U is used every 4 to 6 hours. Usually, the placenta passes within 8 hours after delivery. Manual rupture of the chorioallantoic membrane and exteriorization of the amniotic membrane *per vagina* may be needed to initiate placental expulsion after an elective C-section. Uterine lavage is started soon after the placenta is passed, or simultaneously if it is retained. The uterus is generally lavaged once daily for 3 to 4 days.

Systemic antibiotics and flunixin meglumine are administered for 3 to 5 days after the delivery, depending on the degree of contamination and tissue trauma encountered. Intravenous fluids are administered as needed to maintain adequate hydration and vascular volume and to correct electrolyte imbalances. Abdominal surgery, even without manipulation of the intestines, combined with possible bruising of the small colon or cecum from the dystocia, can result in transient postoperative ileus. Swollen and painful pelvic tissues may lead to retention of feces. The postpartum diet should reflect concern for these potential problems. Water is offered freely. The mare is walked and allowed to graze on green grass the first postoperative day. A bran mash with mineral oil may be beneficial. Discharge instructions include hand walking 2 to 3 times each day, with or without small-paddock turn-out for 3 to 4 weeks.

DYSTOCIA

Dystocia in the mare is one of the few true emergencies an equine practitioner may encounter. Prompt action increases the probability of survival of the foal and decreases the degree of reproductive trauma to the mare.[56,57,60,61] The four procedures to resolve dystocia are assisted vaginal delivery, in which the mare is awake and is assisted in the vaginal delivery of an intact foal; controlled vaginal delivery (CVD), in which the mare is anesthetized and the clinician is in complete control of the delivery of an intact foal vaginally; fetotomy, in which the dead fetus is divided into more than one part for removal from the uterus *per vagina* in an awake or an anesthetized mare; and C-section, in which the fetus is removed through an incision in the uterus.

The goal of dystocia resolution is to deliver a live foal in a manner resulting in a reproductively sound mare.[57] This is usually accomplished at the farm. However, referral hospitals need to be prepared for difficult cases, and the clinician must be able to perform whatever procedure is necessary to resolve any dystocia. Currently, emergency clinicians, who are usually trained as surgeons or internists, tend to perform C-sections at these hospitals. Thus, these clinicians should also be familiar with CVD and fetotomy techniques. No single procedure is right for every situation.

After the mare is in the hospital, dystocia requires prompt action. The mare is anesthetized shortly after arrival to attempt CVD. The hind limbs are hoisted upward until the pelvis is about 3 feet above the ground. Decreased straining, aided by the effects of gravity, and lubrication usually enable resolution of the dystocia. If the foal cannot be delivered within 15 minutes, a C-section or fetotomy should be considered. C-section techniques were described earlier. Fetotomy techniques are described elsewhere.[62-64]

Results of dystocia resolved in a referral hospital in an area with a high concentration of broodmares were reported in 2002.[56] Of 247 cases of dystocia, 71% were resolved by CVD, 25% by C-section, and 4% by fetotomy. At the same hospital, with now more than 700 cases of dystocia at the time of this writing, the distribution of how dystocias were resolved has stayed essentially the same. The method of dystocia resolution varies between hospitals.[56,58,61]

It is difficult to compare short-term survival rates of dystocia mares between different studies. In studies reporting all the procedures used to resolve dystocia, mare survival rates ranged from 82% to 91%.[54,56,61] The mare survival rate following CVD, in the earlier study was 94%.[56] Mare survival rates following C-section to resolve dystocia have ranged from 82% to 91%.[54,56,58,65] Studies focusing on the results following fetotomy performed in a referral hospital or on a farm yielded mare survival rates of 90% to 96%.[63,64]

Duration of dystocia directly affects foal survival. Two studies have shown significant differences in duration of dystocia (from chorioallantois rupture to delivery) between foals surviving and not surviving to discharge from the hospital.[56,61] In one study, the group of surviving foals had a duration median of 60 minutes and the nonsurvivor foals had a duration median of 79 minutes.[56] In this study, 42% of the foals were born alive and 30% were discharged. In another study, the group of surviving foals had a median duration of 71 minutes and the nonsurvivor foals had a median duration of 249 minutes.[61] In this study, 30% of the foals were born alive and 13% were discharged. The median duration was significantly different. In these studies, no foal survived to discharge following dystocia duration of more than 162 minutes. Interestingly, in another study of 33 Fresian mares, the surviving foals had a dystocia mean of 164 minutes and the nonsurvivor foals had a mean duration of 490 minutes. In this study 42% of the foals were born alive and 31% were discharged. It was surmised that the difference resulted from fewer foals engaging in the pelvic canal prior to resolution of the dystocia in this breed.[58]

Increasing duration of dystocia prior to resolution adversely affects fertility of the mare. Live foaling rates are decreased, but can approach normal. Minimizing trauma to the mare's reproductive tract during resolution of dystocia will improve post-dystocia fertility.[55-58,63,64]

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SECTION
X

URINARY SYSTEM

Jörg A. Auer

CHAPTER
63

Diagnostic Techniques and Principles of Urinary Tract Surgery

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PATIENT EVALUATION

Harold C. Schott II

History

Important historical information for horses that may require surgical treatment of the urinary tract includes type and duration of clinical signs, diet, whether other horses may be affected, medications administered, and response to treatment. The most common presenting complaints for horses with urinary tract disease are weight loss, decreased performance, and abnormal urination (stranguria, pigmenturia, pyuria, or incontinence). Other clinical signs vary with the cause and site of the problem and may include fever, anorexia, lethargy, ventral edema, oral ulceration, excessive dental tartar, colic, or scalding or blood staining of the perineum or hind legs (Table 63-1). Although lumbar pain and hind limb lameness have been attributed to urinary tract disease (and may occur with a large renal mass), a musculoskeletal problem is the more common cause of these clinical signs. Decreased performance may be an early presenting complaint for renal disease, possibly a consequence of changes associated with uremia (anemia and lethargy). An occasional horse with urolithiasis or renal neoplasia may have a history of recurrent colic. Prolonged or repeated posturing to urinate and dysuria or hematuria are important findings to implicate the urinary tract as the probable source of abdominal pain in such patients. Obstructive urethrolithiasis in male horses also generally presents as "renal colic," but noteworthy differences from other types of colic are that affected horses typically have the penis dropped and are still willing to eat with this problem.

TABLE 63-1. Historical Factors and Presenting Complaints Associated with Upper and Lower Urinary Tract Disorders

Disorder	Complaints
Upper urinary tract disorders	Decreased performance
	Lethargy
	Partial inappetance
	Weight loss
	Increased thirst
	Polyuria and polydipsia
	Recurrent fever
	Ventral edema
	Oral ulcers
	Excessive dental tartar
Lower urinary tract disorders	Stranguria
	Pollakiuria
	Prolonged or abnormal micturition posture
	Dropped penis
	Distended urethra below anus
	Mass on external genitalia
	Incontinence
	Urine scalding
	Strong urine odor
Upper and/or lower urinary tract disorders	Acute colic
	Recurrent colic
	Abnormal gait
	Hematuria
	Pyuria

When collecting a history, owners should be queried about water intake and urine output as well as changes in frequency, posturing, and appearance of urination. Increased thirst and water intake (polydipsia) may be reported with renal disease, pituitary pars intermedia dysfunction, primary polydipsia, diabetes mellitus, or diabetes insipidus.[1] Astute owners may report increased thirst after exercise or a change in urine appearance, such as a clearer stream, to support polyuria and polydipsia (PU/PD). Owners should also be questioned about any apparent discomfort shown by the horse when posturing to urinate or during actual micturition (i.e., stranguria) as well as abnormal appearance of urine (e.g., pigmenturia, pyuria, or excessive sediment). Pollakiuria (frequent urination) and polyuria (increased urine production) can be confused by an owner and careful questioning is necessary to distinguish between these two problems. Pollakiuria can be seen in normal mares during estrus or with cystic calculi or cystitis in either sex. In contrast, polyuria more often accompanies the previously listed disorders associated with polydipsia. Pollakiuria should also be differentiated from frequent involuntary voiding of urine typical of horses with incontinence. With the latter problem, urine is often passed without the horse posturing to urinate and, in the

early stages of incontinence, may only be observed during exercise. In mares with incontinence, the number of foals delivered should be determined along with any history of dystocia that could have damaged the lower urinary tract. Owners of horses presenting with incontinence should be carefully questioned about possible musculoskeletal or neurologic disorders in the months to years before because incontinence may be a late complication of these primary problems.[2] Further, if excess urine sediment is observed when urine is passed on a flat surface (e.g., when placed in cross ties or a wash stall), accumulation of urine sediment in the bladder should be suspected.

When the presenting complaint is pigmenturia, questions about recent exercise or management changes, as well as when discolored urine is observed, are useful to determine whether the primary problem may be myoglobinuria, hemoglobinuria, or hematuria (Table 63-2).[3] Recent muscle cramping (rhabdomyolysis) during exercise would support myoglobinuria, whereas hemolysis and hemoglobinuria may develop with infectious diseases, exposure to toxins, or as a consequence of immune-mediated hemolysis. Treatment with other medications including rifampin, phenothiazines, nitazoxanide, and phenazopyridine can also cause urine to turn orange, whereas treatment with doxycycline may cause urine to turn dark brown to black. Finally, exposure of equine urine to air typically results in a progressive deepening of color from deep yellow to orange-red to brown. This may be confused with pigmenturia when discolored urine is observed on bedding. This progressive discoloration occurs in some, but not all, urine samples and has been attributed to presence of pyrocatechins in equine urine that change color as they oxidize when exposed to air. In some instances, the urine also turns red after voiding and owners may call when they observe "red urine" on snow.

When pigmenturia is a result of hematuria, passage of red urine throughout urination is consistent with hemorrhage from the kidneys, ureters, or bladder, whereas hematuria at the beginning of urination is often associated with lesions in the distal urethra. Hematuria at the end of urination is usually the result of hemorrhage from the proximal urethra or bladder neck. In fact, the primary rule out for hematuria following exercise is cystolithiasis, whereas passage of several squirts of bright red blood with urethral contractions after the end of urination is nearly pathognomonic for proximal urethral rents in male horses.[4]

TABLE 63-2. Causes of Pigmenturia in Horses

Type of Pigmenturia	Causes
Idiopathic pigmenturia	Oxidation of pyrocatechins in normal urine
	Drug-associated
	rifampin
	Phenothiazine
	Nitazoxanide
	Phenazopyridine
	doxycycline
Myoglobinuria	Exercise-associated rhabdomyolysis
	Complication of anesthesia (crush injury)
	Complication of *Strep. equi* infection
	Clostridial myonecrosis
Hemoglobinuria	Infectious disease
	piroplasmosis
	Equine infectious anemia
	Clostridium spp.
	Intoxication
	Red maple *(Acer rubrum)*
	Onions
	Immunological disorders
	Purpura hemorrhagica
	Neonatal isoerythrolysis
	Adverse drug reactions
	Procaine penicillin G
	Nonsteroidal anti-inflammatory drugs
Hematuria	Urolithiasis
	Urinary tract infection
	Proximal urethral rents
	Neoplasia
	Verminous nephritis *(Halicephalobus gingivalis, Strongylus vulgaris)*
	Idiopathic renal hematuria
	Vascular malformations
	Bladder hematoma (neonates)
	Exercise-associated bladder mucosal trauma
	Intoxication
	Blister beetle (cantharidin) poisoning
	Nonsteroidal anti-inflammatory drugs

Physical Examination

Physical examination findings in horses with upper urinary tract disease may be fairly unremarkable, but subtle changes might be detected. For example, decreased body condition, intermittent decreased appetite, excessive dental tarter, mild ventral edema, and a "fishy" or uremic odor of the oral cavity and skin may be the only outward signs of chronic renal failure (CRF) in uremic horses.[5] Horses with pyelonephritis may have intermittent fevers, lethargy, and decreased appetite, whereas the occasional horse with a renal mass may have altered abdominal conformation (protrusion of the flank on the affected side), mild scoliosis, altered hind limb stance, and hind limb gait abnormalities.[6]

Horses with lower urinary tract disease are often in good body condition with normal vital parameters. Careful examination of the external genitalia may reveal dried blood, a mass, or excessive urine odor. Horses with incontinence typically have urine scalding of the perineum (mares) or of the lower hind limbs (males) in addition to a strong urine odor around the hindquarters. When incontinence is present, it is useful to assess whether the horse appears conscious of bladder distension (becomes restless or uses abdominal muscles to partially empty the bladder) or whether urine appears to be intermittently voided in an unconscious manner. When incontinence is the primary problem, complete lameness and neurologic examinations should be performed to determine whether gait deficits accompany the lower urinary tract disorder. In younger horses with unilateral ectopic ureter, intermittent urine dribbling from the vulva or penis may be observed along with urine scalding. When this problem is unilateral, affected horses are typically reported to also posture and urinate normally.

Part of the initial assessment of horses with suspected renal disease or other disorders causing PU/PD should be to quantify water intake. Daily water intake provided by drinking is normally 45 to 55 mL/kg per day (22.5 to 27.5 L/day for a 500-kg horse) and can be determined by stabling the horse alone and providing a known volume of water.[7] Because water intake can vary with environmental conditions, level of activity, and diet, repeated measurement over several 24-hour periods provides a more accurate assessment of daily water consumption. Quantitative assessment of urine output, which should range between 0.5 and 1 mL/kg per day (6 to 12 L/day for a 500-kg horse), is more challenging to perform. Urine collection harnesses can be applied for 24-hour urine collections, and although horses used for research tolerate these devices fairly well, they have limited application to clinical patients.

Rectal Examination

Rectal palpation should be included as part of a complete physical examination of all horses with suspected urinary tract disease. Initially, the urethra below the anus in male horses should be examined externally as the tail is lifted. Moderate to severe distention often accompanies urethral obstruction, and the offending urolith may be palpated at or below the level of the ischial arch (Figure 63-1). In addition, symmetry of the perineum should be assessed because deformities may be detected in some horses with proximal urethral rents (Figure 63-2). The initial part of the rectal exam involves palpation of the intrapelvic portion of the urethra and urethral sphincter. The latter feels somewhat like the cervix of a mare, only more caudal. Subsequently, the bladder should be palpated to determine size, wall thickness, and presence of cystic calculi, excess sediment, or mural masses. If the bladder is full, palpation should be performed again after bladder catheterization or spontaneous voiding empties the bladder.

If palpating for a suspected cystolith, it is important to remember that dysuria and pollakiuria frequently result in a small bladder that may be entirely within the pelvic canal. In this situation, the bladder and disc-shaped cystolith are best palpated with the hand inserted only wrist deep into the rectum. If the hand is inserted farther forward to search for the bladder in the expected location over the brim of the pelvis, a cystolith can be missed because it may be lying just under the forearm. Most horses presented for incontinence have a large bladder that can easily be made to overflow with pressure placed over the top of the bladder. In contrast, it is nearly impossible to express urine from a horse with a normal urethral sphincter during rectal palpation. Horses with large, atonic bladders often develop sabulous urolithiasis, an accumulation of normal urine sediment in the ventral aspect of the bladder. This sabulous

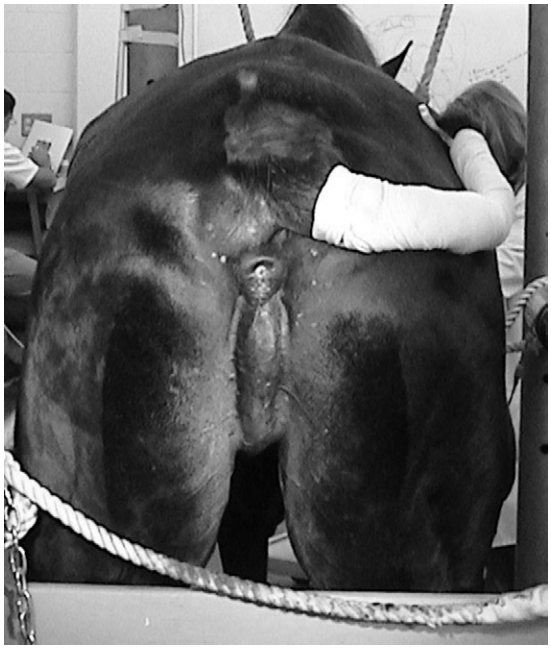

Figure 63-1. Distention of the urethra below the anus in a gelding with an obstructive urethrolith

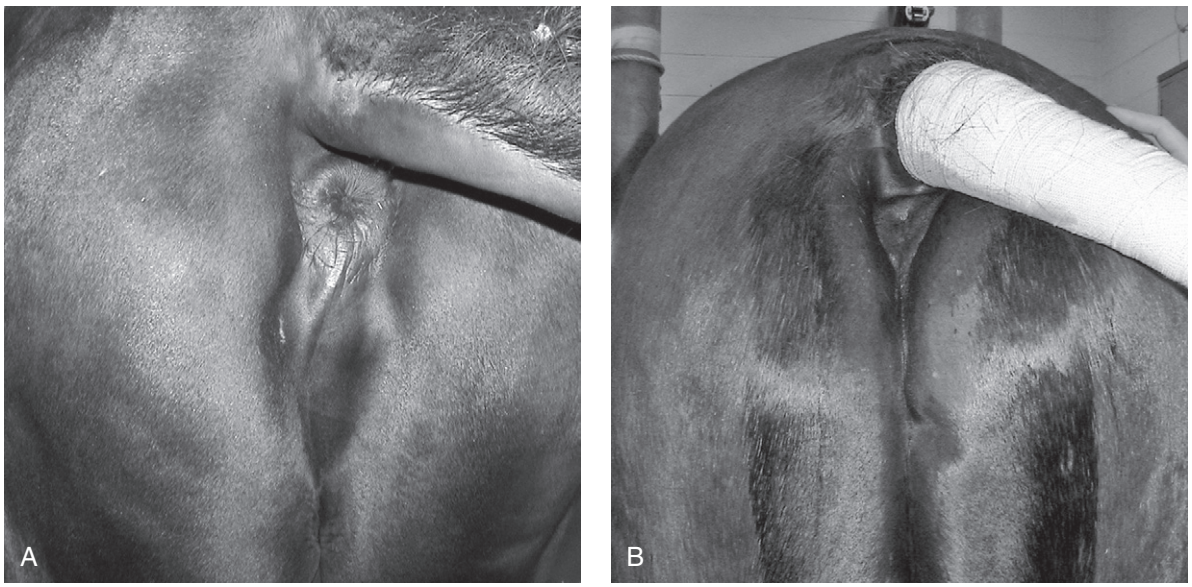

Figure 63-2. Asymmetry of the perineum in two geldings with proximal urethral rents causing hematuria immediately after urination. **A,** Note widening of the perineum. **B,** Note an indentation of the perineum.

sediment can become a large mass and be mistaken for a cystolith.[2] However, two features that can be used to differentiate these problems: (1) the bladder is usually quite large with sabulous urolithiasis when compared to a small bladder with a cystolith and (2) the sabulous sediment can often be indented with firm digital pressure (the bladder may need to be emptied by catheterization to fully appreciate this difference from a cystolith). Last, the apex or cranial portion of the bladder should be palpated to ensure that it is freely movable within the abdomen. Occasionally, the bladder apex remains adhered to the umbilicus, resulting in a bladder that is more tubular and cannot be freely manipulated.

Next, the caudal pole of the left kidney can be palpated for size and texture. The left kidney may be enlarged and/or painful on palpation in horses with acute renal failure (ARF), whereas it may seem smaller, irregularly shaped, and firmer in horses with CRF. It warrants emphasis that these findings are variable and highly subjective. Occasionally, a markedly enlarged kidney, either right or left, may be palpated in cases of polycystic kidney disease or neoplasia. Malposition of one of the kidneys is a rare congenital defect, but I have palpated an apparently normal right kidney immediately above the uterus in a mare. The ureters are generally not palpable unless they are enlarged or obstructed, but the dorsal abdomen (retroperitoneal course of ureters from abaxial to the aorta to the top of the bladder neck) and trigone should be palpated to determine if they can be detected. Dilatation of a ureter (to the size and texture of a garden hose) occurs in some horses with pyelonephritis or ureterolithiasis. In mares, palpation of the distal ureters through the ventral vaginal wall may be more rewarding as they diverge from the bladder neck in a Y-shaped pattern. The distal ureters and ureteral orifices into the bladder can also be palpated in mares at the 2 and 10 o'clock positions between an index or middle finger inserted through the urethra into the bladder and the thumb in the adjacent portion of the vagina. Finally, the reproductive tract in mares and accessory sex glands in stallions should also be palpated to assess whether a reproductive problem could be causing the clinical signs.

Clinical Pathology

Hematology and Serum Chemistry Analysis

A minimum database for a horse with a suspected urinary tract disorder should include a complete blood count (CBC), serum chemistry profile, and urinalysis. An elevated white blood cell (WBC) count and fibrinogen concentration would support an inflammatory or infectious disease process. An elevated globulin concentration further supports chronicity (weeks to months) of an inflammatory response. Mild anemia (packed cell volume 25% to 30%) following decreased erythropoietin production and a shortened red blood cell (RBC) life span with uremia may also be observed in horses with CRF.

On the serum chemistry profile, blood urea nitrogen (BUN) and serum creatinine (Cr) concentrations are the major parameters used to assess renal function, although additional information is provided by electrolyte concentrations. It is important to remember that BUN and Cr are insensitive indicators of decreased renal function because values might not exceed the upper limits of reference ranges until glomerular filtration rate (GFR) is reduced by 75% or more. Although this commonly used percentage is based on studies of subtotal nephrectomy in laboratory animals, it warrants mention that Cr remained normal and body weight was maintained after experimental unilateral nephrectomy in ponies[8] and horses.[9] Therefore, measurement of BUN and Cr is of limited use in evaluating minor changes in GFR. However, when elevated above the upper limit of the reference ranges, small increases in BUN and Cr are sensitive indicators of further deterioration in GFR, because doubling of BUN or Cr can be interpreted as a further 50% decline in remaining renal function (Figure 63-3). Although the term *blood urea nitrogen* concentration is widely accepted, it is important to remember that the actual measurement reported is the urea concentration in serum. Reporting of BUN and Cr also varies among countries. In the United States, BUN and Cr are reported in milligrams per deciliter, whereas in other parts of the world they are reported in standard international units of millimoles per liter and micromoles per liter, respectively. Conversion of BUN from milligrams per deciliter, to millimoles per liter and of Cr from milligrams per deciliter, to micromoles per liter is accomplished by multiplying by 0.357 and 88, respectively.

Azotemia is the term used to describe increases in BUN and Cr detected on a serum chemistry profile; hence, azotemia is a laboratory diagnosis. Azotemia can be prerenal in origin, resulting from decreases in renal blood flow (RBF) and GFR, or it can be the result of primary (intrinsic) renal disease or obstructive disease or disruption of the urinary tract (postrenal failure).[10] The term *prerenal failure* has been used to describe reversible increases in BUN and Cr associated with renal hypoperfusion. Although this term is firmly entrenched in the human and veterinary medical literature, its use likely contributes to a lack of recognition of subclinical renal damage that accompanies a number of medical and surgical conditions. This can be attributed to a large renal functional reserve capacity. In many patients with reversible azotemia and changes in glomerular and tubule function. Integrity can be demonstrated by proteinuria and cast

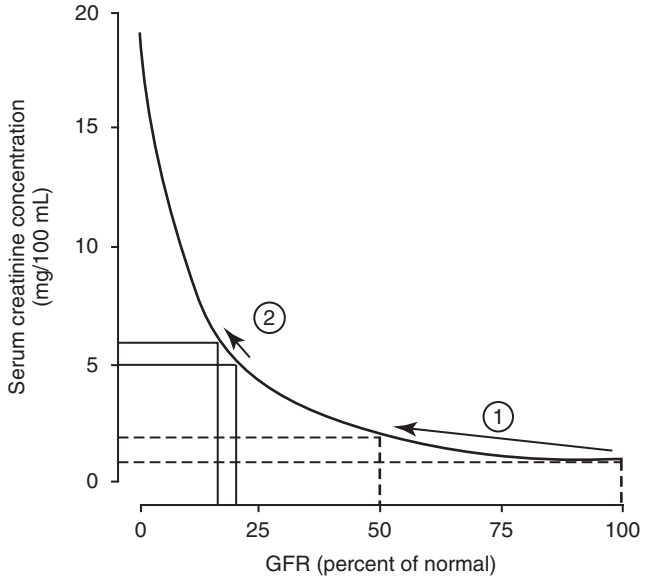

Figure 63-3. Relationship between glomerular filtration rate (GFR) and serum creatinine (Cr) concentration. When renal function is normal, a large decrease in GFR results in a minor increase in Cr *(arrow 1)*. In contrast, when renal function is decreased, as with chronic kidney disease, a much smaller decrease in GFR results in a similar increase in Cr *(arrow 2)*. (From Brenner BM (ed): Brenner and Rector's The Kidney. 8th ed. Saunders, Philadelphia, 2008.)

TABLE 63-3. Incidence of Azotemia (and Associated Mortality) in Horses Presented to a Veterinary Teaching Hospital

	1997		2000	
Examination Data	**Number**	**Percentage**	**Number**	**Percentage**
Horses examined	1902		2289	
Serum chemistry performed at admission*	397	21%	423	18%
Cr ≥ 2.5 mg/dL	82	4.3%	81	3.5%
Cr ≥ 5 mg/dL (mortality rate)	15	0.8% (31%)	19	0.8% (44%)
Cr ≥ 10 mg/dL (mortality rate)	2	0.11% (100%)	3	0.13% (33%)†
Primary renal disease	3	0.16%	2	0.09%

*Assumes that horses that did not have a serum chemistry performed also did not have azotemia.
†The two survivors were neonatal foals with spurious hypercreatininemia.

formation, impaired concentrating ability, and an increase in sodium excretion. Despite the reversible nature of these functional alterations, a degree of permanent nephron loss may occur with prerenal failure and likely explains the finding of microscopic evidence of renal damage in as many as one third of normal equine kidneys.[11] To increase awareness of subclinical renal damage in patients with decreased RBF and GFR, the term *acute kidney injury (AKI)* has been introduced in human and, subsequently, small animal medicine. AKI is defined as an increase in Cr of 0.3 mg/dL or a 50% increase from the baseline value, yet Cr may remain within the reference range.[12] Hemodynamically induced AKI is often associated with oliguria (urine output of less than 0.5 mL/kg for at least 6 hours) whereas urine production with nephrotoxin-associated AKI often remains normal (nonolguric AKI). In addition to changes in Cr and urine output, a number of novel biomarkers of AKI, notably kidney injury molecule-1 in urine, are being investigated as early indicators of renal damage.[13] A similar change in terminology has been adopted in human and small animal medicine for patients with chronic renal disease. Rather than describing patients as suffering from CRF (often an end-stage problem), the term *chronic kidney disease (CKD)* has been introduced to shift attention to the detection of earlier stages of chronic renal disease.[14,15] Although CKD is by nature a progressive disorder, early detection and interventions may slow the rate of progression, thereby prolonging life and, for people, delaying the potential need for renal replacement therapy.

To limit potential nephron damage with renal hypoperfusion, prerenal failure is accompanied by a number of compensatory responses to preserve RBF (autoregulatory response of afferent arterioles) and GFR (increase in filtration fraction due to angiotensin II–mediated efferent arteriolar constriction).[16] However, intrarenal blood flow is not homogeneous with the cortex receiving a substantially greater part of total RBF than the medulla. As a consequence, the medulla, especially the inner medulla, exists in a relatively hypoxic environment even with normal hydration status and RBF.[17] When RBF is reduced, intrarenal production of vasodilative prostaglandins (PGI_2 and PGE_2) is another important response to maintain or even increase medullary blood flow. However, administration of nonsteroidal anti-inflammatory drugs (NSAIDs) nearly abolishes this renoprotective response and can exacerbate the risk of renal damage with renal ischemia. Not surprisingly, the lesion associated with NSAID nephropathy is renal papillary or medullary crest necrosis.[18] Although NSAIDs that preferentially inhibit cyclooxygenase-2 (COX-2) activity (e.g., firocoxib) have been documented to have less adverse effects on the gastrointestinal tract of horses, evidence from other species suggests that the more COX-2 selective NSAIDs offer essentially no protection against NSAID-induced renal injury.[19]

In general, horses with reversible azotemia, covering the spectrum from prerenal failure to mild intrinsic renal damage, tend to have more modest elevations in BUN and Cr than horses with intrinsic ARF or postrenal problems.[20] However, there can be wide ranges of BUN and Cr values for all three categories of azotemia; consequently, specific values do not identify the type of azotemia. The incidence of significant azotemia (defined as Cr ≥ 2.5 mg/dL) in horses that presented to Michigan State University's Veterinary Teaching Hospital during 1997 and 2000 was less than 5% (Table 63-3). Further, the incidence of primary renal disease was less than 0.2%, consistent with azotemia most commonly being a problem secondary to other diseases (e.g., colic, enterocolitis, rhabdomyolysis, intoxications, and others). Of interest, horses with moderate azotemia (Cr 5 to 10 mg/dL) had a mortality rate of 30% to 45%, but the magnitude of Cr elevation did not predict survival in this range of azotemia. Because survival more likely depends on resolution of the primary disease process, it should not be surprising that the magnitude of moderate azotemia did not affect prognosis. In contrast, all adult horses with severe azotemia (Cr > 10 mg/dL) did not survive. In this limited population, only severe azotemia appeared to affect prognosis. In addition to severe azotemia, the change in Cr with treatment also has prognostic value with a reasonable goal being that Cr should decrease by 30% or more during the initial 24 hours of appropriate treatment. As an example, in horses that presented with colic or enterocolitis and a Cr > 3 mg/dL to the University of Georgia Veterinary Teaching Hospital, persistence of azotemia over the initial 3 days of treatment was associated with a greater volume of gastric reflux, abnormal rectal exam findings, and hypochloremia, as compared to horses in which azotemia resolved. Consistent with more severe fluid losses, hypovolemia, and renal damage, horses with persistent azotemia were three times more likely to have a poor outcome.[21]

To further define the type of renal failure in azotemic patients, the BUN:Cr ratio has also been used. In theory, this ratio should be higher for prerenal failure (10 or more using mg/dL units, owing to increased reabsorption of urea with decreased GFR and tubular flow rates) and postrenal failure with uroperitoneum (because of preferential diffusion of urea across the peritoneal membrane) than for azotemia associated with intrinsic renal failure. However, similar to absolute values for BUN

and Cr, BUN:Cr ratios measured in azotemic dogs with naturally occurring diseases were distributed over wide, nondiscriminatory ranges for all three types of renal failure.[22] In horses with acute medical and surgical problems complicated by dehydration and hypovolemia, clinical experience finds that Cr tends to increase (percentage) by a greater magnitude than BUN, leading to a BUN:Cr ratio less than 10:1.[23] In contrast, with CRF the BUN:Cr ratio often exceeds 10:1.[5,24] In addition to differences in excretion and reabsorption of urea and creatinine with decreased RBF and GFR, the preferential increase in Cr with acute disorders may also be related to different chemical properties of urea and creatinine. Urea, a nonpolar molecule, diffuses freely into all body fluids, whereas creatinine, a larger charged molecule, likely requires more time to move out of the extracellular fluid space. The important point is that a sudden decrease in RBF and GFR typically leads to a greater increase in Cr than in BUN, and Cr is the more accurate parameter to follow over time to assess improvement in renal function. As always, exceptions occur and an occasional horse with an acute problem may have a comparatively greater increase in BUN than Cr. In my experience, this is more common in foals and yearlings and may be associated with gastric or upper intestinal bleeding with ulcerative disease. Neonatal foals delivered from mares with placentitis or that experience perinatal asphyxia may have markedly elevated Cr concentrations (occasionally more than 20 mg/dL) after birth with essentially normal renal function. This clinicopathologic finding, termed *spurious hypercreatininemia*, resolves rapidly over the initial 1 to 3 days of life, and normal renal function is supported by adequate urine output and normal serum electrolyte concentrations.[25] With CKD, both BUN and Cr should be monitored over time, and the BUN:Cr ratio may be useful in assessing dietary protein intake because values 15:1 or more suggest excessive protein intake and urea production.[5,24]

Finally, increases in urea and creatinine have different effects on tissues. Specifically, urea is one of the uremic toxins that accumulates with renal failure, and progressive increases in BUN contribute to tissue dysfunction and morbidity, especially at values exceeding 75 mg/dL. In contrast, creatinine has little harmful effect on tissues and should not be considered a toxic compound. Rather, creatinine is simply a marker of renal function that increases when RBF and GFR decline. The reason that Cr is a useful indicator of renal function is that about 1% of muscle creatine is broken down to creatinine daily and the primary route of elimination is in urine (via glomerular filtration with little tubular reabsorption or secretion). Accordingly, as GFR decreases, urinary creatinine excretion also decreases, leading to a progressive rise in Cr.

In addition to screening for azotemia, the chemistry profile yields serum electrolyte, protein (albumin and globulin), and glucose concentrations and muscle enzyme activities. An important difference between prerenal failure and intrinsic renal failure is that electrolyte concentrations should remain normal with prerenal failure, whereas hyponatremia and hypochloremia are characteristic findings in horses with renal disease.[10,20,23] Unfortunately, hyponatremia and especially hypochloremia can also be found in horses with colic or enterocolitis; as a result, these electrolyte changes are not specific for AKI. Serum potassium concentration may be low, normal, or increased with AKI, but significant hyperkalemia (more than 6 mEq/L) is more commonly found with oliguric to anuric ARF or with uroperitoneum. Calcium and phosphorus concentrations vary in horses with renal disease. Hypercalcemia and hypophosphatemia are often found in horses with CKD, especially those fed alfalfa hay, whereas hypocalcemia and hyperphosphatemia may be found with AKI. In fact, the combined findings of azotemia and hypercalcemia are essentially pathognomonic for CKD in horses.[5,24]

Total protein concentration in horses with primary urinary tract disorders is usually normal; however, an occasional horse with a chronic inflammatory lesion (e.g., pyelonephritis, chronic cystitis, neoplasia) has an elevated globulin concentration. With end-stage CRF, intestinal ulceration may lead to hypoproteinemia. Mild hypoalbuminemia may also develop with protein-losing glomerulopathies because albumin tends to pass through the damaged glomerular basement membrane to a greater extent than the higher-molecular-weight globulins. A rare horse with primary glomerular disease may actually develop chronic hypoalbuminemia before onset of azotemia.[26] In horses with AKI secondary to other diseases, total protein concentration is altered more by the primary disease process than AKI. Hyperglycemia secondary to stress, exercise, sepsis, pituitary pars intermedia dysfunction, or diabetes mellitus can result in glucosuria. The renal threshold for glucose in horses has not been well investigated but is likely lower (160 to 180 mg/dL) than in small animals or people. Thus, glucosuria must always be interpreted with knowledge of serum glucose concentration. When pigmenturia is a complaint, measurement of creatine kinase (CK) and aspartate aminotransferase (AST) activities is useful to differentiate myoglobinuria from hematuria or hemoglobinuria.

Acid-Base Balance

Venous blood gas analysis in horses with AKI usually reflects the primary disease process, rather than the secondary renal insult. Thus, horses with colic are often mildly alkalotic because of pain and hyperventilation. With more serious disease and endotoxemia, a variable degree of metabolic and lactic acidosis may be present. With primary renal disease, mild metabolic acidosis may also be detected, but acidosis is usually not severe until marked azotemia (Cr > 10 mg/dL) develops with either oliguric AKI or end-stage CRF.

Urinalysis

SAMPLE COLLECTION

Urine can be collected as a midstream catch during voiding or via urethral catheterization in both sexes. Manual compression of the bladder during rectal palpation may stimulate urination after the rectal examination is completed, especially when a horse is placed in a freshly bedded stall. A fairly practical device to obtain a urine sample from geldings or stallions can be made by cutting off the bottom of a gallon plastic bottle, which is then padded and secured below the sheath (Figure 63-4). The opening of the bottle is covered with a cap that can be unscrewed to collect a urine sample after urine has been voided. In addition, some horses can be trained to void urine on command; often this is most successful following exercise.

Passage of a flexible plastic or rubber catheter via the urethra into the bladder can be easily accomplished in both sexes, although it is generally necessary to sedate male horses for the penis to descend. Bladder catheterization is a clean, but not sterile, procedure because bacteria are present in the vestibule

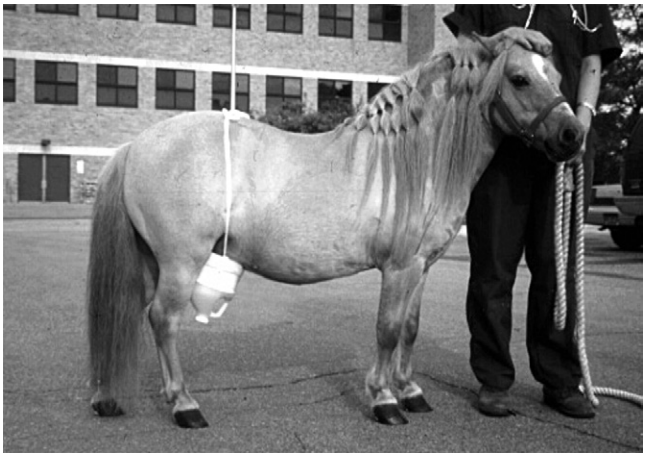

Figure 63-4. A urine collection device for use in male horses and ponies.

of the mare and the distal urethra of male horses. Thus, clean exam gloves can be used rather than sterile surgical gloves for the procedure, but a sterile catheter should be used. The vulva or end of the penis should be cleaned with soap and water and the accumulation of smega in the urethral sinuses dorsal to the urethra (the "bean") should be removed in male horses. Sterile lubricant is applied to the end of the catheter and it is passed into the urethra. In males, mild resistance to passage may be felt as the catheter is passed over the ischial arch into the intrapelvic portion of the urethra (the catheter should be palpable below the anus). Greater resistance is appreciated as the catheter is passed through the urethral sphincter into the bladder. The end of the catheter outside the horse should be plugged with a 60-mL syringe to prevent air from being aspirated into the bladder, because the weight of urine in a partially full bladder can cause it to fall forward into the abdomen and fill further with air through an open catheter. This is not a major problem when it occurs, but it can make aspiration of a sample more difficult because air may have to be evacuated before urine is recovered. Another potential problem in male horses is that the catheter tip can be advanced into one of the seminal vesicle duct openings on the colliculus seminalis on the dorsal aspect of the urethra immediately caudal to the urethral sphincter. If resistance to catheter passage persists after it has been passed over the ischial arch, this problem can be detected by transrectal palpation of the catheter tip going to either side of the bladder neck. When this occurs, the catheter should be retracted to the intrapelvic portion of the urethra and, with the hand in the rectum, fingers can be placed on either side of the catheter to assist in directing it through the urethral sphincter into the bladder. Mares do not have a urethral diverticulum to complicate catheter passage but it is sometimes challenging to find the urethral sphincter on the pelvic floor at the level of the vestibulovaginal orifice. The most common mistake is to insert the gloved hand too far and enter the vagina, passing over the urethral sphincter. In most mares the urethral sphincter can be found with the hand partly inserted as a cone into the vestibule to the level of the knuckles, followed by moving the index or middle finger back and forth under the urethral papilla in an attempt to insert a finger through the sphincter into the bladder. When a finger has been successfully passed into the bladder, the sterile catheter is inserted under the palm and guided along the finger that is already in the bladder. As the catheter is passed, the finger is withdrawn from the bladder and a sample of urine is then aspirated. Mares generally do not need to be sedated for this procedure, but some will be stimulated to void during catheterization; consequently, an assistant should be ready with a specimen cup to collect a sample if the mare urinates. When collecting urine, it is always advisable to fill two urine specimen cups: one can be used for urinalysis and the other submitted (or temporarily saved) for bacterial culture, when indicated.

In mares, urine can also be collected from each side of the upper urinary tract via ureteral catheterization.[27] Indications for this procedure are rare but include a suspected unilateral kidney problem including pyelonephritis or neoplasia. The vulva is prepared in a similar manner but a stiffer, 8 or 10 French polypropylene catheter with a rounded tip and side opening is used. The ureteral orifices can be palpated at the 2 and 10 o'clock positions in the bladder neck. To pass a catheter into the ureter, the index and middle fingers are inserted through the urethra into the bladder with the catheter tip between the fingers (sedation and/or topical anesthesia of the urethral and bladder mucosa may be necessary if the mare reacts to stretching of the urethra). Use of surgical gloves facilitates palpation of the ureteral orifices, and the catheter is advanced with one finger on either side of the ureteral orifice. When the tip has been inserted, it can be helpful to flex the finger tips caudad and grasp the catheter between the fingers and then extend the fingers to guide the catheter 5 to 10 cm farther into the distal ureter. Because urine is normally passed from the ureter into the bladder in "squirts" every 30 to 60 seconds, patience is required because it may take 3 to 5 minutes to collect an adequate volume (5 to 10 mL) of urine from the ureter. After the sample has been collected from one ureter, a new sterile catheter is passed into the bladder to collect urine from the other ureter. In male horses, the only way to collect urine from each ureter is via endoscopic visualization of the ureteral orifices and passage of sterile polypropylene tubing through the biopsy channel of the endoscope into each ureter. When ureteral catheterization is pursued in an attempt to document unilateral pyelonephritis, urine samples collected from both ureters, as well as a sample initially collected from the bladder, should all be submitted for bacterial culture.

GROSS APPEARANCE

Color, clarity, odor, and viscosity should be evaluated at the time of urine collection. Normal equine urine is pale yellow to deep tan and is often turbid as a result of large amounts of calcium carbonate crystals and mucus.[28] It is not uncommon for urine appearance to change during urination or collection with a catheter, especially toward the end of micturition or collection, when more crystals that have gravitated to the bottom of the bladder tend to be passed, causing urine to appear almost milky white.

ASSESSMENT OF URINE TONICITY

Urine specific gravity is a measure of the number of particles in urine and is a useful estimate of urine tonicity. Although determination of specific gravity with a refractometer is quick and easy (reagent strips should not be used to measure specific gravity in horses),[28] it is important to recognize that urine tonicity is most accurately determined by measurement of urine osmolality (U_{osm}) because larger molecules in urine, such as

glucose or proteins, can lead to overestimation of urine tonicity when assessed by specific gravity. Clinically, this is only a problem in patients with diabetes mellitus or heavy proteinuria. Unfortunately, most refractometers have an upper end of the specific gravity scale of 1.035, making it necessary to estimate specific gravity of more-concentrated samples by extrapolation. As an alternative, refractometers with a wider specific gravity scale (1 to 1.06) are available and may be worthwhile to purchase for equine hospitals.

Urine specific gravity or U_{osm} is used to separate urine tonicity into three categories: (1) urine that is more dilute than serum (hyposthenuria or specific gravity < 1.008 and U_{osm} < 260 mOsm/kg); (2) urine and serum of similar osmolality (isosthenuria or specific gravity of 1.008 to 1.014 and U_{osm} of 260 to 300 mOsm/kg); and (3) urine that is more concentrated than serum (specific gravity > 1.014 and U_{osm} > 300 mOsm/kg).[29] Urine of most normal horses consuming dry forage is usually concentrated (two to four times the tonicity of serum) with specific gravity of 1.025 to 1.04 and a U_{osm} of 600 to 1200 mOsm/kg), whereas horses at pasture may have more dilute urine because of the high water content of grass. When deprived of water for 24 to 72 hours, horses with normal renal function produce urine with a specific gravity > 1.045 and an U_{osm} > 1500 mOsm/kg.[30] In contrast, neonatal foals typically have hyposthenuric urine because their diet consists largely of milk.[31] Because the volume of fluid intake as milk by foals up to 60 days of age is nearly fivefold the fluid intake of an adult horse (on an mL/kg basis), healthy foals are polyuric and their urine appears clear with little yellow color. Another consequence of this high fluid intake and associated diuresis is that BUN and Cr values in foals may be near or below the lower limit of the adult reference ranges. Further, although this fluid intake decreases a foal's ability to generate an osmotic gradient in the medullary interstitium, foals can still produce urine with a specific gravity > 1.03 when dehydrated.

Urine tonicity can be used to differentiate prerenal failure from intrinsic renal failure. With prerenal failure, maintenance of urinary concentrating ability is demonstrated by a specific gravity > 1.02 and a U_{osm} > 500 mOsm/kg, and values are often much higher. In contrast, with intrinsic renal failure urinary concentrating ability is lost: specific gravity and U_{osm} are typically <1.02 and < 500 mOsm/kg, respectively, in the face of dehydration.[32] Assessment of urine tonicity can be problematic, however, because it is only valid when performed on urine collected before initiating fluid therapy or administering medications (e.g., α_2-receptor agonists or furosemide) that can affect urine flow and tonicity.[33]

REAGENT STRIP ANALYSIS

The pH of equine urine is usually alkaline (8 to 9).[34] Strenuous exercise or bacteriuria can result in acidic pH, and bacteriuria can also impart a strong ammonia odor to urine because of breakdown of urea by bacteria with urease activity. Feeding large amounts of concentrate generally decreases urine pH toward the neutral value. Similarly, the more dilute the urine sample is, the closer the pH is to 7. Hyposthenuric urine produced by foals typically has a neutral pH and is relatively free of crystalline material. Occasionally, aciduria is detected in a dehydrated or anorectic horse. Although aciduria can be found with metabolic acidosis, many equine patients have hypochloremic metabolic alkalosis accompanied by aciduria. The mechanism for aciduria is likely similar to that described in ruminants with abomasal outflow obstruction.[35] Briefly, after all chloride has been reabsorbed from the glomerular filtrate, further electroneutral sodium reabsorption occurs by exchange with (excretion of) potassium or hydrogen ions. Thus, aciduria is most likely to occur with concomitant hypokalemia or whole-body potassium depletion that develops in horses that have been off feed for several days.

Commercially available urine reagent strips can yield false-positive results for protein when alkaline samples are tested, especially when urine is concentrated.[36] Therefore, proteinuria is better assessed by specific quantification with a colorimetric assay (e.g., assays that are routinely used on cerebrospinal fluid). Urinary protein concentration is less than 100 mg/dL in the majority of normal horses and comparison of the quantitative protein result (mg/dL) to urine Cr concentration (mg/dL) in the form of a urine protein : urine creatinine ratio (UP : UCr) is the recommended test to document proteinuria.[37] Normal horses typically have a UP : UCr < 0.5 whereas significant proteinuria consequent to glomerulonephritis usually results in a UP : UCr > 2. Because proteinuria can also accompany bacteriuria, pyuria, and hematuria or may be found transiently following exercise, an abnormal UP : UCr result must be interpreted in light of these potential confounding factors. Neonatal foals that have acquired good passive transfer of maternal colostral antibodies normally have moderate proteinuria from 24 to 72 hours of life because of excretion of small-molecular-weight proteins in urine.

Normal equine urine should not contain glucose. Although the renal threshold for glucose has not been thoroughly evaluated in horses, early work indicated that it may be lower (160 to 180 mg/dL) than that of small animals and humans.[38] Thus, glucosuria can accompany hyperglycemia associated with the causes described earlier or with administration of dextrose-containing fluids or parenteral nutrition products. In addition, glucosuria may accompany sedation with α_2-agonists or exogenous corticosteroid administration.[39] When glucosuria is detected in the absence of hyperglycemia, primary proximal tubule dysfunction should be suspected. Glucosuria has more often been detected in horses with AKI than in those with CKD.

Unlike in ruminants, ketones are rarely detected in equine urine, even in advanced catabolic states or with diabetes mellitus. A positive result for blood on a urine reagent strip can reflect the presence of hemoglobin, myoglobin, or intact RBCs in the urine sample. Evaluation of serum for hemolysis or elevated CK and AST activities and of urine sediment for RBCs can be rewarding in differentiating between these pigments. Bilirubinuria is occasionally detected on reagent strip analysis of equine urine. Bilirubinuria is associated with intravascular hemolysis, hepatic necrosis, and obstructive hepatopathies. Hepatic disease is further supported by elevated serum bilirubin concentration and increased hepatic enzyme activities.

SEDIMENT EXAMINATION

Sediment examination is an underused diagnostic technique for evaluating urinary tract disorders in horses.[28] Unfortunately, a major limitation is that sediment should be examined within 30 to 60 minutes after collection. To perform sediment examination, 10 mL of fresh urine should be centrifuged (usually in a conical plastic tube) at 1000 rpm for 3 to 5 minutes. The supernatant urine is discarded, and the pellet is resuspended in the few drops of urine remaining in the tube. A drop of sediment is transferred to a glass slide, and a coverslip is applied.

The sediment is first examined at low power to evaluate for casts, and subsequently high-power examination is used to quantify erythrocytes, leukocytes, and epithelial cells and to determine whether or not bacteria are present. Casts are molds of Tamm-Horsfall glycoprotein and cells that form in tubule lumens and subsequently pass into the bladder. They are rare in normal equine urine but are found with acute tubular injury. Casts are relatively unstable in alkaline urine; accordingly, to ensure accurate assessment, urine sediment should be evaluated as soon as possible after collection. Fewer than five RBCs should be seen per high-power field (hpf) in an atraumatically collected urine sample. Pyuria (more than 5 WBCs per hpf) is most often associated with infectious or inflammatory disorders, and normal equine urine should have few bacteria. The absence of bacteria on sediment examination does not rule out their presence, however, and quantitative bacterial culture of urine collected by catheterization should be performed when urinary tract infection is suspected. Equine urine is rich in crystals. The majority of these are calcium carbonate crystals of variable size, but calcium phosphate crystals and occasional calcium oxalate crystals can also be seen in normal equine urine.[40] Addition of a few drops of a 10% acetic acid solution may be necessary to dissolve crystals for more-complete assessment of urine sediment.

URINE ELECTROLYTE CONCENTRATIONS AND CLEARANCES

Urinary electrolyte excretion, reflecting tubular function, can be assessed by measuring urine electrolyte concentrations or can be expressed quantitatively as excretion rates (mEq/min). Horses on an all-forage diet without electrolyte supplementation consume minimal sodium and chloride but large amounts of potassium. As a consequence, their kidneys evolved to conserve more than 99% of filtered sodium and chloride ions. In contrast, potassium ions are not well conserved except during periods of whole-body potassium depletion (i.e., when they are off feed). Normal equine urine typically contains less than 20 mEq/L of sodium, less than 50 mEq/L of chloride, and 200 to 400 mEq/L of potassium, depending on urine tonicity.[41]

Determination of electrolyte excretion rates requires volumetric collection of urine over time, an impractical measurement in most clinical settings. An alternative is to measure fractional electrolyte clearances, comparing the clearance of a specific electrolyte to that of creatinine.[29] A substance that is filtered across the glomerulus but neither reabsorbed nor secreted by renal tubules has a clearance rate similar to that of creatinine and a fractional clearance of 1 (or 100%). In contrast, a substance that is largely reabsorbed by renal tubules after filtration (sodium or chloride) has a lower clearance value than that of creatinine and a fractional clearance less than 1. The fractional clearance of a substance (Cl_A) is calculated by dividing Cl_A by creatinine clearance (Cl_{Cr}):

$$Cl_A/Cl_{Cr} = \frac{\{(\text{urine [A]}/\text{plasma [A]}) \times \text{urine flow}\}}{\{\text{urine [Cr]}/\text{plasma [Cr]}) \times \text{urine flow}\}}$$

which, by rearrangement (cancelling out the urine flow factor) and expressing as a percentage becomes:

$$Cl_A/Cl_{Cr} = \{(\text{plasma [Cr]}/\text{urine [Cr]}) \times (\text{urine [A]}/\text{plasma [A]})\} \times 100\%$$

Although fractional clearance is the appropriate term for this calculation, the term *fractional excretion* is also commonly used. Because urine flow is cancelled out in the equation, only a spot urine sample (not a timed urine collection) needs to be collected. To determine fractional electrolyte clearances, electrolyte and Cr concentrations must be measured in blood and urine samples collected within a few hours of each other. Again, because kidneys conserve more than 99% of filtered sodium and chloride ions, normal fractional clearance values are less than 0.8% for these electrolytes. In contrast, fractional clearance values for potassium are considerably higher (15% to 65%) depending on feed intake.[29] Increases in urine concentration and fractional clearance values of sodium and chloride can be early indicators of renal tubule damage with AKI.[32,42] However, results of these tests must be interpreted in light of diet and treatment because adding salt to the diet or administering polyionic enteral or intravenous (IV) fluids can also increase these values.[33] With CKD, measurement of urine electrolyte concentrations and fractional clearances are of limited diagnostic value because compensatory tubular function with CKD is often accompanied by nearly normal electrolyte excretion.

URINARY DIAGNOSTIC INDICES

In addition to urinary tonicity, diagnostic indices that may also be useful for differentiating prerenal failure from intrinsic renal failure in horses include urine : serum ratios of osmolality, urea nitrogen, and Cr and fractional sodium clearance (Table 63-4). For example, urine : serum Cr ratios in excess of 50 : 1 (reflecting concentrated urine) and fractional sodium clearance values below 0.8% (indicating normal tubular sodium reabsorption) would be expected in horses with prerenal failure, whereas ratios less than 37 : 1 and clearance values greater than 0.8%

TABLE 63-4. Diagnostic Indices that May Be Useful for Differentiating Prerenal Failure from Intrinsic Renal Failure in Horses

Diagnostic Index	Normal Horses	Prerenal Failure	Intrinsic Renal Failure
Urine osmolality (mOsm/kg)	727-1456	458-961	226-495
Urine osmolality : serum osmolality	2.5-5.2	1.7-3.4	0.8-1.7
Urine urea nitrogen : serum urea nitrogen	34-101	15-44	2-14
Urine creatinine : serum creatinine	2-344	52-242	3-37
Urine sodium concentration (mEq/L)	variable*	<20	>20
Fractional sodium clearance (%)	0.01-0.7	0.02-0.5	0.80-10.1

*Urine sodium concentration will vary with the amount of concentrate or supplemental salt added to the diet but is generally less than 20 mEq/L in horses on an all-forage diet.

Modified from Grossman BS, Brobst DF, Kramer JW, et al: Urinary indices for differentiation of prerenal azotemia and renal azotemia in horses. J Am Vet Med Assoc 180:284, 1982, and Seanor JW, Byars TD, Boutcher JK: Renal disease associated with colic in horses. Mod Vet Pract 65:A26, 1984.

were reported in a group of horses determined to have primary renal disease.[32] Although these diagnostic indices can be helpful, the data in Table 63-4 illustrate that renal hypoperfusion is accompanied by a progressive loss of concentrating ability, because ranges for these ratios tend to be lower for horses with prerenal failure than for clinically normal horses. As a consequence, these data also support the concept that progression from prerenal failure to intrinsic renal failure is along a continuum, associated with decompensation of the intrarenal responses to hypoperfusion.[16]

In patients at risk for developing AKI, including horses with serious gastrointestinal disorders and those receiving nephrotoxic medications, additional diagnostic indices such as serial assessment of specific gravity or U_{osm}, reagent strip analysis, sediment examination, urine sodium concentration, and fractional sodium clearance may be useful in identifying significant changes in renal function before the onset of azotemia. Unfortunately, monitoring these parameters is frequently complicated by use of IV fluid support, because a goal of IV fluids is to increase production of less-concentrated urine. Further, IV replacement solutions have a sodium concentration near that of serum (135 to 140 mEq/L) and administration of large volumes (more than 20 L) of IV fluids is associated with a large sodium load that must be eliminated in urine, resulting in urine sodium concentrations that may exceed 50 mEq/L and FCl_{Na} values greater than 1%. In contrast, IV fluid therapy has less effect on reagent strip analysis and changes in urine sediment. Thus, *a practical screening test for documenting tubular damage* is repeated reagent strip analysis for pigmenturia (likely hematuria) and glucosuria. With CKD, the ability to produce either concentrated or dilute urine is lost: affected horses manifest isosthenuria. Administration of IV fluids in an attempt to "diurese" horses with CKD results in a variable increase in urine output, yet specific gravity and U_{osm} remain in the isosthenuric range. The less residual renal function present, the more likely and rapidly IV fluid administration will produce an increase in central venous pressure and, ultimately, edema.

Peritoneal Fluid Analysis

Postrenal azotemia resulting from obstruction or disruption of the urinary tract is usually suspected on the basis of clinical signs, including dysuria and/or renal colic. With bladder rupture, however, some affected foals and adult horses continue to void urine, although progressive abdominal distention usually accompanies development of uroperitoneum. Although the diagnosis is now commonly made via transabdominal ultrasonography, uroperitoneum is best confirmed by measuring a twofold or greater value for peritoneal Cr in comparison to serum Cr.[29]

Imaging Techniques

Numerous imaging techniques are available to examine the urinary tract of the horse. The pros and cons of the various modalities have been well reviewed,[43] and the most rewarding and cost-effective techniques include ultrasonography and endoscopy of the urinary tract. In certain cases, nuclear scintigraphy is a valuable tool because it can provide information on individual kidney function. Textbooks are available with numerous images that provide in-depth review of each of these modalities for investigation of the equine urinary tract,[44-46] so they are only discussed in brief in this chapter. Computed tomography (CT) and magnetic resonance imaging (MRI) are also emerging as useful imaging tools in patients that are small enough (generally less than 200 kg) to fit within the imaging device.

Radiography

Radiography is of limited use to evaluate the urinary tract disease of adult horses because diagnostic radiographs can only be obtained in small foals or Miniature Horses. In smaller patients, intravenous excretory urography can be used to document a nonfunctional kidney or the course of ectopic ureters.[47] However, the procedure may require general anesthesia, and renal elimination of contrast is sometimes difficult to see, leading to inconclusive results.[48] Another approach involves injecting the contrast agent directly into the renal pelvis using a spinal needle under ultrasonographic guidance (pyelography).[49,50] The contrast agent is generally easier to visualize with this approach, and systemic adverse effects of contrast administration are largely eliminated.

Retrograde contrast radiographic studies have also been used in foals with suspected ruptured bladder, ectopic ureter, or urorectal fistula. Radiographic contrast studies can also help to identify strictures or masses in the urethra or bladder, but endoscopy is generally more useful for these problems.

Ultrasonography

Ultrasonographic examination of the urinary tract can be performed transabdominally (kidneys) or transrectally (lower urinary tract).[45,51-55] The right kidney is triangular or horseshoe-shaped and is best imaged transabdominally via the dorsolateral extent of the last two or three intercostal spaces. The left kidney is a bean- to U-shaped organ that lies deep to the spleen. It can be imaged via the last two intercostal spaces or via the paralumbar fossa. Because the left kidney is deeper than the right kidney, it can be difficult to image completely and is best examined with a 2.5- or 3-mHz curvilinear transducer. Occasionally, one or both kidneys cannot be imaged because of gas-filled bowel between the kidney and abdominal wall. Reexamination at a later time is generally required for successful imaging in such cases. The size and shape of the kidneys, intrarenal architecture, and echogenicity of the parenchyma should be assessed systematically. This includes imaging the kidneys in dorsal, sagittal, transverse, and transverse-oblique anatomic planes.[53] With AKI, the kidneys may be normal or enlarged (Figure 63-5, *A*), and abnormalities of parenchymal detail are often not detected. When present, abnormal findings may include perirenal (subcapsular) edema, widening, and subtly increased echogenicity of the renal cortex (see Figure 63-5, *B*). With CKD, especially end-stage renal disease, the kidneys are usually small, have increased echogenicity (the normal left kidney is similar to the spleen), and may have nephroliths or cystic cavities (Figures 63-6 and 63-7). Calculi are often in the area of the renal pelvis and can be differentiated from the normally echogenic renal pelvis because they generally cast an acoustic shadow. In an occasional patient in which the clinical picture is most consistent with AKI, ultrasonography may reveal nephroliths and increased echogenicity in one or both kidneys. These findings support previously unrecognized CKD, with recent exacerbation (so-called "acute on chronic" renal injury), and carry a more guarded

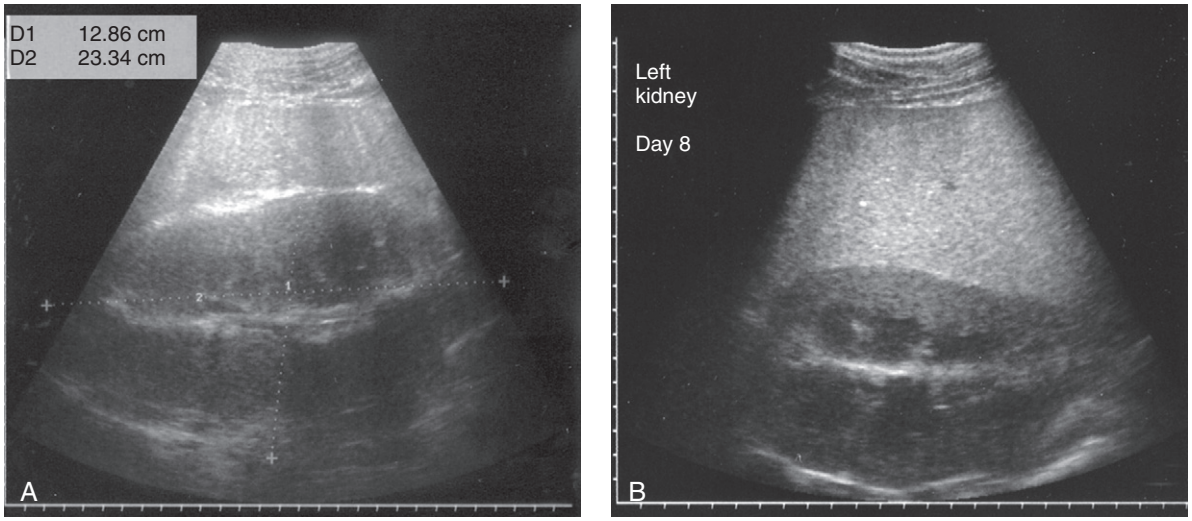

Figure 63-5. Transabdominal ultrasonographic images of the left kidney (deep to spleen) of two horses with acute kidney injury resulting in acute renal failure. **A,** Enlarged left kidney (23.3 cm in length). **B,** Renal cortex is more echogenic than normal.

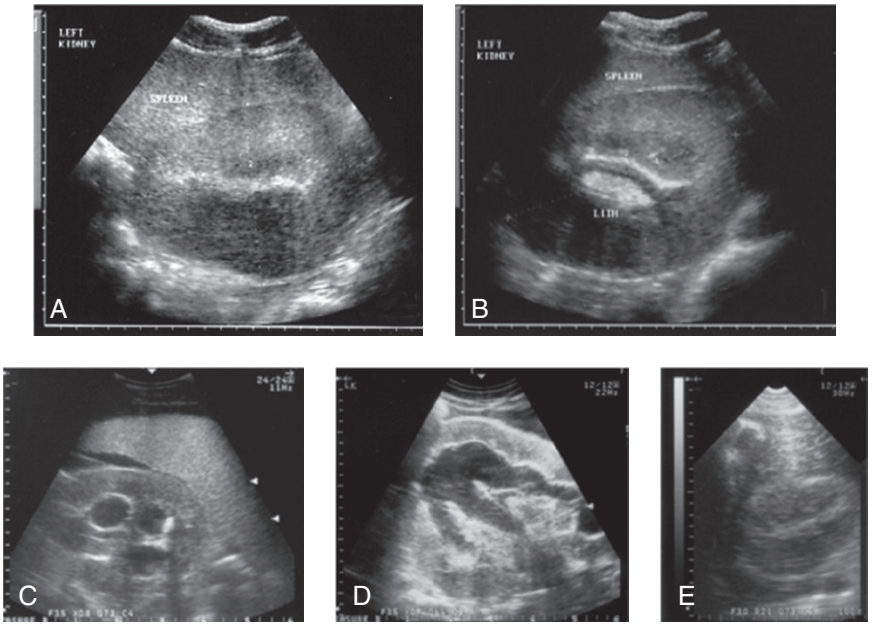

Figure 63-6. Transabdominal ultrasonographic images of the left kidney of two horses with chronic kidney disease. **A,** Left kidney of a yearling with chronic interstitial nephritis that developed 11 months following treatment with an aminoglycoside antibiotic and flunixin meglumine for a leg wound. Note the generalized increase in echogenicity of the renal parenchyma in comparison to the spleen. **B,** Left kidney of the same yearling with the probe directed in a different plane, revealing a large nephrolith adjacent to the renal pelvis. **C,** Left kidney of a stallion with an obstructive ureterolith causing hydronephrosis; note the small nephrolith in the center of the image producing an acoustic shadow. **D,** Left kidney of the same stallion imaged in a plane rotated 90o revealing hydronephrosis consequent to obstructive disease. **E,** Left kidney of the same stallion following relief of ureteral obstruction by electrohydraulic lithotripsy. Note that the kidney is small and the renal parenchyma has a diffuse increase in echogenicity because of renal fibrosis. (From Schott HC: Chronic renal failure in horses. Vet Clin North Am Equine Pract 23:593, 2007.)

prognosis than AKI with normal-appearing kidneys on ultrasonographic imaging.

Imaging of the bladder, urethra, and ureters is best performed transrectally using a 5- to 7.5-mHz linear array transducer commonly used for evaluating the reproductive tract or a 6- to 10-mHz microconvex linear-array transducer. When imaging the bladder, it is important to remember that equine urine, rich in crystals and mucus, is an inhomogeneous, echogenic fluid that can be made to swirl with manipulation of the bladder. Presence of a cystic calculus can typically be confirmed, because calculi have a highly echogenic surface and produce an acoustic shadow; however, ultrasonographic imaging is rarely indicated if the cystolith is suspected on rectal palpation. In contrast, ultrasonography can be a useful tool to evaluate size and echogenicity (homogeneous or heterogeneous) of soft tissue bladder masses. Normal values for bladder wall thickness, distal ureteral, and urethral diameter have been described for normal horses of both sexes.[55] Transrectal ultrasonography can be used

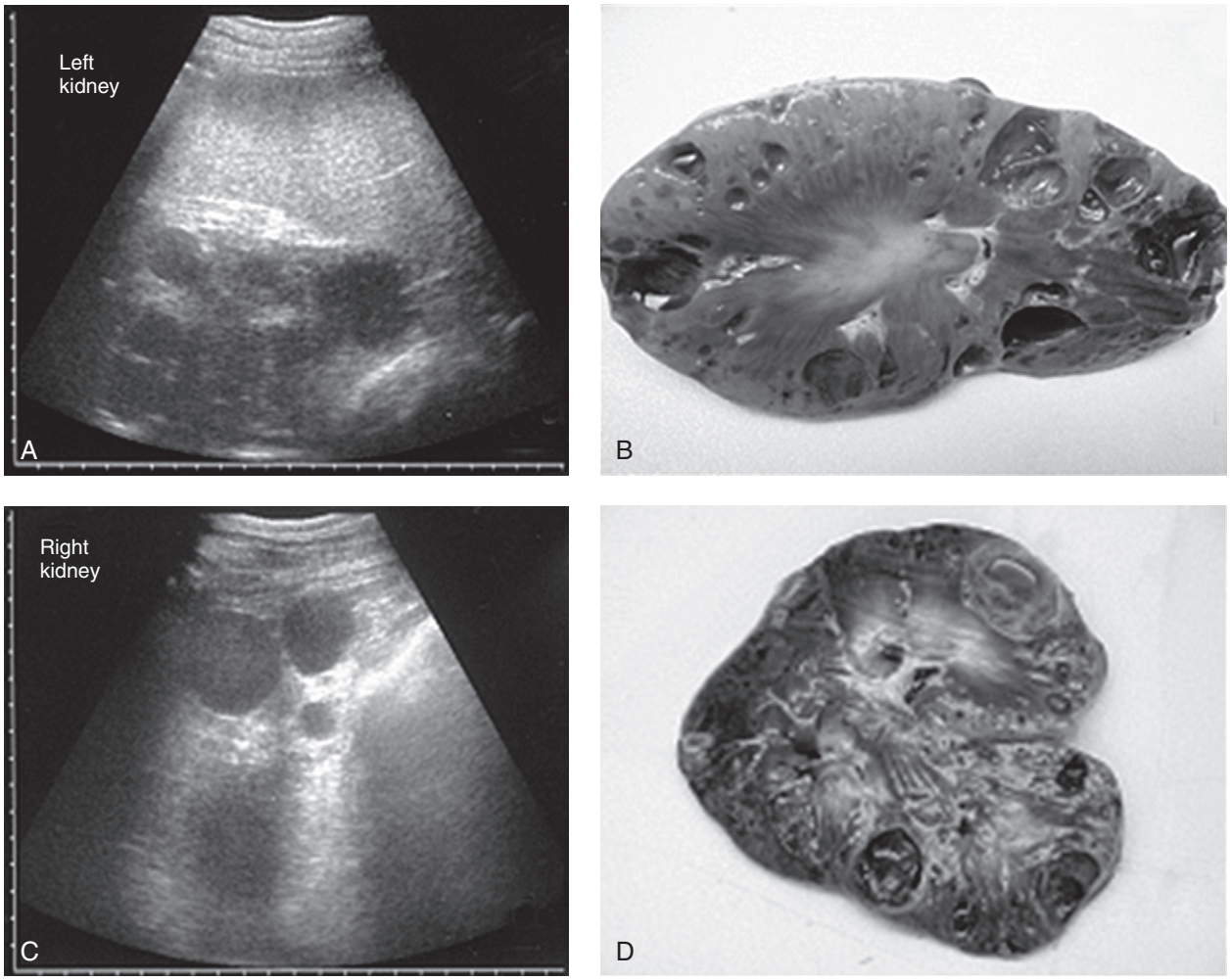

Figure 63-7. Ultrasonographic images of the left **(A)** and right **(C)** kidneys and cross-sectional gross pathology of the left **(B)** and right **(D)** kidneys of a 19-year-old Arabian mare with chronic renal failure consequent to polycystic kidney disease. (From Schott HC: Chronic renal failure in horses. Vet Clin North Am Equine Pract 23:593, 2007.)

to image urine passage from the ureters into the bladder and may reveal disruption or distention of a ureter.[56,57]

Endoscopy

Endoscopic examination of the urinary tract is an extremely useful diagnostic aid when the complaint is abnormal urination.[44,58,59] A flexible endoscope with an outer diameter of 12 mm or less and a minimum length of 1 m is adequate for examinating the urethra and bladder of an adult horse of either sex. As for bladder catheterization, lower urinary tract endoscopy is a clean but not completely sterile procedure; nevertheless, the endoscope should be sterilized before use. Tranquilization of the patient is recommended, the rectum should be emptied of feces, and the distal end of the penis or the vulva should be cleansed thoroughly. The bladder should be emptied by catheterization prior to passing the endoscope, and a sample of urine should be retained, if needed. The endoscope is then passed in the same manner as the catheter, using the air control intermittently to inflate the urethra or bladder. Normal distal urethral mucosa in males is pale pink with longitudinal folds. When dilated with air, the mucosa flattens and a prominent submucosal vascular pattern becomes more apparent as the endoscope is advanced up the urethra to the ischial arch. Passage of a bladder catheter before endoscopy can produce mild irritation of the urethral mucosa that may be seen as a reddish line along the urethra (it should not be mistaken as an abnormality). As the endoscope passes over the ischial arch, the urethra begins to widen into the ampullar (pelvic) portion, and paired rows of bulbourethral gland duct openings are seen along the dorsal aspect. In horses with a complaint of hematuria at the end of urination, consistent with a urethral rent, the caudal aspect of the urethra should be closely examined at the level of the ischial arch for fistulous tracts communicating with the corpus spongiosum penis.[3,4] Advancing the endoscope a few more centimeters and distending the pelvic urethra with air allows visualization of the colliculus seminalis protruding from the dorsal aspect of the urethra just before the urethral sphincter. The joint openings of the ductus deferens and seminal vesicle ducts on the colliculus seminalis should be examined closely, because this can be a site of postejaculation hemorrhage in stallions. Subsequent passage of the endoscope through the urethral sphincter and air distention allows evaluation of the bladder for calculi, inflammation, and masses. Viewing the ureteral openings in the dorsal aspect of the trigone can help determine if the source of hematuria or pyuria is

originating from one or both sides of the upper urinary tract. A small volume of urine should pass from each ureteral opening approximately once every 30 to 60 seconds (more frequent with sedation with an α_2-agonist). Ureteral catheterization to obtain urine samples from each kidney can be performed by passing sterile polyethylene tubing via the biopsy channel of the endoscope. Additionally, biopsy of masses in the bladder or urethra can be performed.

Smaller-diameter endoscopic equipment (less than 6 mm outer diameter) has allowed examination of the distal ureter, using a guide wire passed through the biopsy channel as a stylet to enter the ureter. In addition to allowing examination of the ureter, endoscopes of sufficient length (100 cm or longer) have also allowed advancement to visualize the renal pelvis in mares or via a perineal urethrotomy in male horses (Figure 63-8). In an occasional horse with upper tract disease or chronic sabulous urolithiasis, one or both ureteral orifices are damaged to the point that the normal valvelike openings have become permanently open, allowing a larger-diameter endoscope to be advanced up the ureter to allow visualization of nephroliths in the renal pelvis (Figure 63-9).

Nuclear Scintigraphy

Nuclear scintigraphy is an additional modality that can be used to obtain information about renal anatomy and function.[46] In fact, scintigraphy is routinely used for quantitative assessment of GFR in small animals.[60] Several radiopharmaceuticals can be labeled with 99-metastable technetium (^{99m}Tc), depending on the type of scintigraphic examination being pursued. Further,

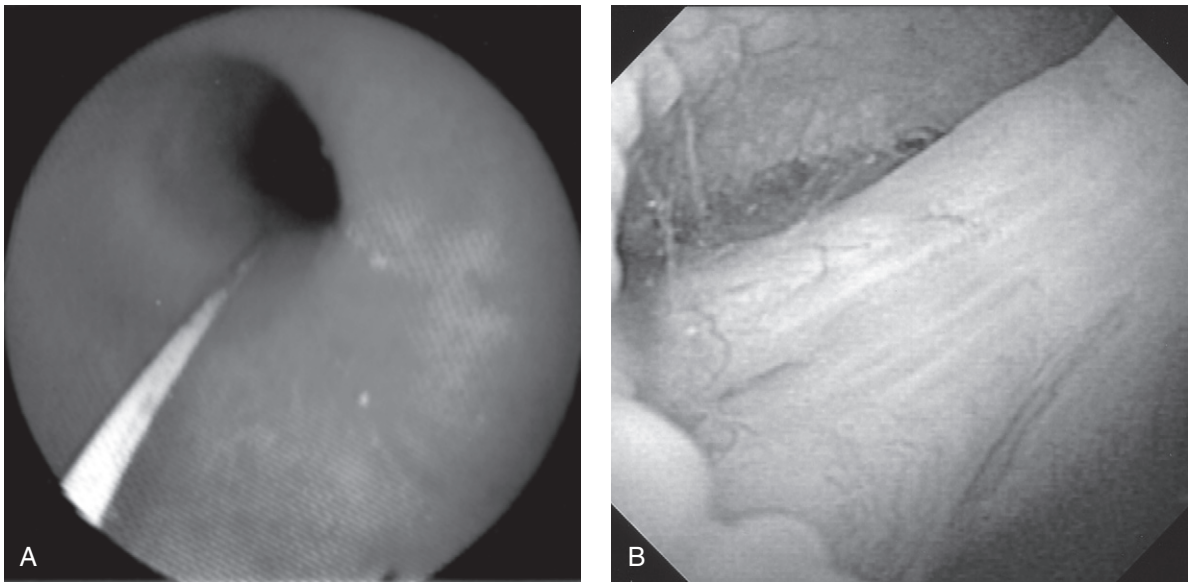

Figure 63-8. Endoscopic images of a normal ureter with a guide wire in place **(A)** and of a normal renal pelvis of a horse **(B).**

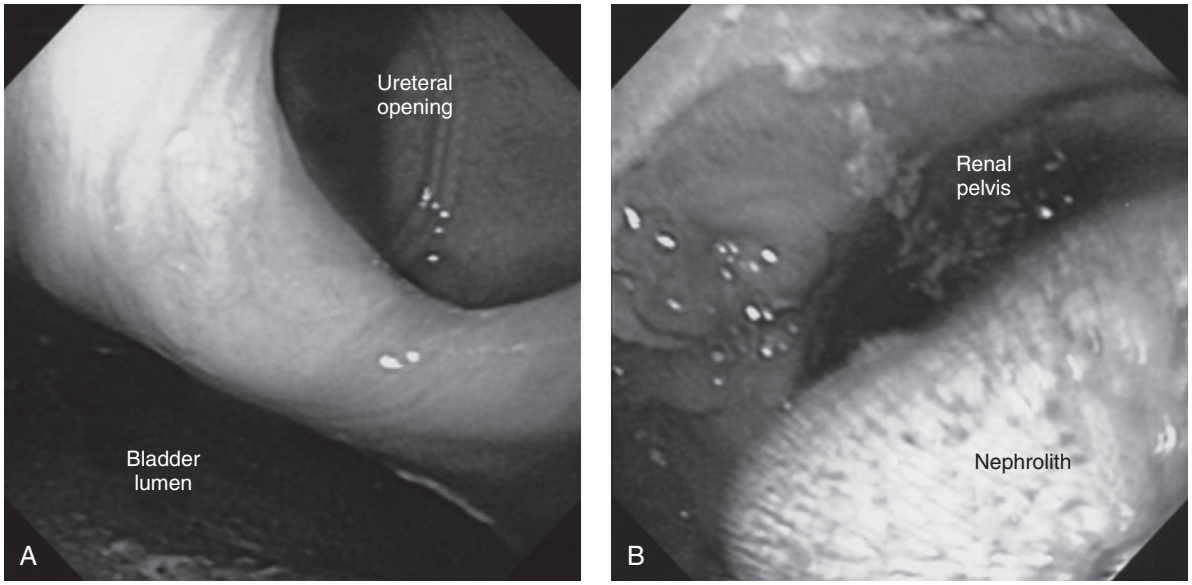

Figure 63-9. Endoscopic images of an abnormal ureteral opening **(A)** and a nephrolith in the renal pelvis **(B)** of a stallion with chronic kidney disease.

labeled radiopharmaceuticals can be used either to generate images or to measure plasma disappearance of radioactivity, to estimate RBF and GFR. In horses, an early study compared measurement of GFR by classic inulin clearance to scintigraphy (using ^{99m}Tc tagged to diethylenetriaminopentaacetic acid [DTPA], similar to inulin in that it is neither secreted nor reabsorbed after filtration). Both plasma disappearance of ^{99m}Tc-DTPA in blood samples collected over time and sequential digital gamma camera images of the kidneys to determine fractional accumulation of the total dose administered were assessed as measures of GFR.[61] Greater variability was found with the gamma camera images, in comparison to GFR values measured by plasma disappearance of inulin or ^{99m}Tc-DTPA, possibly because of the different depths of the two equine kidneys. Renal scintigraphy with gamma camera imaging has also been performed with ^{99m}Tc tagged to glucoheptanate (GH, taken up by the proximal tubule epithelial cells to provide further anatomic detail), and mercaptoacetyltriglycine (MAG_3, nearly completely eliminated during first pass through the kidneys by filtration and tubular secretion) to successfully evaluate renal function in horses with upper urinary tract problems.[48,56,62] With appropriate case selection, renal scintigraphy can provide qualitative information about renal function and is the only method currently available for assessing split renal function (assessing individual kidney function via gamma camera imaging) in horses (Figure 63-10).

Computed Tomography and Magnetic Resonance Imaging

Computed tomography (CT) and magnetic resonance imaging (MRI) provide the most detailed images of the urinary tract of all the available imaging modalities. Unfortunately, both imaging modalities are limited by the patient's size (generally less than 200 kg), the need for general anesthesia, and expense. Nevertheless, the modalities have been used in foals and Miniature Horses and, when coupled with use of appropriate contrast agents, provide the surgeon with excellent anatomical detail of the urinary tract problem (Figure 63-11).[63,64]

SURGERY

J. Brett Woodie

Instruments

The instrumentation that is required varies depending on the procedure that is being performed. Therefore it is beneficial to

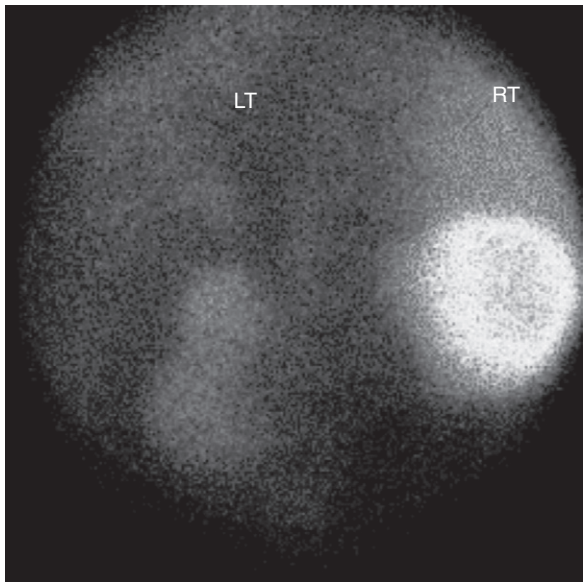

Figure 63-10. Nuclear scintigraphic dorsal image (cranial at *top* and caudal at *bottom*) of the kidneys of a horse with chronic hematuria arising from the left kidney. The study revealed considerably less radiopharmaceutical uptake and elimination by the left kidney and provided support that nephrectomy of the left kidney would not result in a substantial loss of remaining renal function.

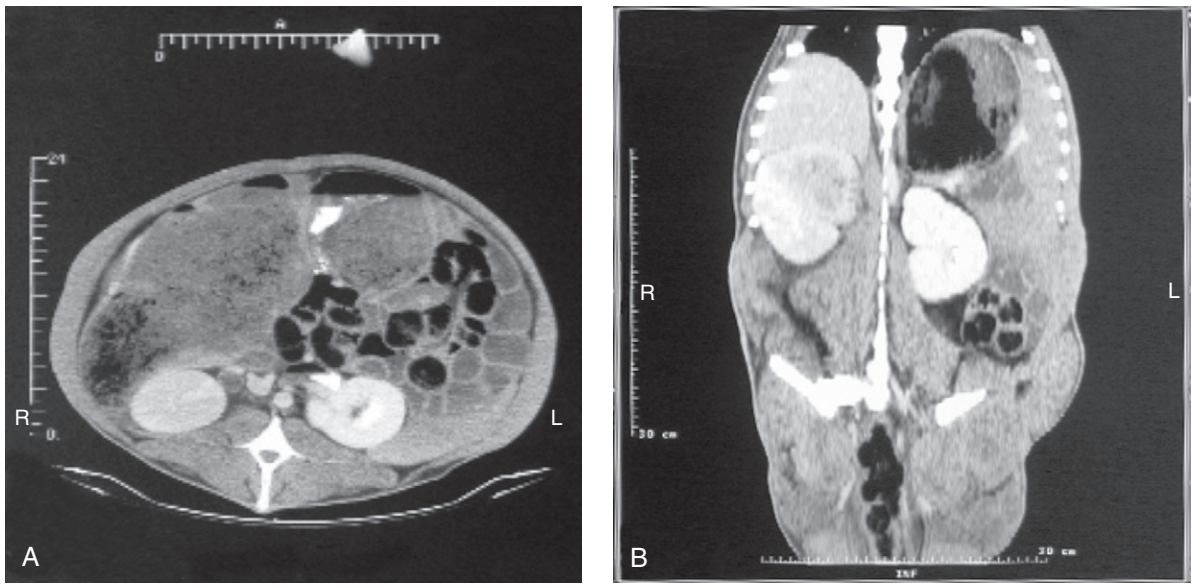

Figure 63-11. Contrast-enhanced computed tomographic images of the kidneys (excretory urography) of a normal Miniature Horse. **A,** The transverse plane image shows nearly homogeneous uptake of the contrast agent in both kidneys. **B,** The reconstructed dorsal plane image provides even greater detail of the intrarenal distribution of the contrast agent within the right kidney *(left side of image).*

have a broad range of long-handled instruments as well as standard-sized instruments. Grasping forceps with delicate teeth and needle holders with fine jaws are necessary to handle the fragile tissues and small-diameter suture needles needed for surgery of the urinary tract.[65] Broad-bladed self-retaining retractors, such as Finnechetto or Balfour retractors, are often essential for effective surgical access to the urinary tract, especially in the adult horse. For electrosurgery, the use of fine-needle electrodes and scalpels is appropriate. Anastomotic procedures and intraoperative stenting of the urethra or ureters can be facilitated by using sterile flexible rubber urethral catheters or silicone or polyethylene cannulas. It has been reported that stents can promote stricture formation in anastomosed ureters. Therefore, if stents are used they should be removed as soon as possible, preferably within 5 to 7 days after surgery.[66,67] Magnifying loupes or an operating microscope and adequate lighting are useful for repairing ureteral defects. Suction with an assortment of suction tips will be required.

Suture Material

The choice of suture material depends on a variety of factors, such as the strength of the tissue, the rate at which the tissue regains strength, the rate at which the suture material loses strength, and the interaction between the tissue and the suture material. Suture materials in a variety of sizes and strengths are required when performing urinary tract surgery. Tissues such as the neonatal bladder are often friable, whereas a bladder that contains a cystic calculus may be thickened. The bladder is considered one of the weakest tissues in the body.[68] Despite this fact, the urinary bladder and ureters have a high regenerative capacity and heal readily.[66,69] These tissues regain nearly 100% of their normal strength within 14 to 21 days.[69] For these reasons, absorbable suture material is an appropriate choice, and the use of nonabsorbable suture material is not indicated.[70]

In fact, nonabsorbable sutures are contraindicated for closure of any structure of the urinary tract. The use of nonabsorbable suture material can serve as a nidus for formation of urinary concretions.[65] As a technical point of urinary tract surgery, no suture material of any type should be placed in such a fashion that it penetrates the urinary epithelium and is exposed to urine.[67,68] Hydrolysis is the mechanism by which synthetic absorbable sutures are absorbed. Hydrolysis may be accelerated if the suture is exposed to alkaline urine, which is commonly found in herbivores or carnivores with urinary tract infections.[71] Absorbable staples provide an alternative method of closure of hollow organs, such as the bladder. To reduce the formation of adhesions, bladders closed with absorbable staples must be oversewn with a continuous-inverting suture pattern.[72] The use of nonabsorbable staples in the bladder is not recommended based on the formation of a urolith in one case report.[73]

A comparative study of bursting strength of rat bladders sutured with 6-0 surgical gut and 7-0 chromic gut suggested that infusion pressures of up to 550 mm Hg are required to induce failure.[74] Voiding pressures of most mammals are considerably lower.[75] The voiding pressures of adult ponies and mares have been determined to be approximately 90 mm Hg.[76] The use of 3-0, 2-0 or 1-0 suture material is adequate to maintain primary closure of incisions in the pressurized portions of the equine distal urinary tract.

Laparoscopy

Laparoscopy offers a minimally invasive method for surgical procedures of the equine urogenital system. Its use has expanded over recent years. Because of the size of the horse and the anatomic location of the urinary tract, traditional surgical approaches may not provide adequate access to complete the surgical procedure. However, a laparoscopic approach may offset these hurdles. Laparoscopy can be performed as a standing procedure or under general anesthesia. The standard laparoscopic equipment and instrumentation (described in Chapter 13) can be used for several surgical procedures of the urinary tract. The list of procedures includes exploratory laparotomy, cystic calculi removal, nephrectomy, renal biopsy, umbilical remnant resection, and urinary bladder repair. Another option is to use hand-assisted laparoscopic techniques. This combines the benefits of laparoscopy with that of an open surgical approach. The hand-assisted techniques offer the advantage of being able to perform manual dissection and retraction. The ability to control hemorrhage is increased as well.

Pharmacologic Considerations

The route of elimation of water-soluble drugs and drug metabolites is through renal clearance. Therefore, the kidneys are exposed to high levels of drugs and drug metabolites. The renal effects of all drugs administered to surgical patients must be considered.[77]

ANESTHESIA

J. Brett Woodie

Anesthetic management of a patient with an impairment of the renal system should be focused on minimizing the time under anesthesia and minimizing hypotension. Renal ischemia can occur during general anesthesia because of systemic hypotension or renal vasoconstriction. It is also important to realize that all anesthetics are likely to decrease GFR. Most decrease GFR by decreasing RBF. Anesthetic agents may indirectly alter renal function through changes in cardiovascular and/or neuroendocrine activity. All fluorinated gas anesthetics are nephrotoxic to some degree.[65] Sevoflurane has cardiopulmonary effects similar to those of isoflurane, and it has the theoretical possibility of creating both hepatic and renal toxicity through the formation of compound A via the interaction between metabolites and CO_2 absorbents.[78,79] Despite the potential for the formation of toxic metabolites, sevoflurane has been used on more than 120 million human patients without one report of induced renal toxicity.[79] However, the effect of compound A formation in the anesthetic breathing circuit of the horse is not known and it would be safest to avoid the use of sevoflurane in patients with kidney disease. Halothane undergoes more hepatic metabolism than other inhalants, but formation of toxic waste products appears to be minimal.[80] Halothane has been a widely used anesthetic agent for horses.[81] Its reduced degree of metabolism renders it relatively less toxic for use in renally impaired horses. Alternatively, isoflurane is useful in critically ill horses and has few renal effects, which are limited to an increase in urine flow and an increase in serum glucose.[82,83]

The kidney is sensitive to hypoperfusion resulting from hypotension. Systemic hypotension while under anesthesia is most likely caused by peripheral vasodilation. Inhalant

anesthetics depress myocardial contractility and cardiac output. Several commonly used anesthetic agents also produce some degree of peripheral vasodilation, which may result in reflex renal vasoconstriction and associated renal hypoperfusion.[84] Renal damage may be avoided by maintaining RBF and minimizing the duration and magnitude of hypotension associated with general anesthesia. Intraoperative administration of balanced fluids enhances perfusion of the kidneys by improving cardiac output. Selective vasopressors, such as dobutamine or dopamine, increase cardiac output and enhance renal perfusion.[85,86] The use of an α-blocker as a premedication reduces the degree of catecholamine-induced vasoconstriction and improves renal perfusion in some anesthetized patients.[87]

Xylazine and detomidine are sedative hypnotic agents commonly administered to horses to facilitate examination. Both of these drugs have a dose-dependent diuretic effect.[88] Horses with uncomplicated obstructive disease of the lower urinary tract that are at risk for acute cystorrhexis could be further compromised by the use of sedatives or tranquilizers with diuretic properties.

ANTIMICROBIAL AGENTS

Harold C. Schott II

Short-term use of perioperative antimicrobial agents is generally indicated for horses undergoing surgery of the urinary tract, and a longer course of treatment is necessary for treatment of urinary tract infection (UTI). The duration of perioperative antimicrobial use may be limited to a single presurgical dose and generally should be discontinued after 24 hours, unless the invasiveness of the surgery warrants a longer treatment course.[89] As a general rule, broad-spectrum coverage against aerobic gram-positive and gram-negative organisms should be provided. Further, potential adverse effects of antibiotics should be considered to determine whether it may be better to avoid use of certain drugs. For example, aminoglycoside antibiotics are best avoided in patients with AKI and CKD, and other drugs may increase the risk of developing enterocolitis in horses that are placed under general anesthesia for urinary tract surgery. Of interest, anecdotes about enterocolitis following general anesthesia and surgery suggest that use of specific perioperative antimicrobial agents may carry differing risks in varying geographical regions; thus, both regional and recent experience should also be used to guide selection of the appropriate perioperative antimicrobial agents.

Selection of an antimicrobial agent for treatment of UTI should be based on culture and susceptibility results of urine or septic tissue débrided at surgery. In addition, practical considerations (e.g., oral versus parenteral medications, frequency of administration, and potential adverse effects) must also be considered when choosing the most appropriate drug to use. It warrants mention that resistance to a particular antimicrobial agent *in vitro* may not preclude successful treatment with the drug *in vivo* as long as high concentrations are achieved in urine. Laboratory reports provide minimum inhibitory concentration (MIC) data for serum, not urine. To determine whether a particular bacterial isolate in urine may be susceptible to urine concentrations of a drug, the actual MIC for some (but not all) organisms can be determined by the laboratory when specifically requested. Similarly, demonstrable susceptibility *in vitro* does not always guarantee a successful response to treatment. For example, *Enterococcus* spp. are often found to be susceptible to the potentiated sulfonamide combinations *in vitro;* however, this pathogen is inherently resistant to these combinations *in vivo*.[90] Recommendations for duration of antimicrobial treatment of UTI in people include 3 days for uncomplicated cystitis (rare in horses) and 1 to 2 weeks for the treatment of upper UTI.[91] Ideally, a voided midstream urine sample should be submitted for bacterial culture 1 to 2 weeks after treatment of an upper UTI has been discontinued.

If the UTI recurs and the same organism is isolated, search for a nidus for persistence of UTI should be pursued (e.g., a urolith or renal abscess) by ultrasonographic examination of the kidneys. Cystoscopy and ureteral catheterization can also be pursued to evaluate whether an upper UTI is unilateral or bilateral. In contrast, isolation of a different pathogen from a recurrent UTI suggests that there may be an anatomical or functional cause of abnormal urine flow predisposing the animal to recurrent infections (e.g., bladder paresis and sabulous urolithiasis). Further, it is not unusual to find highly resistant organisms in the urine of horses with chronic UTIs, especially in those that have bladder paralysis and have been repeatedly catheterized and treated with a variety of antimicrobial agents. In such cases, the antimicrobial agent should be selected based on culture and susceptibility reports of serial quantitative urine cultures. With some problems (bladder paresis and sabulous urolithiasis or pyelonephritis with nephrolithiasis), elimination of UTI is essentially impossible by treatment with antibiotics alone. In these cases, surgical removal of the nidus of infection (i.e., nephrectomy for chronic unilateral pyelonephritis, as long as the contralateral kidney appears normal and azotemia is not present) is the treatment of choice. However, not all persistent infections are amenable to surgical correction (bladder paresis), and long-term (potentially lifelong) antimicrobial treatment may be used with the goal of keeping bacterial numbers "in check" rather than having the goal of eliminating the UTI. For example, I commonly place horses with incontinence, bladder paresis, and persistent UTI on an indefinite course of once-daily treatment with a potentiated sulfonamide because that is the only practical long-term treatment for this type of problem.[2]

Specific Antimicrobial Agents

Penicillins

A single intramuscular (IM) dose (22,000 IU/kg) of procaine penicillin G results in urine concentrations exceeding 60 mg/mL for 48 hours, well above the MIC of many organisms. Thus, frequency of administration of this drug could be decreased to once daily or every other day. Although many of the *Enterobacteriaceae* demonstrate resistance to ampicillin *in vitro*, this drug is concentrated in urine and is often effective against these isolates *in vivo*. The potentiated penicillins (ticarcillin or ticarcillin–clavulanic acid) should be reserved for treatment of horses with UTIs caused by highly resistant organisms (e.g., *Pseudomonas* spp.) and are sometimes selected as an alternative to aminoglycosides in azotemic patients.[92]

Aminoglycosides

The aminoglycosides can be nephrotoxic and should be reserved for the treatment of lower UTIs caused by highly resistant organisms or acute, life-threatening upper UTIs caused by aerobic gram-negative bacteria. It is important to remember that aminoglycoside nephrotoxicity is a cumulative renal insult;

consequently, administration of one or two doses of these drugs is unlikely to precipitate further AKI, even in azotemic patients. However, if azotemia is detected during initial patient work-up, an alternative class of antimicrobials should be selected. Pharmacokinetic and pharmacodynamic studies in adult horses and foals support once-daily administration of both gentamicin (6.6 mg/kg IV) and amikacin (21 mg/kg IV) as the preferred dosage schedule because it improves the bactericidal action (concentration-dependent killing) and lessens the risk of nephrotoxicity.[93,94] Further, there is some evidence that foals may be more susceptible to gentamicin nephrotoxicity than adult horses, providing another reason to consider alternative antimicrobial agents for younger patients.[95]

Potentiated Sulfonamides

Combinations containing the sulfonamide sulfadiazine are preferred for the treatment of UTIs because sulfadiazine is excreted largely unchanged in urine, whereas sulfamethoxazole is largely metabolized to inactive products in the liver prior to urinary excretion.[95] As mentioned earlier, trimethoprim-sulfadiazine combinations (20 mg/kg PO every 12 to 24 hours) are the only practical choice for long-term treatment of horses with persistent UTIs.

Cephalosporins

Cephalosporins are commonly used for treatment of UTIs in other species. However, in horses, the cephalosporins are rarely more advantageous than the penicillins or potentiated sulfonamides. However, ceftiofur has broad-spectrum antimicrobial activity and may be indicated (2.2 mg/kg IM every 12 hours) when urinary pathogens are resistant to other drugs.[92]

Tetracyclines

Intravenous tetracyclines are rarely indicated for the treatment of UTIs in horses. However, there has been a resurgence in use of doxycycline in human and veterinary medicine, and this cost-effective oral antimicrobial agent may be of use for treatment of UTIs caused by susceptible gram-positive isolates. After oral administration (10 mg/kg every 12 hours for 3 days), urinary concentrations reached 145 ± 25 μg/mL 2 hours after the last dose (more than 100 times higher concentrations than in plasma).[96] Bioavailability has not been determined because IV administration of doxycycline causes cardiac toxicity. Caution should be used with this drug because a higher dosage (20 mg/kg PO every 24 hours) resulted in enterocolitis leading to euthanasia in one of six horses in this report.[97]

Fluoroquinolones

Enrofloxacin (5 to 7.5 mg/kg PO every 12 hours) may be a suitable antimicrobial agent for more serious upper or persistent UTIs when the causative organism(s) are resistant to other antibiotics. After oral administration (5 mg/kg every 12 hours), enrofloxacin bioavailability was 60% in horses and the drug was well concentrated in urine.[98] However, fluoroquinolones can damage articular cartilage (particularly in rapidly growing juvenile animals) and their use should, therefore, be limited to adult horses or to foals with urinary pathogens that are not susceptible to other antimicrobial agents.[92]

Other Antimicrobial Agents

The antimicrobial agents discussed earlier should be adequate for prophylaxis for surgery as well as for treatment of most UTIs in the horse. A significant challenge in equine practice is availability of antibiotics that can be orally administered with minimal adverse effects on the gastrointestinal tract and that also have a broad spectrum of activity against many equine pathogens. Although chloramphenicol is not approved for use in the horse, the need for such a drug has also led to a recent increase in chloramphenicol use, and this drug (25 to 50 mg/kg PO every 8 hours) may be indicated for treatment of complicated or persistent UTIs in horses, when supported by culture and susceptibility results. If chloramphenicol is selected, owners and caregivers should be warned about the rare possibility of developing aplastic anemia and should be instructed to wear impenetrable gloves when administering this medication. Further, only formulations that can be dissolved in water or that are prepared as a paste or suspension should be used to limit the risk of inhaling chloramphenicol.

A further challenge in horses undergoing urinary tract surgery in which temporary indwelling urinary catheters or ureteral stents may be used postoperatively is frequent colonization and infection with multidrug resistant organisms, notably *Enterococcus* spp. One antimicrobial agent that may be useful if the UTI persists after catheter or stent removal is vancomycin (7.5 mg/kg IV every 8 hours); however, hospitals should have policies in place to approve and monitor use of this antimicrobial agent, limited to specific cases in which this is the only antibiotic choice on the basis of culture and susceptibility results.[99]

A final medication that is also undergoing a resurgence of use in human medicine and interest in small animal practice for treatment of multidrug resistant UTIs is fosfomycin.[100,101] Pharmacokinetics of this antimicrobial agent after IV, IM, or subcutaneous administration to horses have been reported, and dosages of 10 or 20 mg/kg produced serum and urine concentrations above the MIC for many pathogens.[102] Currently, however, laboratories do not routinely provide a susceptibility report for fosfomycin and I am unaware of the use of this drug in clinical cases of UTI in horses.

ANTI-INFLAMMATORY DRUGS

Harold C. Schott II

Surgical treatment of urinary tract disorders requires short-term treatment with anti-inflammatory drugs to limit postoperative swelling and inflammation and to provide analgesia for the patient. Phenylbutazone and flunixin meglumine, at routine doses, can be used as effective anti-inflammatory drugs and should provide adequate analgesia for most patients. To avoid adverse gastrointestinal and renal effects when using these nonsteroidal anti-inflammatory drugs (NSAIDs), a rule to follow should be to use the lowest effective dosage for the shortest duration that produces the desired clinical result.

The urinary tract presents unique challenges for control of inflammation and pain in the postoperative period. For example, urine may continue to leak through repair sites of disrupted ureters or the bladder. This complication may retard healing of wound margins and result in recurrence of uroabdomen. To minimize this complication, indwelling bladder catheters or ureteral stents may be used in the postoperative period, but their presence may lead to straining. In addition to use of

NSAIDs, administration of phenazopyridine (4 mg/kg PO every 8 to 12 hours) may alleviate lower urinary tract pain in these patients. In people, phenazopyridine relieves burning, irritation, and discomfort, as well as urgent and frequent urination caused by urinary tract infections, surgery, injury, or examination procedures. After renal elimination, the medication acts as a topical local anesthetic on ureteral, bladder, and urethral mucosa. However, phenazopyridine does not have antimicrobial activity and it turns urine an orange color that can stain hands and clothing. Efficacy of the drug should be apparent after the first or second dose, and it is typically administered for only 2 to 3 days. When NSAIDs and phenazopyridine are not effective in providing adequate analgesia, intermittent administration of α_2-receptor agonists or opioids or a continuous-rate infusion of lidocaine (and possibly α_2-receptor agonists and opioids) may need to be considered. In horses in which significant postoperative pain may be anticipated, epidural administration of α_2-receptor agonists or opioids should also be considered to produce prolonged regional analgesia before full recovery from general anesthesia. Refer to recent reviews for further information on these approaches to analgesia.[103-106]

Finally, when a nephrectomy is performed, there is a loss of residual functional nephrons. The reduction in total renal mass should be taken into consideration when formulating an anti-inflammatory and analgesic plan because the risk for nephrotoxicity with NSAIDs may be increased in these patients.

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CHAPTER 64

Kidneys and Ureters

Harold C. Schott II and J. Brett Woodie

ANATOMY

Harold C. Schott II

The urinary system of the horse, like that of most mammals, consists of paired kidneys and ureters, the bladder, and the urethra.

Kidney

The kidneys and ureters are located in the retroperitoneal space. In a newborn 50 kg foal, each kidney weighs 150 to 200 g. In the adult horse (400 to 500 kg), the left kidney weighs 800 to 1000 g and the right kidney is usually 25 to 50 g heavier, although this is not a consistent finding and the reverse relation may be observed.[1] The right kidney is located immediately below the dorsal extent of the last two or three ribs and the first lumbar transverse process. It is shaped like a horseshoe and measures about 15 to 18 cm (6 to 7 inches) in length, 15 cm (6 inches) in width, and 5 to 6 cm (2 to 2½ inches) in height (dorsal to ventral). Craniolaterally, it is embedded in the liver, and its more cranial position in comparison to the left kidney prevents it from being accessible on rectal palpation. The left kidney is more elongated than the right kidney, in the shape of a U or inverted J with the longer arm extending caudad. The cranial pole extends to the level of the hilus of the right kidney. The left kidney is about 18 cm (7 inches) long, 10 to 12 cm (4 to 5 inches) wide, and 5 to 6 cm (2 to 2½ inches) in height. Because of its more caudal location, the caudoventral aspect of the left kidney can be palpated routinely during rectal examination. The blood supply to the kidneys comes from one or more renal arteries branching from the aorta. Accessory renal arteries, which generally enter caudally, may also arise from the caudal mesenteric, testicular, ovarian, or deep circumflex iliac arteries.[1]

The surface of each kidney is covered by a fibrous capsule that is easily peeled from the underlying renal parenchyma. The equine kidney consists of an outer cortex that is slightly narrower than the inner medulla. The cortex is dotted with dark spots: renal corpuscles or glomeruli within Bowman's capsules. In horses, the corticomedullary junction is less distinct than in other species and is typically a deep red that contrasts well against the paler medulla and red-brown cortex. This region undulates between regularly spaced interlobar arteries that subdivide the renal parenchyma into somewhat indistinct lobes, consisting of a wedge of cortex overlying a narrower pyramid of medullary tissue. The cortex extends somewhat deeper into the renal parenchyma adjacent to each interlobar artery, and these cortical projections are termed *renal columns* (not well developed in horses). On a three-dimensional view, renal columns surround or cap the convex base of a renal pyramid, the associated medullary portion of each kidney lobe. Renal pyramids narrow as they extend to their apices at the level of the renal pelvis.[2] A commonly cited anatomy text states that equine kidneys contain a total of 40 to 60 lobes arranged in four parallel rows.[1] However, kidney lobation varies among species with cattle having the most obvious separation of the renal parenchyma into lobes; in carnivores, small ruminants, and the horse they are largely fused, thus making histologic separation into distinct lobes somewhat difficult.[2] The renal pyramids are also fused to a large extent in horses; however, at the innermost aspect of the medulla, the apices of the pyramids are partly separated by invaginations of fibrous connective tissue and smooth muscle extending from the renal pelvis that surround the apex of each renal pyramid. In other species, this smooth muscle has been shown to play an important role because contractions squeeze the apices of the pyramids circumferentially, intermittently forcing urine into the renal pelvis and forcing capillary blood, interstitial water, and electrolytes back into the venous circulation.[3]

Deep to the medulla is the renal pelvis, a funnel-shaped proximal extent of the ureter that is flattened in the dorsoventral plane. The renal pelvis consists of three layers: an external fibrous coat, an intermediate smooth muscle layer, and an innermost layer of transitional epithelium. In some areas the fibrous coat can extend into the renal parenchyma to become contiguous with the connecting tissue of the renal columns (incompletely developed in horses). The muscular layer contains smooth muscle fibers that run in assorted directions, attaching near the site where the fibrous coat joins with the renal columns along the pyramids, and continuing without interruption as ureteral smooth muscle.[3] A prominent ridge of tissue with a mildly concave free edge, termed the *renal crest*, protrudes into the renal pelvis along a craniocaudal axis, opposite to the outflow path of the ureter. The renal crest is a fusion of the apices of many pyramids of medullary tissue in the central aspect of the kidney (Figure 64-1).[2] The distal ends of inner medullary collecting ducts (ducts of Bellini) from the middle portion of the kidney open onto the renal crest to allow urine to exit into the pelvis through the area cribosa. In contrast, the collecting ducts draining nephrons in the cranial and caudal parts of the kidney do not open into the pelvis directly but into two narrow tubular structures termed the *terminal recesses*.[2] These tubular structures are only a few millimeters in diameter but extend 5 to 7 cm (2 to 3 inches) into either pole of the kidney.[2] As the pelvis extends into the ureter, beyond the renal crest, transitional epithelium becomes more prominent and consists of numerous folds (to allow distension and contraction). In addition, the renal pelvis and proximal ureter are lined with both compound tubular mucus glands and goblet cells that secrete thick, viscid mucus that is usually found in the renal pelvis and urine of normal horses.[1,4]

Ureters

The ureters are 6 to 8 mm (2½ to 3 inches) in diameter and travel about 70 cm (28 inches) to their insertions in the dorsal bladder neck, or trigone, close to the urethra. The distal 5 to 7 cm of each ureter courses within the bladder wall. This intramural segment of the ureter functions as a one-way valve to prevent vesicoureteral reflux with progressive bladder

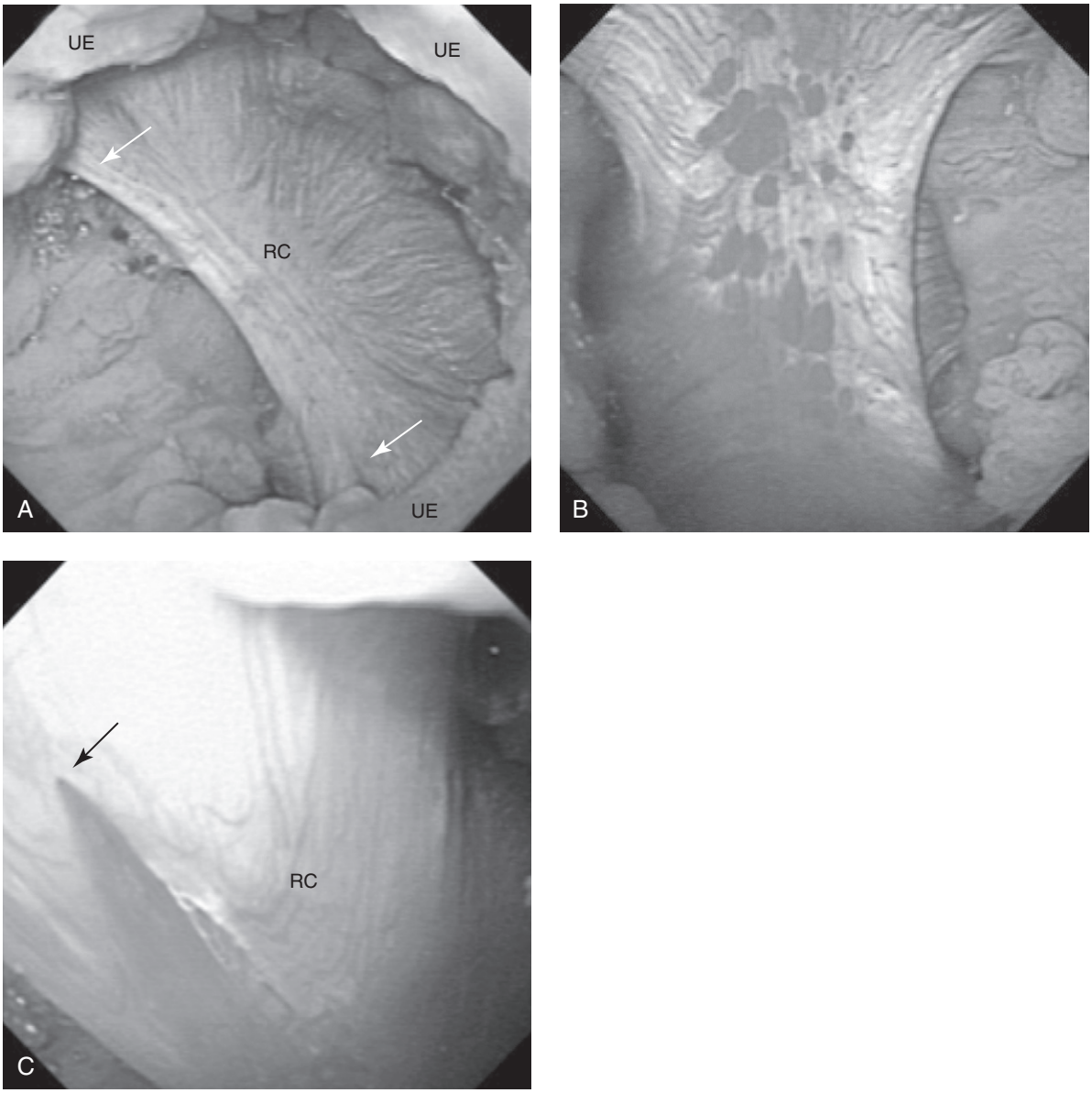


Figure 64-1. Endoscopic appearance of the renal pelvis of the horse. **A,** The renal crest *(RC)* extends from upper right to lower left and uroepithelium *(UE)* of the proximal ureter becomes more prominent toward the periphery of the renal pelvis; entrance of the terminal recesses into the renal pelvis is at the level of the *white arrows*. **B,** Closer view of the renal crest showing phenol red *(dark gray patches)* exiting numerous ducts of Bellini, terminal portions of the innermedullary collecting ducts. **C,** Close-up view over an opening of a terminal recess into the renal pelvis; a gray stream of phenol red–discolored urine is exiting the opening *(black arrow)*.

distention.[1] Ureters are muscular tubes, similar to the esophagus. As a "bolus" of urine accumulates in the renal pelvis, pacemaker activity induces a muscular contraction that propagates the bolus of urine down the ureter to the bladder. With changes in urine flow in other species, the length of the "urine bolus" changes while the rate of transport down the ureter remains the same.[3] Passage of "urine boluses" can be appreciated in horses during either cystoscopy or transrectal ultrasonographic examination. During cystoscopy, streams of urine can be seen to intermittently exit each ureter. These urine streams are asynchronous and vary in volume and frequency with changes in urine flow.[5] During transrectal ultrasonography, forceful passage of urine from the ureters into the bladder can be appreciated by swirling of the echogenic urine in the bladder near the trigone.

Histology

The functional unit of the kidney is the nephron. Each nephron is composed of a renal corpuscle (glomerulus within Bowman's capsule), a proximal tubule (convoluted and straight components), an intermediate tubule (loop of Henle), a distal convoluted tubule, a connecting tubule, and cortical, outer medullary, and inner medullary collecting ducts.[1,2] A study of kidney organogenesis using unbiased stereological techniques found that the equine left kidney contains approximately 10 million glomeruli (for a total of 20 million in both kidneys) and, as in other species, the total number of glomeruli do not increase after birth despite continued growth of the kidney (with increasing glomerular size) until about 1 year of age.[6] Histologically, equine nephrons are similar in most respects to those of other

mammalian species; however, the diameter and epithelial height of the collecting duct segments are comparatively larger. In addition, the equine macula densa (segment of the ascending loop of Henle that lies in close association with the juxtaglomerular apparatus of the afferent arteriole) appears more prominent than that of other mammals.[7] Whether these subtle histologic differences are accompanied by functional differences has not been investigated.

Innervation

Relative to its size, the mammalian kidney has a richer innervation than almost any other organ.[8] Although the neuroanatomy of the equine kidney has not been well studied, autonomic nerves course from the aorticorenal and renal ganglia along the major renal vessels into the kidneys.[1] These nerves are predominantly sympathetic because the kidneys appear to be poorly supplied by cholinergic nerves. Although the best-recognized effect of renal nerves is control of renal vascular resistance (for regulation of renal blood flow over a wide range of perfusion pressures), they also act directly on renal tubules and juxtaglomerular cells. For example, low-frequency stimulation of renal nerves (below the threshold for vasoconstriction) increases proximal tubular sodium reabsorption and renin release by activation of α_1 adrenoceptors.[9] In addition to α- and β-adrenoceptors, renal vasculature is rich in dopaminergic adrenoceptors. Activation of the latter, specifically dopamine type 1 receptors, leads to increased perfusion of the outer renal medulla. Presence of these receptors was the basis for historical use of dopamine (a treatment that is no longer recommended), and recently more specific DA-1 receptor agonists, such as fenoldopam, in an attempt to improve renal blood flow in acute renal failure.[10,11] Renal adrenoceptors can also be activated unintentionally by administration of drugs. A common clinical example is the diuresis induced by administration of the α_2-agonists xylazine and detomidine. Although the diuresis has been attributed to a transient hyperglycemia and glucosuria, the latter is often absent.[12,13] An alternative explanation may be drug binding to α_2-adrenoceptors located on collecting duct epithelium. Activation of these receptors can lead to antagonization of the effects of antidiuretic hormone on cortical collecting ducts, resulting in diuresis.[14]

Autonomic innervation of the ureters is important for ureteral peristalsis. The equine ureteral smooth muscle contains both α_1- and β_2-adrenoceptors, which induce contraction and relaxation, respectively, when activated by norepinephrine.[15] Recent studies of the innervation of the equine ureter demonstrated greater densities of adrenergic neurons in the proximal (renal pelvis) and intravesicular (bladder wall) portions of the ureter.[16] Increased densities in these regions are consistent with the suspected pacemaker activity of the renal pelvis, which initiates ureteral peristalsis, and the sphincter-like function of the distal segment of the ureter.

DISORDERS REQUIRING SURGERY

Harold C. Schott II

Disorders of the kidneys and ureters that may require surgery include congenital anomalies (ectopic ureter and ureteral defects) and acquired disorders. The latter can include nephrolithiasis, ureterolithiasis, pyelonephritis, neoplasia, and ureteral disruption (ureterorrhexis). Rarely, hematuria may be a consequence of vascular malformations for which surgical inteervention may be necessary.

Congenital Anomalies

Ectopic Ureter

Although rare, ectopic ureter is the most commonly described anomaly of the equine urinary tract.[17-34] Ectopic ureters can develop when the ureteric bud (metanephric duct) either fails to be incorporated into the urogenital sinus or fails to migrate craniad to the bladder neck, or when the mesonephric duct fails to regress.[35] In the former instance, ectopic ureters open near the urethral papilla in females or in the pelvic urethra near the colliculus seminalis in males. With the latter, the ureter may open anywhere along the vagina, cervix, or uterus (but only in females because this portion of the mesonephric duct becomes the Wolffian duct system in males).

In horses with ectopic ureters, urinary incontinence is usually apparent from birth. Affected animals are presented for scalding of the hind limbs during the first few weeks to months of life. Horses with unilateral ectopia can also urinate normally, because the other ureter enters the bladder in the appropriate location. Renal function is usually normal, but the affected ureter may be markedly dilated and tortuous (attributed to intermittent obstruction). To determine the site of the ectopic ureteral orifice(s), visual examination of the vestibule and vagina (using a blade speculum) is initially performed to look for intermittent urine flow from the area of the urethral papilla. Endoscopy is helpful in females (while inflating the vestibule and vagina with air and using a hand to form a seal at the vulva) (Figure 64-2) and is required in males to visualize the ectopic ureteral opening. Intravenous administration of a dye (phenolsulfonphthalein [phenol red], 1 mg/kg IV) to discolor urine red may help locate ectopic ureteral openings. As discussed in Chapter 63, intravenous contrast excretory urography has been performed to investigate renal architecture and the course of ectopic ureters; however, detail is often limited and results have frequently been inconclusive. If radiographic imaging is

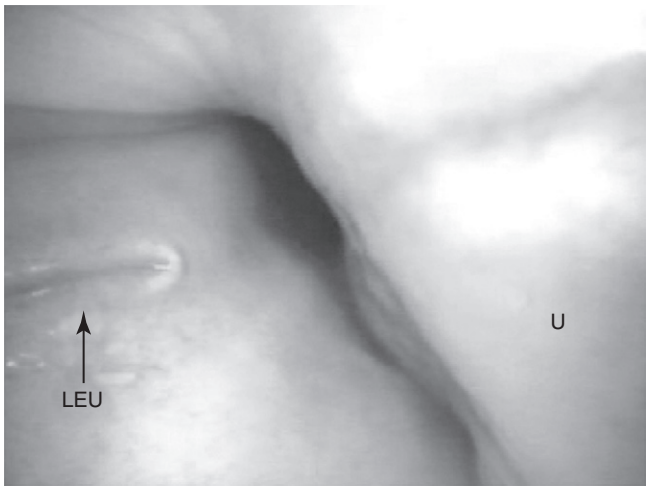
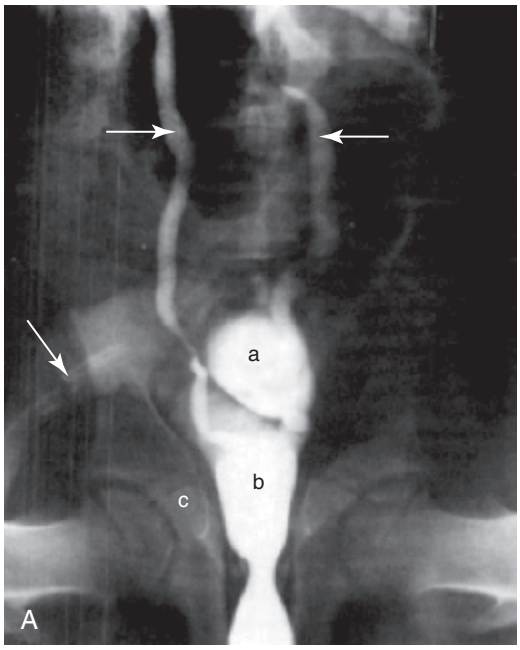


Figure 64-2. Endoscopic image of a left ectopic ureter *(LEU)* opening into the urethral wall *(U)* of a filly. (From Cokelaere SM, Martens A, Vanschandevijl K, et al: Hand-assisted laparoscopic nephrectomy after initial ureterocystostomy in a Shire filly with left ureteral ectopia. Vet Rec 161:424, 2007, with permission.)

pursued, retrograde contrast studies via catheterization of the bladder and ureters or ultrasound-guided pyelography, in which contrast agent is injected directly into the renal pelvis using a spinal needle (Figure 64-3),[31] are more rewarding contrast radiographic techniques, yet they still may not provide adequate information about the distal end of ectopic ureters. If an abnormal ureteral opening can be documented by direct visualization via endoscopy, further information gleaned from contrast radiographic studies is likely to be of limited value, other than documenting ureteral enlargement or possible hydronephrosis. In fact, a study in dogs with ectopic ureters found that endoscopic evaluation was more successful than contrast radiographic studies for locating the ectopic ureteral opening.[36] Currently, I do not recommend contrast radiographic imaging for evaluation for ectopic ureters; rather, endoscopic and ultrasonographic findings are generally preferred to establish the diagnosis and to formulate a surgical plan. In foals that are small enough to fit into the gantry of computed tomography (CT) or magnetic resonance imaging (MRI) scanners, use of these imaging devices, coupled with IV administration of contrast agents, may provide the best overall detail about the course of ectopic ureters from the kidney to the lower urinary or reproductive tract.[34] In a report of 24 dogs with suspected ectopic ureters, CT imaging was superior to fluoroscopic contrast radiographic studies in identifying ectopic ureters.[37] Of interest, cystoscopic and surgical or necropsy findings were considered gold standards for comparison of results in this study. Drawbacks of CT and MRI are that these imaging procedures require general anesthesia and are costly, and currently there is limited evidence that they provide better information than that obtained via endoscopic examination of the lower urinary tract and ultrasonographic imaging of the kidneys. Nuclear scintigraphy was performed in one case of bilateral ectopic ureter to demonstrate an apparent blind end (atresia) of one ureter and a course of the other ureter beyond the bladder.[32] The scintigraphic findings in this case did not agree with results of intravenous pyelography, and they provided limited anatomical detail about the distal end of the ectopic ureter; however, scintigraphy did provide information about the relative function of each kidney in this patient.

Surgical treatment of ectopic ureter has included ureterocystotomy (surgical reimplantation of the ectopic ureter[s] into the bladder with or without tunneling) or unilateral nephrectomy. Before surgery is pursued, it is imperative to determine whether the condition is unilateral or bilateral, which side is affected if unilateral, and whether urinary tract infection (UTI) is present. Further, attempts should be made to rule out other anomalies, especially of the urethral sphincter and reproductive tract. Because the most significant postoperative problem in dogs with ectopic ureters is persistence of incontinence,[38] it is useful to determine whether detrusor and urethral sphincter function are fairly normal, especially with bilateral ectopia. A simple assessment can be made by infusing saline into the bladder and observing whether incontinence develops (sphincter dysfunction) and if the infused fluid is voided spontaneously (detrusor function). More sophisticated assessment can be performed by cystometrography[39-41] and is worthy of consideration because functional abnormalities of the urethra or bladder were found in eight of nine dogs with ectopic ureter in which urodynamic studies were performed.[42]

In 21 cases of ectopic ureter in horses reported in the literature[17-34] and five further cases I saw, 24 of 26 (92%) have been females; however, this lopsided sex distribution may reflect



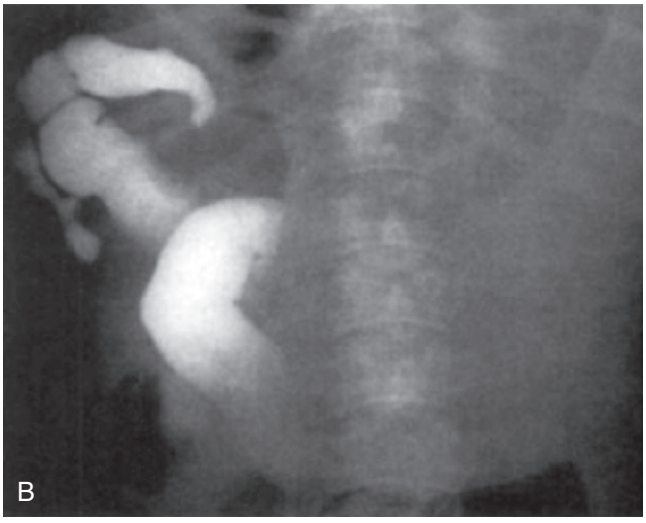


Figure 64-3. A, Ventrodorsal radiographic view of a retrograde contrast-enhanced urethrocystogram in a colt with bilateral ectopic ureters showing the bladder *(a),* pelvic urethra *(b),* and coxofemoral joint *(c).* The *straight white arrow* indicates a catheter within the penile urethra and the *undulating arrows* indicate the ureters. **B,** Ventrodorsal radiographic view of a percutaneous ultrasound-guided pyelogram in a filly with a left ectopic ureter detailing both hydronephrosis and a markedly enlarged and tortuous ureter. Although both approaches provide greater contrast detail than intravenous pyelography, insertion of the distal ends of the ectopic ureters is not well detailed in either study. (**A,** From Modransky PD, Wagner PC, Robinette JD, et al: Surgical correction of bilateral ectopic ureters in two foals. Vet Surg 12:141, 1983. **B,** From Tomlinson JE, Farnsworth K, Sage AM, et al: Percutaneous ultrasound-guided pyelography aided diagnosis of ectopic ureter and hydronephrosis in a 3-week-old filly. Vet Radiol Ultrasound 42:349, 2001, with permission).

that incontinence is easier to recognize in females, because urine entering the pelvic urethra in males may pass retrograde into the bladder. Although a genetic predisposition for ectopic ureter exists for several dog breeds,[43] no breed predilection has been established in horses. However, Quarter Horses and

Standardbreds may be at greater risk: the condition has been found in eight Quarter Horses and six Standardbreds as compared to two Thoroughbreds, two Appaloosas, two Clydesdales, two Shires, one Arabian, one Fresian, one Foxtrotter, and one Warmblood. The condition was unilateral in 17 (left in 11 cases; right in five cases, and not specified in one case) and bilateral in nine. Affected foals should be examined for other anomalies because two foals appeared to have an abnormal urethral sphincter[20,25] and one was a cryptorchid.[19]

Among 20 cases in which surgical correction was pursued, ureterocystotomy was initially successful in establishing a functional ureter in 11 published cases[19,20,24,25,30-33] and two foals seen by me, although four died of postoperative complications.[20,24,31] In contrast, all seven cases treated by unilateral nephrectomy (including two seen by me) had a favorable outcome.[21,22,28,34] Because affected ureters are often dilated and tortuous, surgical reimplantation can be difficult and may not result in a functional ureteral orifice. For example, an attempt to perform a ureterocystotomy in one mare was aborted during the initial surgery because a markedly enlarged ureter was detected; subsequently a nephrectomy was performed several weeks later.[22] A surgically created ureteral opening into the bladder in another case became stenotic and incontinence returned, prompting nephrectomy several weeks after the initial surgery.[32] Consequently, when the problem is unilateral, nephrectomy of the affected side may be the preferred treatment option at present. However, nephrectomy results in a loss of renal functional mass, and with recent improvements in endoscopic equipment, it would seem preferable to pursue endouroscopic laser treatment of ectopic ureters in horses, as is the current treatment of choice in dogs.[44,45] Most reports of ectopic ureter treated by nephrectomy fail to describe what is done with the remnant ureter. In one report, the ureter appeared to continue to pool an exudate that was observed to be passed once or twice daily.[22] Authors of future reports are encouraged to more closely describe whether or not the remaining ureter, which is often dilated and tortuous, is also removed (difficult with a flank approach) or left *in situ*.

Ureteral Defects or Tears (Ureterorrhexis)

Retroperitoneal accumulation of urine and uroperitoneum have been described in seven foals with unilateral or bilateral ureteral defects[46-52] and have been observed in three additional foals by me. These included seven male and three female foals of various breeds (five Standardbreds, two Thoroughbreds, one Belgian, one Oldenberg, and one Appaloosa). Clinical signs including decreased nursing, depression, abdominal distention, diarrhea, and muscle twitching or other signs of neuromuscular irritability, and clinicopathologic abnormalities consisting of hyponatremia, hyperkalemia, hypochloremia, and azotemia, are similar to those seen with bladder rupture but may have a slightly later onset (4 to 16 days of age). Mild protrusion of the vagina may be seen in fillies in which the peritoneum has remained intact. In affected foals, ultrasonographic examination may reveal dilation of the renal pelvis and affected ureter as well as fluid accumulation around the kidneys or farther caudad within the retroperitoneal space. As with ectopic ureters, excretory urography has generally been an unrewarding diagnostic procedure, but contrast pyelography was successfully used to image leakage of contrast agent from a proximal ureteral defect in one report.[52] Contrast radiography has not routinely been pursued because exploratory celiotomy was generally performed shortly after a diagnosis of uroperitoneum was established. Catheterization of the ureters via a cystotomy and retrograde injection of methylene blue allowed localization of the defect(s) during surgery, and four cases, including one seen by me, were successfully treated by suturing the defect around an indwelling catheter.[48,49,52] Although ascending UTI should be an expected complication with a stent, repair of a defect in one foal without use of an indwelling catheter resulted in further urine leakage from the ureter, prompting a nephrectomy 4 days after the initial surgery.[51] Of the five remaining foals, one died consequent to progressive abdominal distention within a few hours after nephrectomy[46] and another foal died after three unsuccessful attempts at surgical repair.[47] Euthanasia was performed in another foal after recurrence of uroabdomen following nephrectomy (because of failure to detect bilateral ureteral defects in one case seen by me) and in two cases in which surgical repair was declined by the owner.[51]

At surgery or necropsy a single defect was found in six foals, bilateral defects were found in four foals, and multiple defects were apparent in one ureter. In most cases the defects were located in the proximal third of the ureter near the kidney. Of interest, distended, tortuous ureters, occasionally accompanied by hydronephrosis, were also described in two affected foals[47,52] and distal obstruction of the ureters at the bladder was suspected in these cases, prompting ureterocystotomy. Although several reports suggest that these ureteral defects may be anomalies of development, the actual cause of these ureteral defects is not known. Traumatic disruption was suggested in the initial report in which histologic examination of the margins of the defect revealed hemorrhage and proliferation of immature connective tissue.[46] A traumatic etiology was further supported by a subsequent report in which histologic examination of the defects revealed absence of transitional epithelium and inflammation in the defect margin in a foal that had been attacked by dogs.[50] Inflammation and granulation tissue also were seen in the apparently obstructed distal ureter in one of the foals with ureteral distention, again suggesting an acquired lesion. Blunt abdominal trauma, as may be sustained during automobile accidents, can cause retroperitoneal accumulation of urine as well as uroperitoneum in humans.[53] Disruption of the ureter is usually near the kidney, and this complication of trauma may not be recognized for several days following injury. In one foal evaluated by me, multiple rib fractures found at necropsy suggested that these ureteral tears could actually be a complication of foaling trauma (Figure 64-4).

Vascular Anomalies

Anomalies of the vascular supply to the equine urinary tract are rare but may result in hematuria, hemoglobinuria, partial ureteral obstruction, or hydronephrosis. Although not described in horses, life-threatening hematuria or hemorrhage into the abdomen could require surgical intervention and possible nephrectomy. A distal aortic aneurysm and associated extrarenal arteriovenous fistula has been described in a 5-month-old colt presented for intermittent hematuria, colic, and lameness.[54] Partial ureteral obstruction and hydronephrosis were observed on the affected side. Intrarenal vascular anomalies, termed *renal arteriovenous malformations,* are similarly rare (reported frequency of 0.04% in humans).[55] Interestingly, these vascular malformations may be silent until later in life, when varying

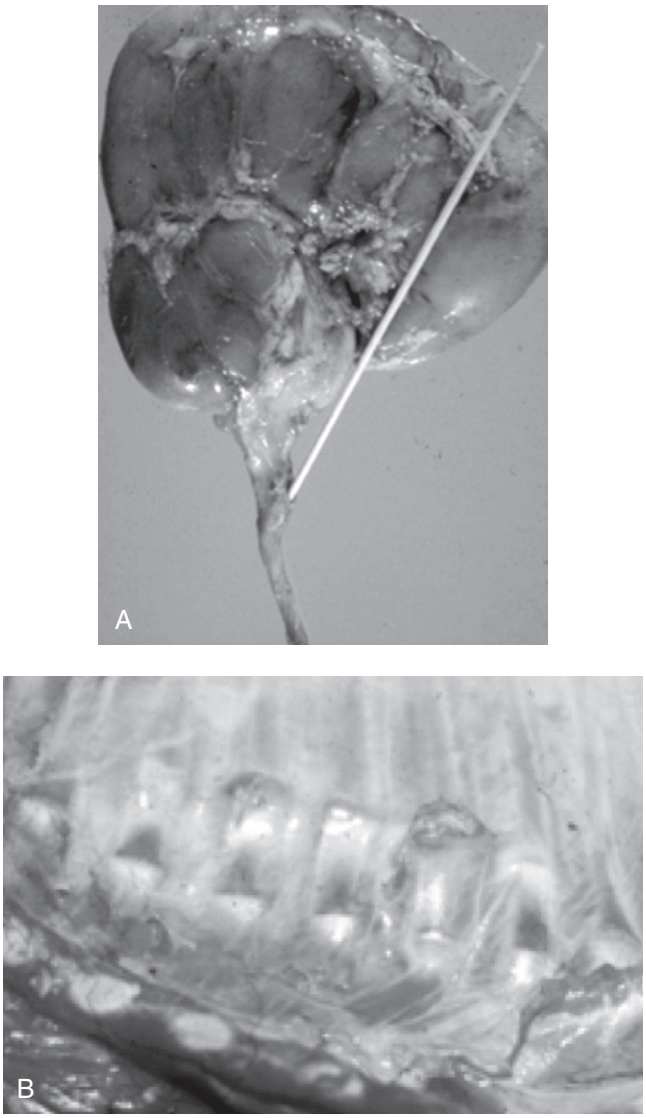


Figure 64-4. A, Kidney and ureter removed from a Standardbred colt with a proximal ureteral defect (or tear) through which a probe is inserted. **B,** Thoracic wall from the same foal showing a series of five fractured ribs, providing support that ureteral defects in foals can be acquired secondary to trauma.

degrees of hematuria and flank pain may ensue. The anomalous vessels are often tortuous and may be focally enlarged and devoid of elastic tissue. Hematuria and hemoglobinuria are thought to arise from areas where the anomalous vessels lie close to the collecting system. With vascular anomalies, an attempt should be made to determine the extent of the defect (unilateral or bilateral) via ultrasonographic examination, contrast radiographic studies, or cystoscopy (visualization that hematuria is coming from only one ureteral orifice). When a unilateral defect is documented in the absence of azotemia, unilateral nephrectomy or selective renal embolization has been recommended to prevent possible fatal exsanguination through the urinary tract[55]; however, conservative treatment may be considered if the urinary tract bleeding is minor and has not resulted in anemia.

A large vascular anomaly resulting in transient hemoglobinuria has been reported in a Quarter Horse colt.[56] Over several weeks, the large anomalous vascular structure spontaneously filled with a thrombus so that specific treatment (a nephrectomy) was not pursued. Occasionally gross hematuria with passage of blood clots can accompany omphalitis or bladder rupture.[57,58] These problems can usually be detected during ultrasonographic examination of the umbilical structures, and tissue echogenicity within the bladder, attributable to a blood clot, can sometimes be imaged.

Acquired Renal and Ureteral Disorders

Renal and Ureteral Calculi

Despite production of urine rich in crystals, urolithiasis is less common in horses than in dogs or cats with reported prevalence ranging from 0.11% to 0.7%.[59,60] In a review of 68 horses with urolithiasis, 15 had uroliths in the kidneys and two had ureteroliths and several horses with cystic calculi also had calculi in the upper urinary tract.[59] Nephroliths develop around a nidus that may be started with a variety of diseases that may damage renal parenchyma.[61] Calcium carbonate and calcium oxalate crystals readily form within collecting ducts and can tightly adhere to damaged renal tissue, forming a nidus for stone enlargement. At present, data on upper urinary tract stones in horses are insufficient to know whether they also develop spontaneously in the absence of tissue damage as in humans and whether they differ significantly in mineral composition from cystic calculi. It has been speculated, but not proved, that prolonged use of nonsteroidal anti-inflammatory drugs can damage renal medullary tissue and increase the risk of nephrolith formation adjacent to or within the pelvis and terminal recesses.[62]

Although nephrolithiasis and ureterolithiasis can be painful conditions in humans, horses often remain asymptomatic, and upper tract stones can be an incidental finding at necropsy.[60] Clinical signs become apparent if obstructive disease develops or bilateral disease leads to chronic kidney disease (CKD).[59,61,62] Nonspecific presenting complaints consistent with CKD including poor performance, lethargy, inappetance, and weight loss are more common than signs of obstructive disease, such as colic, stranguria, and hematuria. In an occasional male horse, a small stone passes down the ureter and leads to urethral obstruction and signs of acute obstructive disease (see Chapter 66). Rectal palpation may reveal an enlarged kidney or ureter (or bladder with urethral obstruction), and ureteral calculi may be palpable in an enlarged ureter.[63] Because normal ureters are generally not palpable on rectal examination, the entire course of the ureters (retroperitoneally along the dorsal abdominal wall to the lateral aspects of the pelvic canal to their insertion at the dorsal bladder neck) should be carefully palpated—an enlarged ureter can be overlooked.

Diagnosis of renal and ureteral calculi is usually made during rectal or ultrasonographic examination (see Figure 63-6). Although ultrasonographic imaging may provide information on the presence, number, and location of calculi, stones smaller than 1 cm diameter can be missed despite complete examination. Other ultrasonographic findings to support upper tract obstructive lithiasis include dilatation of the renal pelvis or proximal ureter and, in longstanding cases, hydronephrosis. Although azotemia generally accompanies bilateral disease, horses with unilateral disease often maintain normal renal function. A quantitative urine culture should be performed in all horses with nephrolithiasis or ureterolithiasis to assess possible concurrent UTI. If stones are available or collected at

surgery, they should also be submitted for culture because they may yield bacterial isolates in the face of a negative urine culture result.[59,61]

Because most horses with nephrolithiasis or ureterolithiasis typically have CKD by the time the diagnosis is established,[59,61,62] few patients are good candidates for surgical treatment. Nevertheless, in a few horses with unilateral disease (without azotemia) or obstructive disease, documented by detection of hydronephrosis on renal ultrasonography or minimal urine production from the ureter on the affected side during cystoscopy, surgery has been effective.[63-67] In one mare, obstructive ureterolithiasis caused apparent lumbar pain, prompting stone removal twice 5 months apart, initially by a ureterolithectomy via a ventral midline approach and subsequently by using a basket retrieval device passed via the urethra into the ureter.[63] However, the mare succumbed to laminitis 3 months later, and bilateral nephrolithiasis was found at necropsy examination. Therefore, further recurrence would have been likely in this case. In a 3-year-old Thoroughbred colt, an obstructive ureterolith was successfully removed via endouroscopically-guided electrohydraulic lithotripsy, and the horse returned to a successful racing career.[64] Unilateral nephrectomy was reported to be a successful treatment for a renal abscess, pyelonephritis, or chronic hematuria associated with a nephrolith in three horses, all of which did not have azotemia and had an apparently normal contralateral kidney.[65-67] Accordingly, with appropriate case selection (lack of azotemia and unilateral disease), nephrectomy can be an effective treatment for nephrolithiasis in horses. In fact, in the absence of azotemia, nephrectomy may be the preferred approach for management of obstructive unilateral renal or ureteral calculi, because removal of the affected kidney should eliminate any associated upper UTI and the potential for recurrence.

Pyelonephritis

Upper UTIs involving the kidneys are rare in horses. The course of the distal segment of the ureters in the dorsal bladder wall creates a physical barrier or valve to prevent vesicoureteral reflux (VUR), a prerequisite for ascending pyelonephritis.[68] Problems that interfere with this barrier and increase the risk for VUR and associated upper UTIs include ectopic ureter or bladder distention, which may occur with bladder paralysis or urethral obstruction. Over time, VUR leads to progressive ureteral dilatation, renal scarring, and infection with commensal organisms. The role of recurrent lower UTI in the development of pyelonephritis is less clear because many cases of recurrent cystitis in other species never proceed to involve the upper urinary tract.[68] Because the kidneys are highly vascular organs, septic nephritis can also develop in association with septicemia in neonatal or adult horses.[69] Unless renal involvement is extensive, the upper UTI may go undetected but could lead to development of nephrolithiasis or CKD months to years later.

Pyelonephritis in horses has been described in association with urolithiasis and chronic cystitis with bladder paresis.[59,68,70] Other causes have included accidental amputation of the penis during castration,[71] foreign bodies in the bladder,[72] and lower urinary tract neoplasia.[73] With pyelonephritis, dysuria is manifested by hematuria or pyuria rather than by stranguria and pollakiuria (as for cystitis). In addition, horses with upper UTIs generally show other clinical signs, including fever, weight loss, anorexia, or depression.[68,74,75] As mentioned in the discussion of renal and ureteral calculi, upper UTIs can accompany nephrolithiasis and ureterolithiasis, and in affected cases it may be unclear which problem developed first.

Diagnostic evaluation includes physical and rectal examinations, urinalysis, and a quantitative urine culture. Careful palpation may allow detection of an enlarged ureter or kidney, although the kidney may also get smaller in long-standing cases. *Actinobacillus equuli*, *Streptococcus equi*, *Rhodococcus equi*, or *Salmonella* spp. are common isolates from horses with hematogenous nephritis.[68,69] In horses with upper UTIs, a complete blood count and serum biochemistry profile should be performed to assess the systemic inflammatory response and renal function. Cystoscopy (to evaluate urine flow from each ureteral orifice) and ultrasonographic imaging of the bladder, ureters, and kidneys are helpful adjunctive diagnostic procedures. Ureteral catheterization (by passing polyethylene tubing via the biopsy channel of the endoscope or by using a No. 8-10 French polypropylene catheter, which can be passed blindly in mares) may allow collection of urine samples from each ureter to distinguish unilateral from bilateral disease.[76]

Treatment for upper UTIs includes a prolonged course of appropriate systemic antibiotics (selected on the basis of susceptibility testing results on isolated pathogens, see Chapter 63). As for upper tract lithiasis, in carefully selected cases with unilateral disease, surgical removal of the affected kidney and ureter may be curative.[65,66] In addition to absence of azotemia, recovery of insignificant numbers of bacteria (fewer than 10,000 CFU/mL) from urine collected from the ureter leading to the unaffected kidney would be valuable information to collect before nephrectomy is pursued

Ureterorrhexis

Ureteral disruption appears to be an extremely rare problem in the adult horse with only three reported cases.[76-78] The cause appeared to be traumatic in a mare following dystocia and in a Thoroughbred gelding after a fall.[77,78] The third case developed iatrogenically during an attempt to enter a ureter transurethrally with a rigid instrument.[76] Unlike in foals, traumatic disruption was more distal toward the bladder and was unilateral in all cases. The postpartum mare presented for abdominal distention because of uroabdomen, and the gelding presented for colic signs with frequent posturing to urinate and uroabdomen was confirmed by a peritoneal fluid to serum creatinine ratio more than 2 in both cases. The diagnosis was made during an exploratory celiotomy in the mare and a ureteral stent was placed without attempting to repair the damaged ureter, because of tissue disruption and fluid (urine) accumulation around the tear. The stent was maintained for 3 weeks and the mare recovered uneventfully. A fluid-filled structure could be palpated dorsolateral to the bladder in the affected gelding, and this case was managed medically (without stenting). He recovered over the next few days with supportive care, and the fluid-filled structure resolved over the subsequent 2 months. In the final case, in which the ureter was traumatically damaged during instrumentation, the tear occurred in the terminal portion of the ureter within the bladder wall and the mare recovered with no specific treatment.

Renal Neoplasia

Renal neoplasms include adenomas, renal cell carcinomas, and nephroblastomas.[79] Renal adenomas are small, well-

circumscribed lesions in the renal cortex that are usually incidental necropsy findings. The most common renal tumor in the horse is renal cell carcinoma or adenocarcinoma.[80-84] These arise from proximal tubular epithelium in most cases. Affected horses usually have nonspecific presenting complaints including poor performance, depression, weight loss, and recurrent colic. Signs that increase suspicion of a primary urinary tract problem include hematuria (94%) and detection of a palpable mass on rectal examination (77%).[84] An occasional horse may have a large enough mass to cause protrusion of the paralumbar fossa or a hind limb gait abnormality and abnormal stance.

Renal cell carcinomas are typically unilateral, and normal renal function is maintained by the contralateral kidney. Although nephrectomy would be the treatment of choice, tumors are typically too large and adherent to surrounding organs by the time they are detected. Accordingly, surgical removal is usually not possible and in one case in which it was attempted, uncontrollable abdominal intraoperative hemorrhage resulted in a decision for euthanasia.[84] Frequent metastases to the lungs and liver are another indication that renal adenocarcinoma is usually not a treatable disease.[84] As an example, in the only other surgical case, a nephrectomy was performed via a flank laparotomy but euthanasia was performed 7 months later because of respiratory difficulty associated with metastases.[83]

In a report of 27 horses with renal adenocarcinoma, only six cases were diagnosed during life (percutaneous biopsy in two and a surgical biopsy in four) but renal ultrasonography was markedly abnormal in all cases in which it was pursued (Figure 64-5).[82,84] Only 2 of 27 horses were discharged from the hospital. The poor prognosis can be attributed to the fact that clinical signs of intra-abdominal neoplasia in horses are often not be apparent until the disease is quite advanced.[85] In one report of renal carcinoma, clinical signs of the tumor were absent until the horse was anesthetized for laryngeal surgery. After an uncomplicated surgery, the horse was repositioned for recovery but died shortly thereafter. Compression of the caudal vena cava by a large renal carcinoma was suspected to lead to a decrease in venous return as the cause of sudden death.[86]

Other neoplastic diseases that may affect the kidneys include nephroblastoma, transitional cell carcinoma, and squamous cell carcinoma.[79,84,85,87] Nephroblastoma (Wilms' tumor) is an embryonal tumor that arises in primitive nephrogenic tissue or in foci of dysplastic renal tissue; the latter tumor types arise from the uroepithelium of the renal pelvis or ureter.[79,87] Neoplastic involvement of the upper urinary tract may also occur with dissemination of lymphosarcoma, hemangiosarcoma, melanoma, or adenocarcinoma arising from other tissues within the abdomen.[88] Although they are not truly cancerous disease processes, mucinous hyperplasia of the renal pelvic and proximal ureteral uroepithelium or ureteropelvic polyp formation can also lead to development of a soft tissue mass in the area of either kidney, ureteral obstruction, and hydronephrosis.[89-91]

SURGICAL PROCEDURES

J. Brett Woodie

Renal Biopsy

Harold C. Schott II

Renal biopsy is a controversial diagnostic procedure but it may provide useful information in select cases.[92-94] Although percutaneous biopsy is considered a reasonably safe procedure when performed with ultrasonographic guidance, it carries risks including perirenal hemorrhage or hematuria and, less commonly, penetration of bowel. In humans, perinephric hematomas are detected by CT imaging in 57% to 85% of patients shortly after biopsy. Microscopic hematuria occurs in virtually all patients for the first couple of days after biopsy, but gross hematuria is observed in less than 10% of patients. Most of these complications are inconsequential, but postbiopsy transfusions have been required in up to 3% of patients, and one death has been reported since 1980.[92] Similarly, in seven normal horses subjected to bilateral renal biopsy, macroscopic (1/7) and microscopic (4/7) hematuria were observed in five and extensive perirenal hemorrhage was found at necropsy examination of four horses subjected to euthanasia 8 hours following biopsy. Evidence of perirenal hemorrhage was still identified at necropsy in a fifth horse euthanatized 9 and 27 days following biopsy (of either side).[94] A recent retrospective study of 151 renal biopsies collected from 146 horses found a complication

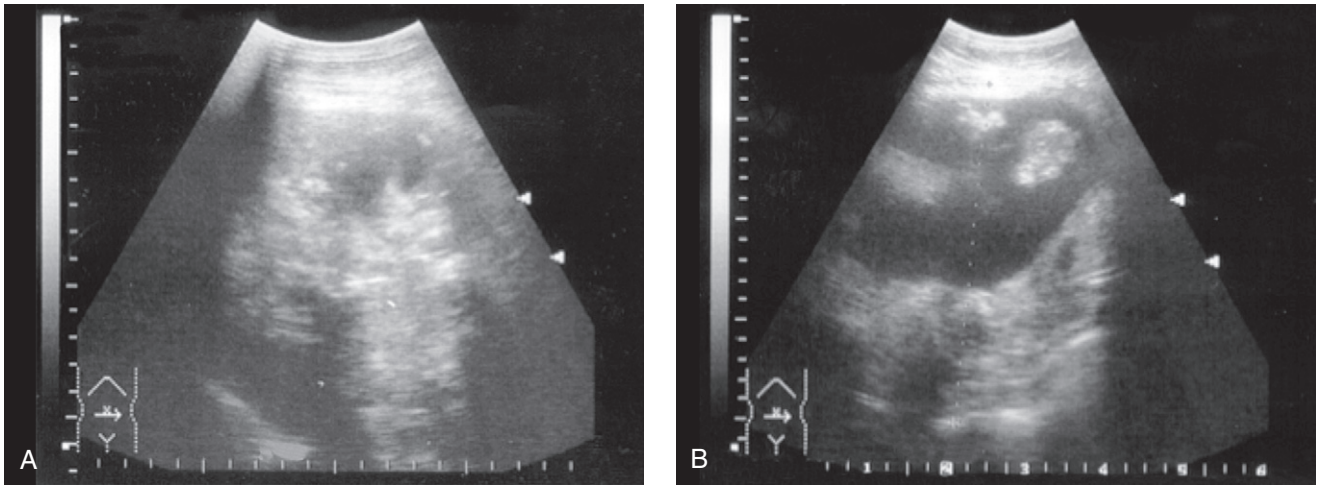


Figure 64-5. Ultrasonographic images of the right kidney of a horse with renal adenocarcinoma. **A,** Little evidence of normal renal architecture remains. **B,** Multiple areas of hypoechoic fluid are apparent within the kidney.

rate of 11.3%: macroscopic hematuria in 3% but a significant decline in packed cell volume in 5%; colic in 4%; one urethral obstruction because of a blood clot in the bladder; one death from hemorrhage; and one abortion of a 60-day fetus in a mare anesthetized for open surgical biopsy.[95] An adequate sample for histopathologic evaluation was collected in 93% of these cases, but the impact of the biopsy results on patient management was not assessed in this report.

Most renal biopsies are collected percutaneously after use of ultrasonography to locate the most appropriate biopsy site (blind biopsy after ultrasonographic localization) and this is the preferred technique in the horse for suspected diffuse renal disease. When a focal abnormality is detected on ultrasonography, laparoscopic-guided or open renal biopsy are more likely to result in collection of the desired abnormal tissue and should be considered. Because renal biopsy is not without risk, the procedure should be approached with caution and pursued only when results could substantially alter the therapeutic plan or prognosis. There is limited information about the impact of renal biopsy results on therapy and outcome of renal disease in humans; however, in one prospective study biopsy results were found to influence physicians' decisions on about half of the cases.[96] In general, renal biopsy is pursued more aggressively in humans with acute renal insufficiency than in those with chronic renal insufficiency, especially when it is difficult to determine the type of renal disease based on results of urinalysis and sediment examination.[92]

In the equine patient, a renal biopsy is performed with the horse sedated and restrained in stocks. With bilateral disease, biopsy of the right kidney is preferred because of its superficial location. After locating the right kidney via ultrasonography, overlying hair is clipped and the site, typically over the 15th to 17th intercostal spaces, is surgically prepared. Local anesthetic is infused subcutaneously followed by a deep stab incision with a No.15 blade to minimize drag of the skin and subcutaneous tissues on the biopsy instrument (a Tru-cut biopsy needle or, preferably, a spring-loaded, triggered biopsy device). Penetration of the needle into the renal parenchyma can often be appreciated by a change in tissue resistance, and the tip of the needle should be advanced about 1 cm deep into the kidney (depth can also be determined via ultrasonographic measurement immediately prior to biopsy). One biopsy sample should be placed in formalin for histopathologic evaluation, and additional samples can be collected for bacterial culture or for specialized microscopic examination, such as by immunohistochemistry or electron microscopy; appropriate sample processing should be determined beforehand by contacting the pathologist who will examine the biopsy samples. Because of the risk of hemorrhage, a minimum number of samples should be collected.

In contrast to humans and small animals, in which the biopsy instrument is angled tangentially in an attempt to collect predominantly cortical tissue, the biopsy needle is generally inserted perpendicular to the equine kidney when the procedure is performed percutaneously. Routine analysis of a renal biopsy in human (and now small animal) medicine typically includes multiple stains of ultrathin sections for light microscopy, immunohistochemistry, and electron microscopy, with an emphasis on evaluation of glomeruli. Unfortunately, no laboratory currently offers detailed evaluation of equine renal biopsies. Additionally, equine renal disease is more often tubulointerstitial in origin than glomerular. When these additional limitations are considered, clinicians should have specific questions in mind before renal biopsies are performed in horses. For example, a biopsy may be useful for establishing a diagnosis of renal dysplasia in a younger horse with CKD or of neoplasia versus infection in a kidney with a focal ultrasonographic abnormality. In the latter instance, a laparoscopically guided or open renal biopsy would have a greater chance of recovering a diagnostic biopsy sample. Specific treatment following a renal biopsy is not required, although patients should be carefully monitored for complications for 1 to 3 days following the procedure.

Nephrectomy

Unilateral right nephrectomy is performed through a right 16th or 17th rib resection or, alternatively, at the 16th and 15th intercostal spaces.[97,98] Depending on the degree of anatomic variation, the most cranial approach can create a plane of dissection across the dorsal aspect of the costophrenic angle and through the diaphragm to the kidney. Although transthoracic approaches have been used successfully, they are more complicated than the right 17th rib resection approach and are not recommended. In the foal, a ventral midline approach can be used for unilateral nephrectomy but in the adult horse this is not feasible.[97]

In the transcostal approach, the horse is placed under general anesthsia, positioned in lateral recumbency, clipped, and prepared for aseptic surgery. Positive pressure ventilation should be available in case the thoracic cavity is entered inadvertently. This complication occurs more commonly on the right side of the horse.[97] A 30- to 40-cm (12- to 16-inch) skin incision is made over the 16th or 17th rib. Dissection is continued through the serratus dorsalis caudalis and the external abdominal oblique muscles to expose the perisoteum of the rib. The periosteum is incised and elevated around the rib, taking care to avoid injury to the intercostal vasculature. A Doyen rib raspatory is helpful for this dissection. The rib is transected 2 to 5 cm distal to the costovertebral articulation using a bone saw or Gigli wire (Figure 64-6). Ventrally, the rib is disarticulated at the costochondral junction. Ronguers and a bone rasp should be used to smooth the end of the bone at the proximal extent of the incision. The medial costal periosteum is longitudinally incised, and the kidney is exposed by blunt dissection through

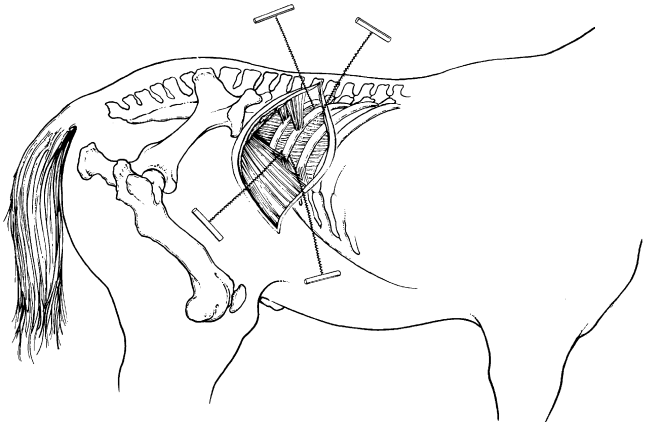


Figure 64-6. Resection of the 17th rib permits surgical access for right nephrectomy or nephrotomy procedure.

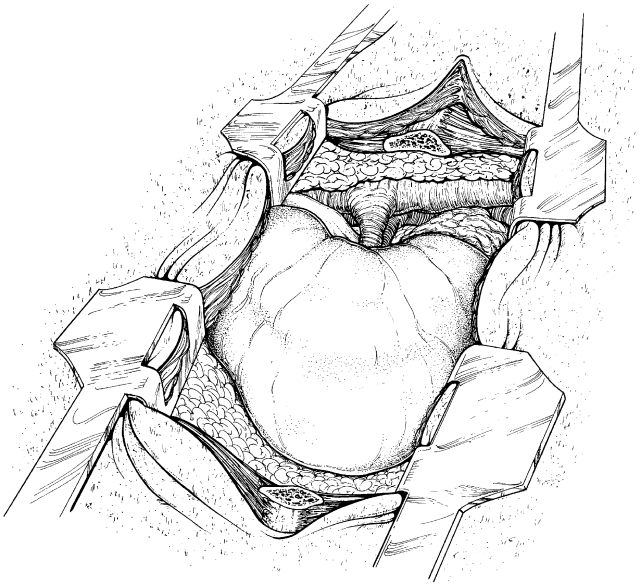


Figure 64-7. All perirenal fat is removed by blunt dissection to permit access to the ureterovascular pedicle.

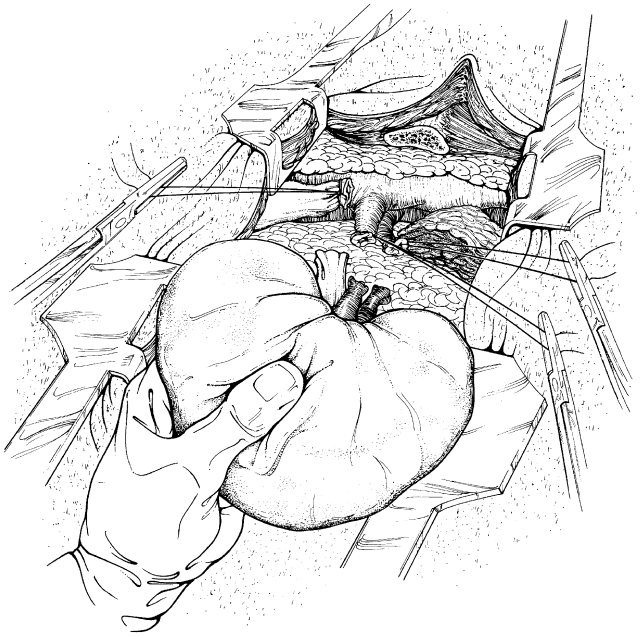


Figure 64-8. The renal artery, vein, and ureter are ligated with transfixing ligatures before resection and removal of the kidney.

the retroperitoneal fat. If additional exposure is required, the incision can be extended ventrad.

The kidney is mobilized by digital circumferential dissection through perinephric fat to expose the ureterovascular pedicle and penetrating capsular vessels (Figure 64-7). A self-retaining retractor is helpful to aid in exposing the kidney. Small capsular vessels and accessory renal arteries should be ligated. Electrocautery can be used if appropriate for the size of the vessel. The ureterovascular pedicle is isolated, and the artery, vein, and ureter are individually double-ligated (Figure 64-8). The use of hemostatic vascular clips or vascular staples facilitates removal of the kidney and provides adequate access for suture ligation of major vascular elements.

After removal of the affected kidney, the renal fossa is lavaged and again evaluated for evidence of hemorrhage. The ureter is mobilized, ligated as far distad as possible, and transected. Resection of the pelvic ureter is not possible when using a flank approach for nephrectomy. Either Penrose or closed suction drains are placed after resection of the kidney to evacuate blood accumulating in the dead space or to manage urine-contaminated tissues.

The periosteum of the rib and deep fascia are closed with a synthetic absorbable suture material placed in a simple-interrupted or simple-continuous pattern. The subcutaneous tissues and skin are closed routinely. Unilateral left nephrectomy of the horse is performed in similar fashion using either a 17th or 18th rib resection or a dorsal flank incision.[21,99,100]

Laparoscopic Nephrectomy by Hand-Assisted Techniques

Laparoscopic nephrectomy in the horse has been described.[101] An ipsilateral flank approach is used to remove the respective kidney. At least three portals are required. The first portal is made between the 17th and 18th ribs at the ventral border of the tuber coxae. The second portal is located midway between the last rib and the dorsocranial border of the tuber coxae. The third portal is prepared approximately 8 cm (3 inches) ventral to the second portal. A 30-cm (12-inch) long, 10-mm (4-inch) diameter, 0-degree laparoscope is used. The procedure for removing the left kidney starts with injection of epinephrine (1 mg in 10 mL of saline) in three or four sites at the dorsal border of the spleen to create splenic contraction. This enlarges the space in which the surgeon has to work. The perirenal fascia is injected dorsally with 20 mL of 2% mepivacaine. A monopolar electrocautery hook blade is used to create a plane of dissection dorsal to the kidney. Perirenal fat is dissected and removed with curved laparoscopic scissors. The hilus of the kidney is carefully dissected free to identify the vessels. Specialized (clockwise and counterclockwise) laparoscopic ligation instruments are used to ligate the renal artery, vein, and ureter, in that order. The area is assessed for hemorrhage prior to making a small flank incision to retrieve the kidney. Complications include pneumothorax and bleeding from accessory renal arteries. This is a technically demanding procedure and requires practice before attempting it on clinical patients.

The first hand-assisted laporoscopic nephrectomy in human medicine was described in 1997.[102] The surgical technique has been described for the left and right kidney in the standing horse.[103,104] Preparation of the horse for this surgery includes withholding feed for at least 12 hours. The administration of perioperative antibiotics and analgesics is at the discretion of the surgeon. The paralumbar fossa corresponding to the kidney that is to be removed is clipped and surgically prepared for aseptic surgery. Administration of a systemic α_2-agonist provides sedation. The paralumbar fossa is desensitized by local infiltration with mepivacaine hydrochloride. Insufflation of the abdominal cavity is not required because of the open approach. A 10- to 12-cm vertical skin incision is made in the paralumbar fossa beginning 5 to 8 cm below the dorsal border of the internal abdominal oblique muscle. The external abdominal oblique

muscle is sharply incised and a modified grid approach is used to gain access to the peritoneal cavity. The peritoneum is sharply incised. A laparoscopic viewing portal is made dorsal to the proximal border of the flank incision. An instrument portal is created 4 to 6 cm cranial and 2 to 3 cm dorsal to the laparoscopic portal.

A 0-degree laparoscope is used for viewing the surgical site, and a hand within the abdomen is used for retraction and blunt dissection. The retroperitoneal space caudoventral to the kidney is infiltrated with 15 to 20 mL of mepivacaine followed by digital massage to facilitate distribution of the local anesthetic throughout the retroperitoneal space. Laparoscopic scissors are used to sharply incise the peritoneum caudal to the kidney. This incision is enlarged by manual dissection to expose the kidney. Careful manual dissection is used to completely identify all of the vascular structures entering and leaving the kidney. The renal artery and vein are double-ligated separately, using size 2 polyglactin 910 with one-handed ties. The vessels are transected using laparoscopic scissors and the kidney is delivered out of the flank incision with the ureter intact. The ureter is double-ligated and transected. The renal vessels are inspected for hemorrhage followed by routine closure of the paralumbar incision in several layers.

The most immediate and severe complication is uncontrollable hemorrhage, which generally originates from a torn accessory branch of the renal artery. Benefits from hand-assisted procedures include: tactile sensation to facilitate dissection, hand retraction, smaller surgical incision, decreased surgical time, and less surgical morbidity, in addition to eliminating the need for general anesthesia.

Nephrotomy

Nephrotomy is not considered a benign or potentially less complicated surgical procedure than nephrectomy. In dogs, it results in a 20% to 50% reduction in renal function.[105,106] In equine patients, nephrotomy is performed less commonly than nephrectomy because indications are fewer and the degree of technical difficulty is substantially greater.

The surgical approach for nephrotomy is similar to that used for nephrectomy. The procedure is technically difficult, given the depth and dimensions of the surgical field and the reduced ability to see the surgical site intraoperatively, complicated by hemorrhage from the penetrating capsular vessels. In canine patients, the approach is through a longitudinal sagittal incision in the convex lateral surface of the kidney.[105,106] To make such an incision, the kidney must be dissected free from the perirenal fat. This disrupts multiple small penetrating capsular vessels that require hemostasis.

After the kidney is mobilized, it may be pivoted about the ureterovascular pedicle to expose the convex surface. Before incising the cortex, major renal vessels are temporarily occluded with noncrushing vascular forceps or Rummel's tourniquets. The renal incision is extended to expose the collecting system and the renal pelvis. Obstructive lesions of the renal pelvis (e.g., renal calculi) are removed, and the collecting system is lavaged. The incision is made just large enough to allow removal of the calculi. Less damage is done to the renal parenchyma if the renal capsule is incised with a blade and forceps are used to bluntly separate the parenchyma.[106] The associated ureter should be cannulated with a size 10 to 15 French polyethylene catheter to ensure patency.

Canine nephrotomy incisions are closed by gently pressing the renal halves together with sustained pressure. The renal capsue is closed with 3-0 or 4-0 synthetic absorbable suture placed in a simple continuous pattern.[106] Although pyelotomy has been suggested as a reasonable approach to the canine renal pelvis, its small size increases the risk of accidental transection of an interlobar artery. Likewise, the close proximity of the renal artery and veins would make pyelotomy a difficult and risky procedure to perform in equine patients.

Ureterotomy

Indications for ureterotomy in the horse are limited principally to obstructive urolithiasis. Presentation of uncomplicated cases of ureteral calculi for surgical treatment is rare. Horses are often chronically affected and have developed some degree of renal pathology. Some horses with sufficient renal mass remain asymptomatic, and the condition is diagnosed as an incidental finding at necropsy.

Horses with identifiable ureteral pathology may be explored through a flank laparotomy, or through caudal ventral midline laparotomy in the mare. Exposure is difficult and extremely limited over the caudal course of the ureter. Typically, lesions are located in the proximal third of the ureter. When lesions can be identified and exposed, the ureter is incised proximal to the obstruction, and the contents are evacuated with surgical suction. The urolith is removed and the ureter is closed in a simple-continuous pattern with small-diameter synthetic absorbable sutures. Silastic tubing may be introduced into the ureter as a stent over which the ureteral repair is performed. When direct surgical intervention is not possible, the use of a grasping basket (Dormia Stone Dislodger) can facilitate closed dislodgment of a ureterolith.[63] The instrument is introduced into the ureteral orifice by direct insertion in mares or under videoendoscopic control using a perineal urethrotomy in males (see Chapter 66). Guidance by rectal palpation is desirable to manipulate the dislodger into a position proximal to the urolith. The dislodger is subsequently opened and retracted to ensnare the urolith and displace it distad using slow, gentle traction.

Repair of ureterorrhexis is approached in similar fashion.[48,77,107] In a case report, successful repair of a traumatic ureteral tear in a postpartum mare was accomplished with an indwelling polyethylene tubing stent (outside diameter, 1.90 mm).[77] The mare had evidence of uroperitoneum and associated electrolyte abnormalities (hyponatremia and hyperkalemia). A ventral midline celiotomy was used; however, because of the accumulation of urine around the ureter and broad ligament, the rent within the ureter could not be identified for primary repair. The surgery was assisted with the use of videoendoscopy of the urinary bladder. This allowed the surgeon to accurately place the stent within the ureter past the presumptive location of the rent. The stent was checked for urine production and subsequently anchored to the vaginal mucosa and perineal skin and kept in place for 21 days. In other reports neonates developed uroperitoneum secondary to a ureteral defect.[48,107] Urine accumulation was also detected in the retroperitneal space. The peritoneum was incised over the swelling and the ureter was exposed. A cystotomy extending from the apex to the neck of the bladder was made. A size 8 French polypropylene catheter was advanced retrograde through the ureteral opening for a short distance and Evans blue dye was injected into the catheter

to locate the defect in the ureter. When the defect was located the catheter was advanced proximal to the defect and the rent was closed using 4-0 or 5-0 suture material. Although a few prosthetic constructs are available for use as ureteral stents in human medicine, the optimal size (7 to 9 French in humans) and material have yet to be determined for the horse and will likely not be determined scientifically because of the rarity of the condition.

Neoureterostomy

Neoureterostomy techniques are used in the horse to manage ectopic ureters. In human and canine patients, vesicoureteral anastomosis has traditionally been the method of choice for treatment of ectopic ureters and selected cases of ureteral avulsion. Complications with these techniques in equine patients prompted treatment of foals by unilateral nephrectomy, assuming that the contralateral kidney was normal.[21,22] Surgical success can be obtained, however, in large horses.

The surgical approach is made through a caudoventral midline incision, extended to the pubis for exposure of the bladder. The viscera are packed off craniad in the abdomen, and the ureter is identified as it traverses the dorsolateral bladder. The ectopic ureter can be identified by retrograde catheterization. If the ectopic ureter is in close proximity to the dorsal bladder, a side-to-side or end-to-side extravesicoureteral anastomosis may be performed to create a new ureteral opening along the dorsolateral cranial base of the bladder. The distal ureter is ligated and dissected free unless it is in an intramural location and surgically inaccessible.

Alternatively, if the ectopic ureter is not in close proximity to the bladder, the ureter may be ligated distad and neoureterostomy achieved by intravesicular anastomosis (Figure 64-9). Tunneling the ureter creates a functional equivalent of a distal ureteral valve, which reduces the likelihood of vesicoureteral reflux.[106] Apposition of the ureteral and vesicular mucosa with 3-0 or 4-0 synthetic absorbable suture material is ideal (Figure 64-10).

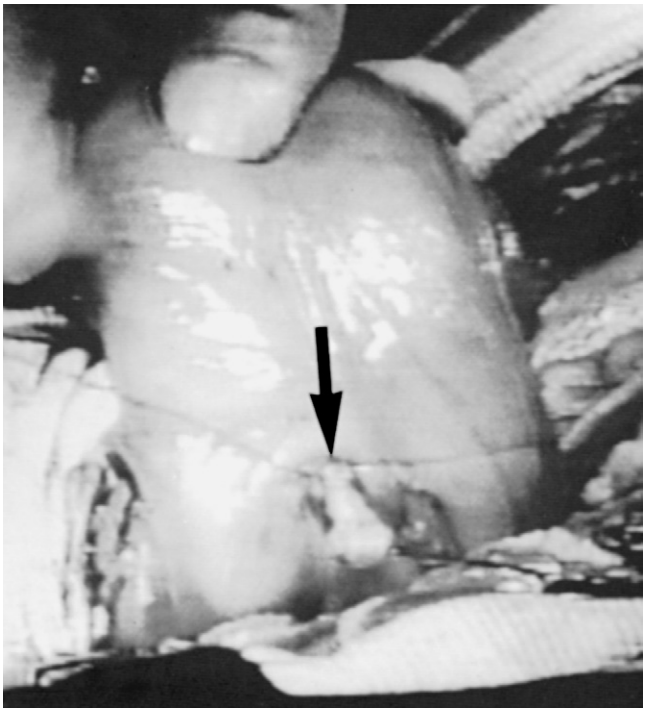


Figure 64-9. Ligatures are placed to secure a ureter *(arrow)* after translocation to the bladder from an ectopic site. The ureter has been tunneled through the seromuscular layer of the bladder to prevent vesicoureteral reflux. (Courtesy Candace Lundin, DVM.)

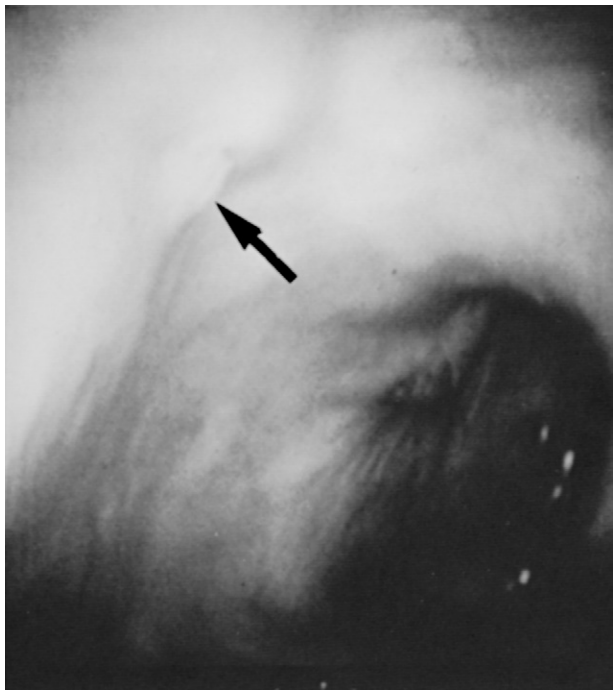


Figure 64-10. Endoscopic appearance of the ureterovesicular anastomosis demonstrated in Figure 64-9. The ureteral stoma can be observed on the dorsolateral wall of the bladder *(arrow)*. (Courtesy Candace Lundin, DVM.)

Aftercare

After all renal surgery, horses should be monitored for adequate water consumption and urine production. The horse should be supported with intravenous fluids. Serum electrolytes and clearance ratios may reveal transient imbalance in electrolyte clearance after nephrectomy, particularly with regard to potassium. Fluid therapy should be adjusted accordingly.

Prophylactic antibiotics are indicated during the perioperative period because this renal intervention is a clean-contaminated surgery. When septic conditions are encountered, antibiotic therapy should be directed by the results of intraoperative culture and sensitivity results. Dosages should be adjusted for the reduction in renal mass when nephrotoxic antibiotics must be used. Depending on the drug, serum levels can be monitored for peak and trough levels to minimize the possibility of renal damage.[108]

Complications

The patient should be monitored for the development of pneumothorax during surgical dissection. Although inherently a part of the transthoracic approach, pneumothorax can develop

when a flank approach using rib resection disrupts the crura of the diaphragm. If this occurs the defect in the diaphragm should be sutured to the adjacent costal musculature to seal the pleural cavity.[97] Access to a mechanical ventilator may be required immediately in the event that the pleural space is opened during the flank approach to the kidney.

Other complications of renal surgery include postoperative hemorrhage and infection. Management of postoperative hemorrhage is based on prevention through application of meticulous surgical technique for isolation and cautery or ligation of contributing and emerging vessels. Assessment of packed cell volume and coagulation defects is appropriate before undertaking renal biopsy or excisional techniques. Surgical drainage is appropriate whenever diffuse hemorrhage, seroma, or bacterial contamination is anticipated.

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CHAPTER 65

Bladder

Harold C. Schott II and J. Brett Woodie

ANATOMY AND PHYSIOLOGY

Harold C. Schott II

The bladder is a highly distensible organ capable of holding 4 L or more of urine in a 500-kg horse. When empty, the bladder may lie entirely in the pelvic canal, and when full it drops over the pelvic rim to extend to the level of the umbilicus.[1] When the bladder is full, the lateral ligaments are easily palpated on either side of the bladder on rectal examination. Their cranial free edges, remnants of the umbilical arteries, are called the *round ligaments of the bladder.*[2] The bladder is covered with peritoneum cranially at the apex and with adventitial tissue in the remainder of the retroperitoneal space. The bladder wall contains two smooth muscle layers: (1) an outer layer of longitudinal to obliquely arranged muscle fibers and (2) an inner layer of transversely or circularly arranged muscle fibers.[2] The outer and inner layers are partly interwoven around the bladder; however, at the dorsal aspect the circular layer becomes external to the longitudinal layer. This anatomical arrangement of muscle fibers has been suggested to allow the bladder to distend to a great extent dorsad as it fills, thereby causing less interference with the intestines. However, it also makes the dorsal wall inherently weak and the likely site of rupture with excessive bladder distention.[3] The outer and inner muscle layers are both arranged longitudinally toward the bladder neck and act in concert to close the internal urethral orifice. The musculature of the bladder wall is continuous with an outer longitudinal and an inner circular layer of smooth muscle surrounding the pelvic urethra. Lateral and ventral to the smooth muscle is the striated urethralis muscle. Together these muscles form the urethral sphincter.[2] The bladder is lined with transitional epithelium overlying a thick submucosa that allows considerable stretching.[1]

Sympathetic innervation to the bladder is provided via the hypogastric nerve, with preganglionic fibers arriving from spinal segments L1 to L4 to synapse in the caudal mesenteric ganglion. Postganglionic fibers supply the bladder (β_2-adrenergic receptors) and proximal urethra (primarily α_1-and some α_2-adrenergic receptors).[4,5] In addition to adrenergic innervation, the equine bladder is also innervated by cholinergic and peptidergic nerve fibers.[6] Parasympathetic innervation originates in the sacral segments of the spinal cord with neurons joining to form the pelvic nerve.[4,5] Somatic innervation of the lower urinary tract is primarily to the striated urethralis muscle sphincter via a branch of the pudendal nerve, which originates from the sacral cord segments (S1 to S2).[1] The smooth muscle of the bladder, referred to as the *detrusor muscle,* is innervated by the parasympathetic pelvic nerve and β_2-adrenergic postganglionic fibers.

The bladder has a *filling/storage phase* as well as an elimination phase.[7] During filling there is an increase in tone of the smooth and striated muscles of the urethral sphincter to maintain continence.

Sympathetic nerve activity is dominant during filling/storage and the detrusor muscle relaxes owing to α-receptor–mediated inhibition of pelvic nerve afferents and stimulation of sympathetic β_2-receptors in the smooth muscle of the bladder. The latter is a reflex response that involves sensory input from bladder stretch and pressure receptors via afferent pelvic nerve fibers to the sacral cord, interneurons in the cord, and pre- and post-ganglionic sympathetic axons in the hypogastric nerve. Relaxation of this muscle allows accumulation of a large volume of urine with little increase in intravesicular pressure. As detrusor muscle fibers reach their limit of stretch, intravesicular pressure starts to rise, and stretch receptors transmit signals via the pelvic nerve and ascending spinoreticular tracks to the pons, cerebrum, and cerebellum. Bladder fullness is sensed, and signals for voluntary micturition are initiated in the cerebrum, beginning the *elimination phase*. These signals are transmitted via brainstem upper motor neurons descending in reticulospinal tracts to sacral parasympathetic nuclei, parasympathetic ganglia in the pelvic plexus or bladder wall, and postganglionic fibers, triggering detrusor contraction. Depolarization waves spread throughout the bladder via tight junctions, resulting in a strong, coordinated detrusor contraction. Concurrent inhibition of the pudendal nerve and hypogastric α- and β_2-sympathetic activity leads to relaxation of the urethral sphincter and facilitates detrusor muscle contraction and elimination of urine. Micturition ends when detrusor stretch receptors cease firing and pelvic nerve efferent activity stops. At that time, sympathetic nerve and pudendal nerve activity resumes for the next storage/filling phase.

DISORDERS REQUIRING SURGERY

Harold C. Schott II

Disorders of the bladder that may require surgery include uroperitoneum, in both foals and adult horses, as well as patent urachus and other neonatal bladder problems. In adult horses, cystolithiasis is the most common surgical disorder of the bladder. Other less common problems that may require surgical intervention include prolapse or eversion and neoplasia.

Uroperitoneum

Uroabdomen can develop as a result of leakage from a ureter (see Chapter 64), the bladder, or urachus.

Foals

In foals, uroperitoneum most commonly develops after bladder rupture during parturition in colts, with prevalence ranging from 0.5% to 2.5%.[3,8-10] The commonly accepted explanation is that a high intravesicular pressure that develops during passage through the pelvic canal during parturition, coupled with occlusion of the urethra, leads to bladder rupture in colts that are delivered with a moderately full bladder. However, presence of posterior urethral valves is the most common cause of lower urinary tract obstruction in male infants and can cause bladder rupture, vesicoureteral reflux, and hydronephrosis, but this

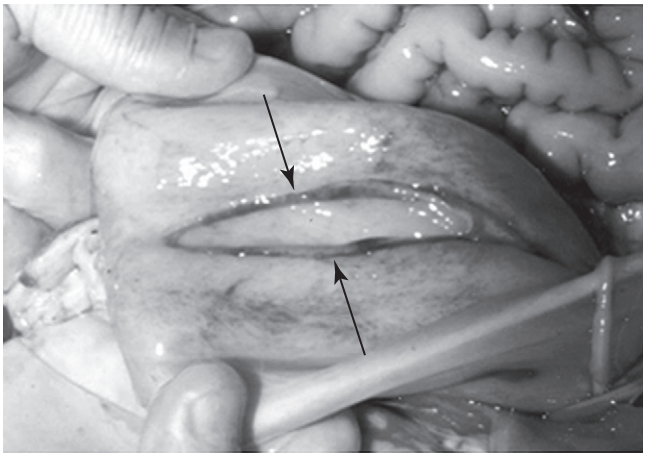

Figure 65-1. Longitudinal bladder tear in the dorsal aspect of a 3-day-old colt *(arrows).* This is the typical surgical finding in a foal that develops uroabdomen consequent to bladder rupture sustained during parturition.

problem has not yet been recognized in horses.[11] Bladder tears are typically 2 to 5 cm in length on the dorsal surface with margins that are hemorrhagic and edematous (Figure 65-1).[3] Surgical exploration also has revealed ventral bladder tears in some foals[8,12] or bladder defects that have smooth margins with no evidence of traumatic disruption,[8,10,13,14] as well as apparent absence of portions of the bladder.[15] Although uncertain, it has been suggested that these latter defects may be developmental anomalies rather than ruptures.

Excessive bladder distention or megavesica has also been described in stillborn[16] and neonatal foals with abdominal distention with or without bladder rupture.[12,17] *In utero* bladder distention suggests lower tract obstruction, but a definite cause of obstruction was not found in these cases. However, an excessively long umbilical cord (more than 85 cm) may lead to intermittent urachal obstruction,[16] but in this instance, urine produced *in utero* should be able to pass into the amniotic cavity via the urethra. Bladder distention causing abdominal distention can be confused with uroabdomen. For example, an enlarged, flaccid bladder was found in a foal undergoing exploratory celiotomy for suspected uroabdomen. Half of the excessively distended bladder was removed and the foal survived.[12] Anomalous fusion of the bladder to the inner umbilical ring, with absence of the urachus, prevented bladder emptying and caused abdominal distention because of megavesica in another report.[18] The bladder was separated from the umbilical ring during surgical exploration to restore normal anatomic and functional integrity of the bladder. Abdominal distention has also been described in an older foal with a markedly distended bladder attributed to an adhesion of the bladder to the urachus.[19] In a colt seen by me, megavesica with uroabdomen was corrected by surgical removal of a large portion of the bladder, but the foal developed recurrent colic after weaning and a narrowing of the distal small colon was found at necropsy. Thus, megavesica remains a poorly understood condition and, when detected, warrants careful evaluation for other congenital anomalies.

Bladder distention is also recognized in foals with perinatal asphyxia syndrome (PAS), especially when recumbent.[12,20,21] Ultrasonographic examination may reveal either an enlarged bladder or incomplete bladder emptying, and both sexes appear to be at similar risk for this problem. Temporary use of an indwelling bladder catheter to keep the bladder empty is useful to decrease the risk of rupture, but ascending urinary tract infection may be a complication. Localized sepsis of the urachus in these patients, as well as foals with septicemia, can lead to urachal urine leakage.[12,20,21] Urine may leak into the abdomen, resulting in uroperitoneum, or externally to cause a patent urachus. Urine can also leak into the subcutaneous tissues and produce a plaque of edema (actually urine) extending from the umbilicus.[8,12,22]

Foals with ruptured bladders sustained during parturition often appear healthy and nurse well for the initial 24 to 48 hours after foaling. Early signs include a decrease in nursing vigor and lethargy followed by progressive abdominal distention and intermittent colic signs.[8-10] Some foals repeatedly posture to urinate, but this behavior can be confused with meconium retention. Subtle differences in straining observed with these two problems may be that colts with uroabdomen have a dropped penis and intermittently pass small amounts of urine, whereas foals with meconium impaction often show more vigorous tail elevation and wagging. The rate at which urine accumulates in the abdomen depends on the size of the defect. Clinical signs with smaller tears, usually in the area of the urachus, may not develop until 3 to 7 days of age.

Clinicopathologic assessment commonly reveals hyponatremia, hypochloremia, and moderate to marked hyperkalemia along with azotemia.[8-10,23,24] These electrolyte alterations may lead to development of fine muscle tremors and neurological deficits.[8,25] Uroabdomen can be confirmed by measuring a ratio of peritoneal fluid to serum creatinine more than 2.[8-10,23,24] More frequently, uroabdomen becomes highly suspect when a large amount of free abdominal fluid is imaged via transabdominal ultrasonography (Figure 65-2), such that confirmatory peritoneal fluid analysis is not consistently pursued.

Because foals with PAS or septicemia are often recumbent, frequency of urination can be difficult to assess, and presence of neurological deficits can be attributed to hypoxic-ischemic encephalopathy or meningitis. Recumbent foals may initially have a dry umbilicus that again starts to leak urine, especially when they are unable to void effectively and have chronic bladder distention. Silent uroabdomen can develop with urachal leakage into the abdomen and may not be detected until abdominal distention becomes apparent. However, this is uncommon because most hospitalized neonates are reassessed once or twice daily with abdominal ultrasonography. As a result, increasing amounts of free peritoneal fluid are often detected before gross abdominal distention develops. In these earlier stages of uroabdomen, alterations in serum electrolyte concentrations and azotemia are less severe, but peritoneal fluid creatinine concentration should still be substantially greater than that of serum.[11,20,21]

Adult Horses

Bladder rupture in adult horses is less common than in foals but has been recognized following parturition[23,26-31] and with obstructive problems including urolithiasis[32-36] and penile hematoma.[37] It has also been observed after blunt abdominal trauma[38] as well as after repair of bladder rupture[39] or treatment of urachal sepsis as a neonate.[40] I also have seen uroabdomen develop following anesthesia for magnetic resonance imaging, and weakness at the bladder apex was a suspected contributing

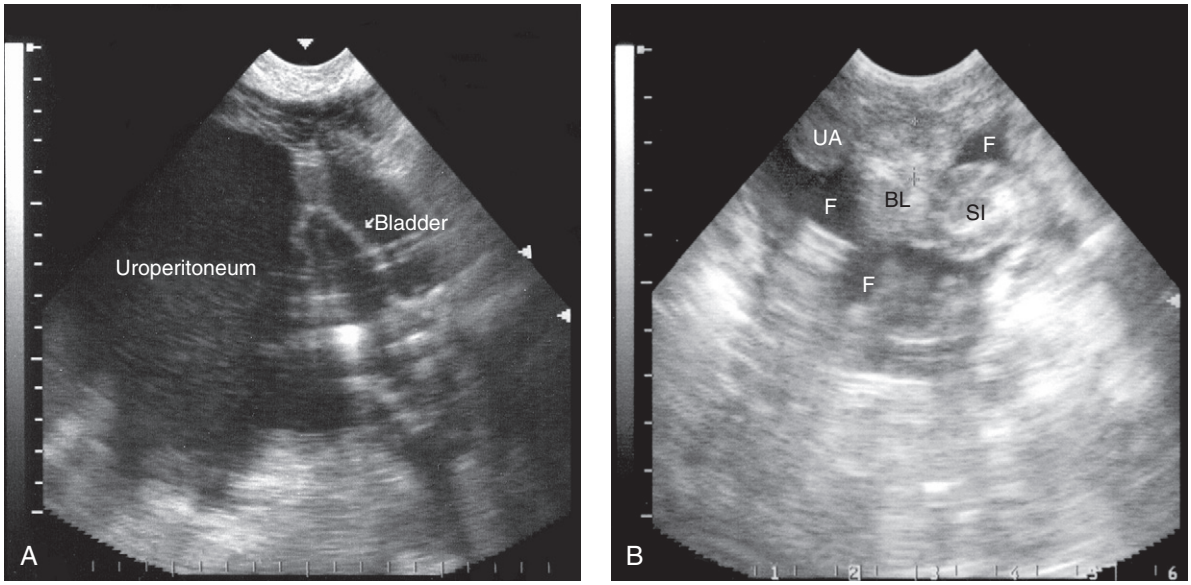

Figure 65-2. Transabdominal ultrasonographic images at the level of the umbilicus in foals. **A,** A large amount of free peritoneal fluid is present in moderate to severe uroabdomen; **B,** Only a modest amount of fluid is visible as would be imaged during the early stages of uroabdomen.

factor. Dystocia or assisted delivery has only been reported in a few mares with postpartum uroabdomen[29,30] and the incidence of this complication of parturition was reported to be 1 in 10,000 births in one large broodmare practice.[31] Clinicopathologic findings of azotemia and alterations in serum electrolyte concentrations in adult horses with bladder rupture are similar to those found in neonates with uroabdomen.

Presurgical Considerations

CORRECTION OF HYPERKALEMIA

Although surgical correction of a bladder tear or resection of the urachus is the preferred treatment for uroabdomen, patients need to be stabilized medically before they are anesthetized for surgical repair. Two main problems that need to be addressed are (1) hyperkalemia, and (2) abdominal distention with urine. Serum potassium concentration is variable with uroabdomen; therefore measurement of electrolyte concentrations is an essential part of case evaluation.[8-10,12,20,21,24] Clinical indicators of significant hyperkalemia ($[K^+]$ > 5.5 mEq/L) can include fine muscle tremors or cardiac arrhythmias.[8,41] When present, hyperkalemia should be addressed by antagonizing the effects of potassium on excitable membranes, redistributing potassium from extracellular fluid to intracellular fluid, and promoting elimination of potassium from the body.[41] Because hyperkalemia raises resting membrane potential, calcium directly and rapidly antagonizes the adverse myocardial effects of hyperkalemia by raising threshold potential, thereby restoring the normal separation between resting membrane potential and threshold potential. In a 50-kg foal with severe hyperkalemia ($[K^+]$ > 6.5 mEq/L), 25 or 50 mL of a 23% calcium borogluconate solution can be added to 500 or 1000 mL of 0.9% NaCl, respectively, and infused over 5 to 10 minutes to rapidly antagonize the effects of hyperkalemia. This should be followed by further administration of potassium-free IV fluids (0.9% NaCl) supplemented with 5% to 10% dextrose. Dextrose administration causes release of insulin that stimulates activation of Na^+/K^+-ATPase pumps to drive potassium back into cells.[41] Dextrose in saline may be all that is needed for treatment of moderate hyperkalemia ($[K^+]$ = 5 to 6.5 mEq/L); however, calcium supplementation would be recommended if a cardiac arrhythmia is detected. Of interest, life-threatening cardiac arrhythmias, including ventricular fibrillation, cardiac arrest, third-degree atrioventricular block, and premature ventricular beats, were reported in nine of 22 foals (41%) with uroabdomen in an early retrospective study.[8] However, arrhythmias have received much less attention in more recent cases series with no mention in one review of 31 cases[20] and description of conduction blocks in only four of 32 foals (12%) in another report.[21] Perhaps this can be attributed to earlier diagnosis of uroabdomen, because ultrasonographic examination has become a routine tool in equine practice. The goal of medical stabilization is to decrease $[K^+]$ to less than 5.5 mEq/L prior to surgical intervention.[8]

DRAINING OF THE ABDOMEN

Drainage of urine from the abdomen also is essential to decrease pressure on the diaphragm to allow adequate ventilation as well as to remove a large amount of potassium from the body. Drainage is most easily accomplished by inserting a large-bore catheter in the ventral aspect of the abdomen (I prefer a 16 French trocar chest tube). However, urine has also been successfully drained via a large-bore tube passed via the urethra and bladder tear into the abdomen in mares.[31] Neonates can be positioned in right lateral recumbency, whereas adult horses are generally drained while standing. In neonates, a stab incision is initially made through the skin and subcutaneous tissue and the chest tube is inserted through the incision, but the tube is used to push the skin about 2 cm to the side before entering the abdomen perpendicularly (not a tunneling procedure). This offsetting of the skin incision and abdominal entry sites limits the risk of omental herniation once the tube is removed. When

a large volume of urine is drained (more than 5 L in a foal or more than 50 L in an adult horse), there is a risk of hemodynamic collapse,[31] and IV fluids (0.9% NaCl) should be concurrently administered during drainage. Foals that have accumulated a large volume of urine in the abdomen may also develop pleural effusion that can complicate ventilation during surgery.[8] Consequently, it is good practice to include an ultrasonographic examination of the thorax at the same time the abdomen is scanned in all cases of uroabdomen. Initiation of broad-spectrum antibiotic therapy to limit the risk of septic peritonitis is also indicated because there is an open conduit from the urethra to the peritoneal cavity. Although accumulation of urine in the abdomen has been incriminated in development of chemical peritonitis,[42] most nonseptic foals and adult horses that undergo successful surgical repair of the bladder have had favorable long-term outcomes without developing abdominal adhesions. Although surgical correction remains the treatment of choice, conservative medical management using an indwelling catheter to keep the bladder small has been a successful treatment in foals with small tears[43] and in an adult horse with uroabdomen in which a distinct bladder tear could not be identified.[44]

Patent Urachus

The urachus is the conduit through which fetal urine passes from the bladder into the allantoic cavity. Normally, the urachus closes at the time of parturition, but incomplete closure is the most common malformation of the equine urinary tract, and a patent urachus occurs more commonly in foals than in other domestic species. Greater than average length or partial torsion of the umbilical cord has been suggested to cause tension on the attachment of the umbilical cord to the body wall. The result can be dilatation of the urachus and subsequent failure to close at birth.[16,19,45] Patent urachus results in a persistently moist umbilicus after birth, from which urine may leak as drips or as a stream. It is important to distinguish a simple or congenital patent urachus, considered a malformation, from sepsis of the urachus, which can also result in urine leakage from the umbilicus within a few hours to days after birth (considered an acquired patent urachus). Local sepsis is often accompanied by more severe illness, including septicemia or other sites of localized infection, particularly of the musculoskeletal system (joints), and may lead to uroabdomen (see "Bladder Rupture," earlier).

Simple patent urachus has been treated with frequent (two to four times daily) chemical cauterization of the urachus with swabs dipped in a concentrated phenol or 7% iodine solution or with silver nitrate applicators. Because the urachus closes spontaneously in many cases, and because these agents desiccate and irritate tissue, predisposing to infection, the rationale for this approach has been questioned.[45] In fact, in a study investigating the effects of various disinfectant solutions on the bacterial flora of the umbilicus of normal foals, the use of a 7% iodine solution was observed to cause rapid desiccation of the umbilical tissue and subsequent development of a patent urachus when the stump fell off a few days later.[46] Consequently, in the absence of apparent infection, no local treatment should be implemented, or it should be limited to a navel dip with 0.5% chlorhexadine solution two to four times a day, but prophylactic antibiotics are commonly administered to affected foals. For acquired patency, likely associated with local infection, broad-spectrum antibiotic therapy is indicated and resolution of the systemic disease may be accompanied by resolution of the umbilical infection and closure of the urachus. Chemical cauterization is contraindicated with local sepsis, as it may increase the risk of urachal rupture and development of uroperitoneum.[47] If no decrease in urine leakage is observed after 5 to 7 days of medical therapy or if ultrasonographic examination reveals abnormalities of multiple structures in the umbilicus,[48] surgical exploration and resection of the urachus and umbilical vessels may be indicated. In a retrospective study of 16 foals treated for sepsis of umbilical cord remnants, six of nine (67%) survived after surgical resection and antibiotic treatment, whereas only three of seven (33%) survived after antibiotic treatment alone.[49] Although this series of 16 foals is often cited in support of surgical intervention, it should be emphasized that this small number of foals were evaluated over a 10-year period (1975 to 1985), during which many aspects of equine neonatal care improved. In a subsequent report of 33 foals with umbilical remnant infections, no difference in survival rate was observed between 23 foals treated with antibiotics in combination with surgical resection or 10 foals treated with antibiotic therapy alone.[48] In the latter report, emphasis was placed on the insensitivity of external examination and palpation of the umbilicus in detecting umbilical remnant infection (normal in 17 foals), in comparison to ultrasonographic examination, and a poorer outcome of cases in which the umbilical vein was involved. As supported by the fact that more than half of the foals in both of these studies were treated surgically, it remains most clinicians' preference to surgically resect infected umbilical remnants, especially with concurrent localized infection of the musculoskeletal system. Although more costly in the short term, surgical treatment allows more-rapid resolution of the problem, decreases the risk that uroabdomen may develop, and usually decreases the overall length of antimicrobial therapy.

Urolithiasis

Bladder stones are the most common type of equine urolith.[50-53] Cystoliths are typically flattened, sphere-shaped stones with a spiculated or smooth surface that cause hematuria, stranguria, pollakiuria, pyuria, or incontinence. In horses there are two basic forms of cystoliths, and both are primarily composed of calcium carbonate crystals.[51,53,54] More than 90% are yellow-green spiculated stones (type 1) that can easily be fragmented (Figure 65-3, *A*). Less commonly, uroliths are gray-white smooth stones (type II) that are more resistant to fragmentation (Figure 65-3, *B*). Type II stones often contain phosphate in addition to calcium carbonate. The crystalline composition of normal equine urine sediment and uroliths is similar: calcium carbonate ($CaCO_3$) in the form of calcite (a hexagonal crystal form of $CaCO_3$) is most common, followed by vaterite (a metastable, hexagonal crystal form in which $CaCO_3$ is partially replaced by magnesium or to a lesser extent by manganese, strontium, and sulfur). Other less common components include aragonite (an orthorhombic crystal form of $CaCO_3$), weddellite (calcium oxalate dihydrate), struvite (magnesium ammonium phosphate hexahydrate), hydroxyapatite, and uric acid.[53-56] Examination of the cut surface of equine cystoliths by scanning electron microscopy reveals a pattern of irregular concentric bands around a core separated by small spherules of crystalline material, suggesting that calculus growth occurs by accretion of crystals already present in normal equine urine on the surface of the growing urolith (Figure 65-4).[54] The gaps between adjacent

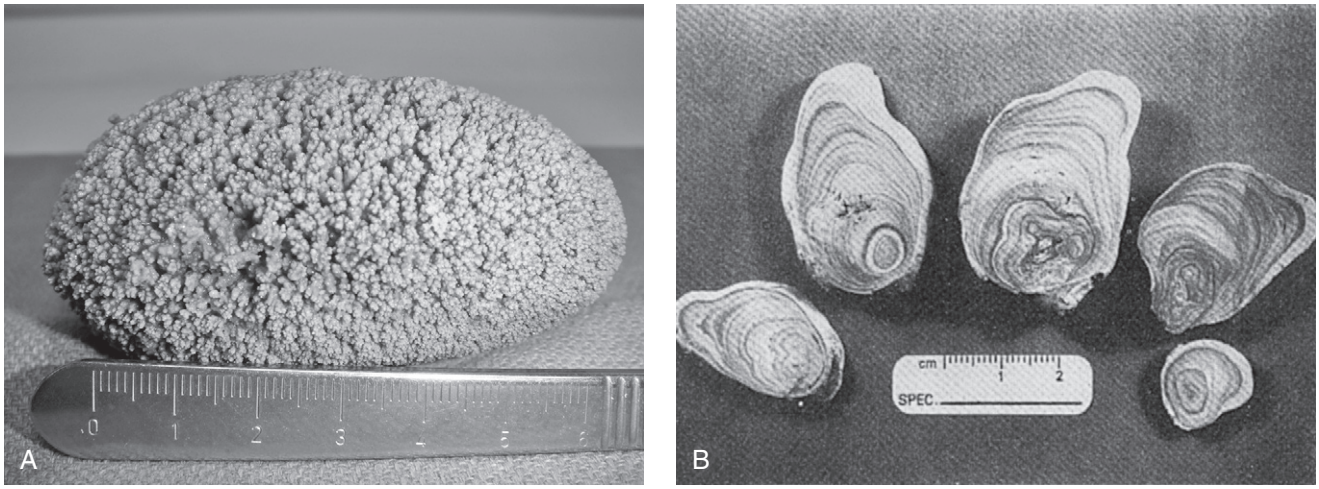

Figure 65-3. Equine cystic calculi. **A,** The more common flattened, sphere-shape type of bladder calculus that is highly spiculated. **B,** The less-common form of gray, smooth-surfaced calculi that can be more irregular in shape. (**A,** Courtesy Julie Rossetto, DVM. **B,** Reproduced from DeBowes RM: Surgical management of urolithiasis, Vet Clin North Am Equine Pract 4:461,1988, with permission.)

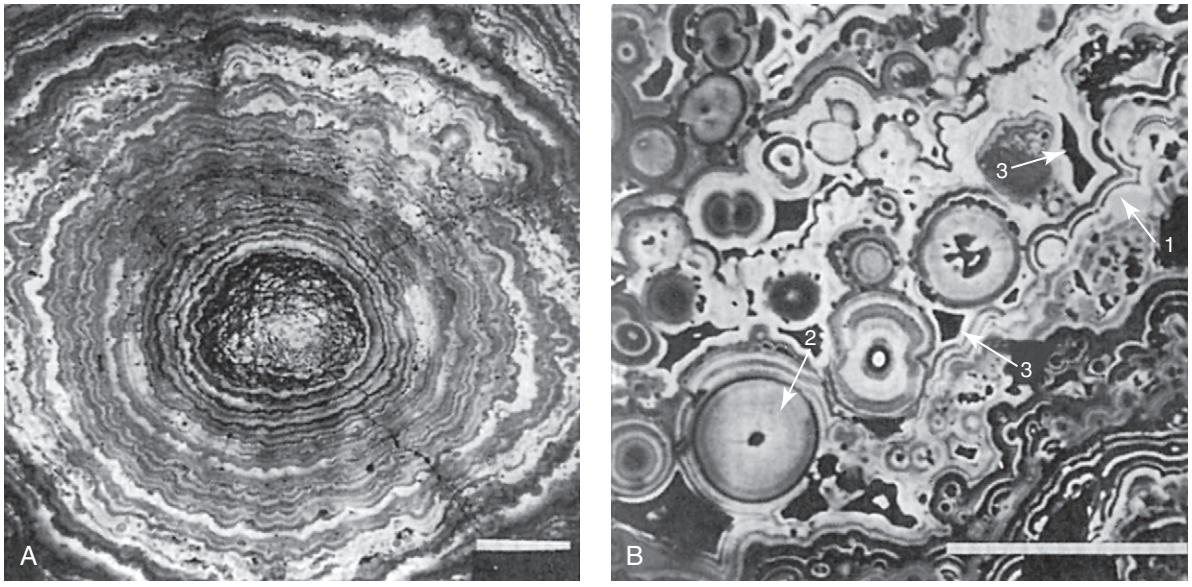

Figure 65-4. Scanning election microscopic appearance of the cut surface of equine cystic calculi. **A,** Lower-power micrograph reveals the intricate pattern of concentric banding around the core (bar = 500 μm); **B,** Higher-power micrograph reveals the ultrastructural features including bands *(1),* spherules *(2),* and primary porosity in black *(3)* (bar = 50 μm). (Reproduced from Neumann RD, Ruby AL, Ling GV, et al: Ultrastructure and mineral composition of urinary calculi from horses. Am J Vet Res 55:1357, 1994, with permission.)

spherules result in porosity of the urolith and explain why some stones are relatively easy to fragment.

The role of urinary tract infection (UTI) in the development of cystolithiasis varies with species. Struvite urolithiasis in humans and dogs appears to be almost exclusively a consequence of UTI, whereas the majority of struvite uroliths in cats and sheep are not associated with infection.[57] In 68 horses with urolithiasis, positive urine culture results were found in only 2 of 19 horses in which urine culture was performed; however, culture of material from the center of 30 calculi yielded positive results from 19 stones (63%), and a variety of bacterial species were isolated.[52] Only 1 of 28 calculi examined in this study contained struvite. The significance of finding bacteria in the center of equine calcium carbonate uroliths remains unknown, and culture of an appropriately collected sample of urine is preferred over culture of a calculus.[58]

The classic presenting complaint for cystolithiasis is hematuria after exercise. An affected male horse may also demonstrate stranguria by repeatedly dropping his penis and posturing to urinate but voiding little or no urine. Although cystoliths are less common in mares, stranguria and incontinence are common presenting complaints. Less-common signs include an irritable attitude, recurrent colic, and loss of condition; one burro presented for recurrent rectal prolapse.[59] Diagnosis of a bladder stone is usually made by rectal palpation. It is important to remember that dysuria and pollakiuria frequently result in a

small bladder that may be entirely within the pelvic canal. As a consequence, the bladder and disc-shaped cystolith are often best palpated with the hand inserted only wrist deep into the rectum. If the hand is inserted farther forward to search for the bladder in the expected location over the brim of the pelvis, a cystolith can be missed because it may be lying under the wrist or forearm. If the bladder is distended, it may be necessary to drain it by catheterization to feel a suspected stone. Bladder catheterization also allows collection of samples for urinalysis and quantitative culture. Because bladder distention is not the expected finding with cystic calculi, sabulous urolithiasis or accumulation of urine sediment with bladder paresis should be considered with a distended bladder, especially if incontinence is present or urine is easily expressed during rectal palpation.[60]

Surgical removal is the treatment of choice for equine bladder stones. An early report described excellent long-term results, without recurrence, after cystic calculi were removed by laparocystotomy in four horses.[61] In contrast, in a larger case series, clinical signs of urolithiasis recurred in 12 of 29 horses (41%) with an interval between episodes of 1 to 32 months. Recurrence of cystic calculi was also greater after treatment by perineal urethrotomy as compared to laparocystotomy.[52] Despite success of dietary management (low protein, phosphorous, and magnesium) for medical dissolution of canine[62] and feline[63] uroliths, dietary management is unlikely to replace surgical treatment of cystic urolithiasis in horses. This can be attributed to the fact that dietary management for small animals has been directed at struvite urolithiasis, and these stones are not common in horses. Nevertheless, dietary management should not be overlooked following cystolith removal to decrease the risk of recurrence. At a minimum, legume hays and dietary supplements containing calcium should be avoided. Additional recommendations may include addition of salt to the diet to increase water intake and urine output as well as allowing access to grass at pasture. Unfortunately, acidification of equine urine is not easily accomplished and is not a routine postoperative recommendation.

Figure 65-5. A, An enlarged bladder filled with a sphere of inspissated sabulous urine sediment at postmortem examination. The mass of urine sediment weighed 5 kg. **B,** The sphere of urine sediment could be cut rather easily with a knife. (Reproduced from Schott HC: Urinary incontinence and sabulous urolithiasis: Chicken or egg? Equine Vet Educ 8:17, 2006, with permission.)

Sabulous Urolithiasis

Another form of equine urolithiasis, termed *sabulous urolithiasis,* has been described in a limited number of horses.[60,64] Sabulous (from the Greek word for *sand*) urolithiasis refers to the accumulation of large amounts of crystalloid urine sediment in the ventral aspect of the bladder (Figure 65-5). This condition is a secondary problem, consequent to bladder paralysis or other physical or neurologic disorders interfering with complete bladder emptying. Affected horses usually present for evaluation of urinary incontinence or hind limb weakness and ataxia, and accumulation of urine sediment in a distended bladder can be detected during rectal palpation. These cases can be confused with cystolithiasis, but an important difference is that the bladder is usually enlarged compared to a small bladder, which is typical for a cystolith. Careful rectal palpation, usually after catheterization to empty the bladder of urine, usually allows indentation of the sabulous mass with firm digital pressure, differentiating the concretion from a true cystolith. Most important, horses with sabulous urolithiasis are not surgical candidates. Rather, treatment involves repeated bladder lavage and antimicrobial therapy for concurrent UTI, but the condition carries a poor prognosis unless the primary problem resulting in bladder paralysis can be resolved.[60,64]

Bladder Displacement

Displacement of the urinary bladder is a rare cause of dysuria.[65-73] In mares, two types of bladder displacement can occur: (1) extrusion (or eventration) through a tear in the vagina and (2) true prolapse with eversion of the bladder (Figure 65-6). Urethral obstruction may also occur with vaginal or uterine prolapse. In male horses, scrotal herniation of the bladder has been described, but this type of bladder displacement is extremely rare.[72] Bladder displacements are typically a consequence of repeated abdominal contractions or straining. Therefore, they are most often associated with parturition and, to a lesser extent, with colic. Perineal lacerations, consequent to trauma or foaling, may lead to extrusion, whereas excessive straining without laceration leads to prolapse with eversion. Because the bladder turns inside out with the latter problem, the diagnosis is established by recognizing the appearance of the bladder mucosa and ureteral openings. Eversion occasionally results in obstruction. Umbilical extrusion or eventration of part of the bladder has also been described in a neonatal filly.[73] The problem was suspected to have developed as a consequence of a urachal tear that allowed eversion of the urachus and part of the bladder. Surgical resection of a portion of the bladder corrected the problem.[73]

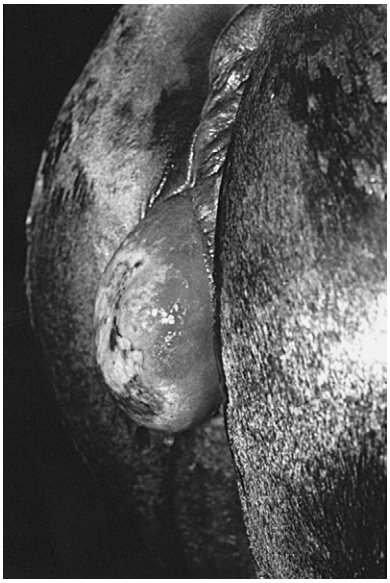

Figure 65-6. Everted bladder in a postpartum 4-year-old Standardbred mare. (Courtesy Warren L. Beard, Manhattan, KS.)

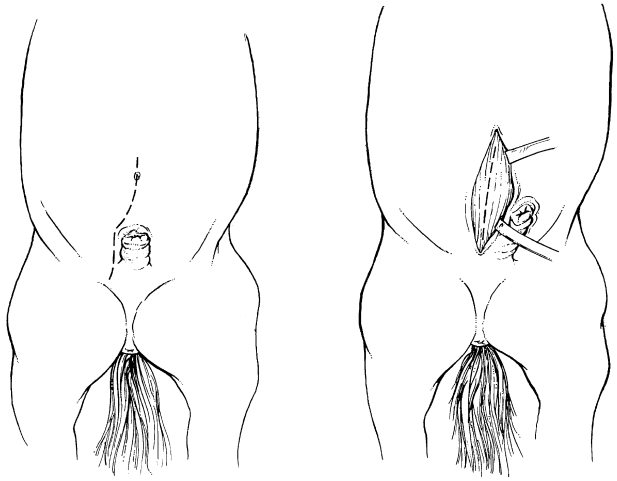

Figure 65-7. A, In male horses or foals, the caudal aspect of the cutaneous midline incision and subcutaneous dissection is directed abaxially to avoid the prepuce and penis. **B,** Then the penis and prepuce are retracted laterally so the body wall can be incised on midline.

Neoplasia

The most common presenting complaint for bladder neoplasia in horses is hematuria; however, straining and urinary obstruction may also occur.[74-79] Unlike dogs, in which transitional cell carcinoma is the most commonly described bladder neoplasm, squamous cell carcinoma has been reported most frequently in horses.[74,77] Other types of bladder neoplasms affecting horses include transitional cell carcinoma, lymphosarcoma, leiomyosarcoma, rhabdomyosarcoma, and fibromatous polyps. Diagnosis of bladder neoplasia can be established by rectal palpation and ultrasonographic imaging of a bladder mass along with endoscopic examination and biopsy. In some instances, masses can also be seen exiting the urethra and vulva in mares. Treatment has included tumor debulking, partial bladder resection, and intravesicular instillation of 5-fluorouracil, but long-term successes have not been reported. The poor prognosis is likely related to extensive bladder involvement by the time clinical signs are initially noted.

Fibroepithelial polyps are another tissue mass that can grow in the bladder or lower urinary tract of the horse. A large polyp mass that developed in the bladder of a pony gelding acted as a ball valve and caused urethral obstruction, bladder distention, and intermittent colic signs.[80] I also have seen vaginal polyps in a neonatal mule that caused confusion with bladder prolapse. Careful examination failed to reveal ureteral openings or urine flow from the abnormal tissue, and passage of an endoscope beyond the polyps revealed a normal urethra and bladder. The polyps were excised and the filly grew without further problems.

SURGICAL PROCEDURES

J. Brett Woodie

Cystorrhaphy

Cystorrhaphy is indicated for disruption of the bladder.[12,19,81-84] The anesthetized patient is positioned in dorsal recumbency. An appropriate-size urinary catheter should be passed through the urethra and secured in the bladder to ensure outflow of urine and to permit intraoperative lavage of the base of the bladder. The prepuce in males is cleansed and closed with sutures or towel clamps to reduce the possibility of incisional contamination. The penis and prepuce can be directed caudad in between the hindlegs so that it is not in the operative field. The abdomen is aseptically prepared for a ventral midline incision. The external umbilicus, if present, should be oversewn with a continuous inverting suture pattern before aseptic preparation of the abdomen.

In the adult female patient, the surgeon should make a 15- to 18-cm (6- to 7-inch) midline incision that extends caudad from a point 2 to 5 cm cranial to the umbilicus. In foals, the incision should be directed abaxially to create a fusiform incision around the external umbilicus for removal. In the male patient, the cranial aspect of the incision is identical; however, the skin and subcutaneous layers of the caudal incision should be directed 2 to 4 cm paramedian to the prepuce (Figure 65-7). The prepuce can be mobilized and retracted to expose the posterior midline for deep incision. If the penis and prepuce has been directed caudad, it may not be necessary to direct the incision lateral to the prepuce.

After the peritoneal cavity has been entered, peritoneal fluid should be cultured and suctioned. The bladder is exposed by maintaining traction on the urachus, if present (Figure 65-8). Balfour or Finochietto self-retaining retractors may be used to facilitate the abdominal exploration. The bladder and urachus should be inspected to identify the site of rupture. The site of rupture in the foal may be in the urachus as well as the dorsal or ventral aspect of the bladder (Figure 65-9).[12] Urachal rupture is often a sequela of urachal infection, especially in septicemic foals (Figure 65-10).[12,84] Tears in the ventral aspect of the bladder tend to run longitudinally toward the neck of the bladder. Accessing the full extent of a ventral defect is often not possible.[84]

If the origin of the uroperitoneum is not evident, the bladder may be distended by retrograde introduction of a sterile solution of fluorescein or methylene blue through the urethral

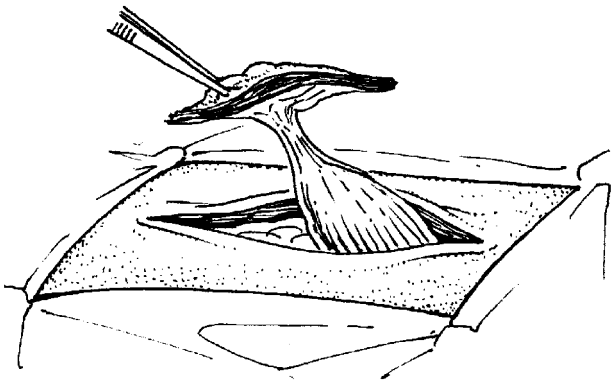

Figure 65-8. After completing the midline incision and mobilizing the umbilicus and urachal remnant from the abdominal wall, the bladder is exposed by careful traction on the umbilicus. When the bladder is exposed, the tissues are retracted caudad.

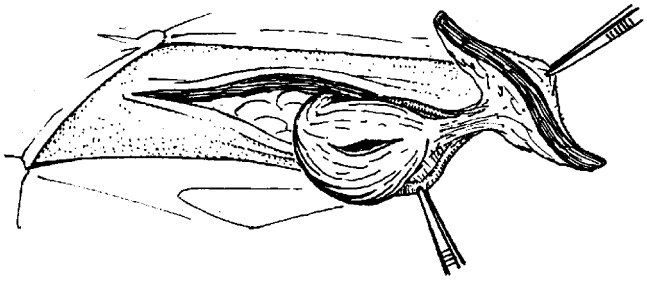

Figure 65-9. The bladder is retroflexed to examine a tear on the dorsocranial border.

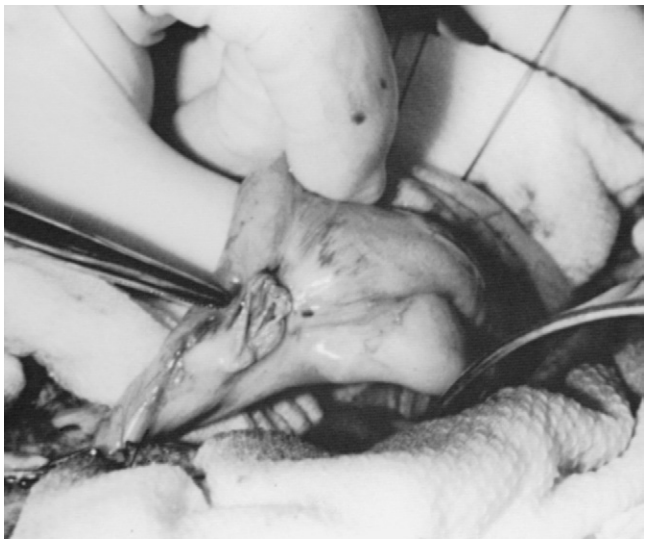

Figure 65-10. A rupture of the urachus (located at the tip of the hemostatic forceps) is readily apparent on inspection of the cranial bladder.

catheter.[12] This method identifies thin areas of the bladder or attenuated areas in the bladder wall that may be leaking, typically on the dorsocranial surface of the bladder.

When the tear is identified, the wound margins are excised (Figure 65-11). Culture and histopathologic evaluation of the

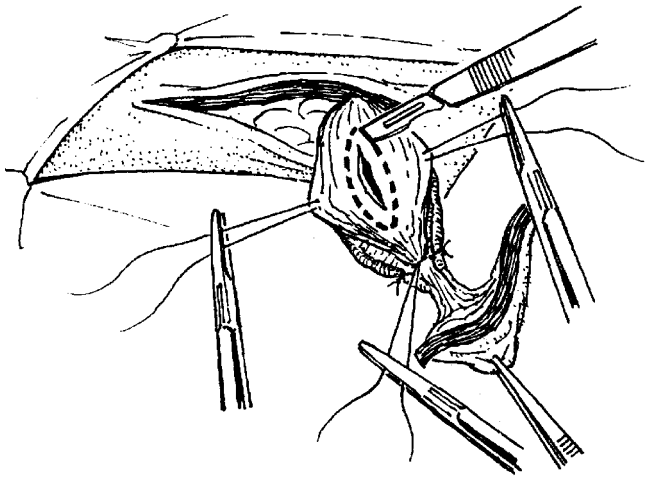

Figure 65-11. Tissues surrounding a tear in the bladder should be excised before primary repair.

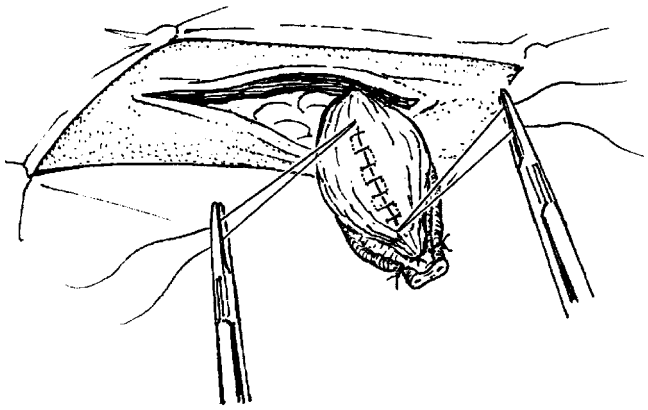

Figure 65-12. A two-layer inverting continuous closure is appropriate for repair of a bladder rupture.

débrided margins of the tear in septic foals should be considered.[12] The use of stay sutures to support the bladder during primary repair is recommended. Surgical closure of the tear should be accomplished in two layers: an interrupted pattern in the first layer, followed by a continuous inverting pattern (Figure 65-12).[83] A double-layer inverting closure is appropriate as well. Small-size (2-0, 3-0) synthetic absorbable suture material on a taper-point atraumatic needle should be used. Care should be taken to avoid suture penetration of the vesicular mucosa, because sutures that penetrate the bladder mucosa perpetuate the formation of cystic calculi in the presence of alkaline urine.[85]

After repair, the bladder should be carefully distended with sterile saline to evaluate potential leakage along the suture line. In a foal in which the urachus is present, the urachus should be resected, cultured, and removed with the umbilicus after primary repair of the tear. Before abdominal closure, the peritoneal cavity is lavaged with warm sterile saline solution. An indwelling abdominal drain may be placed if septic processes are present or anticipated. Despite the irritative nature of urine, clinically significant peritonitis is rare unless concurrent septic disease processes such as omphalophlebitis or neonatal septicemia are present.[12,84] The midline incision is closed with size 1 or 2 synthetic absorbable sutures placed in simple-continuous

fashion. The subcutaneous tissues and skin are closed routinely.

In the foal, it is often advisable to perform a cystoplasty as a means to resolve a tear in the urinary bladder. The viability of the tissue adjacent to the defect is often questionable and does not hold sutures very well, making débridement and closure risky for subsuquent leakage. A cystoplasty procedure would be indicated provided it is possible to resect the apex of the bladder to include the defect.

Urinary bladder repair in the standing mare can also be accomplished by prolapsing the bladder either through a vaginal incision or urethrotomy and sphincterotomy.[30,31] Clinical signs of cystorrhexis are consistent with uroperitoneum, and diagnostic tests should be performed to confirm the presence of a rent. Horses should be stabilized medically and the uroperitoneum should be sterilely drained. Caudal epidural anesthesia is used to desensitize the surgical site. For the vaginal approach, a rigid urinary catheter is placed to identify the location of the urethra and bladder under the vaginal mucosa. A 2-cm incision is made 5 to 10 cm caudal to the cervix. Blunt dissection increases the size of the incision, which facilitates the delivery of the bladder into the vaginal vault. Alternatively, a linear urethrotomy and sphincterotomy can be preformed. A 5-cm incision is made in the urethral sphincter on the dorsal midline. The surgeon places a hand in the bladder and everts the bladder out of the vulva. Care is taken to identify the ureter openings. Rents are closed with a single- or double-layer closure. The bladder is replaced and the vaginal incision or sphincterotomy is closed in a simple-continuous pattern with absorbable 2-0 or 0 suture material.

Cystoplasty

Cystoplasty is the technique of choice for patent or persistent urachus.[12,19,81-84] It can also be used for treatment of bladder rupture. The foal should be anesthetized and positioned in dorsal recumbency. A urinary catheter is placed, and the ventral abdomen is prepared and draped as described earlier (see "Cystorrhaphy," earlier). The external umbilicus should be oversewn with a continuous-inverting suture pattern before aseptic preparation of the abdomen.

A fusiform incision is made around the external umbilicus. This incision can be extended craniad or caudad as needed. The incision is continued as described earlier.

The umbilicus and urachus are dissected free from the body wall. The umbilical vein is double ligated and resected as far craniad as necessary to remove any portion of abnormal umbilical vein. Occasionally, the midline incision must be extended craniad to permit adequate resection of the umbilical vein. The affected urachus may be thickened and distorted. Adhesions may exist between the urachus or its associated vascular structures and the surrounding viscera.[12,84] Care should be taken to identify and transect adhesions that involve the urachus before manipulating the bladder.

Exposure of the bladder is achieved by traction on the urachus. Traction should be applied with caution because of the friable nature of the tissue and the possibility of rupture (Figure 65-13). Exposure of the bladder can be improved by extending the ventral midline incision caudad and/or transecting the ventral ligament of the bladder. Sterile moistened laparotomy sponges can be used to pack off intestines and assist in positioning the bladder. Stay sutures are placed at the ventrolateral

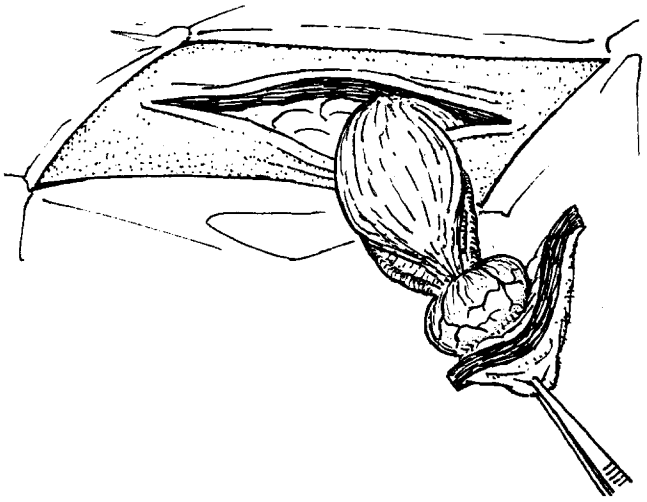

Figure 65-13. After mobilizing and retracting of an umbilical or urachal abscess, the abdomen should be explored for adhesions or additional foci of abscessation.

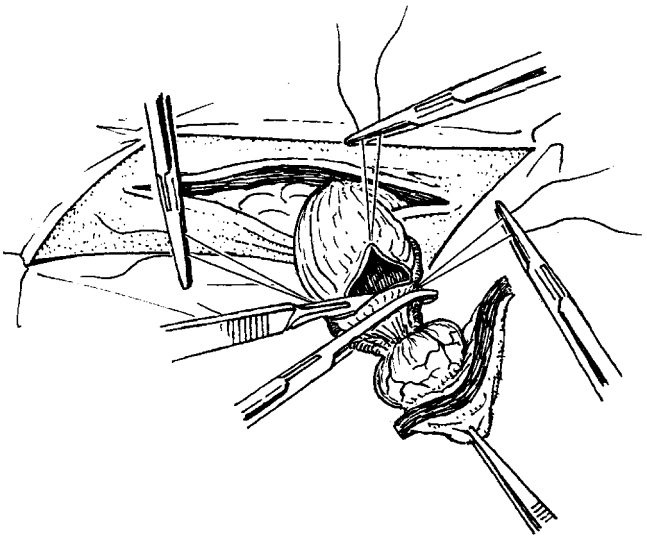

Figure 65-14. Before urachal-umbilical resection, a clamp is applied to prevent spilling urachal contents into the peritoneal cavity. Here, the bladder has been stabilized by stay sutures transfixed as ligatures around the umbilical arteries. The urachus and umbilical tissues are removed by sharp dissection. The bladder is closed by the two-layer inverting continuous suture pattern.

margins of the bladder for additional support and control of the bladder after resection of the urachus. The umbilical arteries should be double ligated with a transfixing suture at the level of urachal resection.

An occluding forceps is used to isolate the urachus and apex of the bladder (Figure 65-14). A transverse incision is made across the apex of the bladder to remove the urachus and vesicular apex (see Figure 65-14). The urachus is removed and submitted for culture and sensitivity testing. Traction is applied to the previously positioned stay sutures to elevate the apex of the bladder and to prevent spillage of urine. Suction is used to remove the residual urine or purulent material from the bladder. Closure is accomplished using a two-layer inverting suture pattern with 2-0 or 3-0 synthetic absorbable suture material,

taking care not to penetrate the lumen. Alternatively, a simple-continuous closure of the first layer may be followed by an inverting suture pattern.[84] The abdomen is lavaged with warm sterile saline before closure. Unless there has been spillage of exudate into the abdominal cavity, an abdominal drain is not necessary. Broad-spectrum antimicrobial drugs are administered routinely for 3 to 5 days because of the potential for abdominal contamination from urachal bacterial contaminants.

Cystotomy

Cystotomy is the surgical treatment of choice for cystic calculi.[50,51,81-83,86] Fasting the horse for 24 to 36 hours reduces the volume of ingesta in the gastrointestinal tract, which can be helpful for this procedure. Positioning of the harse and performing the incision is as described earlier. The large superficial caudal epigastric and external pudendal vessels should be avoided when dissecting deep to the prepuce to expose the caudal midline. In mares, the incision is made on the midline from the umbilicus caudad to the prepubic tendon.

The bladder is identified by palpation in the pelvic canal. Sustained gentle traction is required to exteriorize the bladder (Figure 65-15).[50,51,87] Another method that can be used to facilitate exteriorization of the bladder is allowing the bladder to distend with urine by clamping the urinary catheter while the horse is being prepared for surgery and during the approach to the abdomen. When the surgeon is ready to exteriorize the bladder, the clamp is removed and the bladder is decompressed. The bladder has been stretched by being distended with urine and will be easier to exteriorize. When the bladder is exposed, moistened laparotomy sponges are used to pack off the bowel and elevate the bladder in the surgical field. Large-diameter stay sutures can be positioned at the ventrolateral aspects of the bladder to facilitate control of the cystotomy incision and to reduce urine spillage. A transverse incision is made across the ventral bladder to expose the urolith. Frequently, the urolith is closely adherent to the bladder mucosa, particularly in the case of a type I urolith. The mucosal layer of the bladder must be peeled back from the urolith to permit removal of the calculus (Figure 65-16). The bladder can be lavaged in an effort to remove small fragments of calcular material and blood clots. Irrigation of the bladder by retrograde introduction of sterile saline through the urinary catheter flushes small fragments of the urolith from the neck of the bladder toward the incision, where they can be evacuated by suction.

The cystotomy is sutured with a two-layer closure of synthetic absorbable suture material. Continuous inverting suture patterns such as the Cushing and Lembert patterns are preferred. The sutures should not penetrate the mucosa of the bladder. After closure of the cystotomy, the bladder may be carefully distended with sterile saline to evaluate the closure for leakage. The abdomen is lavaged with sterile balanced saline solution, which is subsequently suctioned off. The midline incision is closed with size 2 or 3 synthetic absorbable suture material in continuous or interrupted fashion. If the caudal limit of the incision was made in a paramedian location, the fascial closure is completed in two layers by suturing the internal and external layers of the rectus abdominis sheath separately. Of the two layers, the external rectus sheath is the more critical to the security of the abdominal closure.[83,84,88,89] The subcutaneous tissues and skin are closed in routine fashion.

Parainguinal Approach

In an adult horse, access to the bladder can be also achieved through a parainguinal approach. This approach has been described in the male horse but can be used in the female patient as well.[90] This approach eliminates the need to reflect the prepuce and reduces the chances of encountering large vessels prior to gaining access to the urinary bladder. The horse is anesthetized and positioned in dorsal recumbency. A urinary catheter should be placed and secured. The inguinal region is clipped and prepared for aseptic surgery. A 12- to 14-cm (5- to 6-inch) skin incision is made parallel and 2-cm axial to the external inguinal ring (see Chapter 34). A combination of sharp and blunt dissection is used to separate the subcutaneous tissues and fat to expose the aponeurosis of the external abdominal oblique muscle. The aponeurosis is sharply incised, parallel to the skin incision for 12 to 14 cm (5 to 6 inches). Retraction is used to gain access to the internal abdominal oblique muscle, which is separated along its fibers. The peritoneum is bluntly entered with the surgeon's finger. The bladder is identified and

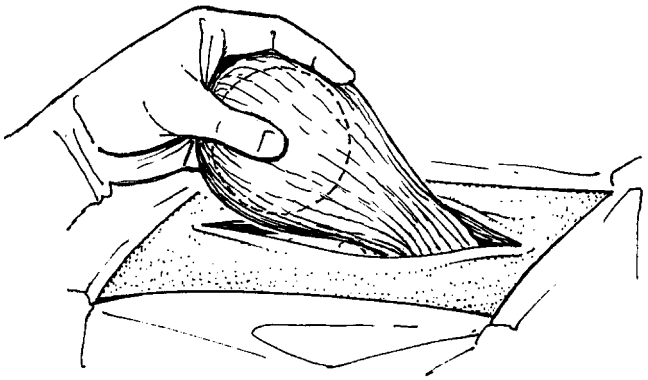

Figure 65-15. The bladder and urolith are delivered into the surgical field by gentle, sustained traction.

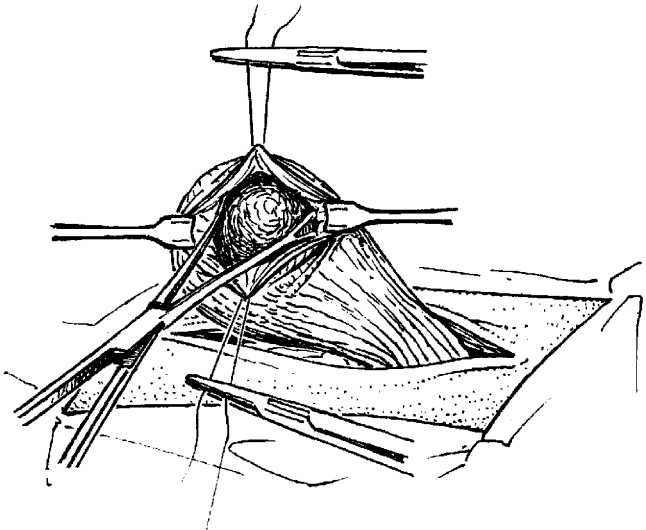

Figure 65-16. The urolith can be grasped with sponge forceps for expedient removal.

exteriorized with steady traction. After the bladder is exteriorized, a moist laparotomy sponge can be wrapped and twisted around the bladder proximal to the urolith. This aids in maintaining exteriorization of the bladder to complete the cystotomy. Following removal of the urolith the bladder is closed in a double layer. Closure of the abdomen is routine, with attention paid to the aponeurosis of the external abdominal oblique muscle. An interrupted cruciate, near-far-far-near, simple-continuous, or a simple-interrupted pattern using No. 2 synthetic absorbable suture material is appropriate for closure.

Laparoscopic Techniques

Laparoscopic techniques have been described for repair of cystorrhexis, persistent urachus, and umbilical infections in the foal.[91,92] Preoperative preparation of the patient including appropriate diagnostic examinations and perioperative therapeutics does not differ from those used for foals undergoing open surgical approaches. The foal is anesthetized and positioned in dorsal recumbency with the pelvic limbs elevated. Intermittent positive-pressure ventilation should be available if needed. The ventral abdomen is prepared for aseptic surgery.

Because the foal's body wall is thin, it is recommended that insufflation be done with a teat cannula or veress needle and that lower (1 to 2 L/minute) insufflation rates be used. The abdominal cavity should be insufflated with CO_2 to a pressure of 10 mm Hg.[92] A 1.5-cm incision is made 5 cm lateral to the ventral midline and 10 to 15 cm cranial to the umbilicus for placement of the laparoscopic cannula. A 30-degree laparoscope is used to explore the abdomen. Additional portals are made 8 to 10 cm lateral and 5 cm cranial to the umbilicus. It is important to avoid damage to the epigastric vessels so that hemorrhage does not obscure visibility.[92] Additional information on laparoscopy can be found in Chapter 13.

For cystorrhexis, urine accumulation needs to be evacuated prior to insufflation. When the laparoscope is placed, the entire bladder should be inspected. Sectioning of the ventral ligament of the bladder using laparoscopic scissors aids in inspecting the bladder. Two working portals are required to resect the edges of the rent and repair the bladder with absorbable sutures using intracorporeal suturing techniques. It is prudent to carefully test the bladder for leaks before deflating the abdomen.

For resection of umbilical remnants, or umbilical remnant infection, the umbilical vein and arteries are isolated with endoscopic scissors and dissected free to facilitate their ligation. A large ligation clip or loop can be used. Several clips may be necessary to ensure proper ligation, especially when structures appear large or pathologically altered. When the umbilical vein and arteries have been transected, a fusiform incision is made around the umbilicus through the body wall under laparoscopic observation. The end of the bladder is exteriorized and resected. A two-layer closure in the bladder is recommended.

Closure of the body wall is routine. Closure of the portals is performed with No. 1 absorbable material in a cruciate pattern in the external fascia of the rectus abdominis or linea alba. The subcutaneous tissues are closed in a continuous pattern. The skin can be closed using a monofilament absorbable material in a continuous pattern, or intradermal sutures can complete the procedure. Overall, good visualization with minimal intervention is obtained with laparoscopic techniques. The use of nonabsorbable linear staples for repair of a tear in the bladder is discouraged, because the staples may serve as nidus for urolith formation.[91]

Laparoscopic techniques for removal of cystic calculi in the adult male horse have also been described.[93] The horse is anesthetized, positioned, and prepared as described earlier. The bladder is lavaged and drained until clear saline irrigation solution is obtained.

A standard umbilical portal is made after insufflation. Five instrument portals are made to complete the procedure. The instrument portals are in a radiant line centered on the sheath. A laparoscopic electrocautery tip is inserted into the cranial right portal and used to make a cystotomy. A suction lavage cannula will be used when the cystotomy is made. A retrieval bag is also placed in the abdomen and positioned below the bladder. The cystotomy must be just long enough to remove the calculus. A retractable metal loop forceps aids in removing the urolith and placing it into the retrieval bag. Suction is used to remove residual debris. The bladder should be closed in two layers using an intracorporal suturing techinque. A double-layer closure is recommended. The bladder is carefully tested for leaks before deflating the abdomen. The umbilical portal is enlarged to retrieve the calculus within the retrieval bag. The linea alba and portal incisions are closed routinely.

Complications of this technique include intraoperative hemorrhage from the instrument portals and obstructed visualization from falciform fat. It should be noted that practice is required to become proficient in using the auto-suturing devices. With proper preparation, cystic calculi can be removed safety and cleanly in the adult male horse.

Lithotripsy

Lithotripsy is a means of fragmenting a urinary calculus into smaller pieces so that the fragments can be removed through a smaller lumen or incision. Lithotripsy can be used to remove cystic calculi through the urethra in mares or through a perineal urethrotomy in male horses.[50] Manual crushing of the calculus has been associated with a high rate of recurrence of urolithiasis.[52] This is most likely because of incomplete fragment removal. Trauma to the urethra is also a potential complication.[52] Based on the high rate of recurrence and potential for urethral injury, other means of fragmenting the calculus should be used if possible.

Laser lithotripsy is an alternative to manually fragmenting a calculus. A pulsed dye laser with a wavelength of 504 nm and a holmium:YAG laser with a wavelength of 2100 nm have been used to fragment uroliths in equine patients.[94,95] The pulsed dye laser causes disruption of the calculus by generating an acoustic wave that is greater than the tensile strength of the crystals in the urolith.[94] The holmium:YAG laser uses photothermal and photoacoustic effects to fragment the urolith.[95] This procedure is performed by using a flexible endoscope that is passed through a perineal urethrotomy to access the bladder in the male patient or through the urethra in the female. Laser energy is applied to the calculus thorough a quartz fiber that has been passed through the biopsy channel of the endoscope. Laser lithotripsy is not a common modality used in cases of equine urolithiasis because of the limited availability of the pulsed dye laser and the high cost associated with renting the appropriate laser equipment. Other drawbacks include long surgery times and the possibility that the laser will not be able to fragment the urolith.[94-96]

Aftercare

Antibiotics are commonly administered after surgery of the bladder. In cases of cystorrhexis or persistent urachus, sepsis represents a risk, so antibiotics are routinely administered to reduce the risk of septic peritonitis and may be continued for several days or weeks postoperatively.[12,49,84] In adult patients with urolithiasis, the risk of urine spillage during removal of cystic calculi is considerable.[51,88] Antibiotics are continued for only 48 to 72 hours unless clinical signs of infection develop.

Postoperative abdominal drainage is indicated if surgical findings suggest the presence of peritonitis or if gross soilage of the peritoneum from an urachal abscess has occurred. Generally, abdominal drainage is not required after cystorrhaphy or cystoplasty. Unless significant spillage with fragments of uroliths has occurred, the need for peritoneal drainage in urolithiasis patients is not necessary. Patients with abdominal drains should remain on prophylactic antibiotic therapy.

Typically, patients are administered low doses of nonsteroidal anti-inflammatory drugs to control postoperative and incisional discomfort. Patients recovering from cystorrhaphy and cystoplasty often require intravenous fluids to correct dehydration and acid–base and electrolyte imbalances. All patients should be routinely monitored for urine output after surgery. Foals that are at risk for sepsis or that are subjected to intensive medical care after surgery are candidates for development of gastric ulceration. Appropriate anti-ulcer and gastroprotectant medications should be given in these cases.

Complications

Clinically significant complications of bladder surgery are rare. The most acute and striking complication is the development of severe ventricular arrhythmias in the anesthetized foal with uroperitoneum.[8,19] Postoperative myositis resulting in death has also been documented.[97] Correction of electrolyte and acid-base status before surgery minimizes the risk of developing these problems.

In foals, contamination of the peritoneal cavity with urine is common and may result in the development of chemical peritonitis.[12] Although most foals with cystorrhexis have a chronic history of uroperitoneum and presumably some degree of chemical peritonitis, the incidence of septic peritonitis is low unless concurrent septic omphalophlebitis or other septic processes are present simultaneously.[12,83,84] In one retrospective study of celiotomy for the treatment of uroperitoneum, several foals had positive culture results for *Mucor* and *Candida* species.[12] All foals with uroperitoneum and concurrent septic disease should be monitored closely for clinical evidence of septicemia or septic arthritis and physitis after surgery.

Adhesions have also been reported as a consequence of abdominal surgery in foals for correction of uroperitoneum.[12] The development of septic peritonitis can result in an increased incidence of adhesion formation, particularly in the caudal abdomen near the bladder.[84]

Contamination of the midline incision with urine or bacteria may lead to the formation of incisional edema and infection. In adult male horses operated on for cystic calculi, preputial edema occurs occasionally,[51] which is generally responsive to anti-inflammatory agents and local wound therapy. Surgical failures of the cystotomy incision after cystoplasty and exploratory cystotomy have been reported, but this complication is rare.[39,98]

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CHAPTER
66 Urethra

Harold C. Schott II and J. Brett Woodie

ANATOMY AND PHYSIOLOGY

Harold C. Schott II

Male

The urethra is the conduit for urine to exit the body and is about 75 to 90 cm (30 to 35 inches) long in a male horse. The intrapelvic (ampullar) portion of the urethra, 10 to 12 cm (4 to 5 inches) long, widens in an elliptical pattern to a diameter of 5 cm (2 inches) across and 2 to 3 cm (1 to 1½ inches) from dorsal to ventral.[1] A rounded dorsal prominence, the colliculus seminalis, is located a few centimeters (about an inch) caudal to the urethral orifice. It is the site of the paired ejaculatory ducts, which are the common openings of the ductus deferens and ducts of the seminal vesicles. The openings of the prostatic ducts are arranged as two groups of small papillae on either side of the colliculus seminalis. The bulbourethral glands open in paired dorsal rows of 6 to 8 ducts each, 2 to 3 cm (1 to 1½ inches) farther caudad from the colliculus seminalis (Figure 66-1).[2] The smaller openings of the ducts of the lateral urethral glands open at the same level on the lateral aspects of the urethra. The end of the urethra terminates at the glans penis, where a urethral process protrudes 1 to 2 cm (0.3 to 0.6 inches) under the tip of the glans. Above the urethral process is the urethral sinus (sometimes referred to as the *diverticulum of the glans*), a bilobed, recessed area that can accommodate the finger tip and is the site where the "bean" of smegma accumulates.[1]

The penis encloses the extrapelvic portion of the urethra and is composed of two vascular, erectile bodies: a larger corpus cavernosum penis (CCP) and smaller corpus spongiosum penis (CSP).[1] The CCP forms most of the dorsal aspect of the penis and the CSP forms a vascular tube surrounding the urethra, largely along the ventral aspect of the penis. The CSP is contiguous at its cranial end with the glans penis and forms a slight enlargement, termed the *bulb,* at the root of the penis at the level of the ischial arch. Except at its origin and end, the urethra is surrounded by a continuous layer of striated muscle outside the erectile tissue. The portion of this muscle covering the intrapelvic urethra (as well as the bulbourethral glands) is the urethralis muscle, and its forcible contraction plays an important role in ejaculation. The bulbospongiosus muscle is a continuation of the urethralis muscle around the CSP surrounding the urethra and extends from the ischial arch to the glans penis. The muscle is thickest at the root of the penis (around the bulb of the CSP) and thins out as it extends to the end of the penis. It acts to empty the penis of blood after ejaculation or after the penis is dropped for urination.[1] Several forceful contractions of the bulbospongiosus muscle help expel residual urine from the urethra after urination. Additional information on this subject can be found in Chapter 60.

Mare

The urethra is about 5 cm (2 inches) long in the mare and its lumen easily accommodates a finger. However, it can be dilated to several times this size if needed for access to the bladder. The external opening lies at the anterior end of the vestibule. The urethral muscle surrounds the urethra throughout its length and is contiguous with the constrictor vestibule muscle.

DISORDERS REQUIRING SURGERY

Harold C. Schott II

Disorders of the equine urinary tract that may require surgery of the urethra include congenital anomalies (rectourethral and rectovaginal fistulas) and acquired disorders. The latter include urolithiasis, soft tissue obstructions (neoplasms or strictures), hematomas, and lacerations. Injuries to the distal penis and urethra are the most common traumatic conditions, especially with breeding accidents in stallions. Geldings and stallions with hematuria and hemospermia as a result of urethral rents are also candidates for surgery, because affected individuals may respond favorably to opening the CSP via a perineal urethrotomy approach.

Rectourethral and Rectovaginal Fistulas

During embryonic development, failure of the urorectal fold to completely separate the primitive hindgut from the urogenital sinus results in a rectourethral fistula in a colt or a rectovaginal fistula or a persistent cloaca in a filly.[3] These anomalies are rare in horses and, when present, are usually associated with atresia

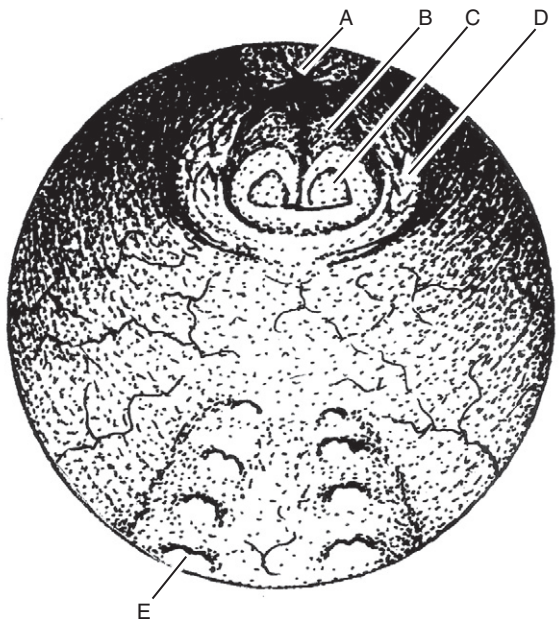

Figure 66-1. Endoscopic illustration of the intrapelvic portion of the urethra of the male horse showing the dorsal surface at the bottom: **A,** Opening to bladder (difficult to see endoscopically). **B,** Colliculus seminalis. **C,** Ejaculatory duct (common opening of the ductus deferens and seminal vessicle duct). **D,** Openings of prostate gland ducts. **E,** Openings of bulbourethral gland ducts.

ani and other anomalies, including agenesis of the coccygeal vertebrae and tail, scoliosis, adherence of the tail to the anal sphincter area, angular limb deformities, and microphthalmia.[4-7] Affected foals are usually presented for atresia ani, although signs of colic and straining may also be observed. Passage of fecal material from the vulva or penis is the sign supporting a fistula. In fillies, rectovaginal fistulas may be detected by digital palpation of the dorsal vestibule and vagina, but in colts a definitive diagnosis usually requires contrast radiographic procedures such as a barium enema or a retrograde urethrogram (Figure 66-2). Surgical correction of atresia ani and fistulas have been performed successfully in several foals, but multiple surgical procedures may be required. Because ascending urinary tract infection (UTI) is common, a sample of urine collected via bladder catheterization (preferably during surgery) should be submitted for bacterial culture. In humans, there is evidence that these anomalies are hereditary; consequently, breeding affected horses is not recommended after surgical correction of the anomalies.

A rectourethral fistula resulting in passage of urine from the anus has also been described in a 3-year-old Thoroughbred gelding.[8] The fistula in this gelding, which was successfully repaired, was thought to be acquired secondary to trauma or straining because no other developmental problems were detected and the edges of the defect were irregular and inflamed when examined with a speculum inserted into the rectum.[8]

Urolithiasis

Surgery may need to be performed on the urethra to divert urine flow in horses with urethroliths that are causing urethral obstruction or to access the bladder for cystolith removal via a perineal urethrotomy (PU). Obstructive urethrolithiasis leads to bladder distention, frequent posturing to urinate, and renal colic.[9-12] It is essentially a male horse problem because mares are generally able to void small stones through the urethra. A persistently dropped penis that may drip urine is also commonly found with obstructive urethrolithiasis. Obstruction can be confirmed by detection of a markedly distended urethra below the anus (see Figure 63-1) and a large, turgid bladder on rectal palpation. Careful palpation of the urethra at the level of the ischial arch may reveal a firm obstructing urolith, although some stones travel more distally into the penis. In horses that have been obstructed for more than 1 to 2 days, bladder leakage or rupture may occur, leading to abdominal distention from uroabdomen. Affected horses typically have a decreased appetite, a large volume of echolucent free peritoneal fluid on transabdominal ultrasonography, and serum electrolyte concentrations typical for uroabdomen (hyponatremia, hypochloremia, and mild to moderate hyperkalemia). Peritoneal fluid creatinine concentration twofold or greater than serum creatinine concentration confirms uroperitoneum.

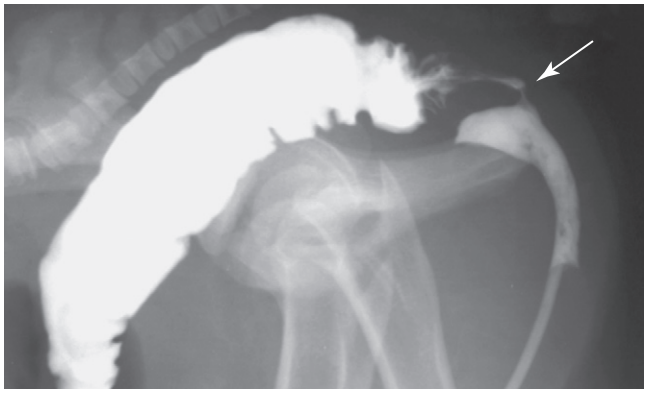

Figure 66-2. A positive contrast urethrogram in a 3-day-old burro that presented with atresia ani and intermittent passage of fecal material from the urethra. A catheter was passed via the urethra and contrast agent was injected, resulting in accumulation of a large amount of contrast agent in the rectum and a lesser amount in the intrapelvic portion of the urethra. A small amount of contrast agent can be seen in the urethrorectal fistula *(arrow)*. (From Reed WM, Bayly WM, Sellon DC (eds): Equine Internal Medicine. 3rd Ed. Saunders, St. Louis, 2010.)

Obstructive urethrolithiasis is an emergency condition (to prevent bladder rupture) that is generally treated by performing a PU into the distended urethra.[9-12] If the offending urolith is at or just beyond the ischial arch, it can often be removed at the time of surgery; however, if it has traveled more distally, removal is more challenging. Urethral distention with hydropulsion through a catheter passed either from the PU site distad or retrograde from the urethral process to the stone (depending on stone location) may be successful in moving the urolith to one of these openings for removal. Urethroliths are composed primarily of calcium carbonate crystals and are characteristically spiculated, allowing them to become embedded in the surrounding urethral mucosa. As a result, the stone may be firmly lodged at the site of obstruction so that hydropulsion and gentle prodding with a catheter tip are unsuccessful in dislodging the stone. In this situation, an alternative is to snare the stone using a device passed through the biopsy channel of an endoscope followed by gentle traction and removal of the stone. When stones cannot be extracted by these methods, endoscopically guided electrohydraulic or laser lithotripsy is another option that can be pursued to fragment the stone.[13,14] If lithotripsy is unavailable, a linear incision through the ventral aspect of the penis and urethra overlying the stone may ultimately need to be performed to remove the stone. Depending on the extent of mucosal and submucosal damage, this incision may be closed or left to heal by second intention.

Radial extracorporeal shock wave therapy (RSWT), using a device commonly used for therapy of musculoskeletal problems (EMS Dolorcast), was reported to successfully fragment obstructive urethroliths in three equids. Using the large-headed probe placed directly on the urethra over the calculus without contact gel, the pressure was set at 2.5 bar, 7 Hz, and 2500 pulses were administered over a 5-minute period. Initially, sandlike sludge was passed out the end of the urethra followed by fragments of the uroliths.[15] Although this procedure was performed on two animals postmortem and bladder rupture was not prevented in the single live horse treated, this novel approach warrants consideration if it can be performed in a timely manner shortly after the patient is admitted and the stone is found.

When the stone has been removed, a catheter should be placed for 5 to 7 days from the PU site exiting the end of the penis to limit potential development of a urethral stricture. In my experience, second-intention healing of PU incisions typically results in dilation of the urethra above the ischial arch, and urethral strictures are more likely a consequence of the damage and associated inflammation at the site where the urethrolith was lodged (Figure 66-3). Broad-spectrum antibiotics should be administered as long as the catheter remains in place to limit the risk of ascending urinary tract infection, and

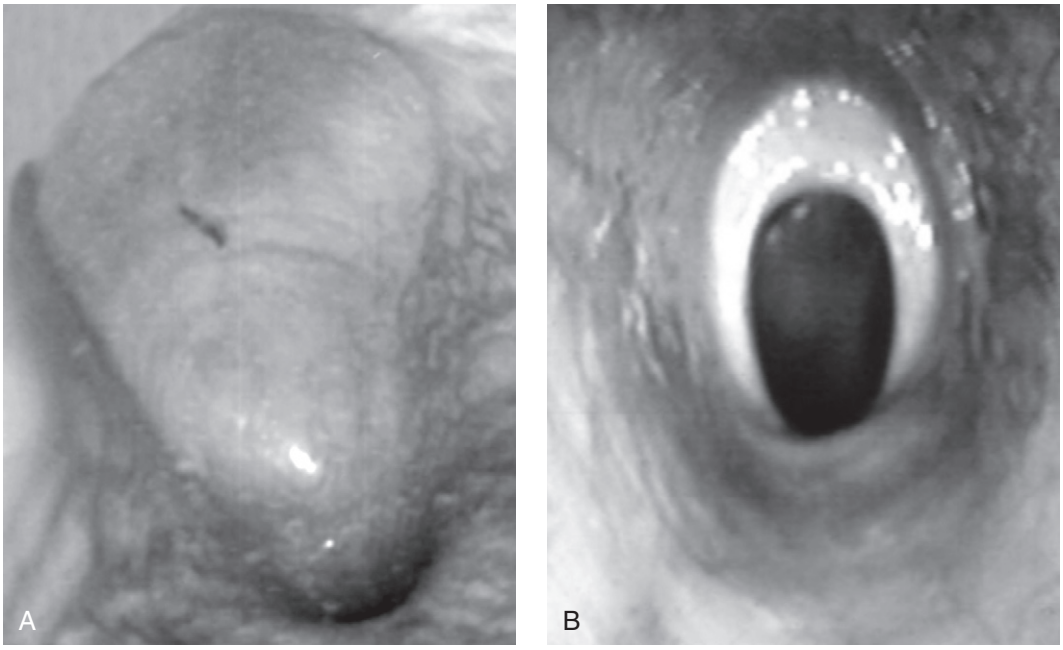

Figure 66-3. Endoscopic images of the urethra revealing distention at the level of the ischial arch **(A)** and a urethral stricture at the site of a previous obstructing urethrolith **(B).** Both images were recorded approximately 1 year following the initial examination.

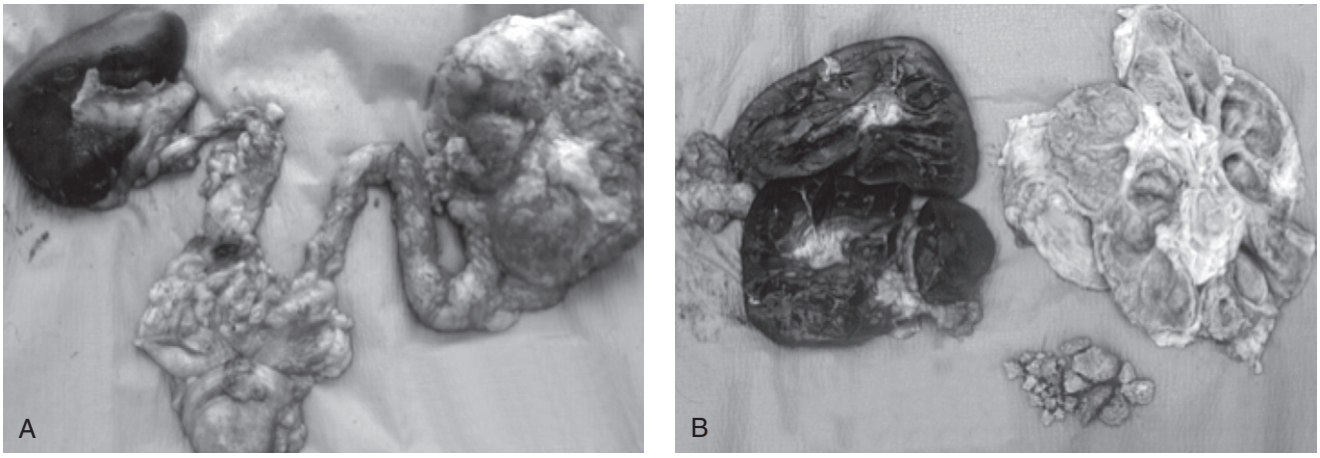

Figure 66-4. Gross pathology photographs of the urinary tract from a 17-year-old Arabian gelding that suffered four bouts of obstructive urethrolithiasis over a 10-year period. The right kidney and ureter were enlarged **(A)** and the kidney parenchyma had been replaced by an abscess containing multiple nephroliths **(B).**

anti-inflammatory drugs are indicated for 3 to 5 days. To limit risk of recurrence, avoidance of forage high in calcium (i.e., alfalfa hay) is typically recommended, although it is important to recognize that all forage provides more calcium than a horse's daily need. Therefore, even when fed grass hay, horses continue to excrete large amounts of calcium carbonate crystals in urine. As described in Chapter 65, use of dietary supplements to acidify equine urine (fewer calcium carbonate crystals form in acidic urine) has not been highly successful.

Over the past decades, I have evaluated several horses with recurrent urethral obstruction due to urethrolithiasis.[16] Further investigation of these cases using transabdominal ultrasonography, cystoscopy, ureteral catheterization, and nuclear scintigraphy has revealed evidence of unilateral upper urinary tract disease including chronic pyelonephritis (to the point of renal abscessation) and nephrolithiasis in many of these patients (Figure 66-4). The most consistent abnormal finding in a series of these cases was an abnormal ureteral opening into the bladder detected with cystoscopy.[16] When affected horses have normal renal function (after acute obstruction and post-renal failure have resolved), unilateral nephrectomy may be the only curative treatment, although I have followed one gelding with a smoldering left-sided pyelonephritis and nephrolithiasis that did not have further problems for over a decade after two episodes of obstructive urethrolithiasis. The important point is that obstructive urethrolithiasis may be a sign of upper urinary tract disease in many horses. Why horses with upper urinary tract disease produce smaller stones that may become lodged in the urethra, as compared to horses with primary cystolithiasis that often have much larger stones, is not known. Nevertheless, in

horses that present with obstructive urethrolithiasis, further investigation for upper urinary tract disease, along with culture of urine and the urolith, may be warranted after the obstruction has been relieved.

Soft Tissue Lesions

Soft tissue lesions involving the urethra include neoplasms, hematomas, and strictures.[17-22] In some parts of the world, parasitic granulomas of the distal urethra also remain a problem.[23-25] However, since the development of avermectin anthelmentics, urethral habronemiasis is rarely observed in well-managed horses. Soft tissue lesions more often result in preputial discharge and dysuria and only rarely cause urethral obstruction. Owners may report a malodorous sheath or diversion of the urine stream and may also observe a growth on the penis when it is dropped for urination. Affected animals may also have urine scalding of the inside of their hindlimbs (males) or perineum (females).

Neoplasia

A recent report of 114 penile and preputial tumors in equids found squamous cell carcinoma (SCC) to be the most common (79%), followed by papilloma (10%) and melanoma (6%).[26] Mean age of affected equids was 19.5 years with no apparent breed predilecton. Common presenting complaints included observation of a penile mass (50%) and malodorous purulent or sanguineous preputial discharge (41%); impaired urination was reported in only 22% of affected animals. The glans penis was the most frequently affected site. In an earlier report of 48 cases of SCC, the glans penis was also the most common site (53% of cases) and the urethral process and urethral sinuses were affected in 28% of cases.[27] Of interest, 75% of affected equids in this earlier report were ponies. Surgical treatment was pursued in 27 animals but recurrence was reported in 9 (33%).[27] In a subsequent report of 45 horses that had surgical resection of penile and preputial SCC, six of 31 (19%) with more than 1 year of follow-up had recurrence, prompting euthanasia in five of these animals.[28] In a more-recent report of 77 horses with penile and preputial SCC, treatment included local excision with or without cryotherapy (15%), partial phallectomy (68%), or penile amputation and preputial resection with retroversion of the penis (17%). Again, recurrence rate approached 30% within 18 months of surgery and was associated with histopathologic score (grade 1 to 3) of the SCC.[29] Taken collectively, these case series clearly demonstrate that SCC is an aggressive, locally invasive neoplasm that warrants a guarded long-term prognosis at initial diagnosis. Larger tumors, evidence of regional lymph node involvement, and a higher histopathologic grade all warrant a poorer prognosis. Because of these high recurrence rates with surgical resection, concurrent treatment with antineoplastic agents is worthy of consideration. Topical application of 5-fluorouracil at 14-day intervals after surgical resection of penile and vulvar SCC resulted in tumor remission (7 to 52 months of follow-up) in eight horses, and another two horses with small penile lesions were succesfully treated with topical application of 5-fluorouracil alone.[30] Similarly, long-term follow up of 573 equids with cutaneous neoplasms treated with a combination of surgical resection and one course of intralesional cisplatin treatment reported a success rate of 88% for SCC, including SCC of the exernal genitalia in 18 males and 15 mares.[31] SCC lesions of the external genitalia of male and female horses were recently shown to contain DNA of a novel equine papilloma virus (*Equus cabillus* papilloma virus-2).[32] Consequently, development of a vaccine to guard against this locally invasive neoplasm may be on the horizon.

Although less common, papillomas (warts) can affect the penis and urethra of younger horses, and melanomas can be problematic in gray horses of all ages.[17,26,33] Infiltration and associated inflammation of preputial tissue may make it difficult for the penis to drop for urination. Urine scalding within the prepuce contributes to further inflammation and development of discharge and a strong urine odor. Papillomas are often self-limiting and regress spontaneously within a few months after appearance; however, some transform into SCC and will need specific treatment.[33]

Melanomas of the prepuce and penis are usually not malignant and are amenable to cryotherapy, surgical removal, or laser ablation.[17,33] However, recurrence may also be a problem with this neoplasm.

Sarcoids can also affect the external genitalia but are more commonly found on the scrotum and prepuce; the penis is rarely affected.[17] Various other neoplasms that may affect the penis and urethra include lymphoma, lipoma, fibroma, basal cell carcinoma, neurofibroma, hemangioma, and adenocarcinoma. For example, a recent report in a mare described a pelvic canal lymphoma that compressed the urethra resulting in dysuria.[18]

Because approach to treatment of preputial and penile neoplasms varies among hospitals and clinicians, comparing treatment outcomes is difficult. As a concequence, a standardized approach to diagnosis and treatment of these lesions has recently been advanced. This approach combines clinical evaluation, including ultrasonography of regional lymph nodes, with histopathology (including grading of SCC) and is worthy of consideration if treatment outcomes are to be improved in the future.[34]

Hematoma

Penile hematomas are an uncommon problem in horses. The diagnosis is made on the basis of history (trauma) and physical examination findings of an enlarged, painful penile shaft. Ultrasonographic evaluation is useful to document size and the characteristic hypoechoic fluid pockets within the hematoma. The injury may cause deviation of the penile shaft and, when severe, may cause urethral obstruction and bladder rupture.[19-21] Trauma to the glans during erection may also cause a rent or rupture of the CSP or CCP into the urethral sinus, leading to penile hemorrhage during breeding.[35] Treatment consists of sexual rest and hydrotherapy, although drainage of the hematoma by needle aspiration can hasten recovery.[21] Temporary placement of an indwelling bladder catheter may be necessary with large hemotomas that can cause partial or complete urethral obstruction. For more information on this condition, see Chapter 60.

Urethral Stricture

Blunt or sharp traumatic injury as well as damage to the urethral mucosa from an obstructive urethrolith can lead to stricture formation, especially following circumferential ulceration of the urethral lumen.[9,22,36] Presence of a stricture may or may not

cause dysuria but likely increases the risk of urethral obstruction. After diagnosis via urethroscopy, phallectomy has been performed, but more recently, laser ablation of the stricture has become the treatment of choice.[22,36]

Urethrorrhexis

Urethrorrhexis, urethral rupture or laceration, is a rare problem in male horses and has not been described in mares, other than iatrogenic sphincterotomy for removal of cystic calculi.[10,37] The problem is most commonly traumatic in origin, either from being kicked or some other form of blunt or sharp trauma. The extrapelvic portion of the urethra is particularly vulnerable to injury at the level of the ischial arch, where it is superficial and relatively unprotected. Urethrorrhexis can also be a complication of urethral obstruction with a urolith and in a recent report in a neonatal colt, the problem was speculated to be a consequence of dystocia.[38] Accidental transection of the urethra during castration has also been described.[39]

Diagnosis of urethrorrhexis can be challenging because horses may have marked soft tissue swelling of the perineum, prepuce, penis, and caudoventral abdomen at the time of presentation. A lack of observed urination should increase suscpicion, as should leakage of serosanguineous fluid from the tip of the urethra. At times, urine may also be observed leaking from the wound. If urine is not detected at presentation, another characteristic finding is rapidly progressive swelling as urine continues to be passed into tissue planes, rather than being eliminated from the body. Rectal palpation is often unrewarding because the bladder will not be distended without obstruction and the intrapelvic portion of the urethra is usually intact.

When open wounds over the urethra are suspected to communicate with the urethral lumen, retrograde infusion of sterile saline through a catheter placed in the distal urethra may result in saline exiting the wound, confirming urethral disruption. Next, urethral mucosal damage can be directly visualized by urethroscopic examination, but it may be difficult to determine whether or not a full-thickness tear of the urethra has occurred. Ultrasonographic examination often reveals fluid accumulation in tissue planes but, again, it may be difficult to confirm actual urethral disruption with this imaging modality, although retrograde distention of the urethra by saline infusion may be helpful during imaging. Retrograde contrast radiography of the penis may be the most definitive imaging modality and also allows determination of the exact site of urethral disruption.

Primary surgical repair of urethrorrhexis is often not possible because urethral margins are irregular and inflamed, unless the disruption was caused by accidental incision. Accordingly, treatment is focused on preventing further tissue damage by urine accumulation (Figure 66-5) by diverting urine flow through a catheter placed into the bladder for 5 to 7 days. Treatment with broad-spectrum antibiotics and anti-inflammatory agents is also indicated to prevent wound sepsis and to limit the risk of a catheter-associated ascending urinary tract infection. Wounds often heal well by second intention, and recovery may be accelerated by partial closure of the urethra.

Hematuria and Hemospermia

Hematuria and hemospermia can be caused by the soft tissue and neoplastic lesions discussed earlier as well as disorders of the accessory sex glands, most notably seminal vesiculitis.[40-42] The latter problem can be challenging to diagnose because there may be no clinical signs other than discoloration of the ejaculate. Catheterization and collection of fluid from each ejaculatory duct for cytology and bacterial culture is required for definite diagnosis.[42] Serosanguineous fluid may also drip from the penis with obstructive urethrolithiasis or urethrorrhexis.

Tears or rents in the proximal urethra at the level of the ischial arch are another cause of hematuria in geldings and hemospermia in stallions.[43] Affected horses generally void a normal volume of urine that is not discolored. At the end of urination, squirts of bright red blood exit the penis, in association with forceful contractions of the bulbospongiosus muscle to empty the urethra of residual urine and to empty the CSP of blood. Occasionally, a smaller amount of darker blood is passed at the start of urination. The condition does not appear painful or result in pollakiuria or dysuria. In actuality, postурination hemorrhage would be a better descriptor for this condition.

Examination of affected horses is generally unremarkable, and laboratory analysis of blood reveals normal renal function

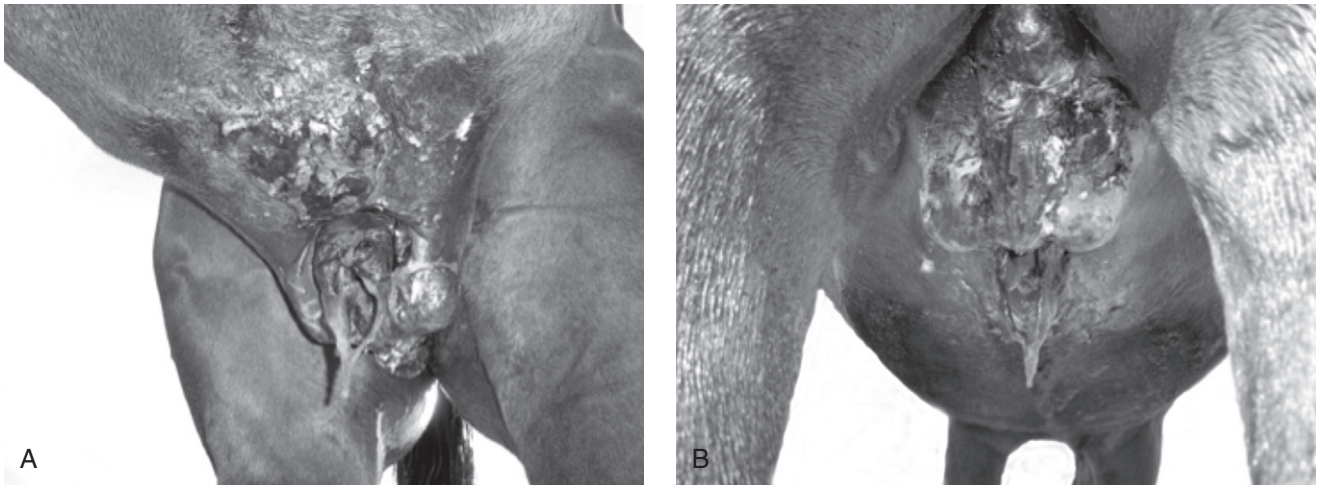

Figure 66-5. Photographs of the front view **(A)** and hind view **(B)** of the prepuce and ventral abdomen of a horse with marked tissue inflammation and necrosis several days following urethral disruption and accumulation of urine in tissues of the perineum, prepuce, and ventral abdomen.

although mild anemia (packed cell volume 25% to 30%) is an occasional finding. Urine samples collected mid-stream or by bladder catheterization appear grossly normal. Urinalysis may have normal results or there may be an increased number of red blood cells on sediment examination, a finding that would also result in a positive reagent strip result for blood. Bacterial culture of urine yields negative results.

Although the pathophysiology of this condition remains unclear, it is likely that the tear or rent develops as a "blowout" of the CSP into the urethral lumen (Figure 66-6). Contraction of the urethralis and bulbospongiosus muscles during ejaculation causes increased pressure in the CSP, which is essentially a closed vascular space during ejaculation. The bulbospongiosus muscle also undergoes a series of contractions to empty the urethra of urine at the end of urination. Hence, the proposed explanation for the bleeding at the end of urination in horses with urethral rents is a sudden decrease in intralumenal urethral pressure after urination while pressure within the CSP remains high.[44] When the lesion has developed, it is maintained by repeated bleeding at the end of urination.

An early report described this syndrome in four Quarter Horse type horses and attributed the post-urination bleeding to ulcers of the proximal urethra that could be seen via urethroscopy.[45] With the introduction of high-resolution videoendoscopic equipment to equine practice, one or more fistulas, rather than ulcers, are typically seen along the dorsocaudal (convex) aspect of the urethra at the level of the ischial arch. Within 1 to 2 weeks after the tear originates (about the time that endoscopy may be pursued), the edges of the urethral mucosal tear have largely healed as one or more fistulas communicating with the vasculature of the CSP (Figure 66-7). External palpation of the urethra in this area is usually unremarkable but can assist in localizing the lesion because external digital palpation can be seen via the endoscope.

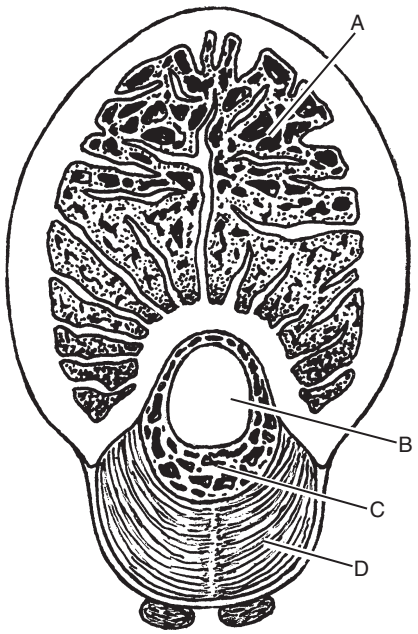

Figure 66-6. A cross section of the equine penis at the level of the ischial arch showing two separate vascular structures: *A,* corpus cavernosum penis; *B,* urethral lumen; *C,* corpus spongiosum penis; *D,* bulbospongiosis muscle.

Interestingly, the majority of geldings with proximal urethral tears have been Quarter Horses or Quarter Horse crosses, which have been free of other complaints. The consistent location of the rents suggests that there may be an inherent weakness of the urethral mucosa adjacent to the CSP at the level of the ischial arch. Some affected horses have unusual perineal conformation (see Figure 63-2), but histologic examination of the penis in a group of normal horses could not demonstrate any anatomical reason for the tears to occur at this specific location.[44]

Because hemorrhage resolves spontaneously in some geldings, no treatment may be initially required. If the problem persists for more than a month or if significant anemia develops (unlikely), a temporary subischial PU approach is made. The incision is extended through the fibrous sheath surrounding the CSP but not into the urethral lumen. The incision creates a "pressure relief valve" or path of lower resistance for blood to exit the CSP after urination. The surgical wound requires a couple of weeks to heal, and moderate hemorrhage from the CSP is apparent for the first few days after surgery. Additional treatment consists of local wound care and prophylactic antibiotic treatment (typically a trimethoprim-sulfonamide combination) for 5 to 7 days. Bleeding should resolve within a week following this procedure.

SURGICAL PROCEDURES

J. Brett Woodie

Perineal Urethrotomy

Perineal urethrotomy (PU) is performed for temporary urine diversion in male patients with obstructive urinary outflow disease or is used to access the bladder to remove small cystic calculi.[9,46-50] In patients requiring chronic urinary diversion, a permanent urethrostomy may be created. It is best to perform a PU or urethrostomy as a standing surgical procedure with the horse sedated and confined in stocks.[9,46,50] The horse should be sedated with detomidine hydrochloride or xylazine hydrochloride. Butorphanol tartrate can be used in combination with either of the α_2-agonists, if necessary. Epidural anesthesia is used to desensitize the perineal region. The rectum should be evacuated prior to starting the surgical procedure and the tail should be tied overhead to the stocks to support the horse if the patient becomes ataxic. The perineal region should be clipped and prepped for surgery. Local anesthetic can be infused to desenstize the skin and superficial tissues if the epidural is not effective. A bladder catheter should be advanced through the urethra to identify the urethra during surgery. Appropriate illumination of the surgical field will be necessary.

A 6- to 8-cm longitudinal midline skin incision is made in the perineum extending from a point 4 to 6 cm ventral to the anus ventrad to just distal to the the ischial arch (Figure 66-8, *A*).[9,46,50] The subcutaneous tissues are divided, and the longitudinal incision is continued deep to divide the paired retractor penis muscles and the bulbospongiosus muscle.[49,50] The incision is continued through the CSP that envelops the urethra. The urethra is exposed by retraction of these muscles. The urethra is identified and stabilized by palpation of the urinary catheter (Figure 66-8, *B*). A longitudinal incision is made along the caudal surface of the urethra, and the mucosa is reflected abaxially. Hemorrhage from the CSP may be copious but subsides spontaneously. It is important that the incision be made

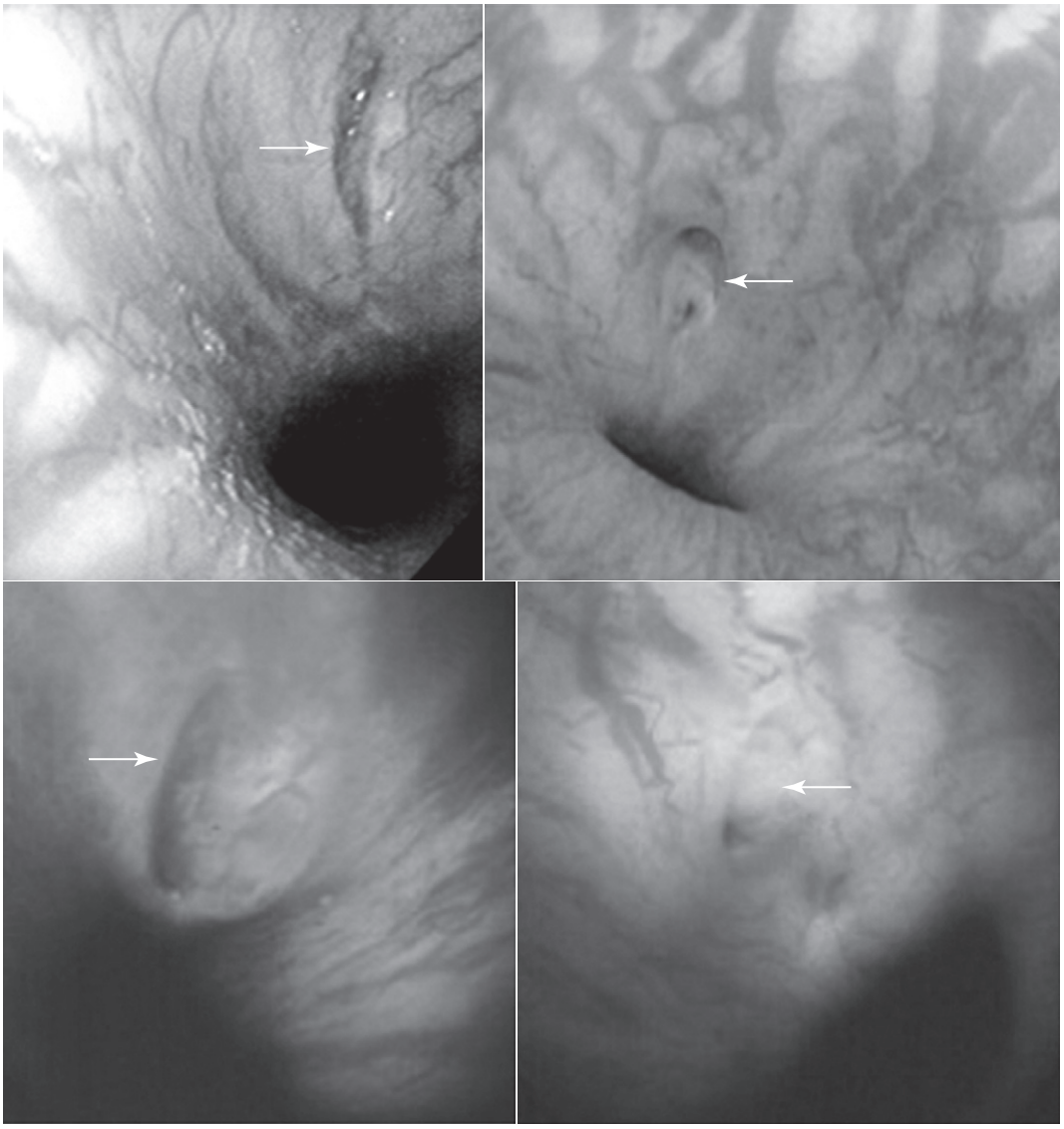

Figure 66-7. Four endoscopic images showing the variable appearance of urethral rents *(arrows)* causing hematuria at the end of urination in geldings or hemospermia in stallions. A consistent finding is that urethral rents are located along the convex aspect of the urethra at the level of the ischial arch.

on the midline and that multiple planes of dissection are not made through the tissues. Edema will form in the tissues if excessive manipulation is required and this will increase the difficulty of the procedure. Intraluminal obstructions such as uroliths may be manipulated and removed by urinary catheters or forceps inserted through the PU incision.[49,50]

For temporary diversion of urine, the wound is managed with local care during the process of secondary-intention wound healing (Figure 66-9). A urinary catheter may be placed through the temporary urethrotomy incision and secured with stay sutures after surgery. Typically, a urethrotomy wound heals within 14 to 21 days with minimal complications.[50] Hemorrhage may occur from the surgical site at the end of urination for up to 2 weeks. The bulbospongiousus muscle contracts at the end of urination, and this increases the pressure in the CSP, causing hemorrhage. If a permanent urethrostomy is desired, the muscles of the ventral penis are sutured along their cut edges with a continuous suture of 3-0 synthetic absorbable suture material to control hemorrhage. The urethral mucosa and skin are approximated using interrupted sutures of 2-0 or 3-0 synthetic monofilament absorbable or nonabsorbable suture material (Figure 66-10). Care must be taken to ensure accurate, tensionless apposition of the perineal skin and urethral mucosa.

Distal Urethrotomy

A distal urethrotomy may be required to remove an obstruction that can not be resolved using endoscopic techniques. Horses should be anesthetized and positioned in dorsal recumbency. Technically, the surgical procedure is similar to that for a PU. If the lesion is sufficiently distal, a tourniquet may be applied to assist with hemostasis and improve intraoperative visualization. Incisions are made over or slightly proximal to the urethral calculus to permit the insertion of grasping forceps. Placement of a catheter to the level of the obstruction can aid in identification of the urethra. Palpation of the calculus may be possible

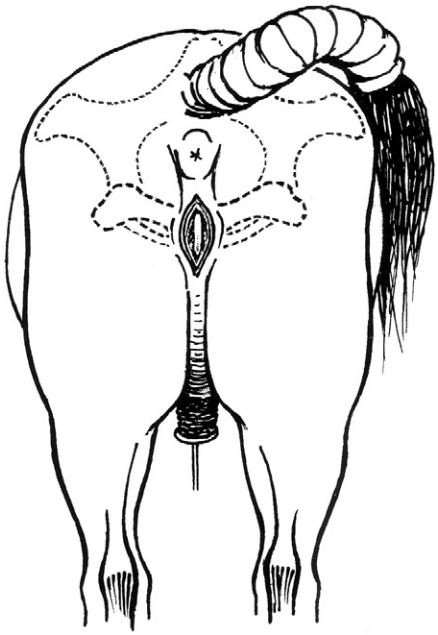

Figure 66-8. The urethral incision is performed immediately dorsal to the ischium to decrease the likelihood of postoperative urine scalding in a horse with a perineal urethrotomy. Preoperative placement of a stallion urinary catheter facilitates intraoperative identification and exposure of the urethra.

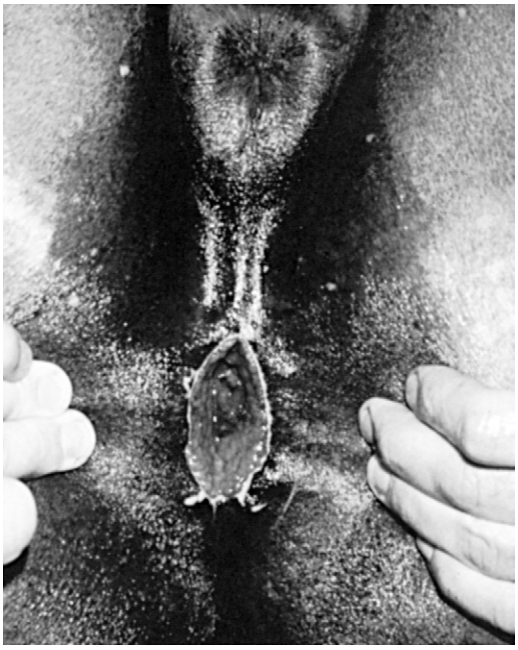

Figure 66-9. Perineal urethrotomy for temporary urinary diversion will heal by secondary intention with minimal complications.

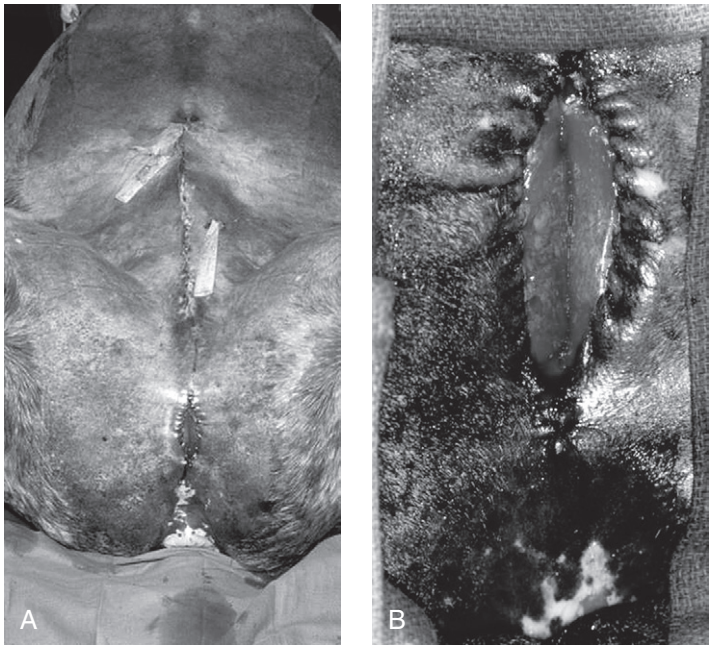

Figure 66-10. A, Perineal urethrostomy for chronic urinary diversion to bypass extensive preputial and penile squamous cell carcinoma. **B,** Accurate apposition of mucosal and cutaneous layers is essential to minimize postoperative complications.

depending on its size. Ultrasonography can assist in localizing the obstruction and guide placement of the incision. After removal of the calculus, the incision is closed in anatomic fashion using 3-0 synthetic absorbable suture material. It is important to accurately reconstruct the CSP and bulbospongiosus muscle to reduce the risk for urine leakage and development of cellulitis. Approximation of the retractor penis muscle provides additional security for the closure.

Urethroplasty

The patient is sedated, restrained, and prepared for standing surgery as described. General anesthesia will be needed if the urethral injury involves the penis distal to the scrotum. Repair of a urethral laceration is consistent with the repair of any other hollow viscus. Careful attention to wound débridement, lavage, and preservation of intrinsic vascular and neural supply is

important. If the urethral laceration is circumferential, an end-to-end anastomosis will be required. Small-diameter (3-0) absorbable suture material such as polyglactin 910 (Vicryl, Ethicon) should be used. The use of an intraluminal stent (urinary catheter) for the repair of a lacerated urethra is considered acceptable.[51]

Aftercare

Postoperatively the patient should be monitored to ensure that urination occurs without difficulty. Antibiotics and nonsteroidal anti-inflammatory drugs should be administered as required. In cases in which urine escaped into the surrounding tissues, the use of surgical drains is required.[39,47]

Traditionally, catheters were left in place after primary repair of a traumatized urethra.[52] However, the literature is unclear whether prolonged maintenance of an intraurethral catheter prevents or promotes stricture formation. More recently, chronic placement of urinary catheters has been discouraged because of locally increased inflammatory response and subsequent stricture formation.[52] Because the value of chronic catheterization is unclear, it is best to remove the catheter as soon as possible.[51-53] Postoperative stricture formation may be minimized by limited duration of catheterization, accurate tissue repair, effective hemostasis, and adequate drainage of the periurethral tissues.[39,47] Foley catheters may be secured in place with stay sutures during the first 48 to 96 hours. The balloon on the Foley catheter should be distended with saline solution rather than air.

Hygiene around the surgical site is imperative. There is a high risk of urine soilage and urine scalding of the hindlimbs and ventral perineum. The urethrotomy and surrounding tissues should be cleaned daily and protective emollients applied. Care should be taken not to disturb the sutures if a urethrostomy procedure was performed. The skin of the ventral perineum, inguinal region, and adductor surfaces of the hindlimbs should be cleansed daily and protective emollients applied.

Complications

Mild tenesmus and discomfort are expected after surgery. The effect of urine contamination of tissues is usually minimal unless tissue trauma is advanced or sepsis is present. Antibiotic and anti-inflammatory therapy is appropriate in such cases. Partial dehiscence of urethrostomy incisions occurs infrequently and may be managed with local tissue therapy, débridement, and delayed closure.[39,47] Stricture of the urethra is a common postoperative finding[8]; stricture is most commonly observed in distal urogenital surgeries such as posthioplasty or subtotal phallectomy.[23] Surgical procedures involving the distal urethra may require spatulation to reduce the likelihood of stricture formation (see Chapter 60).[54] Less commonly, thin veils of urethral mucosa may form in such a fashion as to partially occlude the urethral lumen, requiring further surgical revision.[49]

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SECTION
XI

Diagnostic Imaging

John A. Stick

CHAPTER
67

Radiography

Elizabeth A. Ballegeer and Nathan C. Nelson

Radiography remains the most ubiquitous imaging modality available to the equine practitioner. The importance of radiographs in the evaluation of lameness, as well as areas inaccessible to other modalities because of size or positioning issues, must not be trivialized or forgotten in the age of bigger and better diagnostic modalities.

INDICATIONS

Indications for radiographic examination are multitudinous. The most common reason to obtain equine radiographs is to examine a particular joint or joints following appropriate localization of lameness. Radiographs are also commonly obtained after trauma to a particular body region is witnessed or suspected. Radiographs may be taken after identifying an area of increased bone production on a nuclear scintigraphic study. A swelling seen on a limb or atypical posture may also indicate an area that is appropriate for radiographic examination to either help determine the possible underlying cause or define bony changes. Draining tracts injected with positive contrast material can help define involved structures that may modify therapy or change prognosis. Serial weight-bearing radiographs evaluate and monitor progression of distal phalangeal rotation and/or distal displacement in horses with laminitis. Ataxic horses may have changes seen on cervical spinal radiographs that can suggest spinal cord compression, warranting a more definitive diagnostic imaging technique, myelography. Thoracic radiography is indicated in cases with respiratory or cardiovascular disease. Abdominal radiographs, though rarely performed in the adult equine patient, may be indicated if sand colic or enteroliths are suspected. Neonates with developmentally atretic or mechanically obstructed intestinal loops are more completely evaluated with abdominal radiographs than adults. Radiographs are also the imaging modality of choice for evaluating placement of surgical fixation devices.

X-RAY PRODUCTION AND EQUIPMENT

To better understand the origin of a radiographic image, some underlying principles of x-ray production must be discussed. This begins with the components and function of an x-ray tube.

X-rays are produced when high-energy electrons are accelerated through a vacuum-sealed tube and strike a target called an *anode*. The electrons originate from a small electron cloud surrounding a negative pole *(cathode)*, which is typically a heated tungsten filament (Figure 67-1). The anode is constructed of tungsten alloy to which a positive charge is applied. Because of their negative charge, the accelerating electrons strike the anode, reacting with the nucleus or the shell electrons of the tungsten atoms in the anode. Electromagnetic radiation is released by these interactions, at the wavelength of x-rays (10 to 0.01 nm).

The filament length and anode disc angle may vary, altering the focal spot of the exposure. The focal spot is the area of the anode struck by the electrons generated in the cathode. Some x-ray machines allow selection of a larger or smaller focal spot. Smaller focal spots create better detail in an image, but fewer x-rays can be generated, and the radiographic field coverage may be lessened. Larger focal spots allow higher numbers of x-rays to be generated (which is necessary for thicker areas of the body) but sacrifice radiographic image detail.

As electrons strike the anode, they rapidly decelerate. The lost kinetic energy of the electron beam generates a large amount of heat, which must be removed from the anode. Various mechanisms of heat dissipation exist. Early anodes were stationary tungsten blocks embedded in better-heat-conducting copper; most modern anodes have an anode shaped as a disc. This disc is connected to an induction motor, which rotates the anode, spreading heat along a larger surface. The tube is also housed in an oil bath, which dissipates the radiated heat from the anode. Smaller x-ray tubes (such as portable or dental units) still have a stationary anode. This limits the maximum tube current and number of x-rays that can be generated.

As electrons strike the anode, x-rays are primarily directed at a steep angle away from the anode; however, smaller volumes of x-rays are generated in surrounding directions. The outer layer of the tube housing is lead, which shields the outside environment from stray irradiation produced from the anode. A tube port in this metallic enclosure emits the primary beam in the desired direction. Modifiable ports have movable lead sheets, which collimate the primary beam to a rectangle of variable length and width, though fixed aperture ports are also useful for certain applications, such as dental radiography. A mirror-reflected light within this housing mimics the borders of the primary beam for positioning purposes. The primary beam is also filtered with aluminum, copper, or plastic to remove very-low-energy x-rays that would increase the patient's radiation exposure but are insufficiently energetic to produce a diagnostic image.

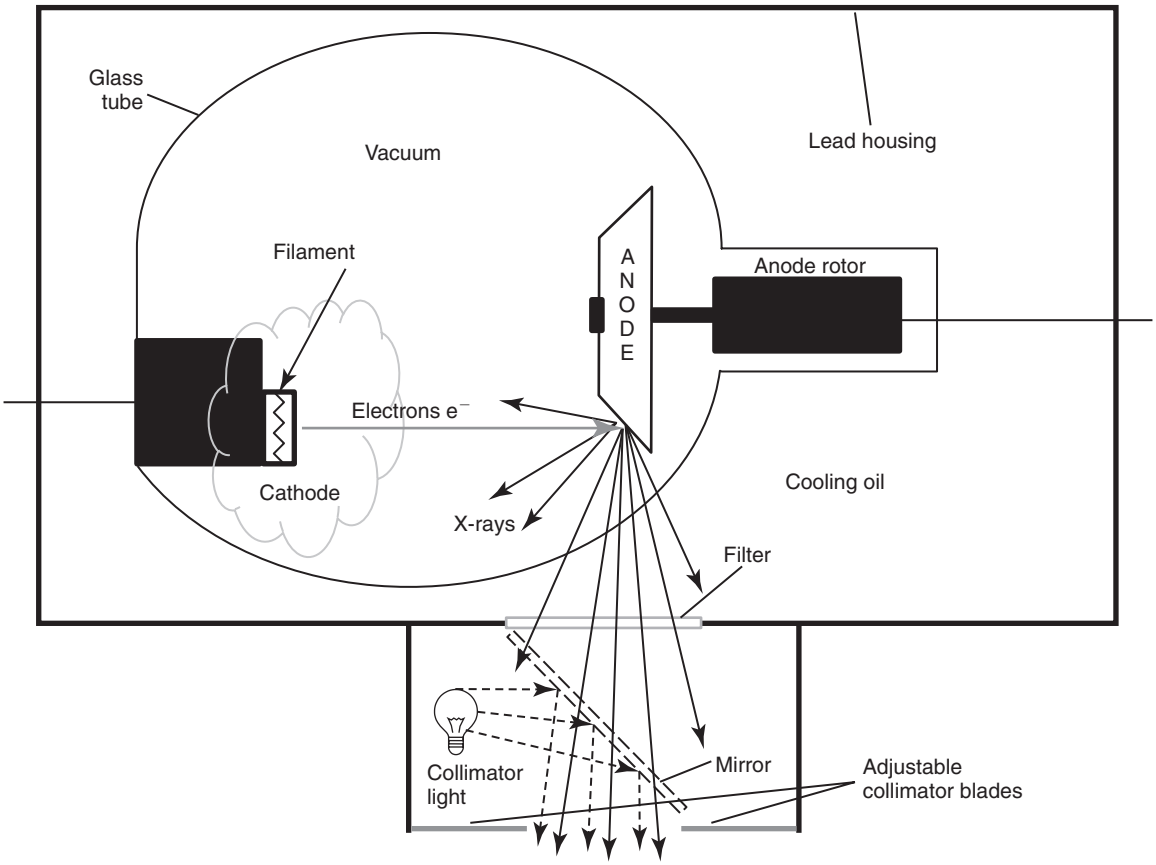

Figure 67-1. Basic x-ray tube configuration; the drawing is not to scale, and the shapes are not strictly accurate.

The energy of x-rays produced is directly related to the modifiable voltage difference between the cathode and anode. This potential difference is usually between 50 and 120 kilovolts (kVp). kVp determines the peak energy of the electron accelerated from cathode to anode, whose energy is then converted into x-ray production. The x-ray beam contains photons of various energies, the mean of which is between one third and one half of the peak energy.

The number of x-rays produced may be increased or decreased by the user depending on imaging needs. A current, measured in milliamperes (mA), is applied through the cathode filament for a selected amount of time, measured in fractions of seconds (s). The product of current and time is mAs and is proportional to the number of x-rays produced.

Both kVp and mAs may be modified to alter the final image quality. Increasing the energy (kVp) of an x-ray beam increases image exposure (resulting in a darker radiographic image) and increases tissue interactions of the x-ray beam. This creates more scatter radiation and more shades of gray on the resulting image, which decreases contrast between different types of tissue (Figure 67-2). Conversely, decreasing kVp reduces overall exposure but enhances image contrast. Increasing the amount of x-rays by increasing the current, exposure time, or both (mAs) will make an image much blacker, but with relatively less tissue interactions than increasing kVp. This creates a very-high-contrast or black-and-white image.

Scatter radiation is always present, both originating directly from the x-ray tube and produced when the beam interacts with matter. Scatter becomes worse with high-kVp techniques, thicker body parts secondary to tissue interactions, or larger fields of view from both the tube and tissue interactions. An increasing amount of scatter degrades the image by adding superfluous radiation that is not related to the imaged anatomy.

The subject of scatter radiation requires brief discussion of grids. Grids improve image contrast by removing some or most of the scatter radiation before it arrives at the image-recording device. Grids are configurations of lead strips divided by radiolucent separators that block radiation that is not oriented parallel to the primary beam. Grids can be focused, which is more common than parallel. *Focused* grid lead strips typically converge slightly on the tube side, and diverge slightly on the film side, in relation to the primary beam x-rays. It is important to note that grids must be centered with the primary beam and be used at a particular distance from the tube, or the angulation of the lead strips will remove primary beam as well as scatter, which is referred to as *grid cutoff*. *Grid ratio* refers to the ratio between the height of the lead strips and the distance between them. The higher the grid ratio, the better the grid functions, although high grid ratio requires a higher technique to create the same exposure of the image. A grid ratio of at least 8 : 1 for techniques with kVp below 90 and at least 12 : 1 for techniques higher than 90 is recommended.[1,2] The *grid frequency* refers to the number of lead strips per unit length; frequencies above 30 lines/cm is recommended.[3] Because grids employ thin linear strips of lead, a normal side effect of use is the appearance of *grid lines* on the final radiographic image.

Typically, grids should be used for body parts that are more than 10 cm (4 inches) thick, because above this thickness,

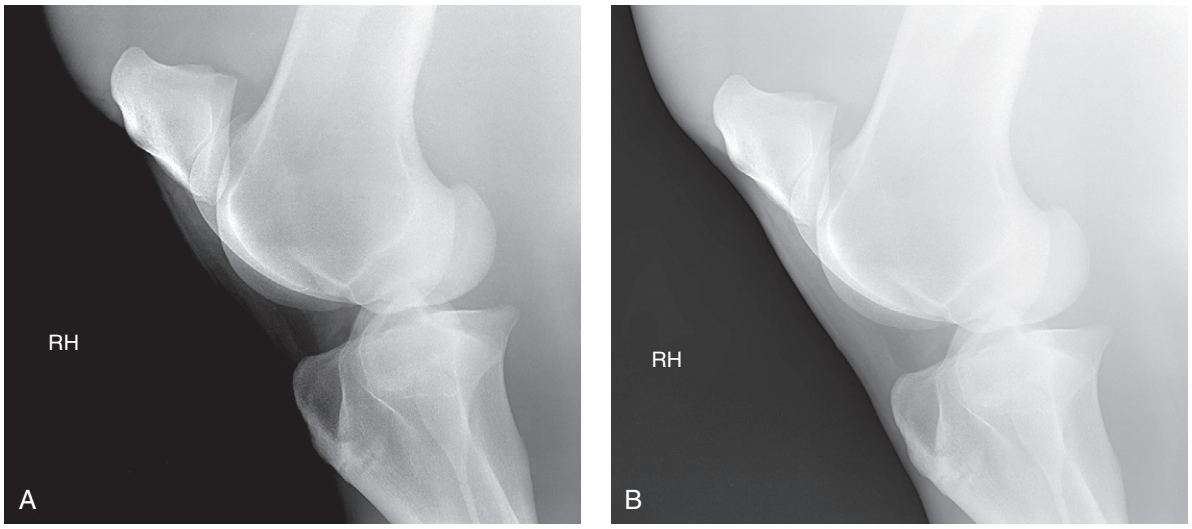

Figure 67-2. A, The lateromedial radiographic view of the stifle of a 3-year-old Thoroughbred gelding simulating high-mAs, low-kVp technique. Note that though the contrast within the bone is great, allowing visualization of trabecular pattern, the large contrast of the image makes soft tissues less visible; **B,** The same radiographic view simulating high-kVp technique. Note the large numbers of grays allowing visualization of soft tissues, differentiation from fat, and the relative lack of contrast within bony structures. Note that the relative brightness of the two images is similar.

significant scatter is produced by tissue interactions. In equine radiography, a grid is often a stationary, separate sheet that must be placed directly against the cassette, but it may be incorporated into the cassette itself. Grids are rarely used for imaging equine distal limbs as the body parts are usually less than 10 cm thick and alignment of the grid with the primary x-ray beam is difficult. Separate grids are somewhat fragile; bending or being stepped on damages the lead strips, resulting in visible white defects on the image.

IMAGE PRODUCTION

After the x-rays have penetrated the patient, carrying with them the differential attenuation that comprises the diagnostic information of the study, it must be transformed into a visible image. This may be accomplished through analog film-screen combinations or through newer digital radiographic techniques.

Film-Screen Combinations

Traditional image formation involves exposing radiographic film to produce an analog picture. The x-ray film is housed within a protective cassette. X-ray film is composed of a plastic base sheet with adherent emulsion on both sides. This emulsion is made of gelatin and contains the silver halide crystals that create the film image.

X-ray film is not particularly sensitive to direct x-ray exposure. To counter this problem, intensifying screens are used. Intensifying screens contain a phosphorescent material that creates either visible or ultraviolet light proportional to the energy and number of x-rays that interact with them. The light emitted by the screens is responsible for the vast majority of x-ray film exposure. This light and the film must be wavelength matched for appropriate exposure. The scintillating crystals in screens were originally calcium tungstate, which has low efficiency of light transformation, and has since been replaced with crystals with higher efficiency in the rare earth group of elements (elements 57 through 71 of the periodic table of the elements).

Screens are described in terms of *fast* or *slow* intensification factors and have numeric values. *Fast* systems (600 to 800 speed) create more light because their phosphor layers are thicker and crystals are bigger and thus more sensitive, but this comes at the cost of resolution. These systems are typically used for thicker body parts, proximal to the carpus or tarsus. *Slow or detail* systems (100 speed) create proportionally less light because of thinner layers with smaller crystals, but they provide much better detail.[1,2] These systems are recommended for detail examinations, such as navicular series or distal phalanx. *Medium speed* systems (400 to 600 speed) are more encompassing for thicker or thinner parts, are more universally used particularly with mobile x-ray units, but may compromise detail in the distal limb.

Image resolution is defined as the ability to discern two adjacent structures on an image; this is limited to more than 0.3 mm in high-speed systems but improves to 0.1 mm with slow/detail systems.[4]

Exposure to light or x-rays reduces some of the silver halide crystals on the film to metallic silver. Not all of the silver is transformed with the light exposure, but it forms small clusters of silver that act as latent image centers, and it catalyzes the reaction to create more metallic silver within the developer. This metallic silver is what creates the blackness on a film.

Full processing of a film begins with (1) the developer step, then (2) fixation, which halts the development and hardens the film emulsion, and (3) washing, which removes the undeveloped silver halide from the film, creating the white portions of the image. The last step in the process is (4) drying, which further hardens the emulsion of the film and allows it to be preserved.[2] All of these steps have precise time requirements, chemical requirements, and, in the case of development, temperature requirements. Automatic processing manages these parameters provided the processor is functioning properly. More room for error and artifact exists with hand development.

Some limitations to film screen systems are inherent to their analog, hard copy acquisition. Radiographic film has a limited

Figure 67-3. Radiographic equipment: **A,** 400-speed film screen equipment with green-based film. **B,** Computed radiography cassette and imaging plate. **C,** AFGA computed radiography processor. **D,** Sound-Eklin Mark IIG direct digital radiography system. Image plate sends the x-ray information transformed into electric signal to a computer for display. (**D,** Courtesy Sound-Eklin Medical Systems, Inc.)

linear response to radiation, which often leads to over- and underexposed areas on the same film.[5] Also, film images cannot be manipulated after exposure. If technique or positioning is incorrect, the study must be performed again, with almost complete duplication of the steps to perform it. The hard copy nature of the film also requires appropriate physical location for storage and physical transportation for another individual to see it. However, benefits include lower initial cost for systems, easy accessibility to previous studies, and usually better quality control on the part of those taking the radiographs.[5,6]

Computed and Digital Radiography

The conversion of x-rays into a visible image may also be completed digitally and viewed on a computer screen. There are multiple ways to achieve this; digital systems are becoming more common in the veterinary medical community and are almost *de rigueur* in the human medical world, and as they are developed by multiple companies for veterinary applications, they have decreased considerably in price. Again, some underlying mechanisms must be understood to help practitioners decide which system is desirable for their circumstances and to understand the tradeoffs between the benefits and costs. Figure 67-3 depicts the different image-formation systems.

Computed Radiography

Computed radiography (CR) applies to systems with photostimulable phosphors (PSP), also known as *storage phosphors*. It was introduced in the 1980s by Fujifilm Medical Systems.[7] The phosphors are usually in the form of barium fluorohalide powders that coat an imaging plate within a light-tight cassette and fluoresce in response to radiation, much as intensifying screens do. However, the image in CR is created through the release of a charge that remains within the phosphors after the initial fluorescence. The imaging plate, placed into an imaging plate reader after exposure and identification, moves from the cassette across a stage with a laser beam, which re-stimulates the phosphor crystals. The crystals then emit visible light that is captured by a photomultiplier tube that transforms the light

energy to an electrical signal. This signal is digitized by an analog-to-digital converter (ADC) and stored. This step is necessary to transform data from a voltage change or pulse into numbers a computer may store and understand.[8] The imaging plate must then be "erased" by exposure to bright white light to release any residual stored charge, before being returned to the cassette.[1,9] Systems may bring the entire cassette into the internal mechanisms of the processor (which may also bring in debris on the surface of the cassette) or just the internal imaging plate. This latent image within the charged crystals is less resilient than that of film-screen systems, and most of it decays within the first hour after exposure.[4,10-12]

Resolution of a CR system is directly related to the scatter of the laser light when it reads the signal from the crystals, which in turn is related to the thickness of the imaging plate.[12] This does not translate into an absolute value similar to film-screen combination, as systems have differing equipment. However, the overall resolution of CR is decreased compared to film-screen combinations (see Figure 67-4 for comparisons). The largest benefit of CR, when compared to film-screen combinations, resides in the linear response of the PSP crystals to exposure, compared to radiographic film, which allows a greater range in technique, resulting in readable images (also known as *exposure lattitude*), especially when coupled with the ability to manipulate the brightness and contrast of images after they are obtained.[1,5]

Indirect and Direct Digital Radiography

Indirect and direct DR systems use digital detectors that translate exposure into digital signals along a panel directly exposed to x-rays and thus are termed *digital radiography*. These systems eliminate the need for a separate image processing unit as used with computed radiography. The difference between indirect and direct DR lies in the presence or lack of an incorporated scintillator, similar to intensifying screens and image plates.

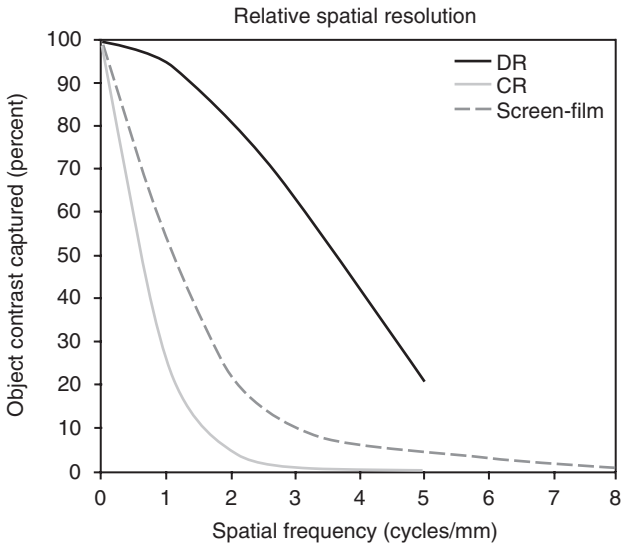

Figure 67-4. Relative resolution capabilities between radiograph modalities, represented as the percentage of an object's contrast recorded by that modality, as a function of size (spatial frequency). Note that discrimination of smaller objects of differing contrast, or increasing spatial frequency, decreases most rapidly with CR system and least rapidly with the DR system. Note this does not take into consideration the resolution of the display system used to view the digital images. *CR*, Computed radiography; *DR*, digital radiography.

Indirect digital radiography (IDR) uses columnar cesium iodide crystals oriented parallel to the primary beam, coupled to a diode layer, which converts light within the differentially stimulated crystals to an electric charge. The charge matrix of the entire array is sensed by an electronic readout system, again converted by an ADC, and stored.[10,13,14] The addition of a scintillating layer adds efficiency to the system, increasing the amount of signal detectable from a measureable amount of radiation,[13] as well as decreasing the blur of signal laterally within the photoconducting layer, though this is also minimized by positively charging the deep side of the layer, attracting electrons.[1]

In contrast, *direct digital radiography (DDR)* omits the scintillation step and uses thin film transistor arrays (TFTs), which directly translate the exposure of an x-ray photoconducting layer (such as amorphous selenium) into a charge, which again undergoes readout, analog-to-digital conversion, and storage.[10,13]

Both of these digital systems make "real-time" images that are displayed within a matter of seconds,[6] unlike CR images, which may take several minutes to be processed. This is a big advantage when retaking radiographs if there are positioning or technique errors. However, digital detectors are expensive, given the number of very small electronic elements that make up a large detector matrix, and are relatively fragile with manipulation. The detectors may be incorporated into positioning tables, or they may be portable and attached to cables. Wireless detector plates are in development, with inherent battery and range challenges. Both IDR and DDR have the similar advantage that CR does over film-screen systems in post-processing manipulation and exposure latitude.

Although the spatial resolution of TFT-based (DDR) systems appears better than film-screen combinations or CR, in reality, the resolution of a digital system is limited by the size of the detector elements and the resulting pixel display (a pixel being one two-dimensional area of data in a digital image), as well as the resolution of the screen upon which they are viewed. Therefore, a very small detector size on a digital system requires more pixels than can be displayed on a monitor, and the advantage over the "lower-resolution" systems is lost.[1]

Charged Coupled Device Cameras

Charged coupled device (CCD) cameras are only mentioned briefly, because they need to be incorporated into a radiographic table and have limited equine application. This system involves a large exposed scintillating layer, which is then minified by an optical lens or fiberoptic coupling devices, a CCD chip that converts the light to electrical charge, and similar analog-to-digital conversion and storage. This system produces a "real-time" image but causes loss of photons in the minification process that leads to increased noise in the image[10,13] and mild distortion of the image at its edges.[6] Despite its shortcomings, this system is much more economical than other digital systems.

Digital System Parameters

A number of parameters are available to assess the performance of a digital radiographic unit, and minimum standards have been established. Each digital system has an inherent spatial resolution, usually expressed as line pairs (lp)/mm, which

directly measures the ability of a system to measure small details. The American College of Veterinary Radiology (ACVR) has recommended that a minimum standard be 2.5 lp/mm; below this, loss of spatial resolution leads to lack of diagnostic information on an image.[15] The American College of Radiology (ACR) has further guidelines for digital systems related to the pixel (length × width of a single element of an image) matrix size. The matrix is the grid of the entire length and width of the image, made of individual pixels. It also has guidelines on the number of gray shades recordable by a device, called *bit depth*. This refers to a binary code used to record digital information, each digit being a *bit*. The ACR recommends a minimum of 2 K matrix and 12-bit depth for digital radiography.[8]

Fluoroscopy

Fluoroscopy is an imaging modality that uses x-rays and differential attenuation to produce a real-time image of anatomy. The image intensifier contains the majority of the electronics used to form the fluoroscopic image, and it receives x-rays emitted by a standard x-ray tube. Within the image intensifier, an input fluorescent screen converts x-ray energy to light, which strikes a photocathode; this creates photoelectrons, which are focused and minimized by a lens as they travel to an accelerating anode. As they travel through this thin anode, they then strike an output phosphorescent layer. This light is then usually imaged by a closed-circuit television camera or converted to a digital signal by an ADC, and the information is stored. The entire image intensifier may be replaced with a system analogous to indirect DR, which presents images in real time and records them as such.[1,2]

The major advantage of fluoroscopy is the ability to watch a dynamic x-ray study in real time. This is essential for the diagnosis of physiological conditions such as dysphagia or airway collapse, and it is a major benefit to watch progress in many contrast studies (see "Image Interpretation," later). Unfortunately, penetration and resolution are limited with fluoroscopy units; very few products are marketed for equine use. One such product offered by GE Healthcare (Figure 67-5) has a capacity of producing 120 kVp/10 mA exposure for fluoroscopy, but this is maximal exposure and may only be carried on for a limited period of time because of tube heat limits.[16]

Digital Image Storage and Display

A fair amount of hardware and software is needed for the digital imaging modalities described earlier. Not only does each imaging device require its own computer but also this computer may be connected via network to a server/archiving device, as well as a diagnostic workstation computer and monitor. This computer must also have the appropriate imaging software to read the archived images. Together, these components form a picture archive and communication system (PACS). Although larger hospitals have a more complicated PACS that communicates with hospital or radiology information systems and may be connected to the Internet, these are not strictly necessary, and a local area network connecting the imaging modality to a server/archiving device represents a PACS in its simplest form.[17]

Typically, the data are stored in digital imaging and communication in medicine (DICOM) format.[18] It should be noted that using this format is voluntary; however, this allows multiple modalities to be stored and accessed in a similar fashion, as well as data compatibility between multiple vendors and hospitals. File size is directly related to pixel size, which in turn is related to the resolution of an image. The smaller the pixel size, the higher the resolution, the larger the matrix (number of pixels high × number of pixels wide) size, the larger the data file. A typical uncompressed radiographic image is 10 to 12 megabytes in size from a high 2048 × 2048 pixel matrix,[8] which can take considerable time to transfer on slower or older networking equipment. The rate at which the network transmits this information is called *bandwidth* and is an important factor to consider.[17] If the network is connected to the Internet, sufficient safety measures to protect security of medical data must be in place, typically in form of a firewall.[19]

Figure 67-5. The large animal fluoroscopy unit from General Electric, at the Michigan State University Teaching Hospital. The x-ray tube is capable of producing high kVp and mA technique and is seen to the *left* of the stocks; the image intensifier, to the *right*, captures the x-ray information and is coupled either to a closed circuit video system or digital detection system for real-time display. The work station seen to the *left* enables the user to see displayed images and capture them as they are taken.

Images may be compressed to save space, though ACR guidelines for these dictate remaining above 20 : 1 ratios to avoid loss of too much diagnostic information.[20,21] Interpretation of images in DICOM format is recommended. Other file formats, such as joint photographic experts group (JPEG), should be interpreted with caution. JPEG compression of an image often exceeds the 20 : 1 ratio, resulting in a loss of detail and image contrast.

A multitude of options exist for data storage and include a variety of discs, solid-state media, and on-site or off-site servers. Each image that is saved must also be backed up to avoid computer error data loss.[22] A full discussion of all of these storage options and computer requirements is beyond the scope of this text but may be found in the referenced articles.[17-20,22]

Typical transfer of images from a PACS occurs on digital storage media, such as CD-ROMs, DVDs, or USB flash drives. Many DICOM PACS include an abbreviated form of DICOM viewing software when the media are transferred. A printer may also be used with digital setup. Most DICOM systems use expensive laser printers that print onto film analogous to radiographic film, although copies may be provided for clients from a standard paper printer.[5] These images are not likely to be of sufficient diagnostic quality for other veterinarians.

Each digital image is also only as good as the monitor used to view it. Lower-quality monitors are employed in exam rooms or in the field, where quality control for positioning and exposure is performed, but higher-quality monitors should be used for primary image interpretation. Medical gray-scale monitors typically have increased brightness, finer pixel matrices, better graphics cards, and are calibrated to a standard gray-scale, over standard color monitors. The major disadvantage to a medical-grade monitor is the significant expense,[23] which can often be $7,000 to $10,000. Color monitors have been shown to have similar diagnostic accuracy to gray-scale monitors, but only if magnification, windowing, and leveling are utilized with software,[24] which can be time consuming and cumbersome. The poor brightness of standard color monitors, which is up to 20 times less than conventional lightboxes and 2 times less than gray-scale monitors, significantly affects diagnostic accuracy.[25-27] A proper darkened environment for interpretation also has considerable effect on length and accuracy of interpretation.[28,29] Clinicians should avoid the tendency to interpret images in the field on small monitors attached to portable digital units suitable only to confirm positioning, or in the hallway on monitors meant for client education.

PERFORMING THE EXAMINATION

The patient should be properly prepared for an examination with adequate sedation in a quiet environment. Dirt should be removed from the haircoat. This is particularly true with preparation of feet for radiographs. Special care must be taken to remove shoes, nails, and debris within the frog or on the hoof wall, and the foot should be packed with petroleum jelly or Play-Doh to remove confusing superimposed gas opacities.

Proper positioning and radiographic technique are the keys to making a radiograph easily interpretable. Standard projections of different body regions are recommended, but special positioning (described later) can be used to highlight a particular area that is normally superimposed over other structures, to visualize fractures tangentially, or to depict soft tissues normally not seen on a radiograph exposed to depict bony structures.

Standardized techniques for specific body parts produce consistent results. Each x-ray machine has specific settings that produce the best diagnostic images, and technique charts made specifically for each machine and imaging capture system are strongly recommended. Having stated this, exposure latitude (or overlap of diagnostic images from differing techniques) of CR or DR systems is much greater than for film-screen systems.

A guide light within the collimator box displays a light field on the patient that represents the area to be exposed by the primary x-ray beam. Superimposed cross hairs are often present to allow the beam to be centered. The collimator light must be recalibrated at regular intervals to make sure the light and primary beam match each other, because the collimator lead sheets and/or mirror reflecting the light may become malaligned with the x-ray beam.

Proper orientation of the x-ray tube to the imaging plate is very important. After the body part being imaged has been centered, the angle of the beam must be carefully observed and adjusted. The tube and x-ray cassette or detector should be parallel to each other, unless a special angled technique is being performed, because this will cause distortion and a lack of tangential imaging of joint spaces. Film focal distance (or distance from the x-ray tube to the film) should be standardized to create consistent images from each chosen technique, with the part of interest directly adjacent to the film. Magnification techniques, which increase patient film distance are possible, and actually decrease scatter radiation reaching the film that may degrade the image, but cause significant unsharpness, or penumbra, of the image, and will not be discussed further here. Lastly, collimation should be adjusted to exclude any areas outside of the primary area of interest.

Study-Specific Positioning

Image projections are named for the path of the x-ray beam. This is confounded by location in the limbs, for which cranial and caudal are used proximal to the carpus and tarsus, but dorsopalmar is used for carpus distally, and dorsoplantar is used for the tarsus distally. Each study should contain at least two orthogonal (90 degrees to each other) projections. This stems from the fact that radiographs are two-dimensional projections of three-dimensional anatomy. To place an object seen on one projection within the third dimension, a second view must be provided. Most commonly, this includes lateromedial and craniocaudal (dorsopalmar or dorsoplantar) projections. This may not always be possible because of anatomical restraints, such as in cervical spine, shoulder, thorax, or abdomen, or it may be performed with great difficulty in an anesthetized horse, such as a ventrodorsal pelvis.

Each projection should have a lead marker placed on the cassette. With distal limbs, this marker indicates which limb (right or left) is being imaged. The marker is usually placed laterally (on craniocaudal and oblique projections) or cranially/dorsally (on lateral projections) with respect to anatomy. Owner's name; horse's age, breed, and sex; exam date; and the hospital name should also be identified clearly.[3] Although digital procedures allow post-processing additions of positional labels, an integrated label used as the image is exposed prevents confusion in labeling a radiograph during the processing step. Labels are particularly important in the limbs distal to the carpus and tarsus, where anatomic differentiation of the lateral and medial surfaces is difficult or impossible.

Many studies require more than two orthogonal projections to provide sufficient information for correct diagnosis. For most studies, this includes oblique projections highlighting the surfaces perpendicular to the path of beam travel. If an angle is included, the degree of angulation refers to the immediately preceding directionality. For example, D45°LPMO means 45% from dorsal toward lateral oblique. See Figure 67-6 for an example of positioning for a dorsolateral, palmaromedial oblique (DLPMO) view of the carpus.

Table 67-1 includes standard radiographic projections for different areas of the equine patient. This is not intended to be an exhaustive reference for all projections that may be needed, but it represents a starting point from which to expand. Particular diseases that occur in each of these areas are covered in their respective chapters elsewhere in this text.

Contrast Studies

Contrast medium adds additional information to radiographic studies involving physiologic processes, or it highlights abnormal communications with the outside environment or between internal structures.

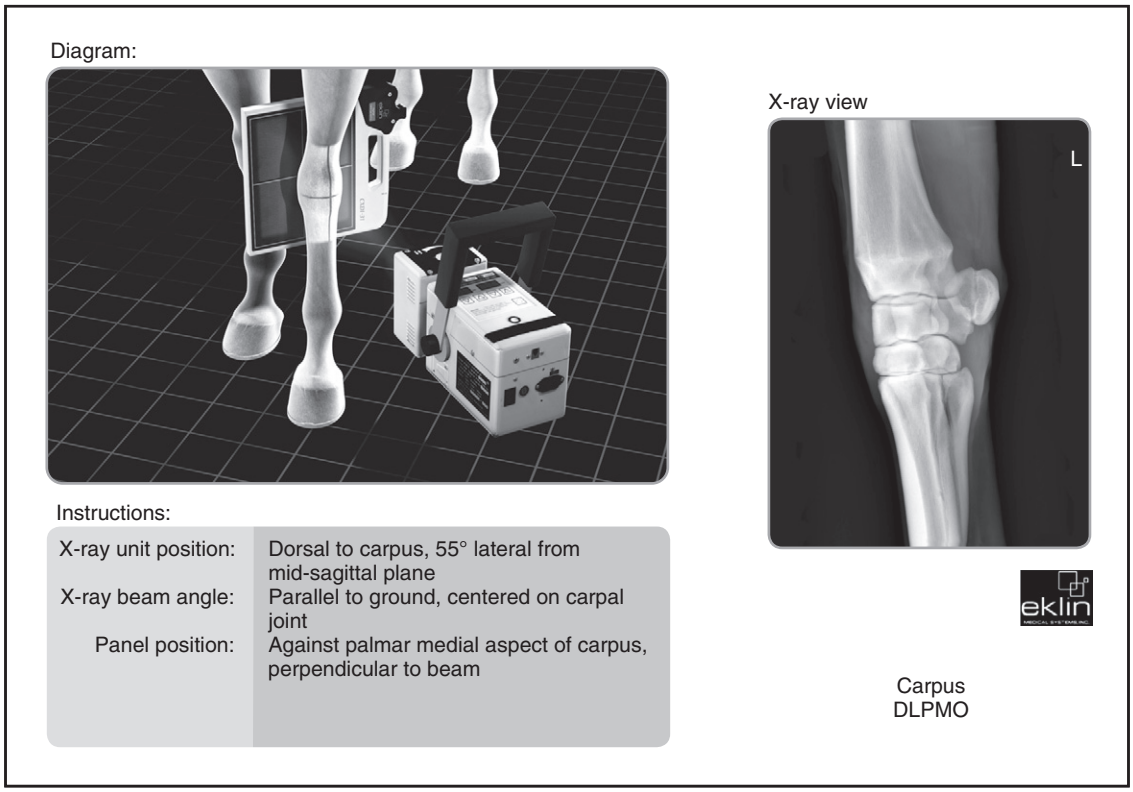

Figure 67-6. Graphic representation of tube and cassette positioning for a carpal 55 degrees dorsolateral palmaromedial oblique *(DLPMO)* projection. Not represented in this image is a holder for the cassette, preferably a metallic frame either supported by a stand or on a long handle that enables the holder to distance himself or herself from both the primary beam and the horse, which will produce scatter radiation. (Courtesy Sound-Eklin Medical Systems, Inc.)

TABLE 67-1. Standard Projections

Imaged Part	Projections	Additional	Considerations
Distal phalanx/ P3	1) Lateromedial 2) Proximal 60° dorsopalmar 3) Flexed proximal 60° D45°MPLO and D45°LPMO	1) Horizontal dorsopalmar 2) Lateromedial with markers placed along dorsal hoof wall or coronary band	Foot placed on radiolucent block, hoof cleaned and packed to remove air from frog, proximal 60-degree views may be taken in upright block (navicular block)
Coffin/DIP joint	See above	Alternate to flexed oblique: proximal 45° D45°LPMO and D45°MPLO	See above
Navicular/distal sesamoid	1) Lateromedial 2) Proximal 65° dorsopalmar 3) Flexed 45-70° (to cassette) palmaroproximal palmarodistal (skyline)		See above; skyline view must be as flexed as possible
Pastern/PIP joint	1) Lateromedial 2) Proximal 30° dorsopalmar	Proximal 30°, D45°LPMO and D45°MPLO	
Fetlock/MCP joint	See above, 3) Proximal 20° D45°LPMO and D45°MPLO	1) Flexed lateromedial, 2) Flexed D45°LPMO and D45°MPLO 3) Flexed dorsoproximal to dorsodistal (skyline)	
Cannon/MCII, III, IV	1) Lateromedial 2) Dorsopalmar 3) D45°LPMO and D45°MPLO	Special angled obliques to highlight a certain surface of MCIII	

Continued

TABLE 67-1. Standard Projections—cont'd

Imaged Part	Projections	Additional	Considerations
Knee/carpus	1) Lateromedial 2) Dorsopalmar 3) D45°LPMO and D45°MPLO	1) Flexed lateromedial 2) Flexed dorsoproximal to dorsodistal (skyline): 35° distal row of carpal bones, 55° proximal row, and 85° distal radius	Very straight dorsopalmar must be obtained for angular limb deformity
Hock/tarsus	1) Lateromedial 2) Dorsopalmar 3) D35°LPMO and P35°LDMO	1) Flexed lateromedial 2) Flexed 65° proximal palmaroproximal palmarodistal (skyline)	Tube placed plantar is often safer than DMPLO
Stifle	1) Lateromedial 2) Caudolateral 45-60°craniomedial oblique	Flexed proximal 60-70° dorsoproximal dorsodistal (patellar skyline)	Sometimes difficult to position lateromedial, good sedation and effort necessary to place cassette
Elbow	1) Mediolateral 2) Craniocaudal		Leg extended to pull away from the thorax/other limb
Shoulder	1) Mediolateral 2) Cranial 45° medial caudolateral oblique		Leg extended to pull away from the thorax/other shoulder
Spine	Lateral centered on areas in succession	Lateral oblique views only if fracture suspected	Lead markers placed on skin can assist in identifying vertebral number
Pelvis	Ventrodorsal; if standing, 10-25° caudal	Lateral in foals, Miniature Horses only	Recumbent in anesthetized horses; if standing, heavily sedated with as much abduction of pelvic limbs as possible
Skull/teeth/nasal/sinuses	1) Lateral 2) Dorsoventral 3) Dorsal (maxilla) or ventral (mandible) 30° lateral obliques	1) Intraoral dorsoventral (maxilla) or ventrodorsal (mandible) 2) Lateral 45° ventral 30° rostrolateral caudomedial oblique for temporomandibular joint[40]	Intraoral views require excellent mouth gag/sedation
Thorax	Lateral views centered: 1) Cranioventral 2) Caudoventral 3) Caudodorsal (usually × 2) 4) If needed, craniodorsal	Ventrodorsal view in foals only	
Abdomen	Lateral views centered at periphery of abdomen, also centrally if a foal		Penetration centrally only in foals, periphery of abdomen in adults
Pre-purchase exam (AAEP and Keeneland Association[41])	Metacarpophalangeal joint: • Proximal 15° dorsopalmar • D30°LPMO • D30°MPLO • Lateromedial (flexed) Metatarsophalangeal joint: • Proximal 15° dorsoplantar • Proximal 15° D30°LPMO • Proximal 15° D30°MPLO • Lateromedial Carpus: • D35°LPMO • D25°MPLO • Lateromedial (flexed) Tarsus: • D65°MPLO • D10°LPMO • Lateromedial Stifle: • Mediolateral • Caudal 20° lateral craniomedial oblique		

AAEP, American Association of Equine Practitioners; *DIP,* distal interphalangeal; *DMPLO,* dorsomedial-palmarolateral oblique; *LDMO,* lateral dorsomedial oblique; *LPMO,* lateral palmaromedial oblique; *MC,* metacarpal; *MPLO,* medial palmarolateral oblique; *PIP,* proximal interphalangeal.

Three types of contrast agents are used. The simplest of these is gas, also called *negative contrast* medium, consisting of either room air or a specific gas such as carbon dioxide, which is added to spaces to separate them from the soft tissues around them. Carbon dioxide in particular, is used for a negative contrast study when wall integrity of a structure is questionable; its high solubility in the blood stream avoids rare air emboli complications from the high, insoluble nitrogen content of room air. Negative contrast studies involve intubation of the structure in question with insufflation with the desired gas, but they are rarely used in equine patients.

The two other types of contrast are considered types of *positive contrast,* meaning they appear as areas of increased opacity on radiographs. The first is barium sulfate suspension, used in gastrointestinal studies. This is usually prepared as a weight-per-volume (w/v) measurement of a certain gram weight of barium sulfate added to a volume of water. For example, a 60% w/v solution of barium sulfate is 60 g of barium sulfate with enough water added to make a total volume of 100 mL. Commercially prepared suspensions come in this fluid viscosity of 60% to 70% w/v, a more concentrated form as barium paste (typically 70% to 100% w/v), creams with even higher concentration, or tablets. Typically, the 60% suspension is appropriate for esophagram studies. For foals undergoing gastrointestinal contrast study, a full gastric volume of this concentration creates a large enough opacity to be overwhelming, and the suspension should be diluted. Concentration may also be represented as a weight-per-weight (w/w) suspension in which a weight of barium is added to a weight of water to obtain a predetermined total weight (e.g., a 20% w/w solution of barium sulfate would be 20 g of barium added to 80 g of water). See Table 67-2 for typical volumes and concentrations for contrast studies. Each of these studies should have survey films of the area of interest taken before administration of contrast medium for comparison.

Barium sulfate should never be used in areas outside of the gastrointestinal tract. Although it is inert, it causes large fibrotic and granulomatous reactions when it enters body cavities or is injected into fistulas. Its inertness, however, is an advantage when barium is accidentally aspirated into the airways.[30] Barium bronchograms were obtained for diagnostic studies, although they are performed less frequently now. With appropriate treatment for pneumonia, the barium is taken up by macrophages and eventually lymph nodes, which will remain radiopaque.[30,31]

The second type of positive radiographic contrast is *iodinated contrast*, used for the majority of intravenous, fistulogram, arthrogram, or myelogram studies. The contrast medium is provided either in ionic or non-ionic form. The ionic form of contrast dissociates into two osmotically active particles, one iodinated benzene ring anion and the second a cation, either sodium or meglumine, whereas the non-ionic form does not. Most forms of both types are hypertonic to blood, but the ionic form is much more so (approximately seven to eight times the osmolarity of blood). Because of the hypertonicity and dissociation to two charged particles of the ionic form, severe side effects secondary to their use may be seen. Most especially, seizures and death may occur when even a small amount is used in the central nervous system (as with myelograms); seizures are still reported with non-ionic forms in myelograms but are usually focal or self-limiting. Ionic iodinated contrast medium may result in severe pulmonary edema and possibly death when aspirated into the alveolar space, and cardiovascular shock when used intravenously in severely dehydrated patients. Intravenous side effects are less pertinent for equine patients than small animals but should be noted if used in foals. The ionic forms, however, are much cheaper than the non-ionic forms, and are safe for use in areas in which fluid influx or efflux is not a life-threatening issue, such as arthrograms or fistulograms, or in properly hydrated foals for intravenous studies. Other notable side effects that occur in other species but have not been reported in horses are acute renal toxicity or hypersensitivity reactions to intravenous contrast.[30]

Radiation Safety

The easiest way to decrease human handler radiation dose is to distance the handler from the animal and radiograph being taken. This is related to the inverse squares law, which states that dose received by an individual is inversely proportional to the square of the distance from the source. In other words, if a person doubles their distance from the tube, the dose they receive is four times less than the original dose; triple the distance, nine times less dose.[32]

Human dose may also be reduced through passive patient restraint with sedation, stocks and restraint, and devices for holding cassettes or image panels while humans are in another room. If a human is in the same room as the animal, proper dosimeter monitoring must be worn.

Lead aprons, thyroid shields, and gloves decrease scatter doses, but do *not* protect handlers from radiation from the primary beam or block all of the scattered radiation. Gloves containing a 0.5-mm lead equivalent only block 88% of 75 kVp technique and only 75% of 100 kVp technique from the primary beam.[33] Aprons containing a 0.25-mm lead equivalent attenuate only 90% of scatter radiation for tube voltages less than 100 kVp. The shielding efficiency increases to 99% with 0.5-mm thickness.[1] Film badges monitor whole-body, thyroid, or gonadal doses of radiation and should be worn outside of protective lead gear. Distal limbs are among the least radiosensitive tissues, and are monitored separately with ring badges, but dose over time can add up quickly with repeated exposures, and effects, including carcinogenesis, often have a very delayed time to onset.[32]

Collimation is another easy way to decrease exposure. Not only does proper collimation decrease the dose to the patient, it reduces scatter radiation produced by tissues outside the area of interest and dose to the handlers. This is particularly relevant with high-radiation techniques used for very thick body parts such as the thorax, abdomen, or pelvis, but it can also occur with stifle or cervical spine radiographs. Scatter significantly degrades the image and adds to human doses. Cavalier treatment of radiation safety is a potentially dangerous practice to undertake; we urge each individual involved in taking radiographs to take avid interest in decreasing their own and their patients' exposure.

IMAGE INTERPRETATION

Initial evaluation of a radiographic image should focus on technical issues. It should be determined if the image is centered on the area of interest, whether it is exposed properly, and whether projections taken are sufficient for diagnosis or if more-specific projections are needed. Image interpretation should proceed in

TABLE 67-2. Contrast Study Parameters

Study Performed	Indications	Contrast and Dose	Study Specifics
Esophagraphy[30]	Dysphagia; regurgitation; suspected esophageal foreign body, stricture, or diverticulum; recurrent aspiration pneumonia	Barium sulfate suspension 60% w/v, orally, volume dependent upon patient swallowing; *not to be used if esophageal perforation is suspected*, use ionic iodinated contrast	Liquid only first phase, mixed with grain second phase, watch with fluoroscopy or take lateral cervical and thoracic films 5-15 sec after swallowing, respectively
Upper gastrointestinal (foal only)[30]	Lack of defecation, suspected intestinal obstruction, suspected gastric ulceration	5 mL/kg 30% w/v barium sulfate suspension through nasogastric tube; *not to be used if gastrointestinal perforation is suspected*, use iodinated contrast (ionic form not recommended if patient is dehydrated)	4-12 h fast prep, (4 h if <2 weeks old), standing right lateral, recumbent bilateral and ventrodorsal films, survey before contrast, then films at time 0, 30 min, 2 h, then 2-h intervals until contrast reaches small colon
Barium colonogram (foal only)[30]	Suspected rectal stricture, sectional hypoplasia, or mass	20% w/v barium sulfate suspension, volume is patient specific but should begin at approximately 5-10 mL/kg; *not to be used if rectal perforation is suspected*	Clean rectum of as much feces as possible, administer contrast through rubber tube with catheter tip or largest Foley available (up to 26 F available)
Dacryocystorhinography[42]	Chronic epiphora, orbital trauma, chronic nonresponsive conjunctivitis, proptosis of third eyelid	Nondiluted ionic or non-ionic iodinated contrast, approximately 5 mL per injection, or until contrast is seen at the medial canthus of eye	Catheterize nasal puncta, take film as injecting the contrast (or duct may not be uniformly filled), *nasolacrimal duct better resolved with CT, but large abnormalities can be seen on radiographs*
Sialography[30]	Swelling or tubular structure suspected associated with salivary system, abnormal ptyalism or discharge localized to a single salivary duct	Nondiluted ionic or non-ionic iodinated contrast, 20 mL/kg, or until sufficient back pressure is felt or contrast leaks around catheter	Catheterize the salivary duct of interest, preferably watched as injected with fluoroscopy, but orthogonal views taken after injection are an alternative, films repeated if complete filling is not seen, *iatrogenic rupture of duct and/or salivary gland are a potential complication*
Fistulography[43]	Draining tract, suspected sequestrum	Nondiluted ionic or non-ionic iodinated contrast, volume is study specific, injected until contrast begins to leak around catheter or sufficient back pressure	Catheterize fistula with Foley if cavity underneath is large enough (to prevent external leakage of contrast), red rubber tube, or intravenous catheter
Arthrography/ bursography/ tenography[43-45]	Lameness isolated to a joint with suspected cartilage or soft tissue abnormalities, foot lameness (navicular bursography), tendon effusion	Nondiluted non-ionic iodinated contrast for bursa or tendon sheath, diluted 1:1 with saline for arthrogram, volume is joint or bursa specific; distend the structure in question until slightly distended and mild back pressure is felt	Limb must be clipped and aseptically prepped, ensure placement in the proper cavity by aspirating joint or bursal fluid or taking a scout radiograph after small amount of contrast injection, better filling of joints obtained if joint is put through range of motion after injection
Selective angiography[46-49]	Laminitis, lameness isolated to the foot, trauma with suspected vascular disruption to foot	Nondiluted (for radiography) non-ionic iodinated contrast injected into specified artery or vein (see right), continuous infusion with pressure injector or single 20 mL injection, 1:1 diluted for CT	Performed under general anesthesia or heavy sedation with local nerve block, selective common digital artery or medial or lateral palmar digital vein infusion for laminitis, and medial palmar artery infusion reported for CT
Excretory urography (foal only)[50]	Suspected ectopic ureters, suspected uroretroperitoneum or peritoneum, renal azotemia	880 mg I/kg of ionic or non-ionic iodinated contrast given intravenously as a rapid bolus; *this should not be done in dehydrated patients, and may cause contrast-induced nephrotoxicity*	For ectopic ureters, prep bladder with pneumocystogram through urethral catheter, take films at time of injection, 5 min, 15 min, and 30 min post injection, more films may be required if poor renal function

a consistent manner, either in a directional fashion from left to right, top to bottom, skeletal then soft tissue structures, and so on, so that all areas of a radiograph are observed and structures or changes are not overlooked. It is very easy to prematurely terminate radiographic interpretation when an obvious lesion is found, missing more obscure lesions that may change or modify a diagnosis or clinical approach to a patient.

The scales of gray from black to white on a radiograph are based upon differential attenuation of x-rays by different tissues and matter. The five basic radiographic opacities are, in ascending order from black to white: gas, fat, soft tissue/fluid, bone/mineral, and metal. This is also the order of x-ray attenuation from least to greatest. Areas on a radiograph are black because they have been exposed to radiation or light; white because they have not. It is important to note that fluid and soft tissue have the same radiographic opacity and cannot be differentiated on radiographs. If no intervening different-opacity object lies between objects of the same opacity, their margins are lost; this is termed silhouetting or border effacement. Summation causes opacities to occasionally be confusing, such as when gas summates with soft tissue, making it appear similar to fat opacity. Orthogonal or additional oblique projections may help sort out this phenomenon. Inherent large changes in the contrast of adjacent tissue, such as mineral (bone), metallic implants, or gas add the most meaningful and interpretable information to radiographs.

Recognition of normal structures is a highly important step; this may require broad experience in looking at multiple radiographs or referencing a textbook or scientific article. When normal structures and opacities are identified, abnormal ones may be recognized. It is important to note that although a consistent pattern should be followed for each type of study, and recognizing abnormalities is based on pattern recognition by the brain, taking away a pattern may make lesions easier to see. The most obvious but not only example of this occurs with rib lesions (Figure 67-7). The eye is accustomed to looking *through* the ribs on most thoracic radiographs; by turning the image upside down, pattern recognition is changed, and the ribs tend to stand out better. Similar situations may be reproduced with windowing and leveling (or hot-lighting) an image to see soft tissues better on orthopedic films.

Certain unique principles apply to equine radiographic interpretation. The bulk of a patient may not allow penetration or positioning for a view, dictating the use of another modality. The weight-bearing status of most appendicular films allows assessment of joint spaces, most commonly narrowing that indicates loss of articular cartilage. The joint space of non–weight-bearing radiographs should be interpreted with caution because the joint space may be made to appear artificially wide or narrow. Gravity is also responsible for distinct fluid-lines in air-filled cavities. The sheer mass of mineral opacity in most bones causes nondisplaced fractures to be masked unless they are very precisely tangential to the fracture line; this may cause need for a multitude of only slightly differently angled projections through the bone, or a "skyline" view. The precise opposite for nondisplaced condylar fractures exists: a view perpendicular to the fracture line is needed. External debris is often present, mimicking lesions. Therefore extra care must be taken to remove this before the study or to identify it as artifactual.

However, there are certain principles of radiographic changes that apply across species differences. Here are a few important facts:

- Sharp bone margins equals acute disease process; rounded equals subacute to chronic and likely inactive; irregular/indistinct equals active
- Bony lysis equals aggressive processes, typically osteolysis/osteomyelitis, but also neoplasia or necrosis; well-defined rounded lucent regions in bone may represent cysts
- Nondisplaced stress fractures may become more lucent before healing because of bone remodeling. This means some fractures may not be evident on films until 5 to 10 days after the event.[34]
- Enthesiophytes or osteophytes represent at least 3 weeks of disease process.[34]
- A lesion should be seen on more than one projection to increase confidence that it is real and not artifactual.

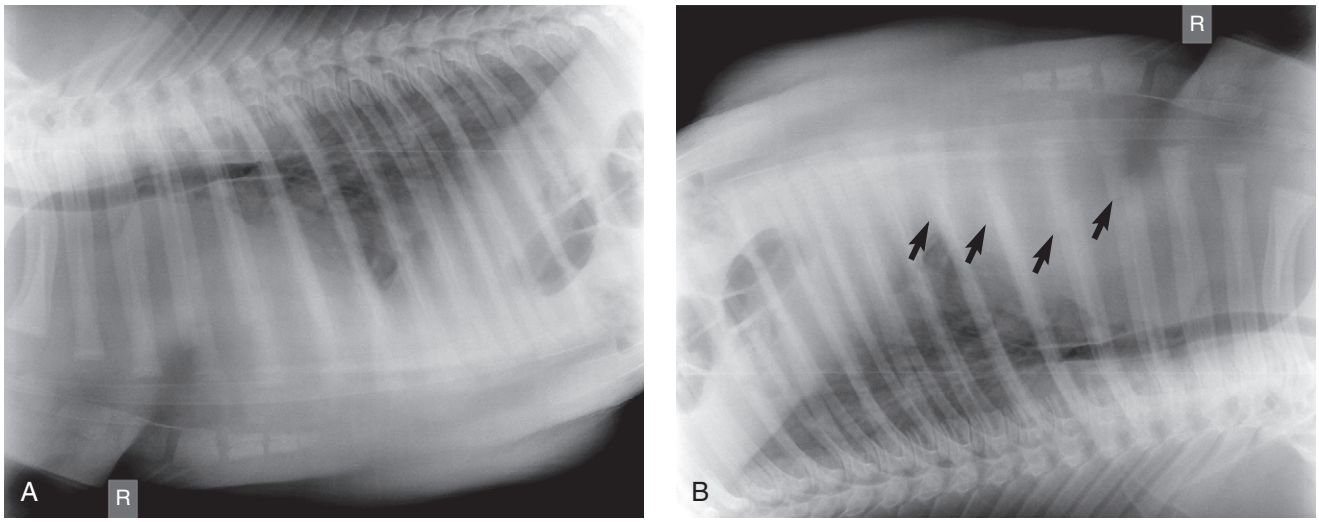

Figure 67-7. A, A traditionally positioned foal thorax view; **B,** The image is flipped upside down, which may depict the ribs more prominent to the viewer after normal positioning pattern is removed. The *black arrows* denote the minimally to nondisplaced rib fractures.

- Radiographs should be interpreted blinded to history or what you *should* find. This will avoid finding "lesions" that fit diagnoses already reached before the examination.

ARTIFACTS

Recognition of radiographic artifacts is important for proper image interpretation, and knowledge of their cause is necessary to facilitate correction and prevent recurrence. Artifacts occur with conventional screen-film radiography and digital radiographic systems. Although some artifacts are common to both modalities, each has unique artifacts as well. A complete review of all radiographic artifacts is beyond the scope of this text, but the most important and common artifacts for screen-film systems and digital radiography systems are discussed later.

Radiographic Screen-Film Systems

Radiographic screen-film systems have a narrow range of appropriate x-ray exposures, with inappropriate technique settings commonly resulting in over- or underexposure. Overexposure occurs when kVp, mAs, or both are too high and results in an image that is generally too dark. This artifact may be corrected by decreasing the mAs by one half or kVp by 10% to 15%. If the resultant image is still overexposed after this correction, the same magnitude correction is applied and imaging is repeated. With underexposure, the resultant image is too light and exposure techniques must be increased (typically by doubling mAs or increasing kVp by 10% to 15%).

The use of grids, though useful to reduce scatter radiation from reaching the film or plate, can also introduce artifact to an image. Particularly the use of focused grids, misplacement from the center of the beam, angling, or using the grid upside-down causes significant artifacts, seen as white lines that travel in a parallel fashion (or grid fashion if the grid is a cross grid) across the image (Figure 67-8).[1,2]

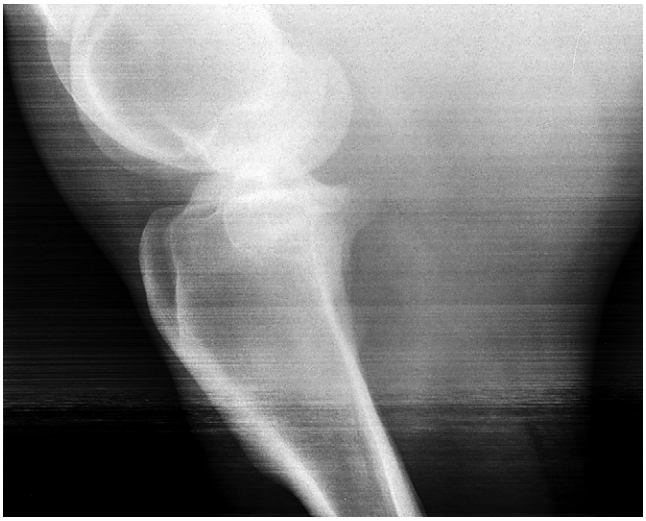

Figure 67-8. *White lines* travel across the image from a decentered grid. If lines occur in a regular, very closely apposed fashion, grids are usually the culprit. Note the *irregular white line* along the upper right side of the image, representing hair in the cassette, as well.

Motion artifact is a common problem that occurs with both film-screen and digital radiography of the equine patient. This artifact is recognized by blurry, indistinct anatomic borders on the resultant radiographic image. The patient's motion during the radiographic exposure is the most common culprit; however, motion of the x-ray tube, particularly if a portable unit is held by hand, can result in identical artifact appearance. Risk of this artifact is greater when examining thicker body parts because mAs is higher and therefore exposure time is longer. Use of appropriate sedation protocols (to minimize patient motion), use of a stand to stabilize the x-ray tube (to minimize tube motion), and using the lowest mAs possible (to reduce exposure time) can all help resolve this artifact.

Radiographic film and intensifying screens are easily damaged with inappropriate handling. Scratches on film remove emulsion and usually result in well-defined white lines. Scratches on an intensifying screen cause insufficient light to be generated, resulting in well-defined white lines on the film where it was not completely exposed.[35] Scratched screen artifact can be differentiated from scratched film by touching the radiographic film. With a film scratch, a divot or physical defect can be felt in the emulsion, whereas with a scratched screen, unexposed emulsion remains on the film (no defect is felt).

Dirt or debris within the radiographic cassette between the intensifying screen and film blocks light emitted from the radiographic screens from exposing the film. This results in abrupt white lines on the radiographic image, which corresponds to the shape of the debris. This can appear similar to scratches on film or a screen, but it may vary in position on subsequent images.[35] Radiographic cassettes and screens should be regularly cleaned using specialized screen-cleaning solution. Harsh cleaning agents may result in iatrogenic removal of screen phosphors.

Inappropriate processing is a common cause of many artifacts, which may occur during the development, fixing, or washing steps. Manufacturer recommendations regarding frequency of chemical replenishment should be strictly followed. Weak chemicals or inappropriate chemical temperatures may result in incomplete development (black areas of film are gray) or fixation (film the cloudiness and color of the emulsion) and cause many problems with image quality. If the wash step is incomplete, fixer material remains on the radiographic film, causing a brown image and characteristic sulfur smell.

Digital Radiography Systems

Digital radiography eliminates problems associated with handling and film processing, but its advent introduces many unique artifacts.[36] Because of their wide dynamic range, digital radiography units can produce high-quality diagnostic images over a larger variation in x-ray exposure techniques than conventional screen-film radiography, but underexposure and overexposure can still occur. With very low exposure settings, the digital image is grainy or pixelated, with low signal-to-background noise ratio. This artifact is properly called *quantum mottle* and can render subtle abnormalities inapparent.[37] Correction requires increasing radiographic exposure settings, typically by doubling mAs or increasing kVp by 10% to 15%.

Digital radiographic systems are more tolerant of inappropriately high exposure settings than screen-film technology, but overexposure artifacts can occur. At very high techniques, areas of the image become completely black and devoid of visible anatomy, a phenomenon called *clipping* (Figure 67-9).[36] This is usually first appreciated at the thinnest or least dense portion

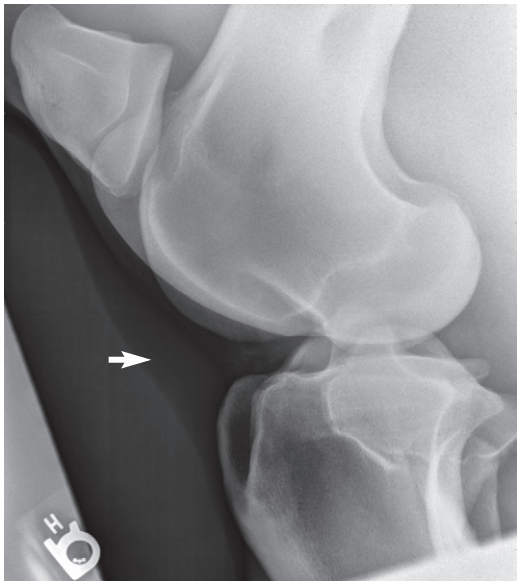

Figure 67-9. Clipping artifact with a digital radiography system. The soft tissues cranial to the stifle joint are rendered black *(indicated by arrows)* from overexposure in this region. Note that the adjacent anatomy is normally displayed and the junction with the clipped area is abrupt.

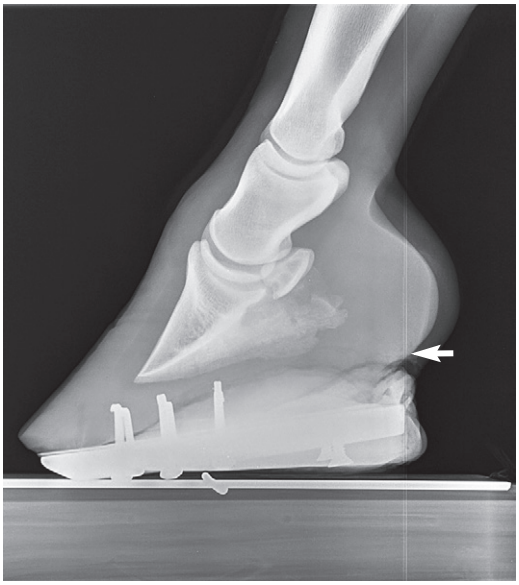

Figure 67-10. Dirty light guide artifact with a computed radiography (CR) system. Two parallel white lines *(arrow)* seen along the right side of the image are caused by debris on the light guide within the CR image processor.

of the patient, and it encompasses larger areas of the image with higher exposure settings.

Digital image processing algorithms inherent to digital radiography units can introduce imaging errors. A process called *unsharp masking* is frequently employed to maintain well-defined anatomical margins on digital images. The imaging computer may perform this process routinely at the time of initial image display without the clinician's knowledge. This processing step usually enhances the digital image, but at very high contrast margins (such as along the margin of a metallic plate), it may artifactually result in a radiolucent halo around the high-density object, simulating implant loosening or infection.[38] This artifact (called *Uberschwinger, rebound,* or *halation* artifact) may be subtle and visible only on close inspection. The development of newer processing software algorithms has reduced or eliminated this artifact, but it may be especially prominent with older imaging systems. Disabling unsharp masking or using an improved processing algorithm (if one is provided by the particular manufacturer) eliminates this artifact.

Dirt or debris may become adherent to the PSP plate in a CR system. During image readout, light emitted by the plate is not collected by the image processor, resulting in a white defect on the image corresponding to the location of the debris.[39] The appearance of this artifact is similar to that generated when debris is present within the cassette with conventional screen-film radiography. Cleaning the PSP plate (using special CR plate-cleaning solution) and interior of the image processor corrects this artifact. Debris may also become lodged on the light guide, the component that collects light emitted by the PSP plate when it is scanned by the laser within the processor (Figure 67-10). This results in a straight, linear white streak, which usually extends from one end of the image to the other in the direction of light guide movement. This is corrected by cleaning the light guide within the image processor.

After PSP plate readout within the digitizer machine, a faint, latent image remains, which the digitizer machine automatically erases by exposure to very bright light. If the original image is severely overexposed or there is an error in the erasure step, the latent image may not be completely eliminated.[39] When that PSP plate is next exposed during radiography of a patient, the remaining latent image may be superimposed on the current patient's anatomy. This results in a double image, which may be very similar to that produced during a double exposure. This artifact can be very subtle and may mimic pathology. If noted, this artifact can be corrected by processing the PSP plate again within the digitizer machine.

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CHAPTER
68

Ultrasonography

Anthony P. Pease and Joana Chaby L.S. Coelho

Diagnostic ultrasonography has revolutionized the quality of medicine for the equine patient. In the last several years, ultrasound machines have become more portable as well as more affordable, and therefore they are more widely available to equine practitioners. The improvements in battery packs and laptop designs have made ultrasonography a convenient modality to use stall-side as well as in the field. Ultrasonography is the only modality that provides real-time evaluation of both soft tissue and, to a limited extent, bone of horses. The purpose of this chapter is to describe how ultrasound produces diagnostic images and explain the indications for equine ultrasonography.

PHYSICS OF ULTRASOUND

Diagnostic ultrasound uses high-frequency sound waves that are outside of the audible range of human hearing to penetrate tissue and reflect back to the transducer, which generates an image. This is accomplished with the use of piezoelectric crystals. These crystals have a unique property that allows them to generate sound when stimulated by an electrical current. Conversely, they produce an electrical current when interacted by sound waves. This electrical current is then converted to a digital image (Figure 68-1).

Various types of ultrasound transducers are available including linear, curvilinear, and phased-array transducers. A common

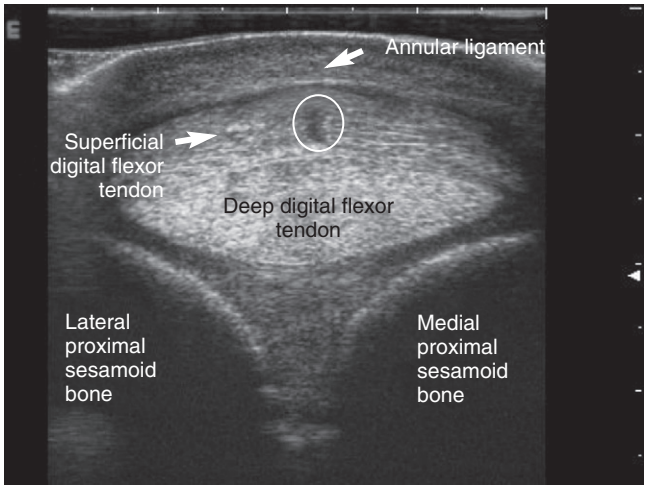

Figure 68-1. Ultrasonography image of the palmar aspect of the metacarpophalangeal joint. Using sound waves, ultrasound provides an image of the ligaments as hyperechoic *(bright)* regions because they reflect sound. Alternatively, bone absorbs sound and therefore only the margin of the bone is seen as a hyperechoic structure with an acoustic shadow seen deep to the bone because of the absorbed sound. A hypoechoic *(dark)* region can be seen within the superficial digital flexor tendon *(circle)*. This loss of the linear fiber pattern represents a tear, where less sound is reflected because of the presence of fluid.

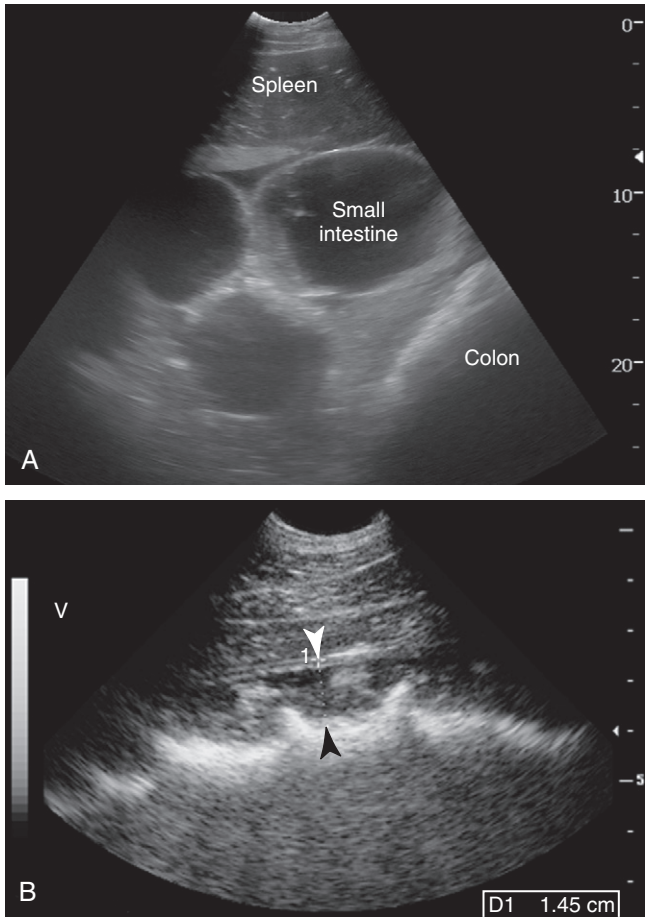

Figure 68-2. A, Image of an obstructed small intestine of a horse using a 4-MHz probe imaging at 20-cm depth. Note the small intestine can be seen in its entirity, but a detailed examination of the wall or evaluation of the spleen seen in the near field is limited. **B,** A colon with a 180-degree volvulus imaged with an 8-MHz probe at 3-cm depth. With the increased resolution and decreased depth, individual wall layers are not seen due to edema rather than poor resolution. The wall has an increased thickness (indicated between the *arrowheads*). However, with the increased resolution, the amount of the abdomen available for review is decreased.

term used to describe a transducer is *sector;* however, this only refers to a transducer that creates a trapezoid shaped image, which all transducers (even the linear transducer with a process called virtual convexing) can do. A *linear transducer* is flat and narrow with approximately 4 cm of scanning surface. The piezoelectric crystals are organized in a linear fashion and emit sound in succession. This characteristic provides the highest resolution, but it makes linear transducers not as useful to image rapidly moving structures (like the heart). Linear transducers are indicated for imaging tendons and ligaments. Specialized linear transducers have the cord attached to the end of the length of the probe to allow transrectal imaging. These transducers can also be used for tendon and ligament imaging as well as imaging the kidneys, left adrenal gland, small intestine, urinary bladder, and uterus. A *curvilinear transducer* is similar to the linear transducer except it is slightly convex, providing a larger field of view. This increased field of view is associated with a slight loss of resolution. A curvilinear transducer is indicated for general abdominal imaging, primarily focusing on the gastrointestinal tract. Because the gastrointestinal tract is superficial, a high-frequency microconvex transducer is used and deeper structures like the liver, spleen, and kidneys are evaluated with the lower-frequency, macroconvex transducers. A *phased-array transducer* has a relatively small rectangular, flat surface. In these transducers, the piezoelectric crystals are arranged in a rectangular shape and send out all the sound waves simultaneously. This allows imaging of rapidly moving structures (like the heart or vessels when using color flow Doppler), and it produces higher-contrast (black and white) images but with less resolution compared to the linear and curvilinear transducers. The flat surface of these probes makes it ideal for imaging between the ribs.

Ultrasound transducers are available in multiple frequencies ranging from 1 MHz to 18 MHz. Image resolution and depth are related to the frequency used. Lower frequencies allow greater sound penetration (deeper structures can be imaged) but offer poorer resolution, whereas higher-frequency sound waves offer greater resolution with decreased image depth. The highest frequency that allows adequate image depth should be chosen to guarantee the best image quality possible. These multifrequency transducers produce the best image at the highest frequency listed and produce a poorer image at the lower frequencies (Figure 68-2).

The high-frequency sound waves used during a diagnostic ultrasound examination are able to penetrate soft tissue and fluid readily but cannot penetrate air and bone. The acoustic impedance of tissues causes these differences in sound penetration. *Acoustic impedance* is the product of a specific tissue's density and the velocity of the sound within that tissue. Sound propagates as a wave based on vibration of the particles in the medium that it is traveling through. This vibration causes compression and rarefaction of the adjacent molecule or cell to propagate the wave over a distance. Because air has molecules that are relatively far apart, sound travels relatively slowly

(approximately 330 m/sec) in air. In soft tissue, the molecules are closer together, which allows sound to travel faster (1540 m/sec). It is this difference in the speed of sound through various tissues that provides a reflective quality to produce an image with ultrasound.

The ultrasound wave travels from the transducer into the tissue and it is reflected from the interfaces between different tissues based on the acoustic impedance of the adjacent tissues. Part of the reflected sound is transmitted through the tissues and received back by the transducers and a portion of the sound is refracted and lost. The strength of the reflective sound is displayed using a gray scale, where black indicates that no sound is reflected and white indicates that a large amount of the sound is reflected. The amount of reflected ultrasound received by the probe depends on the *angle of incidence* (the angle that the sound wave interacts with a structure) of the ultrasound beam transmitted by the probe. Ideally, the transmitted ultrasound beam is oriented perpendicularly to the imaged structure. When the ultrasound beam is not perpendicular to a structure or portion of a structure (such as a tendon), a portion of the reflected ultrasound will be off-angle and will not return to the transducer. As a consequence, a darker (hypoechoic) area will be seen in the image. Thus, during scanning it is essential to maintain the ultrasound beam as perpendicular as possible to the structure being imaged. The appearance of darker (hypoechoic) areas in the image that result from the ultrasound beam not being perpendicular to the structure in question is designated *anisotropism* or *off-normal incidence*. This is especially common when imaging tendons and ligaments. Making sure that the ultrasound beam is perpendicular to these structures can be accomplished by repositioning the probe, moving it closer to the tendon or ligament edges, and by slowly moving the probe in different angles while scanning.

The ultrasound beam that is transmitted by an individual crystal or transducer element has a specific width called *lateral resolution*. *Lateral spatial resolution* is the capacity of the ultrasound to distinguish two points adjacent to each other as separate structures in a direction perpendicular to the beam (along the length of the ultrasound beam). In addition to the beam's inherent width, acoustic principles can be used to narrow the beam further and therefore optimize lateral resolution. The focal point (or focus) controls this optimization of the beam and can be positioned by the operator at any depth level of the image. The ultrasound beam is the narrowest at or immediately above the focal point, which results in optimized lateral resolution at this level. Deeper to the focal point, the ultrasound beam exponentially diverges and therefore the lateral resolution becomes progressively inferior. *Axial spatial resolution* refers to the capability to differentiate two points adjacent to each other as separate structures parallel to the beam (along the width of the ultrasound beam). Axial resolution is determined by the sound wavelength and, hence, by the frequency chosen. Higher sound frequency corresponds to shorter wavelengths and thus better axial resolution. In general, axial resolution is superior to lateral resolution.

After the ultrasound transducer has sent and received a signal, the information needs to be presented on the screen. For any given image, sound can travel through fat, soft tissue, muscle, fluid, blood, and skin. Given that different tissues have different acoustic impedances (the speed that sound travels through the different tissues), the ultrasound image will reflect each tissue type in a different shade of gray on the screen. The depth of a specific structure is then calculated based on the amount of time it takes for the transmitted ultrasound beam to be reflected and received back by the probe. The ultrasound travel time is calculated assuming that sound travels at the same velocity through all tissues. Because soft tissue (versus air or bone) is most abundant in the body, the speed of sound in soft tissues (1540 m/sec) is used as the standard velocity of sound to calculate the depth of ultrasound images.

Knowing how an ultrasound image is produced is the first step to understanding the terminology used in the course of ultrasound: hyperechoic, hypoechoic, isoechoic, and anechoic. All of these terms, except for anechoic (no echoes), are a comparison. Hyperechoic means that the structure is brighter than another structure (preferably on the same image). Hypoechoic means darker than another structure and isoechoic means the structure has the same echogenicity of another structure. This relative echogenicity is important, especially in the abdomen or in tendon lesions to provide a basis for re-evaluation.

Advanced imaging software for ultrasound is also available, such as harmonic imaging, compound imaging, and extended field of view. *Harmonic imaging* can be used to produce a clearer image by reducing artifacts. This requires a specialized probe, but it generates an image from a fundamental frequency that is sent from the transducer. When this frequency interacts with an organ, it creates a resonance frequency that is twice the fundamental frequency. The benefit of this is that the resonance frequency that is used to generate the image is relatively high, giving better resolution, and only travels through the body once, so it is less prone to artifacts. Harmonics can reduce superficial artifacts (e.g., superficial reverberation) as well as side-lobe artifacts, which are produced by weaker sounds that leave the transducer in a direction different from the main beam. *Compound imaging* is the capability to use ultrasound waves sent from multiple angles to evaluate the same region. This is intended to reduce artifacts and give a much more detailed view of a structure, although it is susceptible to blurring with motion. *Extended field of view* allows a complete tendon to be imaged or provides the capability to image a structure or lesion that is larger than the field of view obtained by a given transducer. Three-dimensional imaging and contrast medium ultrasound imaging is also available, but at this time it has very limited use in the equine patient.

ULTRASOUND APPLICATIONS

Because of the noninvasive nature of ultrasound, the transducer can be used on the skin surface, transrectally, intraoperatively, and even endoscopically to evaluate any region of the horse it contacts. Most imaging has been centered on the musculoskeletal system, but abdominal, thoracic, cardiac, and urogenital evaluation are also possible.

To prepare for most musculoskeletal examinations, the hair is clipped with a number 40 clipper blade. Alcohol and/or acoustic coupling gel are applied to establish contact between the skin surface and the probe. Generally, a high-frequency linear probe is used. The use of a stand-off pad is helpful for the evaluation of superficial structures, such as the palmar or plantar annular ligament and the superficial digital flexor tendon in the pastern region (Figure 68-3). A stand-off pad is a gelatin-based pad that is placed between the probe and the skin surface. By increasing the distance between the probe and

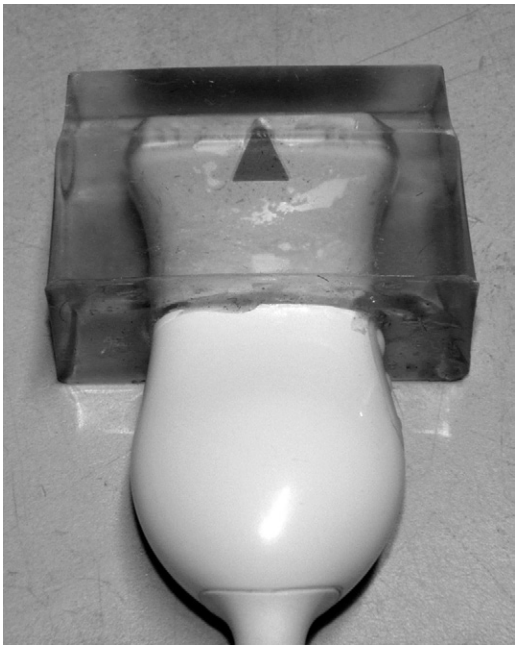

Figure 68-3. A 12-MHz linear ultrasound probe with a stand-off. Note that ultrasound gel is placed within the stand-off to eliminate any air that would be in between the probe and the stand-off. This gelatin pad allows a better contact with a curved surface, such as the equine limb.

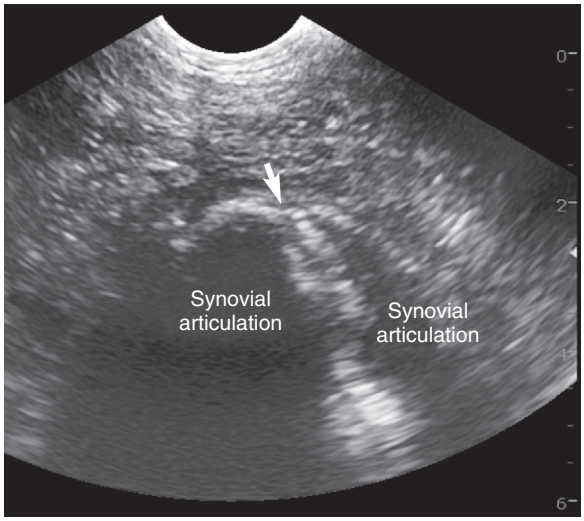

Figure 68-4. Image of the C5-C6 synovial articulation using an 8-MHz curvilinear transducer. The synovial articulations, also called *facets*, are labeled and the *arrow* indicates the joint space.

the skin surface, the near-field artifacts that are seen in the higher portion (near field) of the image are now at the level of the stand-off pad instead of being over the structure of interest.

Cervical Region

Equine cervical degenerative joint disease is a common source of pain and lack of flexibility of the neck in horses. Radiographs can be obtained to assess the proliferation of the synovial articular processes; however, oblique projections are generally needed to determine the side of the proliferation. Additionally, superimposition of the synovial articulations can make radiographic interpretation difficult.[1] High-output x-ray machines with a bucky system and grid are ideal for imaging the neck, although current digital radiographic systems can obtain high-quality images of the cervical regions with portable equipment. Ultrasonography can be used to evaluate the degree of osseous proliferations of the cervical articular facets as well as guide therapeutic injections of corticosteroids and/or hyaluronan into the cervical joint spaces.[2]

The procedure is performed with either a linear or microconvex curvilinear transducer. The cervical articulations from C2-C6 are relatively superficial (approximately 2 cm under the skin) with C6-C7 being slightly deeper (approximately 3 to 5 cm depending on the shoulder musculature). A high-frequency transducer that is greater than 7 MHz provides good resolution with adequate penetration. The transducer can be oriented either craniocaudal[2] or dorsoventral.[3] In my opinion, a dorsoventral approach provides easier landmarks of the cervical articular joint space (Figure 68-4).

In addition to the cervical vertebral bodies, work has been done using ultrasound to evaluate the equine larynx for laryngeal nerve paralysis, dorsal displacement of the soft palate, and inflammatory disease, such as arytenoid condritis.[4,5] Evaluation of the larynx does not require clipping, but a large volume of alcohol is needed to provide adequate contact between the transducer and the skin. During the examination, the hyoid apparatus, vocal folds, arytenoid cartilages, and the cricolateralis, cricopharyngeus and thyropharyngeus muscles can be seen, as well as occasionally the vocalis muscle and saccules of the lateral ventricles.

Shoulder Region

When evaluating the shoulder, one can examine the biceps brachii tendon, bicipital bursa, humeral tubercles, infraspinatus muscle, supraspinatus muscle, and glenohumeral joints. Generally, this examination is performed with the patient in a weight-bearing stance and with the shoulder region clipped as described previously. Ultrasound can be used to guide a needle into the bursal or shoulder joint space for diagnostic or therapeutic purposes.[6]

Evaluating the shoulder joint is generally performed using a microconvex curvilinear transducer. A linear transducer can be used; however, the large footprint of the transducer makes adequate contact with the skin difficult. A high-frequency tranducer should be used (greater than 7 MHz) because the depth that is being imaged is generally between 4 and 6 cm. Application of alcohol followed by acoustic coupling gel provides the best-quality image. As with all body parts, all images should be acquired and evaluated in both the transverse and longitudinal plane. Because of the curvilinear contour of the muscles and tendons in this region, care should be taken to maintain the ultrasound beam perpendicular to the imaged structure to avoid hypoechoic areas in the tendons as a result of anisotropism. Generally, imaging in both planes (transverse and longitudinal) as well as varying the angle of the transducer help to determine if a hypoechoic image persists (representing a lesion) or if it is just an artifact. True lesions should be present in all imaging planes.[7]

Metacarpal and Metatarsal Regions

The palmar metacarpal and plantar metatarsal regions are by far the most commonly examined with ultrasound. Ultrasonography provides valuable information on the tendon and ligaments in these regions. Core lesions can be seen on ultrasound as focal anechoic to hypoechoic regions. The structures normally evaluated in the metacarpal and metatarsal regions are the superficial and deep digital flexor tendons, the suspensory (interosseous) ligament, and the accessory ligament of the deep digital flexor tendon. The proximal suspensory ligament is the most challenging region to examine in the metacarpal and metatarsal regions.[8] Differences in the amount of fat and smooth muscle in the proximal aspect of the suspensory ligament result in normal variations in echogenicity and echotexture of this ligament between horses. Thus, comparison with the contralateral limb is important to confidently differentiate lesions from normal variants of the proximal suspensory ligament. When evaluating any structure in the metacarpal and metatarsal regions, comparison with the contralateral limb is important to evaluate structure size because some normal variation exists between horses. Using magnetic resonance imaging (MRI) as the gold standard for identifying proximal suspensory lesions, one abstract reported that ultrasound had 90% sensitivity and 8% specificity to detect the disease.[8]

Other difficulties of evaluating the metacarpal and metatarsal regions are (1) the narrow width of the palmar or plantar aspect of the limb, which results in difficulty in obtaining a truly perpendicular image of the suspensory ligament at its level of origin (especially in the pelvic limb) and at the level of the proximal sesamoid bones, and (2) a lack of a standard method to determine lesion's location. When imaging the metacarpal and metatarsal regions, ensure that images are obtained as perpendicular to the limb as possible based on the position of the hoof.

Two methods to identify the location of a lesion are (1) a zone method (each zone being about 4 cm in length) (Figure 68-5) or (2) a metric measuring method. In the thoracic limb, the *zone method* starts at the base of the accessory carpal bone or proximal aspect of the calcaneus and extends distad to the level of the ergot. These zones are 1A, 1B, 2A, 2B, 3A, 3B, and 3C. In the pelvic limb, there are nine zones starting at the proximal aspect of the calcaneus. In the pelvic limb, zone 3C is not used but rather zones 3A, 3B, 4A, 4B, and 4C. Zone 1A and 1B are generally used to observe the deep digital flexor tendon and

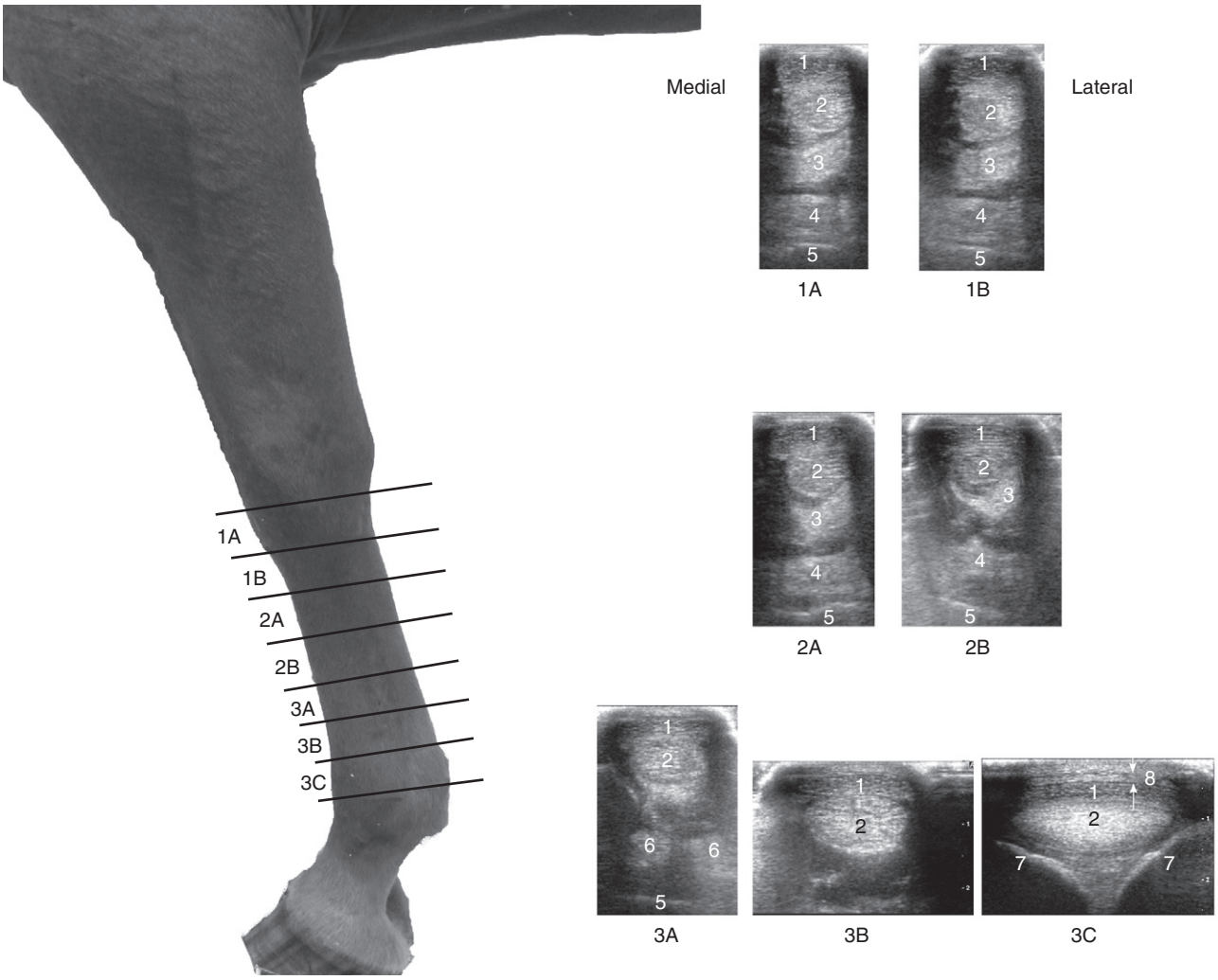

Figure 68-5. The zone method for localizing a lesion on the thoracic limb using ultrasound. *1,* Superficial digital flexor tendon; *2,* deep digital flexor tendon; *3,* distal check ligament; *4,* suspensory ligament; *5,* third metacarpal bone; *6,* extensor branches; *7,* proximal sesamoid bones; *8,* annular ligament.

sustentaculum tali of the pelvic limb. Zones 2A through 4C are similar to those seen in the thoracic limb. The metric measuring method uses a measuring stick or tape and lists the number of centimeters distal to the accessory carpal bone or distal to the tuber calcaneus (point of the hock) as reference to localize the lesion.

Distal Limb

Distal to the proximal sesamoid bones on the palmar and plantar surface of the proximal phalanges, the superficial and deep digital flexor tendons and distal sesamoidean ligaments (Figure 68-6) can be examined. The metacarpophalangeal and metatarsophalangeal (MCP/MTP or fetlock) joints, proximal interphalangeal (PIP, or pastern) and distal interphalangeal (DIP, or coffin) joints can also be imaged. When examining the distal sesamoidean ligaments, the attachment sites of the oblique sesamoidean ligaments can be evaluated as well as the straight sesamoidean ligament. The cruciate ligament of the proximal sesamoid bones can be seen, but are small compared to the other ligaments. In addition, the intersesamoidean ligament, the straight sesamoidean ligament, and the bilobed appearance of the deep digital flexor tendon can be identified. The palmar and plantar structures and collateral ligament of the DIP joint are best evaluated using a high-frequency transducer. A microconvex curvilinear transducer can be used without a stand-off to evaluate the MCP/MTP and PIP joints, though the best-quality images are with a high-frequency linear transducer and stand-off pad.

The collateral ligaments of the MCP/MTP and PIP joints can be evaluated along its full length. Each collateral ligament of the MCP/MTP joint is formed by two distinct structures, the long (more superficial) and short (deeper) collateral ligaments.[9] These two ligaments have slightly different orientation and therefore must be imaged individually. When both the long and the short collateral ligaments of the MCP/MTP joint are in the same image, one can appear more hypoechoic compared to the other because of the difference in fiber direction of the ligament and anisotropism. They will have a normal echogenicity when imaged individually or compared to the contralateral side. The proximal attachment of the collateral ligament of the DIP joint can also be evaluated ultrasonographically. The distal aspect of the collateral ligament of the DIP joint is encased by the hoof capsule, which prevents penetration of the ultrasound beam. The MCP/MTP, PIP, and DIP joints can also be evaluated for the presence of osseous fragments and increased synovial fluid.[10]

Pelvis and Coxofemoral Region

Evaluation of the pelvis with radiography has been described in the equine patient. However, because of the large amount of musculature and the superimposition of the contralateral side of the pelvis, high radiographic settings and oblique angles can make this evaluation difficult in the standing horse. General anesthesia can be used with a specialized table, but the risk of recovering a horse with a possible pelvic fracture makes the potential for exacerbating the fracture not worth the benefit of the diagnostic test.[11] However, ultrasonography is noninvasive and provides a real-time evaluation of the soft tissue and bone surface of the pelvis. A lower-frequency transducer is needed[11] (3 to 5 MHz, macroconvex curvilinear transducer) to obtain the depth necessary to evaluate the coxofemoral joint, whereas a higher-frequency transducer can be used to evaluate the ileum. Indications for pelvic ultrasonography include horses that have a suspected pelvic fracture, hematomas, joint distention, osteoarthritis, and coxofemoral joint luxation. The examination can be performed both transcutaneously and transrectally to evaluate the pelvis and coxofemoral joints[12] (Figure 68-7). Transcutaneous and transrectal ultrasound examination of the sacroiliac joint has been described but never reported.[13] The use of extended view imaging has also been described as a method to

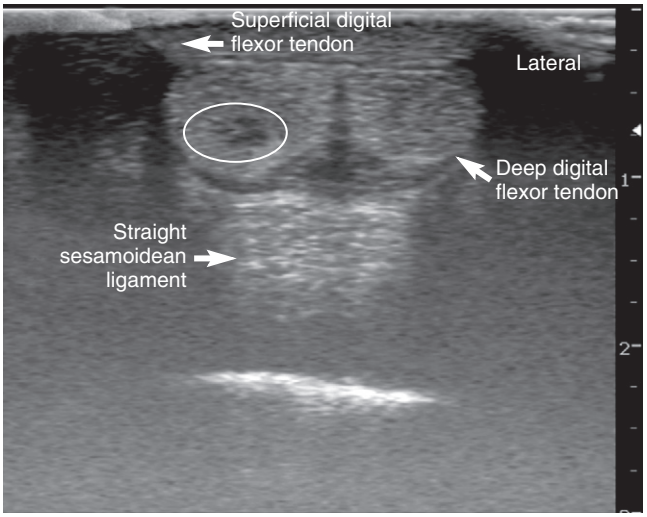

Figure 68-6. Ultrasonographic image of the palmar proximal interphalangeal (pastern) region with the straight sesamoidean ligament and deep and superficial digital flexor tendons identified. In the medial lobe of the deep digital flexor tendon, a focal lesion appears hypoechoic *(circle)*.

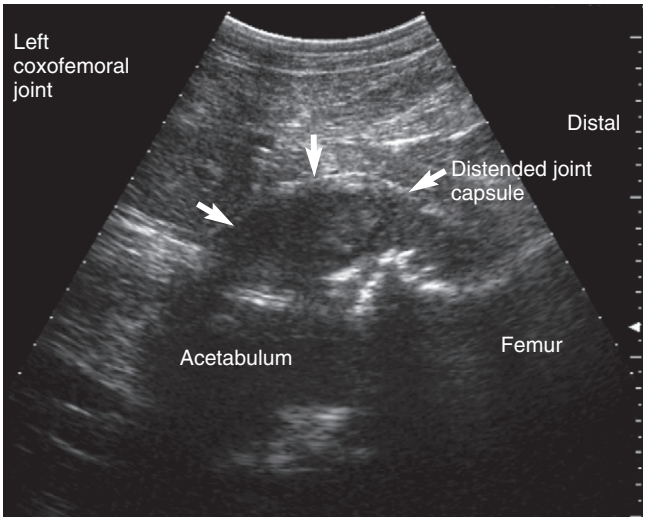

Figure 68-7. A transverse image of the left coxofemoral joint of an adult horse with pain localized to the hip. The large anechoic region is a distended joint capsule and the irregular margin of the femoral head is secondary to osteophyte formation. Ultrasound-guided centesis of the joint space was performed and synovial fluid was obtained that indicated degenerative change rather than infection.

show anatomic information, such as the symmetry of the tuber sacrale with pelvic fractures and to determine if the sacroiliac joint is involved.[10]

Femorotibial Region

Evaluation of the femorotibial region is primarily performed using radiography. However, there are currently many indications for ultrasonography because of the abundance of supporting soft tissues. Ultrasonography can provide valuable information about the three joint spaces (femoropatellar, lateral and medial femorotibial) as well as the menisci and collateral ligaments.[14] A linear transducer can be used to evaluate the joint and even evaluate the trochlear ridges for evidence of osteochondrosis lesions.[15] The patellar ligaments and long digital extensor tendons can also be evaluated in both transverse and longitudinal orientation.

The menisci should have a uniform appearance and the body of the medial meniscus can be observed cranially as it attaches to the tibial tuberosity (Figure 68-8). To image the cranial attachment to the tibial tuberosity (the cranial meniscal-tibial ligament), a microconvex, curvilinear transducer needs to be used to provide a small footprint that allows the ultrasound beam to be perpendicular to the fibers of the cranial meniscal horn. Imaging the insertion of the medial meniscus is important because tears can arise from this region. Additionally, increased joint fluid in one or multiple joint spaces can be seen with meniscal damage.[16]

Abdomen

Ultrasonography of the abdomen is severely limited by the size of the abdomen and the presence of gas primarily within the colon and cecum.[17] Despite this limitation, ultrasonography is extremely useful to evaluate the small and large intestines for displacement or ischemia caused by strangulating lesions. Normal small intestine has good motility and has a wall thickness of approximately 3 to 4 mm.[18] Distinct wall layering of the gastrointestinal tract is routinely seen when using a high-frequency transducer (8 to 10 MHz) (Figure 68-9). A high-frequency microconvex curvilinear transducer provides a large field of view adequate to image most of the abdomen. To image the liver or kidneys, a lower-frequency (3 to 5 MHz) macroconvex transducer can provide more penetration; however, this is done at the expense of image resolution.

Focal ultrasound scans have been recommended for horses that present with acute abdominal pain (colic).[17,19] In these horses, specific sites are scanned to assess specific causes of acute abdominal pain. The term *fast localized abdominal sonography in horses (FLASH)* has been used to describe the evaluation of the equine abdomen not as a complete unit but at focal sites around the abdomen to limit the scanning time to approximately 15 minutes. These sites allow the evaluation of the colon wall thickness for colon torsions (ventral, just caudal to the xiphoid)[17] and right dorsal colitis (right 10th intercostal space)[20] as well as the small intestine (inguinal regions) and the region of a left dorsal displacement (nephrosplenic entrapment; dorsal, left 17th intercostal space).[19] This concept is discussed further in Chapter 36.

CONCLUSION

Ultrasonography is an invaluable tool for surgical planning and evaluation of various equine injuries. From using it on a soft tissue swelling to look for fluid pockets or foreign materials, to evaluating tendons for chronic lameness and imaging the thorax or abdomen for fluid, ultrasonography not only provides a diagnostic modality but can also be used to guide needles for centeses and therapeutic interventions. Because of its portability, lack of invasiveness, and low upkeep costs, diagnostic ultrasound can provide valuable information in patients with both chronic and acute conditions. The largest limitation of ultrasonography is the fact that the image and examination is primarily dependent on the skill of the user, the user's knowledge of anatomy, and allowing adequate time for a thorough evaluation.

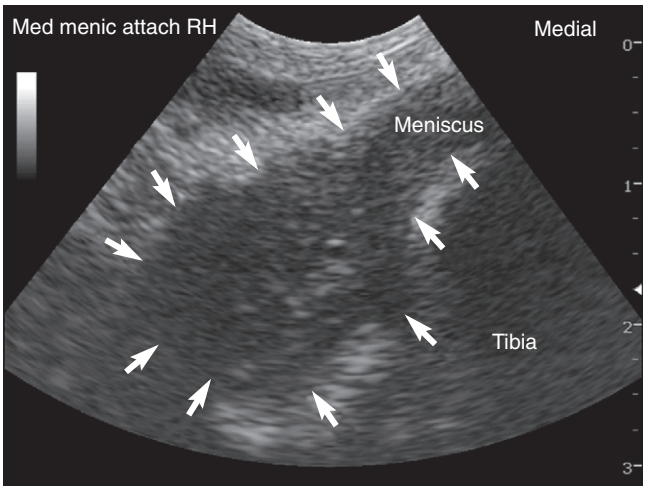

Figure 68-8. Ultrasonographic image of an abnormal appearance of the cranial meniscal-tibial ligament (which attaches the cranial aspect of the medial meniscus to the tibial tuberosity). The ligament should be hyperechoic and uniform in size, but this ligament is large and hypoechoic *(outlined by arrows).*

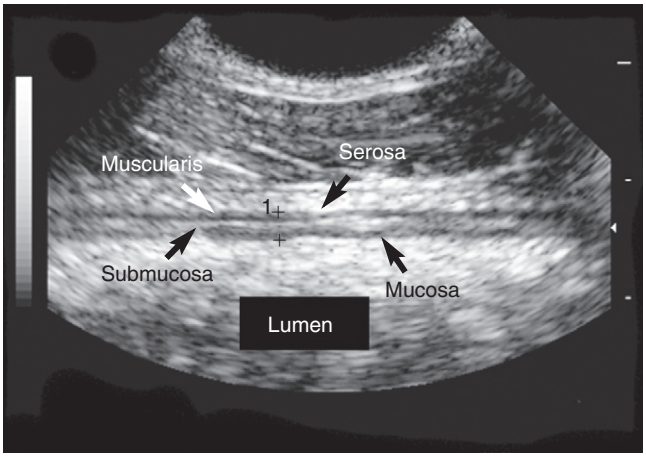

Figure 68-9. The normal colon wall layering including the serosal layer, muscularis layer, submucosal layer, mucosal layer, and the lumen. This image detail is obtained using an 8-MHz probe and imaging at 4 cm in depth.

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CHAPTER
69

Nuclear Scintigraphy

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Nuclear scintigraphy studies are based on the detection of emissions from a radioactive substance introduced into the body. Scintigraphic studies employing a radioactive substance with affinity for bone (bone scans) account for the vast majority of nuclear medicine procedures in the horse. Radiography and ultrasonography, the traditional imaging modalities employed to investigate equine lameness, provide superb anatomic detail. In contrast, bone scan images are poor in anatomic detail but provide information reflecting the physiology of diseased tissues. As a result, scintigraphy may provide definitive information early in a disease process or be a more sensitive supplemental test when traditional imaging methods are equivocal.[1,2] This chapter primarily focuses on bone scan studies, discussing various aspects of the imaging equipment, radiopharmaceutical physiology, imaging artifacts, indications, and some normal anatomic variations for which understanding is necessary for proper image interpretation.

PRINCIPLES

The radioactive substance administered in a nuclear scintigraphy procedure is called a *radiopharmaceutical*, which contains a radioactive atom (called a *radionuclide*) bound to a pharmaceutical compound. Technetium-99m (^{99m}Tc), the most common radionuclide used in nuclear scintigraphic examinations, is a radioactive form of the element technetium and emits a gamma ray with energy of 140 kiloelectron volts. Gamma rays and x-rays are indistinguishable, differing only in their method of production, with gamma rays formed by the breakdown of the nucleus of an atom (as with the natural decay of ^{99m}Tc) and x-rays formed by the ionization of the electron cloud of an atom (as within the anode of an x-ray tube). Detection of gamma rays emitted by ^{99m}Tc is the basis for scintigraphic image formation.

The pharmaceutical component of the radiopharmaceutical determines its biological activity and localization. For skeletal scintigraphy (bone scan), the most common pharmaceuticals are the polyphosphonates, usually methylene diphosphonate (MDP) or hydroxymethylene diphosphonate (HDP). MDP is more readily commercially available than HDP.[3] These compounds are preferentially absorbed by bone, allowing detection of skeletal lesions.

Although less commonly used than MDP and HDP, other pharmaceutical compounds or cells can also be coupled to ^{99m}Tc. Renal and hepatic function can be quantified through use of ^{99m}Tc-mercaptoacetyltriglycine and ^{99m}Tc-mebrofenin, respectively.[4,5] ^{99m}Tc-denatured albumin remains within the vascular bed and can be used to acquire images of vascular anatomy.[6]

^{99m}Tc-hexamethylpropyleneamine oxime (^{99m}Tc-HMPAO) aggregates in white blood cells and can be useful in highlighting areas of active inflammation and infection.[2]

Radiopharmaceuticals are generally purchased through a radiopharmacy and delivered on-site immediately prior to a study. Because ^{99m}Tc decays rapidly, a radiopharmacy must be located nearby for quick delivery. For practices with a high scintigraphy caseload without convenient access to a radiopharmacy, a ^{99m}Tc generator may be employed. These generators contain molybdenum-99 (^{99}Mo), a longer-lived parent radionuclide, which decays to ^{99m}Tc. The ^{99}Mo is separated from the ^{99m}Tc by flushing sterile saline through the generator once or twice a day, creating a solution of sodium ^{99m}Tc-pertechnetate ($Na^{99m}TcO_4$).[7] The $Na^{99m}TcO_4$ may then be bound to the pharmaceutical on-site, using an available kit. Molybdenum generators are generally useful for 1-2 weeks.[7]

As the most commonly used radiopharmaceuticals in equine patients, the biokinetics of ^{99m}Tc MDP and HDP are well known. Following intravenous injection, an initial vascular phase follows the course of the radiopharmaceutical through blood vessels.[2] The radiopharmaceutical then rapidly crosses the vascular endothelial lining to distribute in the extracellular fluid.[3,8] Bone binding subsequently occurs, with maximum uptake at about 60 minutes after injection. These substances bind to exposed hydroxyapatite, with the amount of uptake determined by the rate and extent of osteoblastic activity in bone at the time of administration. In normal bone tissue, proteins line much of the surface of the inorganic matrix, preventing excessive polyphosphonate binding. In areas of bone damage and bone proliferation or in very young animals, more hydroxyapatite is exposed, resulting in greater bone uptake of MDP or HDP. Blood flow is important for delivery but does not affect bone uptake unless severely decreased.[3] By 3 hours after injection, the ^{99m}Tc MDP concentration in normal tissues is proportional to their calcium content, with low concentrations in muscle (approximately 0.005% calcium) and high concentrations in bone (14% to 24% calcium).[8] This dynamic interaction between vascular, extracellular, and bone uptake continues, with relatively greater bone activity and less soft tissue activity over time.

Both ^{99m}TcMDP and HDP are primarily cleared in the urine, with some excretion into saliva and sweat. As a result, the kidneys and urinary bladder are prominently seen on a normal bone scan, occasionally obscuring the anatomy of the caudal spine, pelvis, or proximal hind limbs.[9] Superficial contamination may also occur if voided urine accumulates on the distal limbs (Figure 69-1).

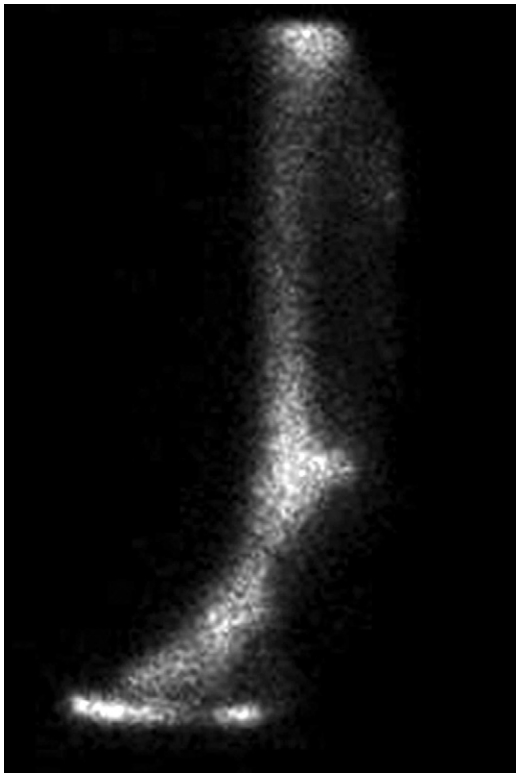

Figure 69-1. Urine contamination on the bottom of the foot. If superimposed over normal anatomy, urine contamination may mimic pathology.

The Gamma Camera

A gamma camera (also called a *scintillation camera* or *Anger camera*) is needed to detect gamma rays emitted from the patient and transpose the data into a clinically useful image that demonstrates areas of radiopharmaceutical uptake. The camera has multiple components that detect gamma rays by converting their energy into an electronic signal sent to an imaging computer (Figure 69-2).

The collimator is a thick lead sheet located on the outermost surface of the camera housing. It is perforated with many small holes that selectively allow only gamma rays that are nearly perpendicular to the collimator surface to enter the camera, blocking off-axis gamma rays and preventing them from being detected by the camera and degrading the image. Without the collimator, the gamma camera does not produce a meaningful image. The gamma rays passing through the collimator form a two-dimensional anatomic image of the patient's anatomy. Different collimator types exist, but the low energy, all purpose (LEAP) collimator is the standard used for routine equine scintigraphic procedures with ^{99m}Tc. High resolution collimators are available, but they attenuate more gamma rays than LEAP collimators and subsequently require longer imaging times. The collimator on most gamma cameras can be removed, but care is necessary as the detector components underneath are easily damaged.

Under the collimator assembly, a sealed large-diameter, flat, fluorescent crystal (usually thallium-activated sodium iodide) approximately 7 to 10 mm thick is located. When gamma rays emitted by the patient strike the crystal, a focal flash of light is emitted (scintillation), with an intensity proportional to the energy of the incident photon. The light from this flash then strikes one or more photomultiplier tubes that are coupled to the crystal. These tubes convert the light energy from the crystal to an electronic signal. An imaging computer records the location where each gamma ray struck the crystal, using signals triangulated from multiple photomultiplier tubes, producing x and y coordinates for each gamma ray recorded by the camera.

EXAMINATION PROCEDURE

The injection of the radiopharmaceutical should always be performed through an indwelling intravenous catheter, which is thoroughly flushed after injection to ensure that the patient receives the entire dose. The radioactivity administered is

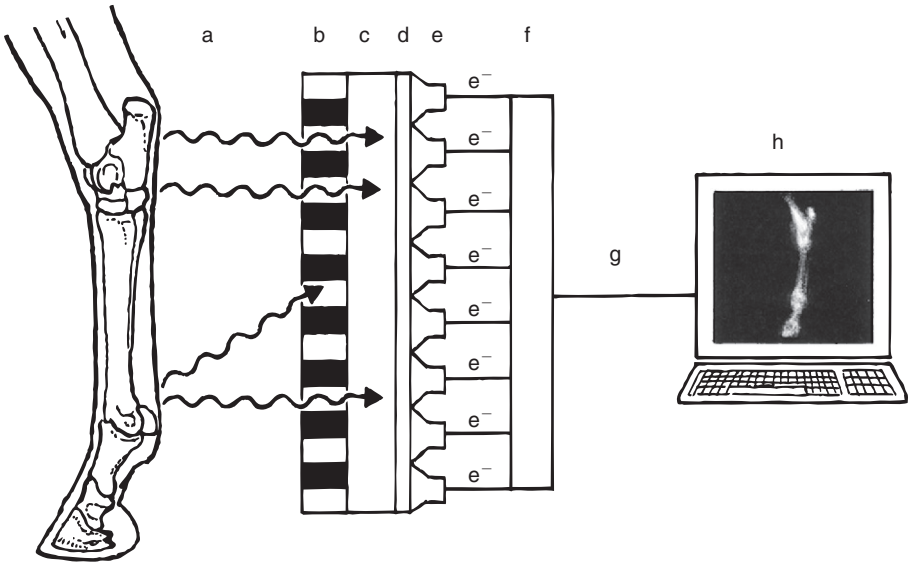

Figure 69-2. Cross-sectional schematic of a gamma camera. Gamma rays *(a)* are produced by the patient and cause scintillation events within the NaI[Tl] crystal *(c)*. The collimator *(b)* prevents nonperpendicular gamma rays from being detected. Photomultiplier tubes *(e)*, which are coupled *(d)* to the crystal, convert the scintillation light to an electric signal that is analyzed *(f)* for positional data *(g)* to create an anatomic image displayed on the imaging computer *(h)*.

measured in the international unit of megabecquerels (MBq) or the traditional unit of millicuries (mCi), with 1 mCi equivalent to 37 MBq. Doses are calibrated for a specific injection time of day, i.e. a dose may be designed for injection at 10 AM. The physical half life of ^{99m}Tc is approximately 6 hours, meaning that if the original dose is 100 mCi, then at 6 hours only 50 mCi will remain. It is therefore important that the dose be administered near the scheduled injection time to achieve the desired level of injected radioactivity and a clinically useful image. A typical dose for an average adult horse is between 120 and 200 mCi 99mTc MDP or HDP. Because a large percentage of radiopharmaceutical is rapidly excreted by the urinary system, the effective half life of ^{99m}Tc within the animal is much shorter than its physical half life.

A complete skeletal scintigraphy study comprises three phases: vascular, soft tissue, and bone. Vascular phase (also called phase I) images are acquired immediately after radiopharmaceutical injection (for 1 to 2 minutes) and demonstrate vascular anatomy before the radiopharmaceutical enters adjacent soft tissues. An image is acquired every 2 seconds, allowing a dynamic view of the body region of interest that may be viewed in series or as a composite image. The short duration of this phase necessitates that the patient be in position in front of the gamma camera when the injection occurs and allows examination of only one body area.

Soft tissue phase (phase II or pool phase) images are acquired when the radiopharmaceutical has entered the extracellular space but has not yet been taken up by bone. This typically occurs between 3 and 10 minutes after injection and is completed at the time bone uptake occurs. Bone uptake may be present by 2 to 3 minutes after injection, especially in young animals or where high bone remodeling is present, or it may be delayed 10 to 20 minutes in older animals.[3]

If bone phase (phase III or delayed phase) images are to be acquired, the onset of imaging is delayed for a longer period. Because of its more rapid bone uptake and increased soft tissue clearance, bone phase HDP imaging may begin approximately 1 hour after intravenous injection, whereas MDP imaging typically begins at 2 hours.[3] Although maximal bone uptake may occur earlier, waiting a longer period allows clearance of MDP or HDP from the soft tissues, giving better images of the bone with less confounding soft tissue uptake.

If the pelvis or upper pelvic limbs will be imaged, furosemide is injected intravenously approximately 60 to 90 minutes prior to imaging. By the time of imaging, the horse should have emptied its bladder, removing excreted radiopharmaceutical that can prevent interpretation of the pelvis or proximal limbs. Protective covers and bandages on the feet and distal limbs, which are removed prior to imaging, protect these areas from radioactive urine contamination.

Sedation is recommended during image acquisition. The amount of sedation used should be sufficient to prevent spontaneous patient motion, but should not make the patient ataxic or wobbly, which can result in significant motion artifact.

Image acquisition may be continued for a fixed time period (time based) or until a certain number of gamma rays are counted (count based). Regardless of method, acquiring a minimum number of gamma ray counts is critical to image quality. In general, distal limb images require at least 75,000 to 100,000 counts and those of the upper limbs and spine require 200,000 to 300,000 counts for satisfactory image quality. Imaging for 60 seconds usually satisfies these count requirements, but 90-second images result in higher counts and often better image quality.

The computer divides the surface of the gamma camera into a row/column matrix of small blocks. Because x and y coordinates for a gamma event are recorded by the gamma camera, the computer bins the event into a block of the matrix.[2] Typically, a 256×256 matrix is employed, although matrices as low as 64×64 have been used. By making each individual block smaller, higher-dimension matrices provide better spatial resolution but require more counts for adequate image quality and thus longer imaging times (Figure 69-3). Matrices with dimensions exceeding 256×256 (i.e., 512×512) exceed the inherent resolution limits of the gamma camera, providing no additional benefit but requiring higher count numbers and longer imaging times.

Whenever possible, orthogonal projections of each body area are acquired, with oblique projections occasionally necessary. For instance, when imaging the pelvis, right lateral, left lateral

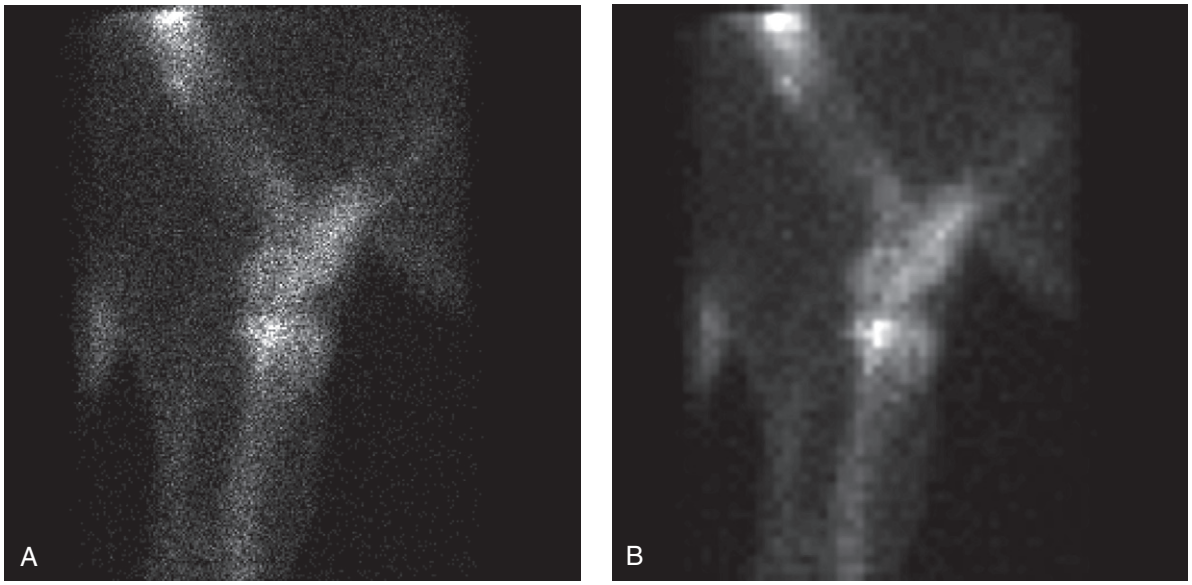

Figure 69-3. Bone phase images of the right stifle acquired with a 256 × 256 matrix **(A)** and a 64 × 64 matrix **(B).** The 64 matrix has a more pixilated appearance and decreased spatial resolution compared to the 256 matrix.

and dorsal projections are routinely acquired, but oblique projections (with the surface of the gamma camera at approximately 30 degrees to horizontal) of the left and right sides of the pelvis provide less anatomic superimposition and can be especially helpful in evaluating the sacroiliac area. For the caudal thoracic and lumbar regions, an oblique angle of approximately 50 to 65 degrees from horizontal is employed, allowing the camera to be closer to the spine than it would be on lateral projections.[10] Dorsal projections of the thoracic limbs or plantar projections of the pelvic limbs include both legs in the same imaging field, allowing direct comparison of the amount of activity within each limb. Because many gamma cameras cannot be placed entirely flush with the ground, the horse can be made to stand on a platform or wooden box to acquire images of the feet.

Flexible or rigid lead sheets should be placed between the left and right limbs when acquiring lateral images, or between the thoracic and pelvic limbs on dorsal and plantar images to prevent *shine through* of the limbs not in the region of interest. For imaging of the spine and pelvis, images should be acquired from both left and right sides. Because of the thickness of the horse and the amount of overlying soft tissues, lesions on the far side of the animal, away from the gamma camera, may not be visible because of gamma photon attenuation by the body.

INDICATIONS FOR NUCLEAR SCINTIGRAPHY

The indications for bone phase (delayed) scintigraphy are many, but in general a bone scan should be seen as a supplemental imaging modality and not a replacement of the basic lameness examination and workup. Bone phase scintigraphy is indicated when:

- The site of lameness cannot be determined because of unclear results of gait evaluation with local anesthesia
- Lameness is localized to a particular area, but injury is not detectable with radiography or ultrasonography
- Multiple limb lameness is present
- Lameness is suspected to originate from the upper limb or pelvis in areas not easily accessible radiographically
- A fracture is suspected (especially a nondisplaced or of a fissure type), but not seen on radiographs
- Lameness is intermittent[1]

Soft tissue phase imaging can be helpful for identifying regions of acute inflammation seen with muscular and tendinous pathologies. Acute inflammatory processes lead to increased regional blood flow and capillary permeability, causing greater radiopharmaceutical uptake in the soft tissue phase. This technique is most useful in the distal limb for lesions such as suspensory desmitis, because the large amount soft tissue surrounding the proximal limb often makes interpretation difficult. Ischemic tissue may be detectable as regions of decreased radiopharmaceutical uptake.

Although classically part of the complete three-phase bone scan, vascular phase images are rarely employed in equine studies, even though they can be helpful to assess the degree of vascular perfusion to an anatomic region.[11] Indications for vascular phase scintigraphy include horses with unusual hindlimb lameness in which there is suspicion of aortoiliac thromboembolism[12,13] and instances in which ischemia or disruption to blood flow is suspected in the distal limb after frostbite, degloving injuries, fractures, or severe lacerations.[2,11] Vascular phase images may be used as part of the complete three-phase bone scan for early detection of osteomyelitis, demonstrating increased regional blood flow to an area.

Rarely employed, radiolabeled leukocytes (with ^{99}Tc-HMPAO) do merit discussion because they can be helpful in imaging orthopedic or soft tissue inflammatory disease. Scintigraphy using ^{99m}Tc diphosphonates demonstrates regions of active bone turnover resulting from infection, fractures, degeneration, and other causes. By selectively labeling leukocytes, ^{99m}Tc-HMPAO allows better discrimination of inflammatory conditions from other causes of bone remodeling and delineates

areas of soft tissue inflammation.[11] Perhaps the most promising indication for radiolabeled leukocyte imaging is the evaluation of orthopedic implants in which infection is suspected but not confirmed by culture, radiography, or diphosphonate bone scanning results.[11,14] ^{99m}Tc-HMPAO has also been helpful to determine the appropriate duration of antibiotic therapy and may be useful for locating sites of infection in animals with fever of unknown origin or persistent leukocytosis.[2] Although early reports using radiolabeled leukocytes in the equine have been promising, few studies exist to determine the true validity of this technique.

INTERPRETATION PRINCIPLES

Correct interpretation of skeletal scintigraphy studies requires familiarity with all factors that can affect radiopharmaceutical uptake, including technical details of image acquisition, the patient's age and use, breed, results of lameness work up, and therapies.[15] Large, well-defined areas of increased radiopharmaceutical uptake (IRU) may be easy to detect and interpret; however, mild to moderate areas of IRU provide greater difficulty, especially for the inexperienced observer.[1] It is important to recognize that increased radiopharmaceutical uptake on a bone scan does not necessarily reflect bone pathology, nor is it synonymous with pain, but it must be interpreted relative to clinical signs and the findings of other diagnostic tests.[1]

Familiarity with normal anatomy and skeletal physiology is helpful when viewing bone scan images. Young animals, because of higher osteoblastic activity, have increased generalized skeletal uptake compared to older animals. The activity in the area of the physes may be particularly intense and varies with the age of the patient. Some physes close scintigraphically at the time of radiographic closure (like the distal femoral physis at 2 to 2.5 years) whereas others may have IRU even after radiographic closure (like the distal radial physis, which may have IRU up to 42 months but closes radiographically at 24 to 32 months).[16] This discrepancy may be explained by the fact that the IRU in the physeal region may not actually represent physeal uptake, but may instead result from histologic architectural changes that expose hydroxyapatite crystals to circulating diphosphonate binding within the adjacent metaphysis and epiphysis, which continue to remodel after physeal activity declines.

Areas of IRU can be present in any horse depending on its stress history and may not be a source of clinical lameness.[15] For example, jumping horses are predisposed to IRU in the phalanges, Standardbreds in the third carpal bone and proximal sesamoid bones, and Thoroughbreds in the distal palmar and plantar third metacarpal and metatarsal bones. Focal intense IRU may be seen on the proximocranial aspect of the tibia in association with middle patellar ligament desmitis, but an identical pattern has been seen in cases of distal limb lameness (where lameness was abolished after blocking the distal limb). In these cases, lameness may have caused increased stress on the patellar ligament, resulting in increased uptake.[17] It must be remembered that an area of IRU is not necessarily a source of pain causing lameness, and normal RU does not mean a lesion is not present that can cause pain or lameness.[17]

In racing horses, the distal third metacarpal bones typically have higher incidence of IRU compared to jumping horses. This is especially seen in Thoroughbreds. Uptake in the dorsal cortex of the metacarpus in Thoroughbreds is unique and may not be associated with lameness. It is likely that IRU reflects a continuum ranging from adaptive stress remodeling to nonadapative modeling that results in lameness.[15]

The effects of perineural anesthesia and joint blocks on the soft tissue and bone phases have been studied. No effect of intra-articular anesthesia is seen on bone phase images, but it can cause some mild IRU on soft tissue phase images. This effect depends on the joint involved and generally persists for 2 to 4 days, but it sometimes lasts as long as 14 days after injection. This is most pronounced in the larger joints of the proximal limb.[18] Similarly, perineural anesthesia has a minimal effect on bone phase images. In the soft tissue phase, low palmar and high palmar blocks result in the greatest focal activity, which are most prominent 1 day after injection and persist to at least 17 days after administration.[19] IRU on bone scan images may be seen after perineural anesthesia more proximally in the limb, where the blocks may involve skeletal muscle, which shows significant sensitivity to the irritant properties of local anesthetics. Focal myositis and necrosis have been reported after local anesthetic injection, resulting in IRU in muscle that persists on bone phase images.[19]

Absence of blood flow on the vascular phase images implies a complete or near complete failure of delivery of radiopharmaceutical, which may result from tourniquet injuries, vascular infarcts, or severe lacerations.[20] Pathophysiologic increases in blood flow, as may be seen with regional inflammation or anesthetic administration, may increase the regional count density.[3] Soft tissue and bone phase images should still be acquired, as collateral circulation may have developed.

The amount of radiopharmaceutical that localizes on the soft tissue phase depends on vascular delivery, autonomic tone in the local area, rates of diffusion into and out of the local extracellular fluid, and the size of the local extracellular fluid space.[3] Any disease process that expands the extracellular fluid compartment will result in increased uptake of ^{99m}TcMDP on soft tissue phase images.[8] Increased soft tissue uptake of radiopharmaceutical usually results from local inflammation, which increases blood flow and vascular permeability with increased delivery and radiopharmaceutical leakage. Early bone uptake can occur as soon as 5 minutes after injection (as mentioned before, particularly in young patients), so soft tissue phase images should be acquired rapidly and interpreted with caution. The clinician should also be cautious in interpreting soft tissue uptake in areas where severe bone uptake is also seen in the delayed bone phase, as early bone uptake may be seen in these regions mimicking soft tissue pathology. Soft tissue phase imaging is likely most effective for soft tissues of the lower limb, as the findings are usually subtle and can be obscured by the significant muscle mass of the proximal limb. If imaging of the soft tissues without the possibility of confounding bone uptake is desired, administration of ^{99m}Tc-pertechnetate (radionuclide in suspension, unlabeled to any pharmaceutical compound) can be performed.

Persistent IRU in soft tissues on bone phase images may be caused by focal dystrophic mineralization, which may occur at sites of chronic inflammatory, necrotic, or infarcted tissue.[9] Uptake may also be seen in tissues that are not obviously mineralized on radiography, particularly in horses with rhabdomyolysis (typing-up syndrome).[21] This occurs as the result of diphosphonates binding with calcium salts secondary to muscle necrosis and influx of calcium into cells, promoting bone tracer uptake.[9] This form of soft tissue IRU is often seen as linear

uptake pattern in muscles on delayed phase images, but it may involve a large muscular region (Figure 69-4).

Poor bone uptake or persistent soft tissue uptake is occasionally encountered, especially of the limbs distal to the carpus and tarsus, and may affect one or more limbs *(cold limb)*.[22] This manifests as a generalized decrease in bone uptake distal to the carpus and tarsus, the metacarpophalangeal or metatarsophalangeal region, or only involving the feet, and may be asymmetric.[22,23] A positive correlation between increasing environmental temperature and better perfusion of the distal limbs and bone uptake of radiopharmaceutical is known to occur.[22] The precise cause is uncertain, but this phenomenon may result in missed lesions or confused interpretation.[22] It has been shown that 15 minutes of exercise at a trot and canter significantly increases hind limb perfusion and uptake of radiopharmaceutical into bone.[22] Bandaging prior to bone scan imaging appears to have no effect on bone radiopharmaceutical uptake.[22] It is therefore recommended that horses be lunged for approximately 15 minutes prior to injection of the radiopharmaceutical, except when a fracture is suspected.[22]

The severity of bone radiopharmaceutical uptake may help determine its cause. Osteomyelitis and fractures tend to have the most intense IRU, with osteoarthritis often more mild (Figure 69-5). Fracture and osteomyelitis uptake is usually intense because of a large increase in blood flow and osteoblastic activity.[24] Overlap in this distinction exists, as enthesopathy

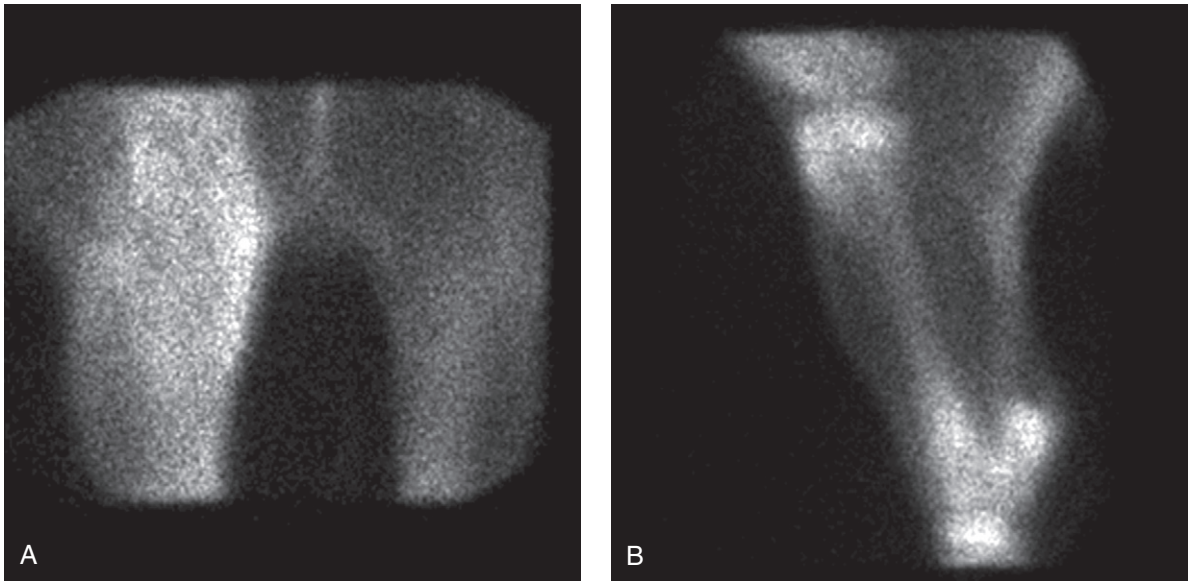

Figure 69-4. Plantar **(A)** and lateral **(B)** bone phase projections of the tibial region in a 7-year-old Quarterhorse examined for swelling of the left proximal hind limb acutely after performance. Intense radiopharmaceutical uptake is seen through the soft tissues despite delayed images (2 hours after injection), consistent with rhabdomyolysis.

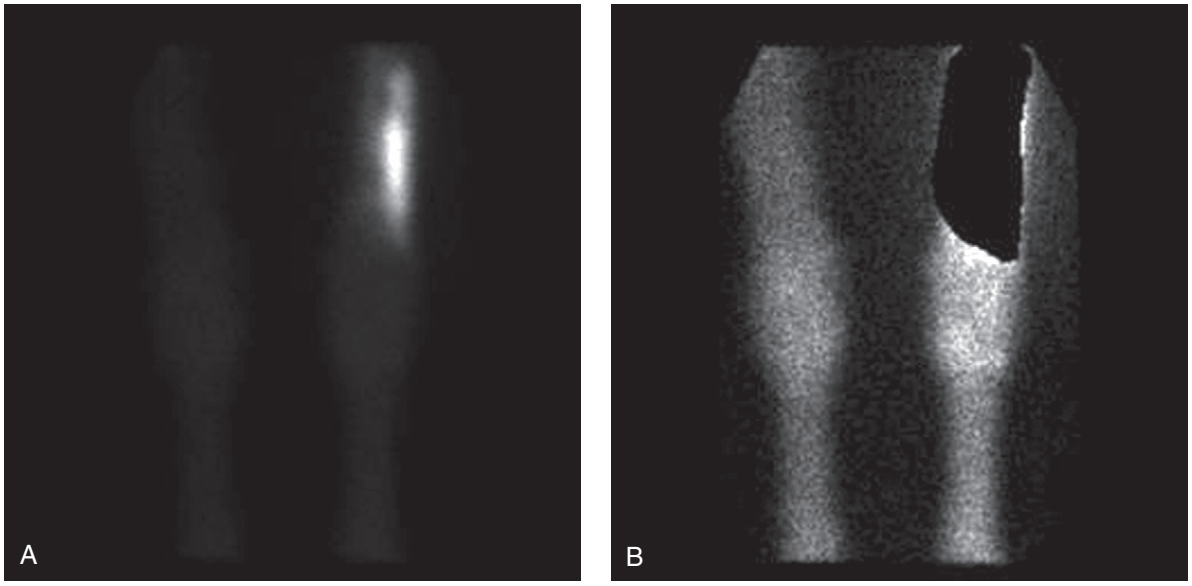

Figure 69-5. Dorsal projections of the carpi, with a fracture of the left distal radius. A digital mask is applied on image **B.** The severe radiopharmaceutical uptake is characteristic of a fracture, causing the normal bone of the distal left limb and the entire right limb to be difficult to see when the mask is not applied. No abnormalities were seen on radiography of this area prior to the bone scan procedure.

and osteoarthritis may also have very intense uptake, which may be focal. Some conditions, such as osteochondritis diseccans and subchondral bone cysts, may have little associated osteoblastic activity, resulting in little or no radiopharmaceutical uptake.[3] When imaging fractures, the time since injury is important to consider. Chronic and subacute fractures (more than 2 days) have more uptake than acute fractures. Fractures less than 24 hours old may have little to no uptake, as osteoblastic activity has not yet increased beyond levels seen in adjacent bone. The pattern of uptake may also be helpful to determine the cause of IRU, with linear uptake along the cortical surface often occurring with stress remodeling and uptake occurring along joint margins in cases of osteoarthritis.

ARTIFACTS AND IMAGE QUALITY

Imaging artifacts may arise from problems with the imaging technique, the gamma camera, or the radiopharmaceutical. A complete review of imaging artifacts and quality control is beyond the scope of this chapter, but the most common causes of poor image quality are discussed.

Poor Image Resolution

Poor image resolution (blurry, indistinct anatomic images) is a common imaging artifact with many causes. Image resolution decreases as the patient moves farther from the surface of the gamma camera (Figure 69-6). Increased distance from the camera allows nonperpendicular, off-axis gamma rays to traverse the perforations of the collimator into the gamma camera, resulting in a blurry image. Ideally, the skin surface of the horse should be within 1 to 2 cm (½ to 1 inch) of the gamma camera face.

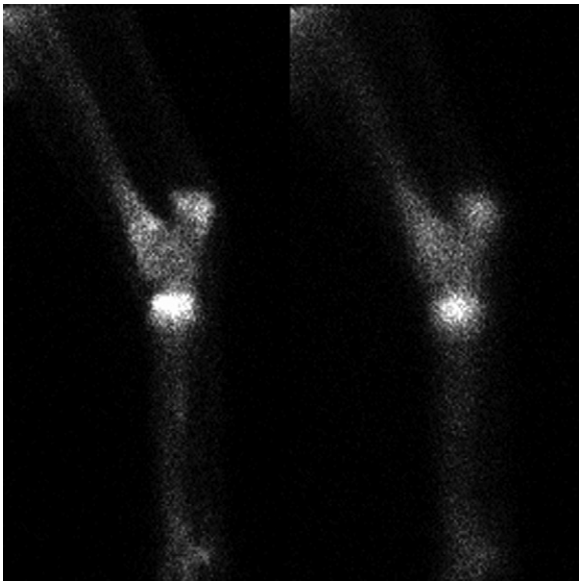

Figure 69-6. Increasing distance between the subject and gamma camera has a negative impact on image quality. The image on the *left* was acquired with the gamma camera 2 cm from the patient and margins of different structures are distinct. The image on the *right* was acquired with a distance of 30 cm between the patient and the gamma camera face. Margins of bone are blurry and indistinct. Moderate IRU at the level of the tarsometatarsal joint is secondary to degenerative joint disease.

Poor image resolution can also be seen with patient motion. Even subtle swaying of the patient can be enough to cause a blurry image. Motion of the gamma camera head during an acquisition can also decrease image resolution and is a problem with older, less-stable gantry-mounting mechanisms. Patient or gamma camera motion artifact can be corrected to some extent by motion-correction software packages. Systems with this capability acquire a series of 1- to 2-second long imaging frames. After the series is acquired, the computer analyzes and corrects for motion-between frames, then sums the frames to create the final bone scan image. This software should be seen as an additional method to improve image quality and does not take the place of patient sedation and restraint.

Gamma Ray Count Density

Adequate *gamma ray count* density is critical to image quality. Adequate numbers of counts ensure that small or subtle lesions are visible and that anatomic margins are well defined. An examination is a tradeoff between achieving adequate numbers of counts for an image and duration of the imaging examination. Acquiring higher count numbers improves spatial resolution but increases the probability of patient motion and total examination time. Many horses will not stand still for more than 60 to 90 seconds, resulting in motion that degrades the image.

Time-Based Acquisition

Time-based acquisition has advantages over count-based acquisition. If the urinary bladder or urine contamination is in the field of view, these highly radioactive areas may cause premature termination of image acquisition if count-based imaging is being used. If lateral images of the right and left limbs are to be compared, time-based acquisition ensures that an area of increased uptake in one limb does not prematurely terminate image acquisition, preventing an "apples to apples" comparison of the two limbs.

Impurities in the Radiopharmaceutical

Persistent soft tissue activity can be caused by impurities in the radiopharmaceutical. The most common impurity is ^{99m}Tc-pertechnetate unbound to any pyrophosphate compound. This impurity arises when air enters the vial during radiopharmaceutical creation, causing oxidation of the reducing agent. Because ^{99m}Tc-pertechnetate acts as an iodine analog (with similar size and valence), uptake in the thyroid gland on bone scan images should alert the clinician to this possibility. Poor bone uptake may also be present. This problem is prevented through careful handling of the radiopharmaceutical kit. Routine quality control assays to the finished radiopharmaceutical product should also be performed.

Gamma Camera

The gamma camera is complex, with many independent parts that may cause artifacts. As examples, photomultiplier tubes may fail or require calibration, the NaI[Tl] crystal may crack, or the camera may be calibrated to the wrong gamma ray energy. The sheer number of potential problems with the physical equipment emphasizes the need for routine quality control

practices such as visual and quantitative assessment of gamma camera field uniformity, tests of linearity and spatial resolution, and calibration of the gamma camera to the appropriate gamma ray energy. Details of these tests and their recommended frequency are available from equipment manufacturers and independent resources.[25]

RADIATION SAFETY AND PERSONNEL EXPOSURE

Principles of modern radiation safety are based on the concept of ALARA, which stands for *as low as reasonably achievable*. Although some radiation exposure to personnel is unavoidable during a scintigraphy procedure, ALARA requires that all reasonable methods should be employed to lower radiation dose to humans and patients.

Time, Distance, and Shielding

Time, distance, and shielding are the three primary methods to achieve ALARA. The fewest personnel necessary should be in the imaging room with the patient, and those present should limit the time spent there to the absolute minimum. Increased distance from the patient can be a very effective method of limiting personnel exposure. Radiation exposure decreases by the square of distance from the source (called the *inverse square law*). Therefore, moving twice as far from a patient decreases radiation dose to personnel by a factor of four, moving four times farther away decreases dose by 16, and so on. When possible, positioning aids are used to maintain patient position without the need for personnel to directly support the patient. Finally, when feasible, radiation shielding should be employed. Prior to injection, the dose should be shielded with a lead syringe casing. During the examination, lead body and thyroid shields (0.5-mm lead equivalent) provide personnel dose reduction factors of approximately four to six.[26] Because of their close proximity to the horse, personnel restraining the horse receive as much total dose as those drawing up and injecting the radiopharmaceutical and operating the imaging equipment.[27]

Excretion

Excretion of many radiopharmaceuticals occurs in body waste. The diphosphonates such as ^{99m}Tc-MDP have high, fairly rapid excretion in the urine, with 70% of the dose excreted through the kidneys within 4 hours.[9] Based on radiation measurement rates for bone scan studies, the biggest radiation hazard to personnel performing these studies is from the radioactive urine and not the horse itself.[28] Latex examination gloves, boot covers, and protective clothing should be worn at all times when dealing with the radiopharmaceutical, the patient, a contaminated stable, or biological waste.[28] Compared to straw, wood shavings are a superior bedding material for the isolation stable because they are more absorbent and result in less drainage of urine and greater control over the area of contamination.[28]

Dosimeters

Personnel dosimeters must always be worn when dealing with scintigraphy studies. Film badges are a common type of monthly or quarterly recording system for radiation dose. These badges contain a small emulsion of radiographic-type film, which becomes exposed to radiation during a time period. Radiation dose is then estimated based on the film exposure. Ring badges are also used to monitor exposure to the hands, which can be significantly higher than that to the body because of the proximity of the fingers to the patient and injected dose. These contain a small lithium fluoride crystal, which stores radiation energy in a manner that can be measured later.

Patient Isolation

Patient isolation is enforced after the imaging procedure has been performed. Release criteria vary by state, but most patients receiving a bone scan are able to be released the following day. Stalls housing the patients should be monitored for radioactivity and cleaning should be delayed until sufficient time has passed for radioactive decay. Geiger-Mueller detectors or ion chambers are used to monitor for radioactive spills or excretions that must be isolated or cleaned.

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CHAPTER 70

Computed Tomography

Jennifer Kinns and Anthony P. Pease

Computed tomography (CT) has been used in human clinical medicine since 1972 and in veterinary medicine since the early 1990s. Its use in equine practice was initially limited to very few institutions, but reports of CT in musculoskeletal and craniomaxillofacial imaging are now widespread. CT uses x-ray beam technology to create tomographic slices of the patient's anatomy. The modality has superior contrast resolution compared to radiographs, allowing fluid and soft tissue to be differentiated. It also eliminates the problem of superimposition that can render radiographs of complex anatomic structures difficult to interpret. Although CT is not as good as magnetic resonance imaging (MRI) for imaging soft tissues because of an inferior contrast resolution, it is the modality of choice for imaging bone.

The use of CT in the horse can be limited by availability, the need for general anesthesia for most systems, and sometimes by the size of the patient. Availability of CT is increasing, and with secondhand systems readily available for purchase from human hospitals, the cost of setup can be relatively low. Many university hospitals and tertiary referral centers now have equine CT capabilities. Anesthesia is necessary for positioning in standard systems. The CT opening, or *gantry*, is a horizontally oriented short-bore cylinder, requiring the horse to be dorsally or laterally recumbent. The length of scan time is very quick, however, and anesthesia times can be kept short. Patient weight and size can limit the ability to perform a CT scan. A specially designed equine CT table is necessary; it must support the weight of the patient, integrate with the existing CT table, and allow the anatomy of interest to move through the CT gantry. The diameter of this gantry opening generally ranges from 50 to 85 cm (20 to 34 inches), and imaging of limb and torso structures above the carpus or tarsus is therefore limited in adult horses.

Standing CT is possible. At the time of writing, it is only described as a prototype for CT of the head and for *ex vivo* imaging of the foot[1,2] but if successful its use is likely to become more widespread.

INDICATIONS

CT evaluation is recommended for a variety of surgical conditions. It can be of particular use in accurately delineating lesions of complex anatomic structures, such as the skull. It is the best modality for evaluating bone and is the method of choice for assessing intracranial hemorrhage in acute trauma.

CT has been used extensively in studies of the equine head.[3-5] Sinonasal disease is a common clinical presentation for which radiographs often provide only limited information and CT can be very useful.[6,7] The CT appearance of dental disease has been described, and the modality is particularly useful for surgical planning in this region,[7] especially with three-dimensional reconstruction. CT is also particularly beneficial in evaluating the temporomandibular and temporohyoid joints, delineating infectious, degenerative and traumatic processes that may be difficult to evaluate radiographically.[8-10] Patients presenting with centrally referable neurologic signs may also be evaluated with CT. Although MRI is the modality of choice for brain imaging, CT can provide important diagnostic information, such as the presence of a mass effect, hemorrhage, or abnormal contrast enhancement, and it is more rapidly acquired than MRI images. CT evaluation of external mass lesions of the head allows a more thorough evaluation of the associated soft tissue and bone involvement than radiographs alone do.

Computed tomography can be used in evaluating appendicular musculoskeletal disease, and it is particularly useful in accurately delineating bone lesions, particularly those associated with trauma. In almost all cases, CT should be used as an adjunct to survey radiographs when additional information is necessary. Clinical use has been described in association with fractures of the middle phalanx[11] and tarsus.[12] In the future, CT may be useful as a method for the early diagnosis and monitoring of stress-associated bone injury.[13] The ability to perform volume-rendered, three-dimensional reconstruction should also make the modality ideal for accurate evaluation of angular

limb deformity. Degenerative bone lesions may be evaluated with CT,[14,15] and this imaging technique has provided excellent detail of new bone formation in horses with proximal suspensory desmitis that has proved refractory to conservative treatment.[16] CT can be used to evaluate musculoskeletal soft tissue, such as the distal deep digital flexor tendon,[17] providing an alternative to MRI when use of the latter is limited by availability.

BASIC PHYSICAL PRINCIPLES AND EQUIPMENT

Hardware

The basic components of the CT scanner are the image gantry and the CT table. The gantry contains the x-ray tube, x-ray detectors, and the data acquisition system; the CT table is an integral part of the system that moves the patient through the scanner at a specific and predetermined speed. Rotation of the x-ray tube within the gantry allows radiographic projections to be obtained at intervals around the patient, and the data obtained from these projections allows construction of an image. The x-ray detectors measure the intensity of the x-ray beam after it has passed through the patient and the data are used to calculate the degree of attenuation of the x-ray beam at each interval. Movement of the patient through the gantry then allows this process to repeat at predetermined locations, resulting in a *slice* image at each location. Certain helical systems operate with a moving gantry rather than a moving table (see later) although the effect is the same.

An image is formed by the mathematical reconstruction of the data acquired by the detectors, generally using a technique called *filtered back projection*. There is a short time delay between scanning and image display while the integral computers perform this function. Various specific algorithms can be applied to the data set to emphasize certain features. In *bone imaging*, for example, spatial resolution can be maximized using a bone, or high-frequency, algorithm. Spatial resolution is defined by the ability to differentiate two points that are close together. Improved spatial resolution therefore allows better definition of fracture margins, for example. For *soft tissue analysis*, a low-frequency algorithm is more appropriate to maximize contrast. Contrast resolution is defined by the ability to differentiate two structures that are similar in attenuation. Improved contrast resolution therefore allows better visualization of changes in tissue structure. Many modern machines allow the application of different algorithms to the data after acquisition, so the patient is only scanned once.

Axial and Helical Scanning

Older CT systems acquired data through *axial scanning*, also called *computed axial tomography (CAT) scan*. Data would be acquired around the patient's anatomy, then the table would slide forward a specified intervening distance and the process would be repeated. This stop-start method takes time, as the gantry had to be stopped after each slice was acquired to allow the electronic cables to be retracted. *Helical scanning* removes the time constraints of axial acquisition; the table moves at a constant speed as the data are collected at intervals around the patient. It is possible because of *slip-ring* technology, meaning that wires are not required to provide power to the x-ray tube, which eliminates the coiling limitations of cables at the end of each slice acquisition, allowing the gantry to rotate continuously. Most modern scanners have both axial and helical capabilities. Helical scanning is much faster and is appropriate for scanning any large area or for angiographic studies.

Specific helical CT systems are now available that allow movement of the gantry rather than the table. They run on rails with a fixed table, and although custom table construction is still required for equine use, a gantry width of up to 80 cm (32 inches) increases the possibilities for equine scanning. As the horse can be kept in one position, it is possible to maintain position with partial suspension on the hoist (Figure 70-1).

Pitch is an important concept in helical scanning, which must be understood by any CT operator. To create the image in helical scanning, a mathematical *interpolation* step is used to fill in the gaps in the data set created by patient motion. This does not have an impact on the quality of the image unless the patient is moved a large distance relative to the gantry rotation. The term *pitch* is therefore used to quantify the movement of the CT table relative to the gantry rotation, and is described by the equation:

$$\text{Collimator Pitch} = \frac{\text{Table movement (mm) per 360° rotation of the gantry}}{\text{Collimator width (mm)}}$$

A pitch of 1 is essentially the same as axial mode acquisition, the gantry rotates 360 degrees around the slice and no data are lost. A pitch of less than 1 implies over-scanning, and the additional information acquired is not likely to markedly improve the diagnostic quality of the image. Pitches of greater than 1 improve the efficiency of scanning and by definition reduce scan times; pitches of up to 1.5 are commonly used where at least 66% of the gantry rotates around a given slice and the remaining 34% is extrapolated from the slice before and the slice after.[18]

Single and Multiple Detector Array Scanning

CT systems are also available as single detector array or multiple detector array scanners. With *single detector array scanners* the

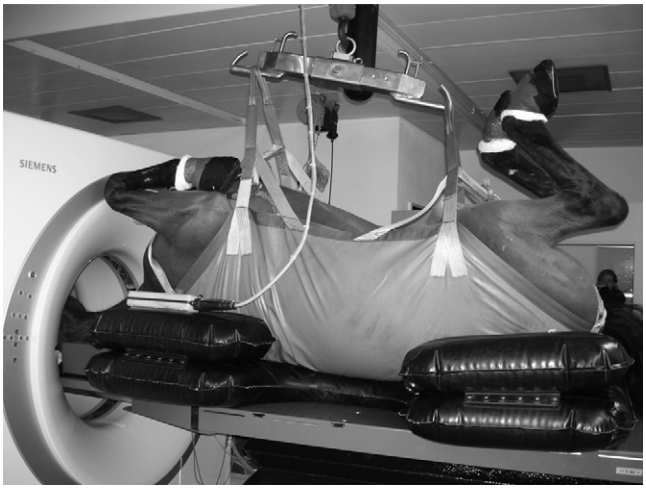

Figure 70-1. Positioning of a horse for scanning in a helical CT system with a moving gantry. As the horse can be kept in one position (without the need for table movement), this position can be partially maintained on a hoist.

slice thickness is determined by opening or closing a collimator. When *multiple detector arrays* are used the slice thickness is determined by the width of each detector in the array, and this can improve spatial resolution. For the same volume of data, multiple detector array scanners reduce tube heating because of increased efficiency, which can avoid tube cooling delays when scanning a large area. The speed of data acquisition is also much faster when multiple detector arrays are used because more than one slice is obtained during one tube rotation. Therefore, multiple array scanners reduce the chance for patient motion and are better for time-dependent angiographic studies.

Choice of Slice Thickness

In single detector array systems the slice thickness is determined by adjustment of lead collimators, whereas in multiple detector array systems it is determined by the number of detectors from which data are grouped together. The choice of slice thickness is important, because it contributes to the contrast and spatial resolution of the study. CT images acquired as thicker slices have a greater number of x-ray photons that are detected. This greatly increases the available signal and improves the inherent contrast of the image. Conversely, thinner slices improve spatial resolution and reduce partial volume averaging (see "Artifacts," later). The choice of slice thickness depends on a number of factors. Tube heating can prohibit the acquisition of thin slices through a large volume, and the size of the anatomy to be scanned may therefore limit the acquisition to thick slices. In general though, thin slices are chosen when spatial resolution is important (such as distal limb bone imaging) and thick slices are chosen when soft tissue contrast is important.

Image Formation

The tomographic slice is an image of a slab of patient anatomy. Like radiographs, it still represents compression of a three-dimensional volume in to a two-dimenisonal image, but the third dimension (slice thickness) can be less than 1 mm and is uniform across the image. The image is composed of pixels, each one representing a volume, or voxel, of tissue. The objective measure of attenuation of the x-ray beam is the Hounsfield unit (also called a CT number). Specific tissues have characteristic levels of attenuation (Table 70-1), and measurement of the Hounsfield units can therefore be used in part to crudely determine tissue type (fluid versus soft tissue) and to measure levels of contrast medium enhancement.

TABLE 70-1. The Characteristic Hounsfield Units (CT Number) for Different Tissue Types

Tissue Type	Characteristic Hounsfield Units
Bone	1000
Fat	–90
Muscle	50
Hemorrhage	70
Gray matter	40
White matter	50

Artifacts

Several CT-associated artifacts can cause diagnostic difficulty, and many of these are accentuated when imaging larger structures.

Beam Hardening

Beam hardening refers to dark or light bands across the anatomy caused by changes in the intensity of the x-ray beam. CT and radiography both use a polyenergetic x-ray spectrum, with the kVp referring to the peak energy of the beam. When the x-ray beam passes through the patient, lower-energy x-rays are attenuated more than those of higher energy. Where the beam encounters a highly attenuating tissue, such as bone, the lower-energy photons are removed and the beam that continues is of higher energy or hardened. Artifacts are produced (Figure 70-2) because rays from some projection angles are hardened more than others, confusing the reconstruction algorithm.

Motion Artifacts

Motion artifacts occur if the patient, or part of the anatomy, moves during image acquisition. Small movements lead to blurring and larger movements can lead to double images, because that part of the patient is scanned in more than one slice.

Partial Volume

Partial volume is a problem with any modality that compresses three-dimensions in to a two-dimensional image. As each pixel on the image represents a voxel (volume) of tissue, more than one tissue type may be represented. This is not usually a problem, but it can lead to misinterpretation if the edge of a curved surface of bone is included in an area of soft tissue (Figure 70-3). The Hounsfield unit measure of that pixel will be an inaccurate representation of tissue content, and the subjective appearance could lead to a misdiagnosis of pathology. Thinner slices limit partial volume artifacts, and careful viewing

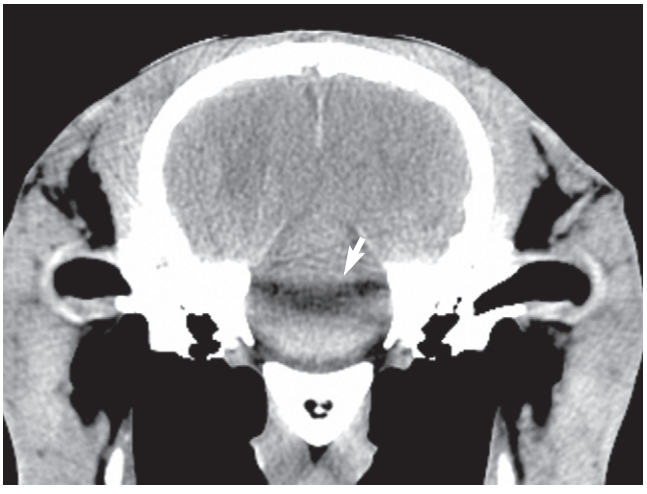

Figure 70-2. Beam hardening artifact in the caudal fossa region of the equine skull. The thick hypoattenuating band across the brainstem *(white arrow)* is an artifact and would prevent evaluation of the parenchyma in that region.

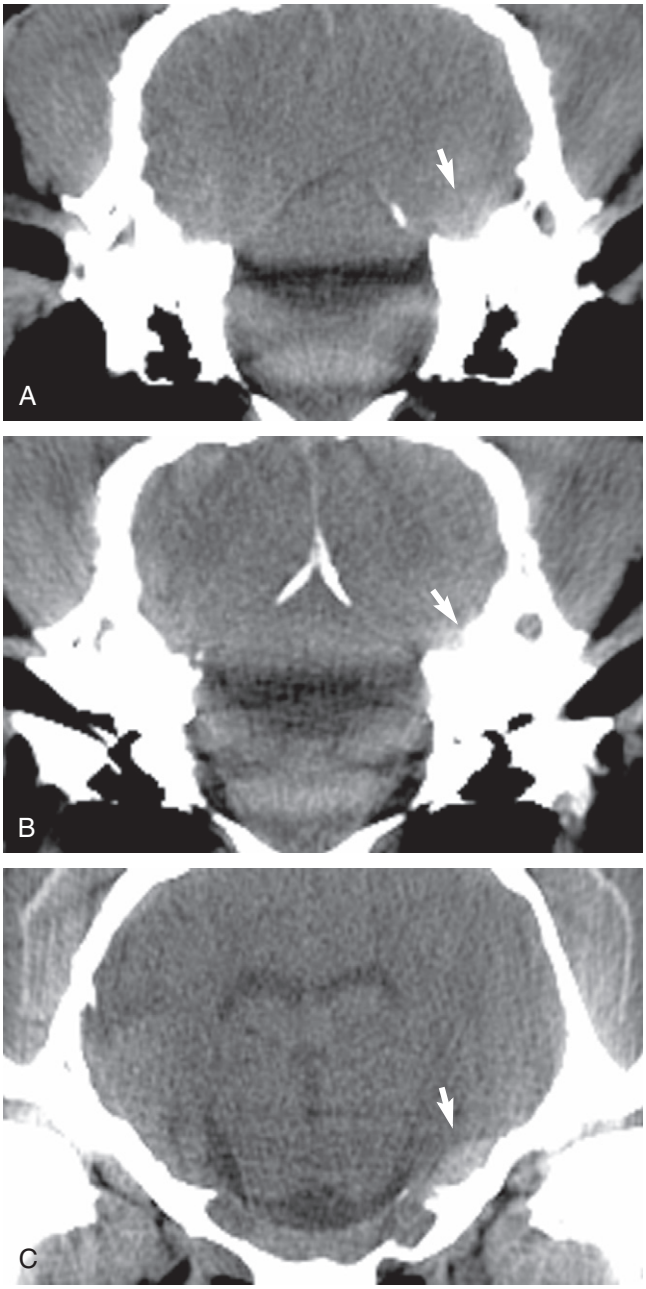

Figure 70-3. A, Partial-volume artifact. The hyperattenuating region *(arrow)* in the ventrolateral aspect of the temporal lobe could be misinterpreted as a parenchymal lesion. **B,** The adjacent slice shows that the curved bone in this region extends axially, and the hyperattenuating appearance was therefore likely due to the presence of both soft tissue and bone material in the projected voxel (the three-dimensional volume of tissue that is compressed in two-dimensions on the image slice). **C,** A true hyperattenuating lesion in a similar location. This represents a subdural hematoma. It can be differentiated from partial-volume artifact by the sharply demarcated margins and absence of an equivalent appearance on the opposite side.

of transverse slices and two-dimensional reconstructions usually identifies the true anatomy.

The equine caudal fossa presents a specific problem in CT. The occipital and petrous temporal bones cause beam hardening, leading to streaks across the region, and partial volume causes inappropriate hyperattenuation of regions in the brain

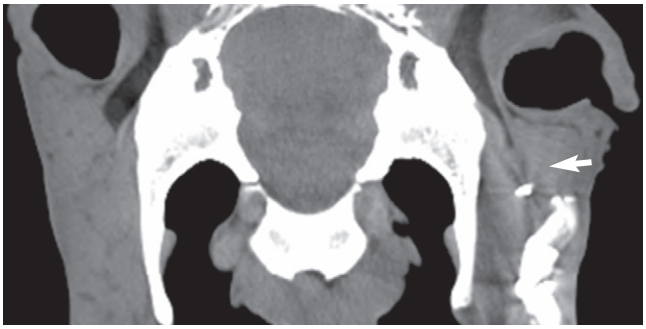

Figure 70-4. Artifact associated with regional injection of nondiluted contrast medium. There is marked streaking artifact *(arrow)* around a contrast-filled blood vessel. Metallic implants create a similar though more marked artifact.

stem. Acqusition with thin slices that are reformatted as thick slices for viewing can help reduce the problem.[19]

Metallic Artifacts

Metallic artifacts occur if there is any metallic implant within the imaged anatomy. Although this is not usually a problem in equine imaging, a similar artifact can occur if nondiluted contrast medium is used for fistulography or regional perfusion studies (Figure 70-4). Severe streaking artifacts occur. A combination of factors lead to the artifact; beam hardening and partial volume are present in addition to the fact that the computer cannot interpret information from material as dense as metal. A number of software corrections are available to limit this and are manufacturer specific.

Use of Contrast Media

Contrast media can be used in CT in a number of ways. For all contrast procedures, a non–contrast medium–enhanced scan must be obtained initially as a baseline. Most typically, contrast medium is used to evaluate tissue perfusion. After intravenous injection of an iodinated contrast medium, tissue that has a vascular supply will enhance. Pathological enhancement, as seen with inflammation or neoplasia, will be greater than that of surrounding normal tissue, although certain tissues such as lymph nodes or salivary glands have a higher normal degree of enhancement because of their blood supply or affinity for iodine. To evaluate contrast enhancement, a time delay of at least 5 minutes is recommended before the postcontrast scan is obtained to facilitate recirculation of contrast. A dose of at least 200 mL of 300 mg/mL iodinated contrast medium is recommended. This is significantly less than the dose used for small animal imaging, but it is typically adequate for evaluating perfusion. The typical small animal dosage of 80 mgI/kg is not technically feasible because of the time necessary for injection, and the likelihood of side effects with a full dose is unknown.

CT Angiography

CT angiography refers to the use of contrast to highlight the vasculature, with the timing of the CT scan set to highlight arterial or venous contrast. This technique has been used in the equine foot.[17] It can be used to define the arterial and venous vascular anatomy of a region for diagnostic purposes or prior

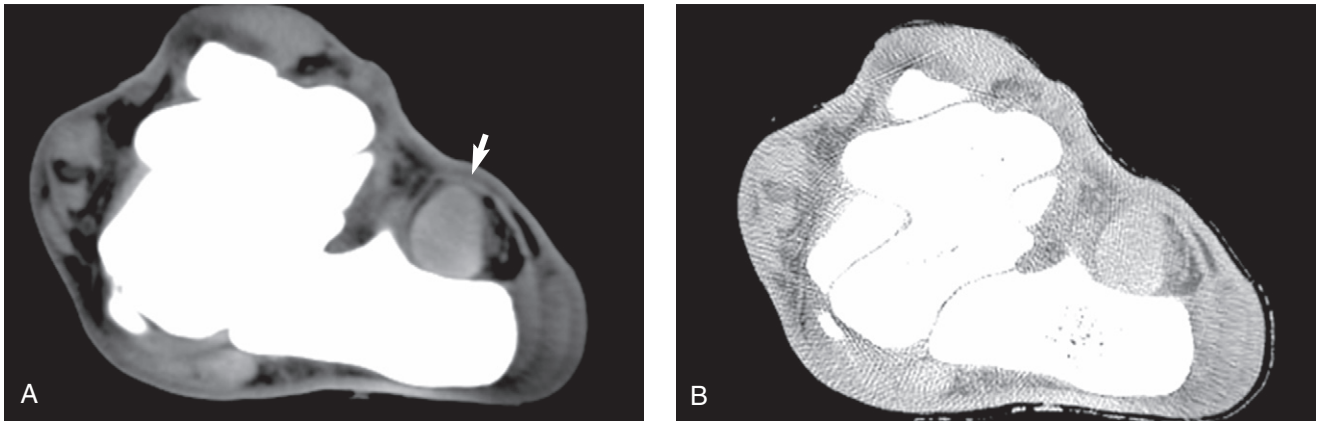

Figure 70-5. A, Transverse image of the carpal canal acquired with thick slices and a soft tissue algorithm demonstrating good delineation of the flexor tendon *(arrow).* **B,** This is the same region acquired with thin slices and a bone algorithm. The increased noise on the image makes it more difficult to differentiate tendon from surrounding tissue, and any lesion could be missed.

to surgery, and to differentiate vascular anomalies, such as arteriovenous fistulas from other vascular lesions. If a peripheral vascular injection is used, an initial test bolus is usually required, from which the timing of the arterial or venous scan is calculated using a dynamic scan obtained in one location. Alternatively, the vascular region of interest may be specifically catheterized, thus avoiding the time delay and dilution associated with peripheral injection. If specific catheterization is used, contrast medium should be diluted to 50 to 100 mgI/mL to avoid excessive artifact.

CT Fistulography

CT fistulography can be a useful method to evaluate the path of a draining tract and involvement of critical structures. For the skull in particular, being able to view the anatomy without superimposition, is a great advantage. Use of a Foley catheter, where possible, can help avoid leakage of contrast medium. Contrast media should be diluted with saline or sterile water to obtain a concentration of 50 to 100 mgI/mL to avoid excessive artifact, and CT acquisition is then performed immediately after injection to the point of back pressure. This procedure is most useful in conjunction with intravenous contrast enhancement, because inflammation of the tract seen with the latter may provide better delineation of the extent of the lesion.

Dacryocystography

Dacryocystography is the use of a contrast medium to evaluate the nasolacrimal system. CT-dacryocystography can be useful in horses presenting with chronic epiphora or facial and orbital trauma. The nasolacrimal duct is catheterized, and diluted iodinated contrast is injected, followed by an immediate CT scan.[20]

INTERPRETATION PRINCIPLES

Image Viewing

CT studies are viewed using different window widths and levels to highlight different types of structures. The *window level* refers to the Hounsfield number around which the other shades of gray are centered. For soft tissue viewing, this should be about 50, whereas for bone viewing it is approximately 500. The *window width* is the number of shades of gray that are portrayed on an image. The human eye can only visualize 30 to 90 shades of grey; a CT image has many more subtle differences in grey scale (typically 4,096), but they will not be detected. To visualize soft tissues, the number of shades of gray on the screen are decreased so that subtle tissue differences become more apparent (Figure 70-5). When contrast is maximized in this way, it is at the expense of spatial resolution, and bone margins are not well defined. For bone imaging, spatial resolution is maximized by increasing the window width to about 2000, at the expense of soft tissue contrast (Figure 70-6). All CT studies should be viewed using both bone and soft tissue settings in order that lesions are not missed or overinterpreted. Suggested window settings for different structures are listed in Table 70-2. Bone windowing is not the same as using a bone algorithm for data reconstruction. Where maximum spatial resolution is necessary, both a bone algorithm (at the data processing stage) and a bone window (at the viewing stage) should be used.

TABLE 70-2. Suggested Window and Level Settings for Viewing Different Regions of Interest

Tissue Imaged	Window	Level
Bone	2000	500
Brain	150	40
Soft tissues	400	50

Reconstruction in Multiple Dimensions

One of the advantages of CT imaging is that images can be reconstructed in multiple two-dimensional planes after image acquisition. This is referred to as *multiplanar reconstruction (MPR)*, which allows reconstruction of a two-dimensional image in any plane. Sagittal, dorsal, and oblique plane reconstructions aid in defining the location and specifications of a lesion. They are particularly useful for fractures or luxations, where the transverse slices alone could miss a lesion if the fracture line is parallel to the transverse axis. MPR reconstruction is recommended for all musculoskeletal or dental imaging as an adjunct to transverse image viewing. The quality of the

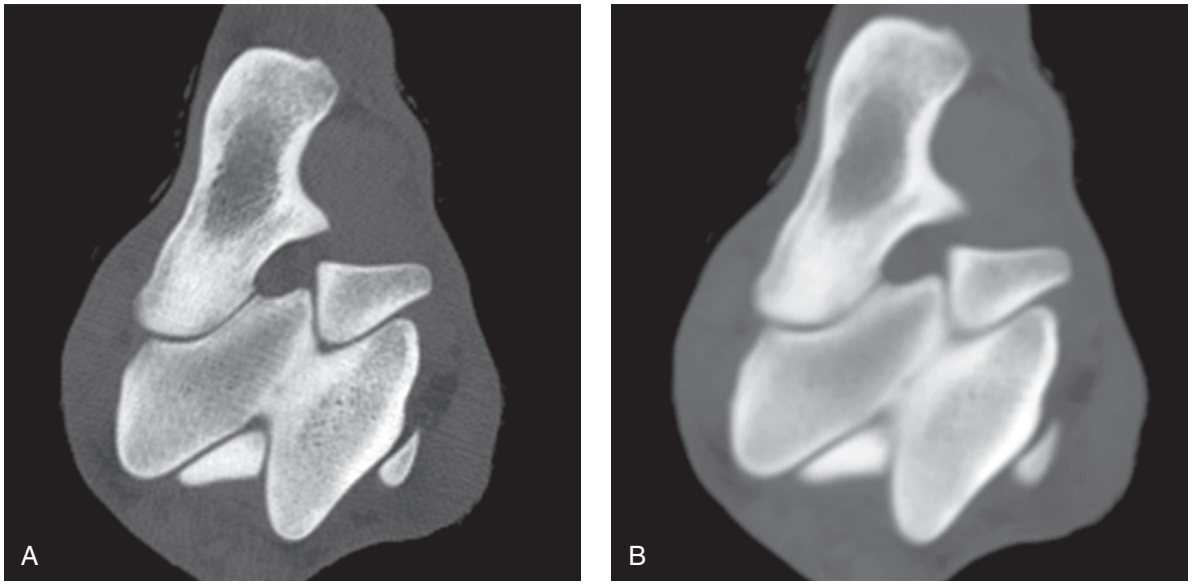

Figure 70-6. A, Transverse image through the tarsocrural joint acquired with thin slices and a bone algorithm. There is exquisite detail of the bone. **B,** The same image acquired with thick slices and a soft tissue algorithm. Although it is viewed with a bone window, the spatial resolution is not as good, resulting in blurred margins and a loss of trabecular detail.

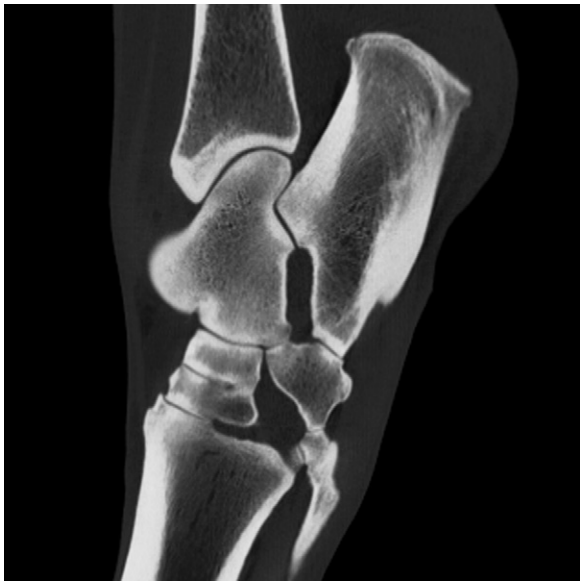

Figure 70-7. A sagittal reconstruction of the tarsus.

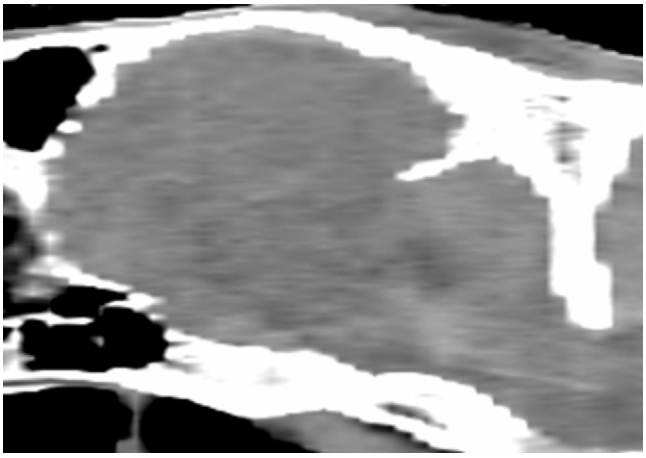

Figure 70-8. A sagittal reconstruction of a thick slice helical acquisition of the caudal skull with significant stair step artifact.

reconstruction depends on the slice thickness used for acquisition and the use of helical or axial modes. Thinner slices always produce a smoother reconstruction with better spatial resolution in the selected planes (Figure 70-7). Helical reconstructions, particularly when acquired as thick slices, are susceptible to a *stair step* artifacts, where the surface of the reconstruction is interrupted by multiple regularly spaced fluctuations (Figure 70-8). This can be reduced by using overlapping slices.

Three-Dimensional Volume Rendering

Three-dimensional volume rendering can be a very important tool in surgical planning (Figure 70-9). It allows visualization of the anatomy in a lifelike perspective, and it can be used to understand lesion orientation. It can also be important in explaining lesions to the client for whom transverse slices of anatomy are harder to understand. Three-dimensional reconstruction should not be used as a substitute for evaluation of MPR or transverse images in diagnosis. This is because a smoothing and shading algorithm is used, which can make small fracture lines or nondisplaced fracture fragments difficult to impossible to see. The resolution of the three-dimensionally rendered image is poor, and superimposition is limiting.

Curvilinear Reconstruction

Curvilinear reconstruction methods allow the operator to select a curved reconstruction plane to follow a structure of interest, thus flattening that portion of the anatomy that could not otherwise be visualized in one plane. In imaging the cranial cervical

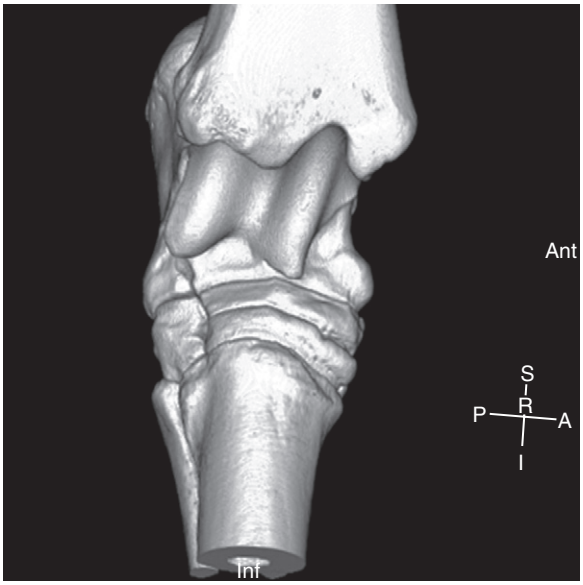

Figure 70-9. A volume-rendered three-dimensional reconstruction of the tarsus.

region, for example, the spinal cord could be flattened such that it appears on one dorsal plane image, allowing better evaluation of lateralizing compression.

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CHAPTER 71

Magnetic Resonance Imaging

Joana Chaby L.S. Coelho and Jennifer Kinns

For the past several years, magnetic resonance imaging (MRI) has been increasingly used as a diagnostic tool for investigation of equine lameness. MRI has numerous advantages compared to other imaging modalities. Unlike radiography and ultrasonography, MRI allows concurrent detailed evaluation of osseous and soft tissue structures. Additionally, MRI allows evaluation of soft tissue structures that are not accessible with ultrasonography, such as several structures within the hoof capsule and the intraarticular ligaments of joints, such as the carpus and tarsus. MRI provides physiological information that, unlike nuclear scintigraphy, is anatomically specific. The soft tissue contrast and detection of fluid within bone are superior with MRI compared to computed tomography (CT). In contrast to CT, the evaluation of cartilage with MRI does not require intraarticular contrast injection. When compared to radiography, CT, and ultrasonography, MRI has reduced spatial resolution. However, this apparent limitation is greatly outweighed by MRI's excellent contrast resolution. The exceptional ability of

MRI to distinguish different tissues is because several parameters related to tissue's biochemical environment can be manipulated to accentuate tissue differences. MRI can identify lesions not detectable by other imaging modalities[1-5] and often adds information about lesions that have been previously diagnosed. Equine MRI should be considered when the cause of lameness cannot be determined based on clinical examination and other imaging modalities, specifically radiography and ultrasonography. The number of sequences and planes necessary to produce a diagnostic study mean that imaging one anatomic region can take up to 45 minutes. As horses are either under general anesthesia or standing (still) under sedation, localization of the lameness is essential so that the MRI examination can be limited to a specific anatomic region.

EQUIPMENT, PHYSICS OVERVIEW, AND SAFETY

Equipment and Logistics

The main components of any MRI system are the magnet, gradient coil, radiofrequency generator, and computer, the last of which controls the interplay among the other components and the reconstruction, storage, and display of images.[6] The strength of a magnet's main magnetic field is measured in tesla (T), where 1 T equals 10,000 gauss (G), which is 20,000 times the strength of Earth's magnetic field.[7] Both high-field (more than 1 T) and low-field (less than 1 T) MRI systems are currently used to image the equine limb. Either of these systems must be built in a room shielded from extraneous radiofrequencies that otherwise would interfere with the scan. Caution must be taken with electronic equipment that is brought into the shielded room because it may degrade image quality. All horses undergoing MRI must have their horseshoes and nails removed prior to conducting the study to avoid artifacts (see "MRI Artifacts," later). Support tables for equine MRI are custom built with nonferrous material. Thick plywood or synthetic material and stainless steel can be used. Plenty of padding is important to provide adequate support.

Anesthesia equipment should contain as little ferromagnetic material as possible and should be placed in the room far enough from the magnet to not interfere with the magnetic field. All high-field and most low-field MRI systems require the horse to be under general anesthesia. An MRI examination of both feet takes approximately 60 minutes. Additional time is required for anesthetic induction and positioning of the horse in the magnet. Complications from general anesthesia have not been observed at institutions where numerous horses have undergone MRI since 1997.[8] However, the scan should be limited to a specific anatomic location. Scanning more than one region on each limb not only increases scanning time, it also requires extra time to reposition the limb in the coil and the horse in the magnet. Therefore, prolonged anesthesia can be avoided by isolating the source of lameness to one region. Some low-field MRI systems allow the horse to be imaged while standing under sedation.

Motion artifact is an important limiting factor of the diagnostic quality of the images acquired with these systems. Low-field systems have lower signal-to-noise ratio compared to high-field systems, which translates into lower-resolution images. To increase signal-to-noise ratio when using low-field systems, the scanning times must be longer, which further increases the chance of motion artifacts. Furthermore, the field of view is relatively small when using low-field systems, which limits the size of the anatomic region to be imaged on each scan.

The *configuration of the magnet* is the factor that limits the anatomic region that can be imaged. Most high-field systems have a closed-bore (tubelike) configuration, which makes the length of the bore and diameter of the gantry the limiting factors. Most bores are 50 to 60 cm (20 to 24 inches) in diameter; one system currently in the market has a 70 cm (28 inches) diameter gantry. The open vertical low-field magnets are limited by the dimension of the vertical gap (where the imaged region is positioned), which is relatively small (usually 45 cm [18 inches]). Both high- and low-field (with the horse under general anesthesia) systems allow imaging of the distal limbs up to and including the carpus and tarsus, and the equine head. Most high-field systems are not able to image the equine neck, although the wide-bore configuration magnets can image as caudal as the fourth cervical vertebra in smaller horses. Certain open low-field systems can also image the neck up to this level. The width of the shoulders prevents imaging of the more caudal cervical region. A limited number of short-bore wide-gantry high-field systems can image the equine stifle, if the horse meets specific measurement criteria.[9] The caudal cervical spine, thorax, and abdomen of an adult horse cannot be imaged. Low-field standing systems also allow imaging of the limb up to the carpus and tarsus. However, motion artifacts usually render nondiagnostic images.

For an MRI examination of the head or limbs, adult horses are positioned in lateral recumbency on a wheeled custom-built table that is placed next to the back of the magnet (opposite side from the magnet's table). Foals under 90 kg can be positioned in dorsal recumbency on the magnet's built-in table.[10] Unlike in most CT scanners, the customized support table for adult horses remains stationary during the MRI.

When using any MRI system, it is important to position the region to be imaged in the isocenter of the magnet because image quality deteriorates as the area being imaged is farther away from the isocenter. Positioning the region of interest in the isocenter can be a challenge, especially when using closed-bore, high-field systems. The horse's body is pushed as close as possible to the magnet and the imaged limbs are pulled into the bore of the magnet with ropes that are applied at the level of the pastern. Additionally, removing the built-in table from the magnet's bore may give more room for the limbs to be pulled farther into the magnet. To ensure that the limb being imaged is correctly labeled as right or left, vitamin E capsules can be taped on the lateral aspect of the hoof. For example, one capsule can be used for the right foot and two for the left. The vitamin E capsules show as rounded homogeneous high-signal intensities (Figure 71-1).

Physics Overview

Signals generated during MRI come from hydrogen protons. Hydrogen protons are the basis of MRI because they are much more abundant in the body than other atoms and have favorable magnetic properties.[7] During an MRI exam, the hydrogen protons are placed under the influence of a strong magnetic field. The protons are excited by an applied radiofrequency (RF) pulse. When the RF pulse is removed, the protons relax and through this process emit a signal, which is used to create an electronic image. The proton's relaxation occurs through three different processes: T1 relaxation (or T1 recovery), T2 relaxation

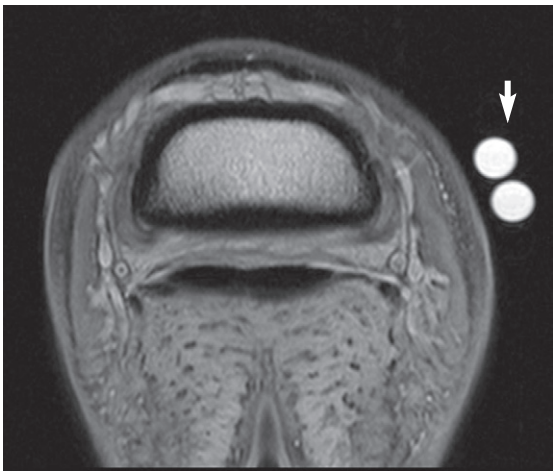

Figure 71-1. Transverse proton density image of the distal limb, at the level of mid second phalanx showing two vitamin E capsules *(arrow)* taped to the lateral aspect of the limb. The normal bilobed deep digital flexor tendon is seen palmar to the second phalanx. (Courtesy Dr. Russell L. Tucker, Washington State University, Pullman, WA.)

(or T2 decay) and T2* (read *T2 star*) decay. T1 recovery refers to the relaxation achieved through energy released from the protons into their environment. T2 decay results from the energy transfer between the protons themselves. These two relaxation processes depend on the environment that surrounds the protons. Thus, protons in different tissues have different relaxation properties. T2* decay depends solely on field inhomogeneities. Field inhomogeneities are inherent to all magnets, being greater in magnets with open (versus closed) bores and in magnets with wider (versus narrower) bores.

In MRI, differences in tissue contrast result from differences in the various tissues' relaxation properties (T1 and T2 relaxation), proton density (PD), and parameters that are operator dependent. Magnetic resonance pulse sequences that emphasize T1 relaxation properties of the tissues are designated T1-weighted and sequences that emphasize T2-relaxation properties of the tissues are called *T2-weighted*. The image contrast in PD-weighted sequences depends solely on the number of hydrogen protons present in the tissues.

Repetition time (TR) and echo time (TE) are key parameters for creating image contrast that can be manipulated during the MRI. TR is the time (in milliseconds) between the application of the RF pulse and the start of the next RF pulse. TE is the time (in milliseconds) between the application of the RF pulse and the peak of the signal received by the coil to produce the image. Both parameters affect MRI contrast because they provide varying levels of sensitivity to the relaxation properties of different tissues. TR and TE affect contrast on T1- and T2-weighted images, respectively. Images with short TR are T1-weighted, whereas images with long TE are T2-weighted. PD images use long TR and short TE, so the effects of T1 and T2 relaxation, respectively are minimized and the number of protons present in the tissue is the main factor contributing to image contrast.

The ideal parameters to image a specific anatomic region vary among different magnets. Thus, scanning protocols must be optimized for each individual MRI system.

Spin echo (SE) and gradient echo (GRE) pulse sequences are the two fundamental types of MRI pulse sequences, both of which are used for equine imaging. The two major advantages of GRE sequences are shorter acquisition time, which reduces artifacts resulting from motion and time under general anesthesia, and volumetric (three-dimensional) acquisition, which allows thinner contiguous slices (1.5 mm compared to 3 to 4 mm with SE) to be obtained without compromising signal-to-noise ratio.[11] GRE sequences are used in standing MRI systems where motion artifacts and relatively low signal to-noise-ratio are of most concern. On the other hand, GRE sequences have poorer contrast resolution compared to SE sequences, because of increased susceptibility to field inhomogeneities.[12] Fast (or turbo) SE sequences (versus conventional spin echo sequences) are often used for equine imaging to decrease acquisition times. However, these sequences still require longer acquisition times than do GRE sequences.

Different MRI pulse sequences highlight different types of tissues and lesions. In one examination, various *combinations of pulse sequences* can be used depending on the imaged region and type of lesion that is suspected. Magnetic resonance pulse sequences commonly used to image the equine limb are fast (or turbo) SE (T2, PD, or T1), short tau inversion recovery (STIR), and GRE (T2*-weighted, PD or T1) sequences. (Because GRE sequences do not cancel out magnetic field inhomogeneities, unlike SE, GRE sequences are called *T2*-weighted* instead of *T2-weighted*.)[11] *PD* and *T1* sequences are useful for demonstrating anatomic detail and are especially useful to evaluate tendons and ligaments. On *T2 sequences*, fluid has high signal intensity (white). Given that tissues that are diseased often have increased water content and consequently have a high signal on T2 images, they can be easily identified with this sequence. Most lesions within the bone, such as edema-like lesions and osseous cystlike lesions, result in high signal intensity on most MRI sequences. To most easily identify these lesions, sequences using fat suppression techniques are used. Fat suppression can be achieved by using STIR sequences (a spin echo sequence) or by using a technique called *fat saturation*.[11-13] In both STIR and fat-saturated images, bone has low signal intensity (black). Thus, pathologic changes that result in increased signal intensity within the bone are easily depicted with these sequences. The main advantage of fat saturation is that it can be applied to SE and GRE sequences.[11] On the other hand, fat suppression in STIR sequences is more complete than in fat-saturated sequences.[13]

Safety

Contrary to imaging modalities, such as radiography and CT, that use ionizing radiation, there is no evidence that there are any long-term or irreversible biological effects associated with the magnetic fields used in MRI.[11,14] However, other hazards do exist when using MRI. Given the strong magnetic fields and the strong force that they exert on ferromagnetic metal objects brought close to them, rigid safety procedures must be used. Ferrous objects (such as clippers, hoof knives, and oxygen tanks) must not enter the magnet room because they can become harmful or even lethal projectiles. Prop materials (such as sand bags) should be inspected because some are not filled with sand but with steel shot, which is highly magnetic. Credit cards and magnetic media can be erased by the magnetic field and should not be taken near the magnet. Objects may be tested with a hand-held bar magnet before being taken into the room. Personnel must be educated about potential hazards, and signs

should be attached to all entrances to the magnetic field (including the fringe field) to deter entry of people who may be wearing pacemakers, have metallic ferrous implants, or are carrying ferromagnetic objects.

INTERPRETATION PRINCIPLES

Clinical MRI has been used to evaluate multiple structures of the equine limb including tendons, ligaments, bone marrow, joints, and articular cartilage. On an MRI examination, regardless of whether the lameness is unilateral or bilateral, both limbs are often concurrently imaged. Because of the high level of symmetry between the right and left equine distal limbs,[15] imaging both limbs allows subtler lesions to be more confidently identified and most normal variants to be correctly interpreted.

On MRIs, high signal intensity refers to brighter areas (white) and low signal intensity refers to darker areas (black), intermediate signal refers to structures with various shades of gray. The terms *hyperintensity* and *hypointensity* can also be used to designate areas of higher and lower signal intensity, respectively. Cortical bone has low signal intensity on all sequences. Medullary bone has high signal intensity on most sequences because of the high fat content of the bone marrow. In general, tendons and ligaments have low signal intensity in all sequences.

Similar to CT, the data acquired with MRIs are tomographic (acquired slice-by-slice) although, unlike CT, MRI slices can be obtained in any anatomic orientation without loss of image quality. Orthogonal slice planes (sagittal and transverse) are routinely performed. Additional planes oriented through specific anatomic parts can help identify certain lesions.

MRI examinations result in a large number of serial images to be evaluated. Interpretation of MRIs requires thorough knowledge of the regional anatomy, as well as an understanding of pathologic processes and physics of MRI. The remainder of this chapter focuses on the principles of interpretation of MRI of specific anatomic regions of the equine limb with a brief reference to MRI of the equine brain.

INTERPRETATION OF MAGNETIC RESONANCE IMAGES

The Foot

The region most commonly examined with MRI is the foot. MRI can identify osseous and soft tissue lesions in the foot that are not depicted by other imaging modalities.[2,5,16-19] Today, because of MRI, it is recognized that the soft tissues within the hoof wall play a major role in the various pathologies encountered in the navicular syndrome.[2,5,8,20-22] Affected structures that are commonly recognized on the MRI examinations of horses that present with palmar foot pain are the distal sesamoid (navicular) bone, podotrochlear bursa, deep digital flexor tendon, distal sesamoidean impar ligament, and collateral sesamoidean ligaments. Several of these structures are often concurrently affected.[2,5,20,21,23-25] The higher sensitivity and specificity of MRI for lesions in most structures within the foot allows more-specific therapeutic protocols to be prescribed earlier in the disease process.

Distal Sesamoid Bone and Podotrochlear Bursa

MRI can reveal abnormalities of the distal sesamoid bone that are not detectable with radiography and provides additional information in horses with radiographic changes. Changes of the distal sesamoid bone that can be seen with MRI and often not on radiographs include (1) medullary cavity changes (abnormal increase or decreased signal intensity), (2) flexor cortex erosions, and (3) fragmentation of the distal margin of the distal sesamoid bone, at the origin of the impar ligament (Figure 71-2) and at the distal angles of the distal sesamoid bone. Distal sesamoid bone lesions are most often seen concurrently with different combinations of lesions of the deep digital

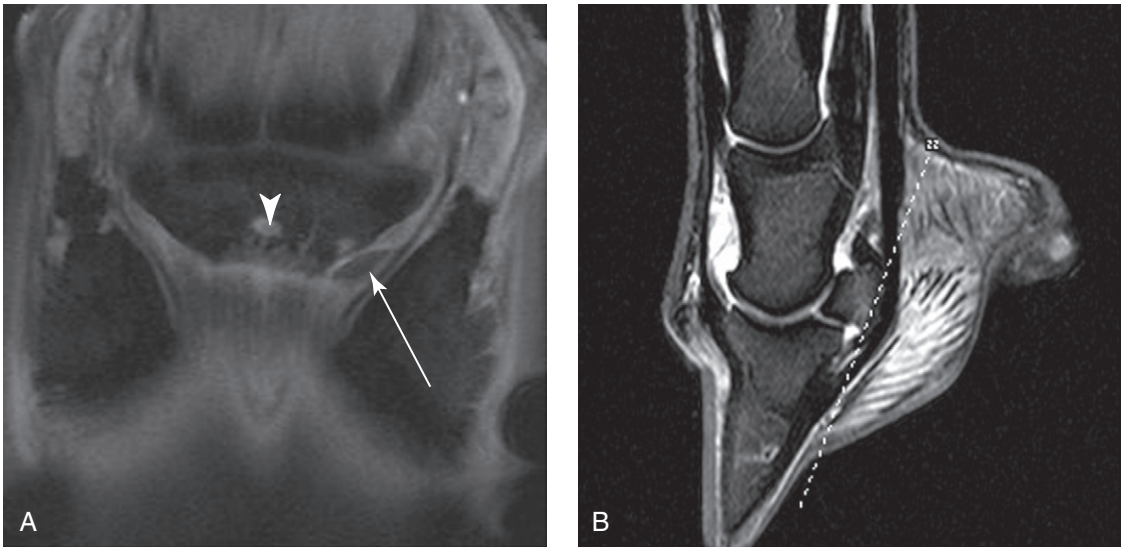

Figure 71-2. A, Dorsal gradient echo image (GRE) of the distal sesamoid bone. Note the fragment next to the distal margin of the distal sesamoid bone at the origin of the distal sesamoidean impar ligament *(arrow).* Also note the enlarged rounded distal synovial invaginations *(arrowhead);* **B,** Sagittal short tau inversion recovery (STIR) image of the foot showing the plane image from which **A** was obtained. (Courtesy Dr. Russell L. Tucker, Washington State University, Pullman, WA.)

flexor tendon, collateral sesamoidean ligament, and distal sesamoidean impar ligament.

The majority of horses with palmar foot pain or navicular syndrome have *high signal intensity* within the medullary cavity of the distal sesamoid bone on STIR and fat-saturated images.[2,5,21] In addition, more focal hyperintensity is often seen in the region of the synovial invaginations when these are enlarged (Figure 71-3). On histopathology, the areas of high signal intensity within the medullary cavity of distal sesamoid bones of horses with advanced "navicular disease" correspond to areas of fat degeneration and necrosis, osteonecrosis, fibrosis, hemorrhage and edema.[15,21] Concurrent *low signal* within the medullary cavity on PD, T1, and T2 sequences may, or may not, be present (Figure 71-4). Regions of low signal intensity within the distal sesamoid bone can result from chronic blunt trauma and represent areas of decreased bone marrow fat content and replacement of marrow trabecular bone with compact trabecular bone.[19,21]

Regions of altered signal in the distal sesamoid bone can also be found in the proximal, distal, and palmar/plantar aspects of the bone. Distal sesamoid bone flexor cortex erosions are seen on MRI as areas of intermediate or high signal intensity along the cortical bone.[19,26] A normal synovial fossa present in the middle third of the sagittal ridge of the distal sesamoid bone may be seen on sagittal MRI as a smooth concavity in the flexor margin of the bone.[27] This should not be interpreted as an erosion or degeneration of the palmar fibrocartilage.

Podotrochlear bursitis, characterized by distention of the bursa with fluid with or without proliferation of the soft tissues, can be seen on MRIs. Fluid within the bursa has a high signal intensity on T2 and PD images. In the normal bursa a small amount of fluid is seen in the proximal and distal recesses. Most commonly fluid is not seen in the bursa between the distal sesamoid bone and the deep digital flexor tendon, and if fluid is seen in this region it is only a thin line.[28] An increased amount of fluid within the podotrochlear bursa is more easily identified in its proximal recess, between the collateral sesamoidean ligament and the deep digital flexor tendon. The distal recess of the bursa does not distend as much as the proximal, and larger amounts of fluid must be present to separate the distal sesamoid bone from the deep digital flexor tendon.[29]

Deep Digital Flexor Tendon

Deep digital flexor tendon injury is an important cause of lameness in horses with foot pain.[15,17,21-23,30,31] On MRI, the normal deep digital flexor tendon has regular margins and uniform low signal intensity (black) on all standard sequences. In some horses, on T1 images, a stippled appearance may be present in the tendon because of small intermediate signal intratendinous foci. These foci correspond to normal loose connective tissue septa.[28] In the distal limb, the deep digital flexor tendon is formed by two symmetric rounded lobes separated by a well-defined midline sagittal high-signal-intensity septum.[28,32] From the level of the proximal distal sesamoid bone distally, the tendon becomes progressively flatter (in a dorsopalmar/plantar direction) and at its insertion, it is crescent shaped with minimal lobe separation.[32] Because of magic angle effect, high signal intensity is commonly seen in the distal aspect of the deep digital flexor tendon when pulse sequences with short TE are used (see "MRI Artifacts," later).[33-35]

Deep digital flexor tendon lesions are easiest to identify on transverse images because of the loss of symmetry in shape, size and/or signal between the two lobes of the tendon. The majority of the lesions within the deep digital flexor tendon are seen as increased signal intensity on T2 and PD images. Deep digital flexor tendon lesions can be (1) core lesions (focal high signal intensity) (Figure 71-5), (2) parasagittal splits (linear parasagittal increased signal intensity) (Figure 71-6), or (3) dorsal irregularities or abrasions (irregularities of the dorsal margin) (Figure 71-7). Enlargement of one lobe or both tendon lobes is often seen concurrently with these lesions, but significant tendon enlargement has only been found at the site of core lesions.[32] The type of lesion or combination of deep digital

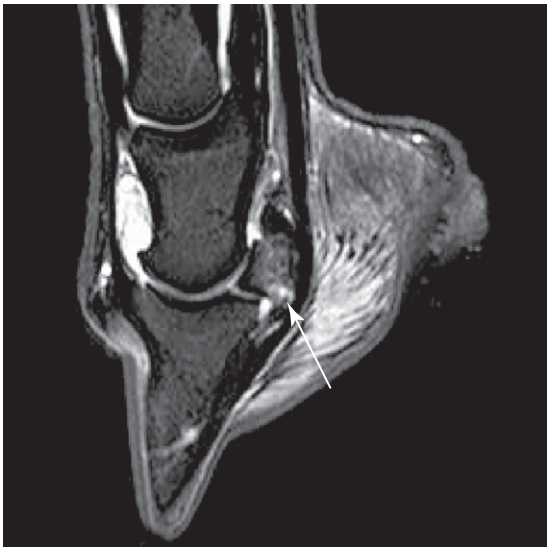

Figure 71-3. Sagittal short tau inversion recovery (STIR). The distal sesamoid bone is diffusely hyperintense. In addition a more focal hyperintensity is present in the region of the enlarged synovial invaginations *(arrow).*

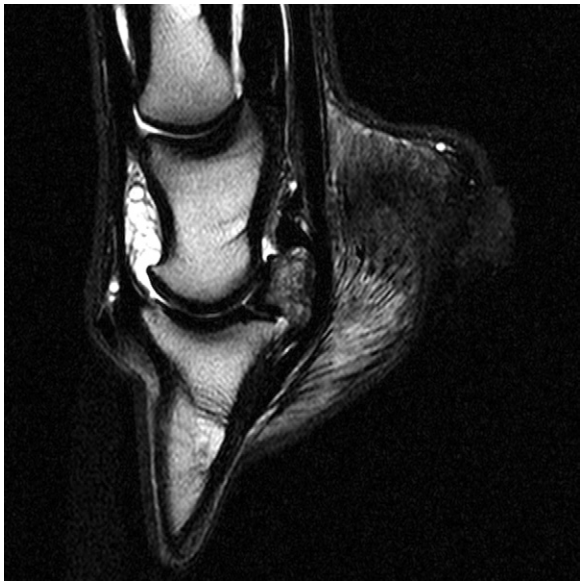

Figure 71-4. Sagittal T2-weighted image of the same foot as Figure 71-3, showing diffusely decreased signal within the distal sesamoid bone.

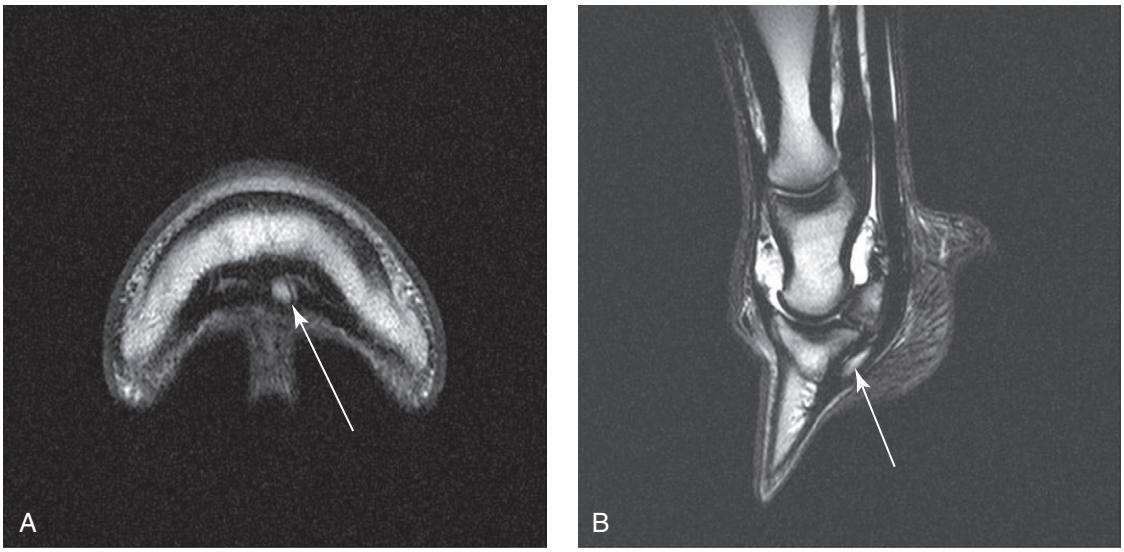

Figure 71-5. A, Transverse T2-weighted image at the level of the insertion of the deep digital flexor tendon onto the distal phalanx. Note the focal high-signal-intensity lesion *(arrow)* at the insertion of the deep digital flexor tendon onto the distal phalanx. **B,** Sagittal T2 image showing the same lesion. This lesion is not caused by magic angle effect given that this image is obtained using a long echo time (TE). (Courtesy Dr. Russell L. Tucker, Washington State University, Pullman, WA.)

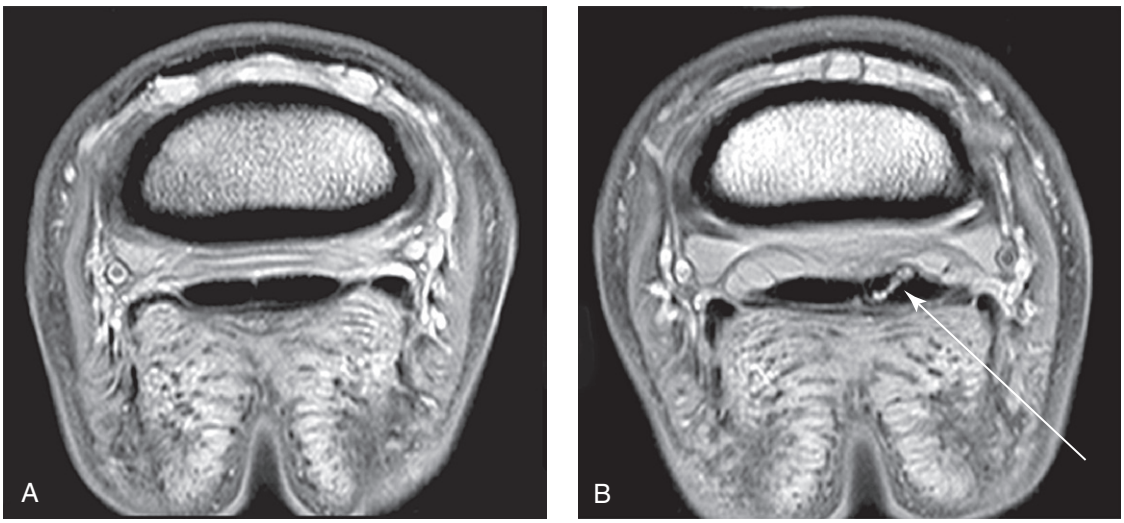

Figure 71-6. A, Transverse proton density image of the distal limb at the level of the proximal aspect of the second phalanx. Note the high signal-intensity parasagittal split through the lobe of the deep digital flexor tendon *(arrow)*. **B,** The normal contralateral limb is shown for comparison.

flexor tendon lesions varies depending on their anatomic location.[21,23] Lesions of the distal deep digital flexor tendon can occur proximal to the distal sesamoid bone, at the level of the collateral sesamoidean ligament and proximal recess of the podotrochlear bursa, at the level of and distal to the distal sesamoid bone. Horses may present with a single lesion or concurrent lesions at different sites. Deep digital flexor lesions may or may not be associated with distal sesamoid bone changes in horses with heel pain. Histopathology of lesions of the deep digital flexor tendon, identified with MRI, in horses with advanced distal sesamoid disease[21] and in horses with lameness of at least 2 months' duration[30] abolished with a palmar (abaxial) nerve block, revealed degenerative changes of the deep digital flexor tendon without evidence of inflammation. Because the appearance of degenerative lesions of the deep digital flexor tendon is similar to the ones reported in other studies (without histopathology confirmation) as tendinitis, the term *tendinopathy* is considered to be more appropriate when describing lesions of the deep digital flexor tendon localized to the foot. Although it is likely that inflammation is present at some point soon after the initial injury, its MRI appearance has not been correlated with histopathology findings. Adhesions of the deep digital flexor tendon can occur to the collateral sesamoidean ligament and distal sesamoid bone.[15,27] Adhesions of the deep digital flexor tendon are most easily seen when fluid is injected into the podotrochlear

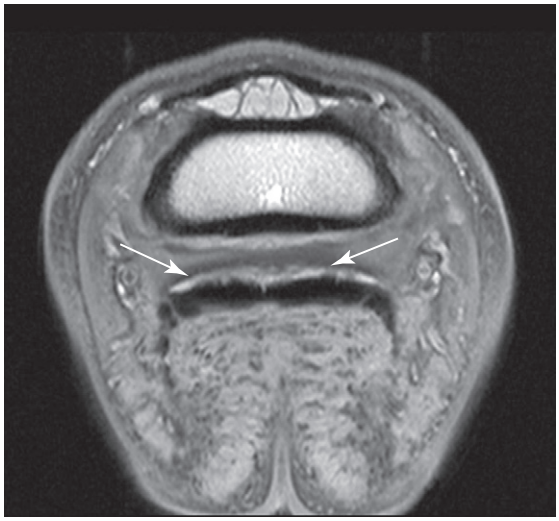

Figure 71-7. Transverse proton density image of the distal limb, at the level of mid middle phalanx. Note the irregularities of the dorsal margin of both lobes of the digital flexor tendon *(arrows).* (Courtesy Dr. Russell L. Tucker, Washington State University, Pullman, WA.)

bursa.[36] However, because of time constraints and logistics, this may not be practical in the clinical setting. Tendon adhesions in and around the podotrochlear bursa may benefit from débridement during bursoscopy.[37]

Distal Sesamoidean Impar Ligament

The distal sesamoidean impar ligament can be easily seen on MRI because of the high signal intensity of the synovial fluid within the distal interphalangeal joint and distal recess of the podotrochlear bursa, which outlines its dorsal and palmar borders, respectively.[28] The normal distal sesamoidean impar ligament is symmetric with smooth margins, and it has a striated signal intensity pattern with higher signal intensity compared to the deep digital flexor tendon. Increased signal intensity within this ligament should be interpreted with caution because of its fan shape and synovial in-pouchings between fiber bundles.[15] Moreover, when subject to magic angle effect its signal is homogeneously increased (see "MRI Artifacts," later).[28] Thickening of the ligament can be seen with or without signal intensity changes. Lesions of the distal sesamoidean impar ligament are often associated with abnormalities of the distal sesamoid bone, involving not only the distal margin but also the proximal margin and medullary cavity. Osseous changes at the origin of this ligament include entheseous new bone formation and avulsion fractures. Fragmentation at the origin of the distal sesamoidean impar ligament can be clearly seen when a thin slice plane is oriented proximad to distad over the distal sesamoid bone, perpendicular to its distal margin (see Figure 71-2).

Collateral Sesamoidean Ligament

The collateral sesamoidean ligament is obliquely oriented, which makes its full evaluation more difficult. On transverse images, the ligament is formed by two thin branches (lateral and medial), which originate from the dorsal distal aspect of the proximal phalanx. Proximal to the distal sesamoid bone, the branches merge with the ligament's body. At this level, the ligament has a narrower central body and symmetric lateral and medial branches (Figure 71-8). On T2 images the collateral sesamoidean ligament has signal intensity similar to the deep digital flexor tendon. Normal ill-defined symmetric increased signal intensity is commonly seen within the ligament's branches just proximal to the point where they merge with the ligaments' body. Injury of the collateral sesamoidean ligament can be present by itself; however, most commonly it is associated with distal sesamoid bone and/or deep digital flexor tendon lesions.[5] Lesions of the collateral sesamoidean ligament can be seen as thickening of its body and/or braches, diffuse or less commonly focal increased signal intensity, and loss of separation from the deep digital flexor tendon (see Figure 71-8). Entheseous new bone formation and avulsion fractures can be seen at the insertion of the ligament onto the distal sesamoid bone.

Collateral Ligaments of the Distal Interphalangeal Joint

The normal collateral ligaments of the distal interphalangeal joint have mildly heterogeneous low signal intensity on T2 images, and mild variations in signal intensity can be present in sound horses.[38] Increased signal intensity from magic angle effect can be seen in the collateral ligaments of the distal interphalangeal joint (see "MRI Artifacts," later).[38-40] To assess the size, the collateral ligament can be compared to the other collateral ligament in the same foot and with the collateral ligament in the contralateral foot. Focal or diffuse increased signal intensity, poorly defined margins, and osseous changes at the attachment sites of these ligaments can be seen in lame horses. Osseous lesions are commonly seen concurrently with ligament lesions. Osseous lesions are more common at the ligaments' insertion onto the distal phalanx than at their origin.[41,42] Also, the most severe lesions have been identified in the distal aspect of the ligaments,[41] which are not accessible ultrasonographically. Histopathology of the collateral ligaments of horses with lameness of more than 2 weeks' duration revealed mostly degenerative changes with little evidence of inflammation.[41] Given that the MRI appearance of degenerative and inflammatory processes is similar, the term *desmopathy* (versus desmitis) is considered more accurate to describe MRI changes of these ligaments.

Distal Digital Annular Ligament

The distal digital annular ligament is mostly located within the hoof capsule. This makes its ultrasonographic evaluation difficult. On MRI, this ligament is seen as a discrete thin structure with homogeneous low signal intensity. Distal digital annular ligaments with focal or diffuse thickness greater than 2 mm and heterogeneous signal intensity are considered abnormal.[43] Adhesions between this ligament and the deep digital flexor tendon may be seen with high-field MRI. Horses with distal annular ligament desmopathy may benefit from tenoscopic surgical transaction of the ligament.[43]

The Distal Sesamoidean Ligaments

Desmopathy of the distal sesamoidean ligaments is a cause of lameness that has been most commonly diagnosed with ultrasonography. The medial and lateral distal oblique sesamoidean ligaments are symmetric and on MRIs have low signal intensity,

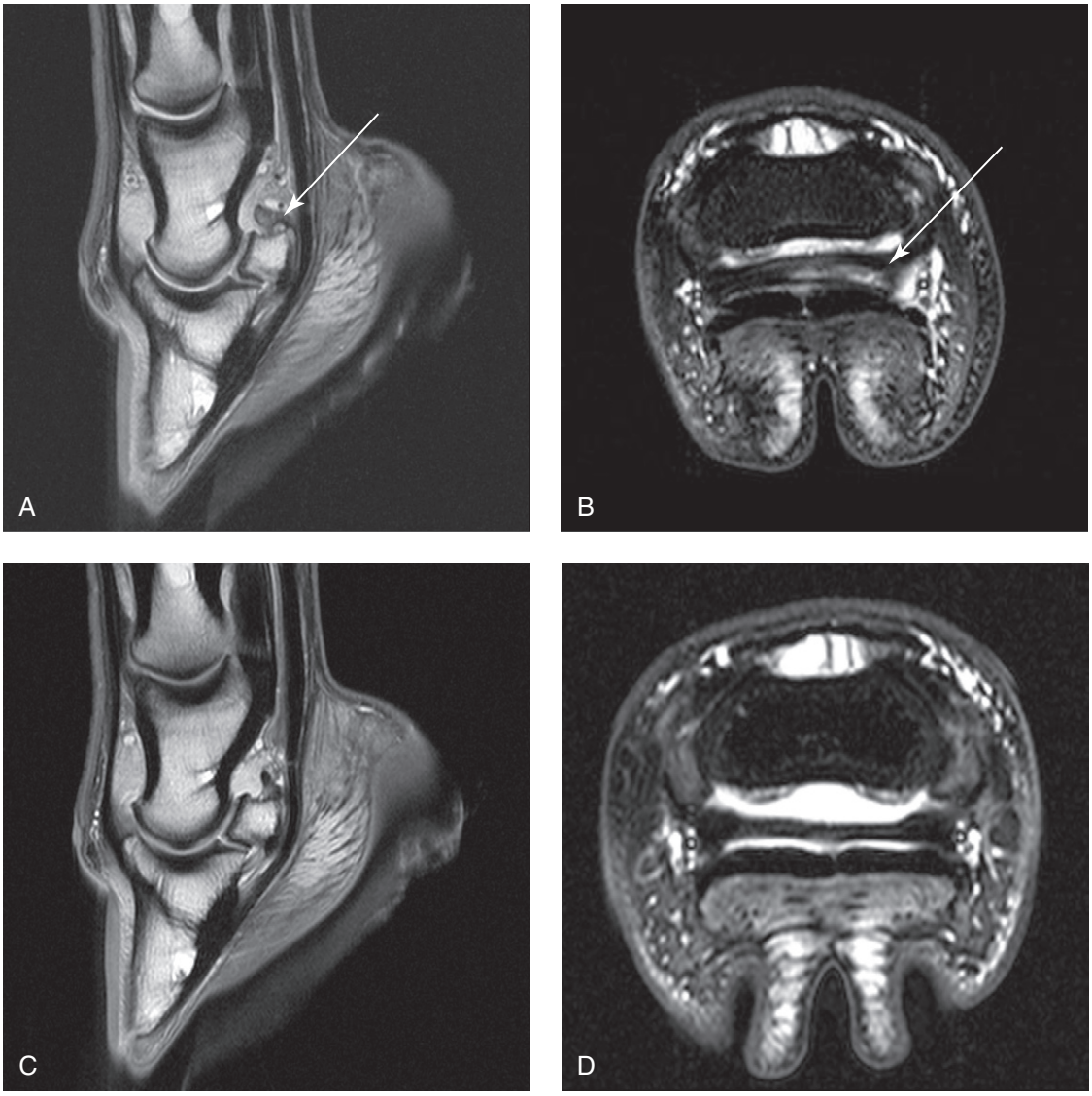

Figure 71-8. A, Sagittal proton density (PD) image of the foot. The collateral sesamoidean ligament is thickened with high signal intensity *(arrow)* close to its insertion onto the distal sesamoid bone. **B,** Transverse short tau inversion recovery (STIR) image at the level of mid second phalanx. The collateral sesamoidean ligament is thickened *(arrow)* with increased signal intensity consistent with desmopathy. Sagittal PD **(C)** and transverse STIR **(D)** images of the contralateral foot of the same horse as **A** and **B,** showing a normal collateral sesamoidean ligament. (**C** and **D,** Courtesy Dr. Russel L. Tucker, Washington State University, Pullman, WA.)

with several thin linear intermediate to high signal areas throughout it giving it a heterogeneous appearance.[44] Because of the variation between the amount of heterogeneity in the distal oblique and straight sesamoidean ligaments among horses, comparison with the contralateral limb (if normal) is important. As magic angle effect can be observed in the oblique sesamoidean ligament when using standing MRI systems, care should be taken when interpreting increased signal intensity within this ligament (see "MRI Artifacts," later).[40] Magic angle effect has not been reported in the oblique distal sesamoidean ligaments with high-field systems. However, because magic angle effect occurs in several other ligamentous and tendinous structures with both high- and low-field systems, it is likely that the same happens with the distal oblique sesamoidean ligament.

The Metacarpal and Metatarsal Region

Suspensory Ligament

As seen with ultrasonography, the appearance of the proximal aspect of the suspensory ligament on MRI is variable among horses.[45,46] This variability is attributed to the differences in the amount, pattern, and location of muscle and adipose tissue within the ligament.[46] The muscle and adipose tissue within the ligament are seen as areas of intermediate to high signal intensity and are unequally distributed between the medial and lateral lobes. In addition, the lobes of the proximal suspensory ligament are asymmetric in size and shape. Because of these variations, comparison with the contralateral limb (if normal) is essential for accurate interpretation of the proximal suspensory ligament. Injury of the suspensory ligament can be seen as

abnormal signal, enlargement and indistinct and/or irregular borders.[47] MRI allows evaluation of the abaxial margins of the suspensory ligament, which cannot be easily seen ultrasonographically. High-field MRI allows identification of adhesions between the suspensory ligament and exostoses of the second metacarpal bone.[48] In normal limbs a small space of intermediate to high signal intensity, corresponding to loose connective tissue, is present between the suspensory ligament and the second metacarpal bone. Adhesions are seen as loss of this space, which is filled with low signal intensity.[48] The loss of the normal space between the suspensory ligament and the second metacarpal bone, in conjunction with suspensory ligament changes and splint bone exostoses in a horse with chronic or recurrent lameness localized to the proximal metacarpal region, is highly suggestive of adhesions between the suspensory and the splint bone. These horses will likely benefit from surgical release of the adhesions and partial splint bone osteotomy.[48] The low signal intensity between the suspensory ligament and the second metacarpal bone should, however, be interpreted with caution because osseous proliferation and an enlarged suspensory ligament could result in a similar appearance. MRI has demonstrated that some fibers of the lateral lobe of the suspensory ligament originate from the proximal fourth metacarpal bone in normal limbs.[45] Also, compared to the medial lobe and the second metacarpal bone, the lateral lobe of the suspensory ligament is overall closer to the fourth metacarpal bone, and in some areas it contacts the bone. Therefore, adhesions between the suspensory ligament and the fourth metacarpal bone may be more difficult to assess than adhesions to the second metacarpal bone.

Accessory Ligament of the Deep Digital Flexor Tendon

Large individual variation exists in the MRI appearance of the accessory ligament of the deep digital flexor tendon. In a large percentage of horses, areas of heterogeneous signal intensity may be present.[45] Comparison with the contralateral limb is important so as not to overinterpret these areas.

Cartilage and Bone

On MRI, articular cartilage has a uniform high signal intensity adjacent to the low signal intense subchondral bone. Articular cartilage injury can be seen as loss of the normal high signal intensity, corresponding to defects or thinning. These changes are more easily identified on three-dimensional GRE sequences because they allow thin slices to be obtained without loss of resolution. It can be difficult to confidently identify cartilage lesions when no concurrent subchondral bone lesions are present. This is because the articular cartilage is thin and several artifacts can create signal changes that are not related to injury. These artifacts include motion, volume averaging, and magic angle effect. High-field MRI has moderate sensitivity in identifying full-thickness cartilage erosions in the metacarpophalangeal joint, and these lesions are often underestimated.[49]

MRI is sensitive for detecting subchondral bone sclerosis and subchondral bone osseous cystlike lesions. Osseouscystlike lesions are seen as intermediate to high signal intensity focal areas on T2, GRE, and fat-suppressed sequences (Figure 71-9).[3,4,17] These lesions may be surrounded by a low signal intensity rim.

Areas of increased signal intensity within trabecular bone are commonly designated bone bruise or bone edema. These lesions have been reported in the horse's distal sesamoid bone, proximal metacarpal region, and carpal bones and are best seen on fat-suppressed sequences (STIR and fat-saturation sequences). On histopathology of human knees with osteoarthritis, bone bruises represent bone marrow necrosis and fibrosis, edema, and hemorrhage.[50] Edema and hemorrhage represent a minor component of these lesions.

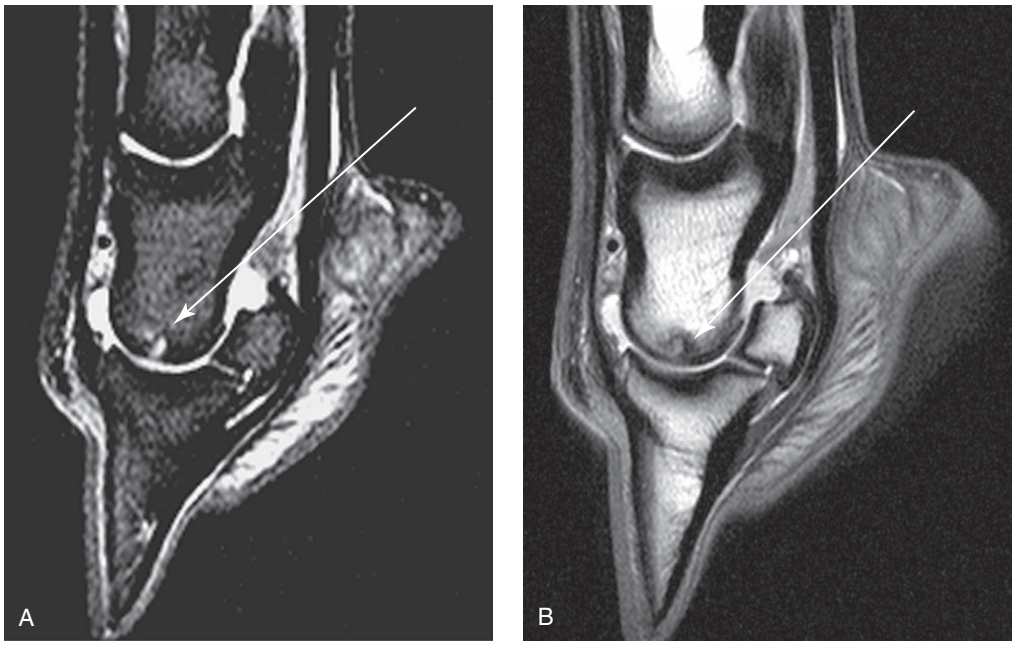

Figure 71-9. A, Sagittal short tau inversion recovery image of the foot. Note the focal high signal intensity in the subchondral bone of distal second phalanx consistent with an osseous cyst-like lesion *(arrow).* **B,** Sagittal proton density image shows a rim of hypointensity surrounding the subchondral bone lesion consistent with sclerosis *(arrow).*

The Head

MRI is the gold standard modality to image the brain in humans and animals. The use of intravenous contrast media is indicated when imaging the equine brain. The contrast material used with MRI is a gadolinium derivate. Doses of 0.02 mmol/kg in adult horses and 0.1 mmol/kg in foals of gadolinium-diethylenetriamine penta-acetic acid (gadolinium-DTPA) have been reported for imaging the brain.[10,50] However, an optimal dose of intravenous gadolinium contrast in horses has not been established. T1 images are used for evaluating contrast enhancement. Many congenital, toxic, inflammatory or infectious (including equine protozoal encephalopathy), and neoplastic processes affecting the brain can be identified with MRI.[10,51-53]

MAGNETIC RESONANCE IMAGING ARTIFACTS

Magnetic Susceptibility Artifact

To avoid magnetic susceptibility artifacts from ferrous material, prior to undergoing MRI, horses must have horseshoes and nails carefully removed and the feet should be cleaned. Because even small pieces of metal can cause a substantial artifact, a 60-degree dorsopalmar/plantar oblique radiographic projection of the distal phalanx should be obtained prior to the MRI examination to identify small fragments of nail that may be left behind after shoe removal. Also, materials that have ferrous content should not be used to pack the foot for radiography. For example, blue Play-Doh is produced using a ferrous blue dye, and it should be avoided in horses that may have subsequent MRI performed. Susceptibility artifact appears as areas of signal void (black) surrounded by high signal intensity (white), often accompanied by various degrees of anatomic distortion (Figure 71-10).[12] GRE sequences are subject to more severe susceptibility artifacts than SE sequences.[6,12]

Magic Angle Effect

It is important to recognize magic angle effect because it can mimic lesions in tendons and ligaments. In horses, magic angle effect can occur in the distal aspect of the deep digital flexor tendon and collateral ligaments of the distal interphalangeal joint in low-field and high-field systems, standing, or under general anesthesia.[33-35,38-40] Magic angle effect has also been reported in the distal sesamoidean impar ligament using a high-field system[28] and in the oblique sesamoidean ligaments with a standing system.[40] Normal tendons and ligaments, being built of tightly bound collagen fibers, have low signal intensity (black). When using MRI sequences with a short TE (T1, PD, T2*), increased signal intensity is seen within tendons and ligaments in regions where the collagen fibers are aligned at 55 ± 10 degrees with the magnet's main magnetic field (B0). This increase in signal intensity is referred to as *magic angle effect*, and it does not correspond to a pathologic change within the tendon or ligament (Figure 71-11). Magic angle effect can occur in any imaging plane, be it sagittal, dorsal, or transverse.[33,35,39] To differentiate a hyperintensity caused by a true lesion from magic angle effect, sequences with a long TE (T2) must be used. High signal intensity regions (hyperintensity) within one of the tendinous or ligamentous structures mentioned earlier, on sequences with long TE, likely represent a true lesion. The presence of other concurrent changes, such as altered size, shape, and irregular margins, can further corroborate the presence of a true lesion.

Zipper Artifact

All magnet rooms are shielded to eliminate the interference from local radiofrequency broadcasting stations or from electronic equipment that emits an electromagnetic signal that could interfere with the MRI signal. Zipper artifact is caused by leakage of electromagnetic energy into the magnet room. Leakage can be caused by electronic equipment brought into

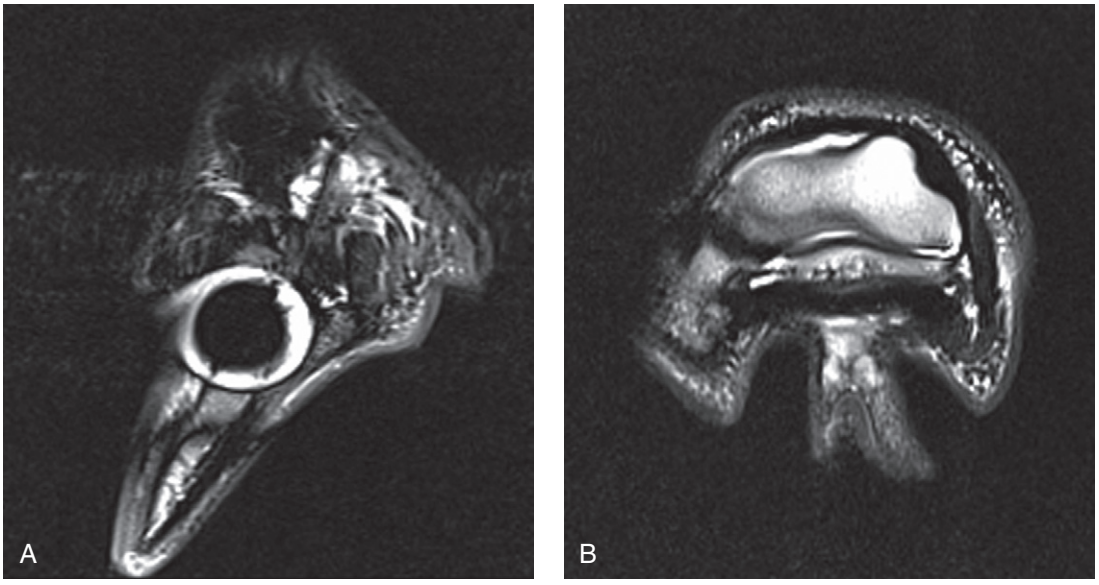

Figure 71-10. A, Sagittal short tau inversion recovery image of the foot, showing a round signal void *(black)* surrounded by a high signal intensity *(white)* rim in the region of the distal phalanx. This appearance is caused by severe magnetic susceptibility artifact because of an incompletely removed shoe nail; **B,** Transverse T2-weighted image of the same foot, at the level of the distal phalanx. Note the anatomic distortion of the distal phalanx because of a magnetic susceptibility artifact (*left side*).

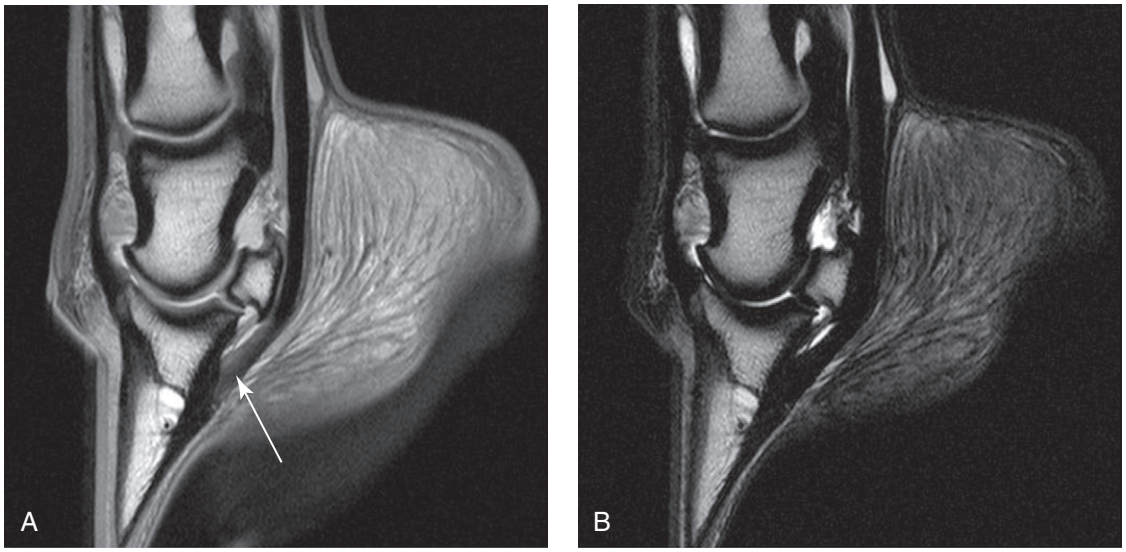

Figure 71-11. A, Sagittal proton density image of the foot. Note the increased signal intensity within the deep digital flexor tendon (DDFT) *(arrow)* distal to the distal sesamoid bone because of magic angle effect. **B,** Sagittal T2-weighted image of the same foot. Because of the long echo time of the T2 sequence, the hyperintense region in the distal DDFT is no longer seen. (Courtesy Dr. Russell L. Tucker, Washington State University, Pullman, WA.)

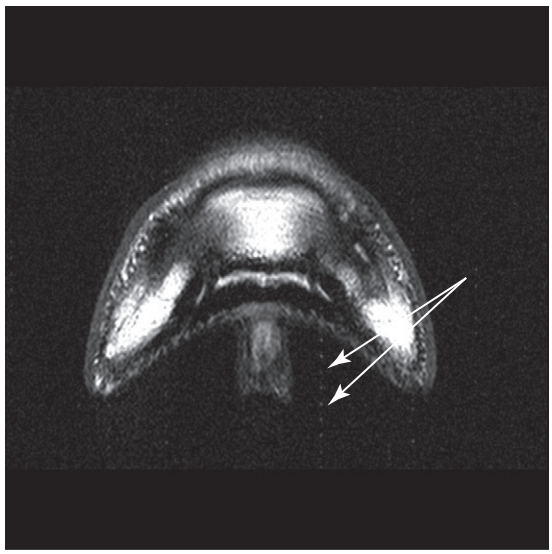

Figure 71-12. Transverse T2-weighted image of the distal third phalanx, at the insertion of the deep digital flexor tendon. Note the small (pinpoint) high signal intensity dots oriented vertically in the image because of zipper artifact *(arrows).* This artifact is caused by extraneous radiofrequencies entering the magnet's room when the room's door is opened during scanning. (Courtesy Dr. Russell L. Tucker, Washington State University, Pullman, WA.)

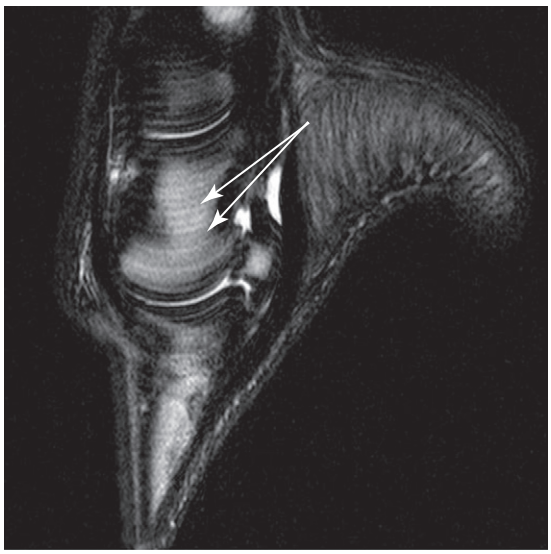

Figure 71-13. Sagittal T2-weighted image of the foot. Note the repeated hypointense curved lines *(arrows)* that repeat themselves through the first and second phalanges. These lines are caused by motion artifact. (Courtesy Dr. Russell L. Tucker, Washington State University, Pullman, WA.)

the magnet room or when the door of the room is opened while the scan is in process. This artifact appears as a region of increased noise with a width of 1 or 2 pixels that extends as lines of white and black dots in the frequency encoding direction (Figure 71-12).[6]

Motion Artifact

Motion artifact appears as blurring of the image and ghosting (the same region repeated multiple times) (Figure 71-13).[12] Motion artifact is particularly a problem with standing magnets. In horses under general anesthesia, respiratory motion can result in substantial movement of the upper limb. Respiration-related motion can be minimized by placing sandbags on top of the distal limbs.

Volume Averaging Artifact

Each pixel on an MRI represents a voxel or three-dimensional volume of tissue. Voxels can include more than one tissue type,

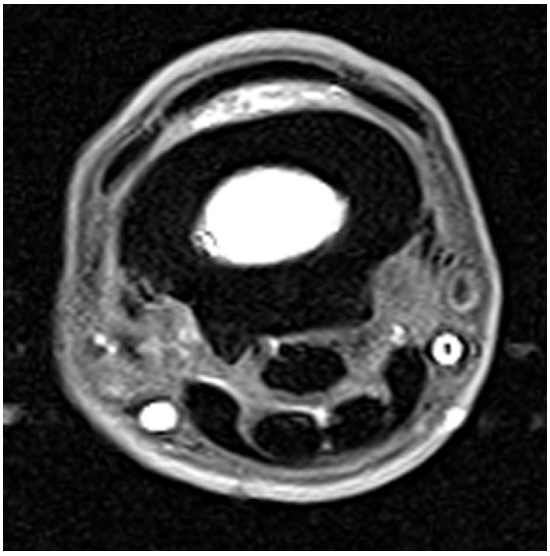

Figure 71-14. Transverse T1-weighted (T1) image of the proximal aspect of the proximal phalanx. Note the ghosting of the palmar digital veins (*arrows*) due to flow artifact.

and the intensity of the signal represents an average of those tissue types. Volume averaging should be considered at the edges of all structures. If the structure adjacent to the one being evaluated is, for example, a joint with fluid, the high signal of the fluid may average with the edge of the tendon, ligament, or bone, making it appear that a lesion is present within that structure. Volume averaging is more likely to occur with thicker slices.

Flow Artifact

Flowing blood is a specific source of motion artifact. Flow artifact is seen as ghosting of the vessel in the phase encoding direction (Figure 71-14). To remove flow artifact from an area of interest, the phase encoding direction can be changed.

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SECTION
XII

Musculoskeletal System

Jörg A. Auer

CHAPTER
72

Diagnostic Anesthesia

Anton E. Fürst

During a lameness examination, diagnostic analgesia is carried out to assist in determining of the anatomic site of lameness. It is generally used in horses that are obviously lame or that show a positive response to a flexion test or other provocation tests. A detailed discussion of diagnostic analgesia can be found in textbooks dealing mainly with lameness.[1,2] Perineural anesthesia is preferred over intra-articular anesthesia because it creates less morbidity.

TECHNICAL ASPECTS

General Considerations

Injectable solutions, needles, and syringes must be sterile. It is advisable to use new needles for each injection, and the gauge of needle should be as small as practicable, so the patient will not react adversely. Diagnostic anesthesia of the distal limb up to the carpus or tarsus is carried out with the affected limb held in a non–weight-bearing position by the clinician, if possible, whereas the limb is usually bearing weight for proximal analgesia. For temperamental horses, the limb may be held up for analgesia of the proximal regions as well.

Mechanism of Action

Pain stimuli caused by an injury are transmitted to the central nervous system by peripheral nerves, mainly thinly myelinated A delta and nonmyelinated C fibers. Local anesthetics block nerve conduction by preventing the increase in membrane permeability to sodium ions that occurs when nerves are stimulated. Local anesthetics exert their effect by inhibiting the rapid inward flow of sodium ions, thereby inhibiting depolarization of the membrane. This is achieved by the dissociation of local anesthetic, which alters the electrical forces across the membrane. The order and degree of neural blockade correlate with the diameter and type of nerve fiber and the degree of myelination. As a local anesthetic takes effect, the first nerve fibers blocked are the B fibers; the A delta and C fibers are next, and the last nerves blocked are A gamma, A beta, and A alpha.[3] Hence, sensory nerves are the first to be desensitized, followed by sympathetic and eventually motor nerves.[3] For more details on pain management see Chapter 23.

Choice of Local Anesthetic Agent

Mepivacaine is the local anesthetic agent of choice for perineural and intrasynovial analgesia.[3] Its duration of action is 2 to 3 hours, and it causes less tissue irritation than other local anesthetics. Once a bottle of local anesthetic is opened, the efficacy of the drug diminishes rapidly, so it should be used within a few days. Generally, 2% solutions (20 mg/mL) of local anesthetic are used. As a rule, because of the irritant properties of anesthetic agents, as little as is necessary to obtain anesthesia should be injected. In severely inflamed or abnormal tissues, a higher concentration or amount of local anesthetic is commonly needed to achieve the desired effect. This can occur because local anesthetics penetrate membranes less effectively in an acidotic area, such as an infected wound or joint.[3] Additionally, diffusion of the anesthetic to its intended site of action is diminished in edematous or scarred tissues, and the anesthetic effect is therefore less reliable.

Adrenalin at a low concentration is sometimes added to a local anesthetic when it is used for perineural anesthesia to induce a certain degree of vasoconstrictionin the area.[4] Although it prolonges the duration of analgesia, adrenalin also imparts a more selective analgesia. Therefore, adjacent structures including other nerves and ligaments are affected to a lesser degree.[3] However, the disadvantage of adrenalin is that severe swelling may occur 1 day later. Adrenalin may be responsible for the occurrence of skin necrosis and white hair, which are sometimes observed after diagnostic analgesia.

Perineural Anesthesia

Long or dirty hair should be trimmed with a No. 40 clipper blade, and all injection sites should be thoroughly cleaned with antiseptic soap and gauze sponges. A combination of 70% alcohol and chlorhexidine is often used for this purpose. Aseptic preparation of the skin is required before injection of synovial spaces. The clinician's hands must also be clean because they are used to palpate and locate the nerves under the skin immediately prior to injection.

Synovial Anesthesia

Anesthesia of synovial spaces is performed only after thorough cleaning of the relevant area. Hair should be clipped and the skin cleaned first with a surgical scrub, such as chlorhexidine, followed by a disinfectant as described previously. The skin should be cleaned for 5 to 10 minutes. Clipping the hair has two advantages: it aids in cleaning the skin and in identifying the injection site. However, in two studies, infection rates after intrasyovial anesthesia were not decreased by clipping or shaving the hair.[5,6]

Anesthesia of synovial spaces requires sterile gloves, needles, and syringes. Generally, the synovial space is injected using an 18-gauge (1.2-mm–diameter) or 20-gauge (0.9-mm–diameter) needle. Large-gauge needles are better for collecting synovial fluid, but horses tolerate their placement less well than smaller-gauge needles. The needle is inserted alone without the syringe attached. Depending on the temperament of the horse, a twitch may be used for restraint. This is particularly useful when needles are inserted through large muscle masses—for example, over the shoulder, elbow, and hip joints. A twitch also should be used when injecting complex joints, such as the stifle. The operator should always use a competent assistant to restrain the horse, so that sudden movements, which may result in damage to the joint cartilage or needle breakage, are prevented. When the needle is to be inserted over a large distance (shoulder or hip joint), the skin over the injection site should be anesthetized using a small amount of anesthetic and a small-gauge needle. For certain procedures, a spinal needle is used because it is flexible and less likely to break. After injection of the synovial space, the horse is walked to promote distribution of the anesthetic agent.

Needle Positioning for Intrasynovial Anesthesia

For most synovial spaces, the needle is correctly positioned when synovial fluid flows spontaneously out of the hub of the needle. Sometimes, the needle must be rotated several times until synovial fluid flows out. When this fails, aspiration of synovial fluid may be attempted, although this rarely works because the synovial villi become lodged in the tip of the needle, thereby blocking the flow. Injection of air often produces a characteristic bubbling of synovial fluid in the joint, and a larger amount of injected air sometimes results in backflow of foamy synovial fluid. Another indication of correct needle placement is minimal resistance to the injection of local anesthetic. In special cases, the joint can be radiographed after intrasynovial injection of contrast medium (see Chapter 67).

Animal Restraint

Whenever possible, horses should not be sedated for diagnostic anesthesia even though the risk of injury while working on the hind limbs is high. A twitch applied to the nose is an excellent restraining method and makes diagnostic anesthesia easier and safer. However, sedation may be necessary in intractable horses. Detomidine (0.002 mg/kg intravenously) is ideal and can be reversed.[7] Alternatively, acepromazine (0.002-0.01 mg/kg intravenously) may be used. The use of α_2-agonists for sedation is not recommended because the horse may react violently when the needle is inserted. Additionally, these drugs have two other effects that may be confusing during a lameness evaluation: they induce some analgesia and they produce ataxia.

EVALUATION OF ANESTHESIA

Testing

The clinician must determine whether diagnostic perineural anesthesia has achieved the desired effect and the nerve in question has been desensitized before re-evaluating lameness. A pointed instrument, such as a ballpoint pen, can be used to assess the sensitivity of the skin and deeper structures including tendons and ligaments. A convenient device consists of a stick approximately 1 m long, with a blunt nail positioned at one end. The nail can be conveniently placed on the region of temporarily neutralized sensory innervation of the horse to test the efficacy of analgesia without bending down. Hoof testers, joint flexion, and digital pressure (Tables 72-1 and 72-2) are also used to evaluate desesitization of the nerve or joint. Lameness can be evaluated only when the nerve or joint have been correctly desensitized.

Onset and Duration of Effect

Depending on the site, lameness should be re-evaluated 5 to 50 minutes after the injection of local anesthetic. Not waiting long enough leads to false-negative results. Local anesthetic effects begin within a few minutes after injection, but it takes variable periods of time for the horse to become sound. The first re-evaluation of lameness should always be done 5 to 10 minutes after injection. Before performing the next nerve block in the distal limb, the clinician should wait a minimum of 20 minutes, and for the proximal limb, a minimum of 45 minutes. Generally, the horse is re-evaluated 5 to 60 minutes after analgesia of a synovial space, although this time depends greatly on the joint—for example, it takes as little as 5 minutes for desensitization of small synovial spaces, such as the distal interphalangeal joint, and up to 60 minutes for the desensitization of large synovial spaces, such as the stifle joint. The goal of intrasynovial analgesia is desensitization of the nerve fibers of synovial structures. Studies show that additional structures outside the synovial space are also desensitized.[8,9] This occurs because of the close proximity of large nerves to the synovial space and the rapid diffusion of the anesthetic agent into the surrounding tissue. Therefore, the selectivity of joint analgesia comes into question; in a cadaveric study, it was shown that 15 minutes after injection of mepivacaine with an added radiodense dye into one synovial space, mepivacaine was present in adjacent synovial spaces.[10]

Interpretation

Lameness is re-evaluated after each injection of anesthetic. The horse should be walked and trotted "in hand" on a hard flat surface. All the tests (flexion, moving the horse in a tight circle, hyperflexion, extension) that were positive in the initial examination should be repeated. The horse can also be lunged on soft ground if required (see later). The response to diagnostic anesthesia can be negative, positive with residual lameness, positive, or positive with lameness in the contralateral limb. A positive response with residual lameness may be difficult to interpret. The most difficult interpretation is that of a lameness that gradually improves after each injection. It must be emphasised that an explanation must be found for each improvemet of the lameness after each anesthestic injection. The results of diagnostic anesthesia may be incorrectly assessed, yielding false-positive or false-negative results.[11,12] Negative results, in particular, must be carefully interpreted; failure to desensitize a nerve may be caused by an individual variation in the course of a nerve in some horses or by loss of efficacy of the local anesthetic agent. Furthermore, excessive diffusion of the anesthetic agent may lead to erroneous results. Certain types of lameness may become less pronounced during the evaluation, as a result of a warming-out effect (some arthritic conditions

Text continued on p. 1006

TABLE 72-1. Perineural Analgesic Blocks of the Forelimb

Number	Nomenclature	Targeted Nerves or Region	Injection Site	Gauge (G) of Needle and Amount of 2% Local Anesthetic	Testing the Analgesia	Desensitized Nerves or Region	Differential Diagnosis, Remarks
F1 (Figure 72-1)	Distal palmar digital nerve block, palmar digital block	Lateral and medial palmar digital nerves	The needle is inserted subcutanously directly over the nerve, just proximal to the cartilages of the foot	24 G, 2 mL per site	Deep pain is assessed with hoof testers; loss of skin sensation proximal to the heels	80% of the hoof and a large part of the digit	Navicular disease; disorders of the DIP and PIP joints; fissure fractures and fractures of distal, middle, or proximal phalanx; pedal osteitis; abscess in the hoof capsule; pododermatitis, laminitis, hoof cracks, tendinitis, and insertional desmitis of the deep digital flexor tendon cysts in the distal, middle, or proximal phalanx
F2 (Figure 72-1)	Proximal palmar digital nerve block, basis sesamoid block, mid pastern ring block, pastern nerve block	Lateral and medial palmar digital nerves and dorsal branches	The needle is inserted in the mid pastern region directly over the nerve and subsequently advanced farther dorsally; the nerve is easily palpated	24 G, 3-5 mL per site	Hoof testers are used to assess entire hoof; loss of skin sensation dorsal to the coronary band	100% of the hoof, the dorsal pastern region	The same as F1, but structures further dorsal are desensitzed
F3 (Figure 72-1)	Abaxial sesamoid block, mid sesamoid block	Lateral and medial palmar digital nerves and dorsal branches	The needle is inserted subcutanously directly over the nerve on the abaxial side of the proximal sesamoid bones	24 G, 3 mL per site	Loss of skin sensation over the pastern region	Same as F2 plus parts of the metacarpophalangeal joint: foot, digit, metacarpophalangeal joint, proximal sesamoid bones	Often performed in combination with other nerve blocks; otherwise rarely performed because interpretation is difficult

F4 (Figure 72-1)	Low metacarpal nerve block	Medial and lateral palmar metacarpal nerves	With the limb held up, the needle is inserted between MCIII and the distal free end of MCII and MCIV, respectively, and advanced subcutaneously, axially, and subfascially, *or* the needle is inserted just at the distal aspect of the MCII/MCIV and directed axially, where the metacarpal nerves course more superficially.	22 G, 5 mL per site	Loss of skin sensation in the MCP area	Parts of the phalangeal region and parts of the MCP joint and the proximal sesamoid bones	Predominantly performed in conjunction with other more distal nerve blocks
F3, F4 (Figure 72-1)	Low four point, abaxial sesamoid block and low palmar metacarpal nerve block, low palmar anaglesia or block, low palmar ring block, distal metacarpal nerve block	F3 and F4	F3 and F4	22 G, 5 mL per site	Forceful flexion of the IP and MCP/MTP joints; loss of skin sensation distal to the injection site	The same as F3 and F4	The same as F3 and F4
F5 (Figure 72-1)	High palmar nerve block	Lateral and medial palmar nerves	Local anesthetic is first deposited subfascially on the lateral aspect, immediately proximal to the digital flexor tendon sheath between the deep digital flexor and the suspensory ligament. The needle is then advanced and the injection is repeated on the medial aspect of the limb.	20 G, 5 mL per site	Loss of skin and tendon sensation distal to the injection site	The same as for F3 plus parts of the flexor tendon sheath, parts of the superficial and deep flexor tendons, and the suspensory ligament	

Continued

TABLE 72-1. Perineural Analgesic Blocks of the Forelimb—cont'd

Number	Nomenclature	Targeted Nerves or Region	Injection Site	Gauge (G) of Needle and Amount of 2% Local Anesthetic	Testing the Analgesia	Desensitized Nerves or Region	Differential Diagnosis, Remarks
F4, F5 (Figure 72-1)	High palmar metacarpal nerve block, mid four point, mid palmar ring block, middle metacarpal nerve block	F4 and F5	F4 and F5	20 G, 5 mL per site	Partial loss of skin sensation distal to the injections	The same as F4 and F5	The same as F4 and F5
F6 (Figures 72-1 and 72-2)	Subcarpal palmar nerve block	Lateral and medial palmar nerves	The needle is inserted between the suspensory ligament and the deep digital flexor tendon 2 cm distal to the CMC joint	20 G, 5 mL per site	Pressure applied to the flexor tendons	Parts of the flexor tendons	
F7 (Figure 72-2)	Lateral and medial palmar metacarpal nerve analgesia or infiltration of the origin of the suspensory ligament	Lateral and medial palmar metacarpal nerves	The needle is inserted axial to the splint bones just abaxial to the suspensory ligament, 2 cm distal to the CMC joint, and advanced to the palmar cortex of MCIII	20 G, 3-5 mL per site	Pressure applied to the origin of the suspensory ligament	Proximal part of the suspensory ligament, parts of the carpal joints, carpal canal, intermetacarpal ligament	Aseptic procedure is required because synovial structures (e.g., the CMC joint and the carpal canal) may be penetrated
F6, F7 (Figure 72-2)	High four point, high palmar ring block, subcarpal block, proximal metacarpal nerve block	F6 and F7	F6 and F7	20 G, 5-10 mL per site	Loss of sensation in the area distal to the injection site	The entire limb distal to the injection site and parts of the carpal joints and the carpal flexor tendon sheath	Disorders of the suspensory ligaments, metacarapal ligament, MCIII, carpal joint, carpal flexor tendon sheath, metacarpal fascia; aseptic procedure is required because synovial structures are penetrated

F8 (Figure 72-2)	Lateral palmar nerve block	Parts of the median and ulnar nerves	The needle is inserted in the distal third of the groove over the medial aspect of the accessory carpal bone until the needle contacts the bone	22 G, 3 mL	Pressure applied to the distal sesamoidean and suspensory ligaments should elicit no response	Suspensory ligament, carpal canal	Insertion desmopathy of the suspensory ligament
F9 (Figures 72-2 and 72-3)	Ulnar nerve block, ulnar block	Ulnar nerve	With the limb held up or bearing weight, the needle is inserted between the flexor carpi ulnaris and the ulnaris lateralis muscles, 5 to 10 cm proximal to the accessory carpal bone on the caudal aspect of the antebrachium, and advanced to a depth of approximately 2 cm	20 G, 10 mL	Loss of skin sensation along the lateral aspect of the carpus to the MCP joint	Parts of the suspensory ligament, carpal canal, carpal joints, deep and superficial flexor tendons	
F10 (Figure 72-3)	Median nerve block	Median nerve	Caudal to the radius, just distal to the superficial pectoral muscle, approximately 4 cm deep	20 G, 10-20 mL	Loss of skin sensation distal to the injection site	Parts of the suspensory ligament, carpal canal, carpal joints, deep and superficial flexor tendons, distal radius	
F11 (Figure 72-3)	Cutaneous nerve block, cutaneous antebrachial block	Medial cutaneous antebrachial nerve	On the cranial and caudal aspects of the accessory cephalic and cephalic veins, between the carpus and elbow	20 G, 10 mL		Only skin innervation	
F9, F10, F11 (Figure 72-3)	Triple block	Ulnar nerve, median nerve, and medial cutaneous antebrachial nerve	F9, F10, F11; see above	F9, F10, F11; see above	F9, F10, F11; see above	Loss of sensation to all parts of limb distal to the mid radius	

CMC, Carpometacarpal; *DIP*, distal interphalangeal; *IP*, interphalangeal; *MC*, metacarpal nerve; *MCP*, metacarpophalangeal; *MTP*, metatarsophalangeal; *PIP*, proximal interphalangeal.

TABLE 72-2. Perineural Analgesic Blocks of the Hind limb

Number	Nomenclature	Targeted Nerves or Region	Injection site	Gauge (G) of Needle and Amount of 2% Local Anesthetic	Testing the Analgesia	Desensitized Nerves or Region	Differential Diagnosis, Remarks
H1 (Figure 72-4)	Low plantar nerve block, plantar digital block	Digital plantar nerve	The same as F1	24 G, 6 mL	The same as F1	The same as F1	
H2 (Figure 72-4)	Mid pastern ring block, pastern nerve block	Digital plantar nerve and its dorsal branch; deep branch of the fibular nerve	The same as for F1 plus lateral and medial to the long digital extensor tendon	22 G, 16 mL	The same as F2	The same as F2	
H3 (Figure 72-4)	Abaxial sesamoid block, basis sesamoid block	Digital plantar nerve and its dorsal branch; deep branch of the fibular nerve	The same as for F1 plus lateral and medial to the long digital extensor tendon	22 G, 12 mL	Loss of skin sensation below the injection site	The same as F3	
H4 (Figure 72-4)	Low metatarsal nerve block	Medial and lateral plantar metatarsal nerves	The same as F4	22 G, 6 mL	The same as F4	The same as F4	
H5 (Figure 72-4)		Superficial and deep fibular nerves	Lateral and medial to the long digital extensor tendon In the groove between MTII and MTIII	22 G, 6 mL	Partial loss of skin sensation below the injection site	Skin below the injection site	This anesthesia is never carried out on its own
H3, H4, H5 (Figure 72-4)	Low plantar ring block, distal metatarsal nerve block	Medial and lateral plantar nerves, medial and lateral plantar metatarsal nerves, dorsal metatarsal nerves	H3, H4, H5; see above	22 G, 15-20 mL	Forceful flexion of the interphalangeal and MTP joints	All structures distal to the injection sites: hoof, interphalangeal joints, MTP joint	
H6 (Figure 72-4)	High plantar nerve block	Medial and lateral plantar metatarsal nerves	The same as F5	22 G, 12 mL	The same as F5	The same as F5	
H4, H5, H6 (Figure 72-4)	Middle plantar ring block, middle metatarsal nerve block	H4, H5, H6	H4, H5, H6; see above	22 G, 15-20 mL	Forceful flexion of the interphalangeal joints	All structures distal to the injection sites: hoof, interphalangeal joints, metatarsophalangeal joint, and parts of the tendons	
H7 (Figure 72-4)	Subtarsal nerve block	Medial and lateral plantar nerves	The needle is inserted 3 cm into the subcutaneous and subfascial tissue, distal to the tarsometatarsal joint axial to MTII and MTIV, respectively	20 G, 2 × 5 mL			

H8 (Figure 72-4)		Medial and lateral plantar metatarsal nerves	The needle is inserted 3 cm distal to the TMT joint axial to MTII and MTIV until contact is made with MTIII	20 G, 2 × 5 mL			
H5, H7, H8 (Figure 72-4)	High plantar ring block, proximal metatarsal nerve block	The same as H7 and H8	The needle is inserted 3 cm distal to the TMT joint axial to MTII and MTIV until contact is made with MTIII; the needle is withdrawn and a second depot is injected subfascially; the needle is advanced subcutaneously farther dorsally to the long digital extensor tendon	20 G, 6 × 5 mL	Forceful flexion of the interphalangeal joints; deep palpation of the tendons	All structures distal to the injection sites, hoof, interphalangeal joints, MTP joint, most of the tendons, part of the TMT joint, part of the tarsal sheath	
H9 (Figure 72-5)	Analgesia of the tibial nerve, tibial nerve block	Tibial nerve; the nerve lies in a groove between the calcaneal tendon and the deep digital flexor tendon	The needle is inserted 8 cm proximal to the calcaneus, from medial (or lateral); the anesthetic should be injected into a larger region subfascially. Caution is advised because horses can react violently to the injection. It is preferable to pick up the ipsilateral forelimb. The nerve can be palpated as a firm cordlike structure with the tarsus flexed.	20 G, 20 mL	Loss of skin sensation on the plantar surface of the tarsus	Plantar structures of the tarsus	
H10 (Figure 72-5)	Analgesia of the fibular nerve, fibular nerve block	Superficial and deep fibular nerves; fibular nerve	The needle is inserted approximately 10 cm proximal to the tarsal joint, between the long digital extensor muscle and the lateral digital extensor muscle. First, 10 mL is injected subcutanously, and then the needle is inserted slightly caudal to the tibia. Analgesia of the deep fibular nerve is not always satisfactory; the superficial nerve can be easily palpated.	20 G, 2 × 10 mL	Loss of skin sensation on the dorsal tarsus and dorsolaterally on the tarsus and metatarsus		
H11 (Figure 72-5)	Analgesia of the saphenous nerve	Saphenous nerve	Cranial and caudal to the saphenous vein, approximately 15 cm proximal to the tarsus	20 G, 2 × 10 mL	Loss of skin sensation dorsomedially on the tarsus and metatarsus		
H9, H10, H11 (Figure 72-5)	Triple block	H9, H10, H11; see above	H 9, H10, H11; see above	H9, H10, H11; see above		Entire distal hindlimb, tarsus, and distal tibial region	Useful analgesia to differentiate disorders located distal and proximal to the tarsus

MT, Metatarsal nerve; *MTP,* metatarsophalangeal; *TMT,* tarsometatarsal.

are less painful as exercise progresses and the horse is said to "warm out" of the lameness), or the lameness may be overshadowed by agitation of the patient. Sometimes, the horse must be re-evaluated the next day, and all perineural injections repeated.

PRECAUTIONS

Interpretation of the results of perineural anesthesia in horses being treated with systemic antiinflammatories and analgesics can be difficult. Therefore, diagnostic analgesia should not be performed until the effects of the systemic analgesics have worn off.

Horses with Suspected Incomplete Fractures

In severely lame horses (e.g., with tendon rupture, bone fracture), the cause can usually be diagnosed without diagnostic anesthesia, and in these cases, the area is merely radiographed or examined ultrasonographically. In selected cases, diagnostic analgesia may be carried out before or after radiography to confirm a diagnosis. However, incomplete fractures can progress into complete fractures with displacement after diagnostic anesthesia. Therefore, the horse with a suspected incomplete fracture should be walked or trotted in a straight line, in hand, for only a few strides to determine the effects of the anesthetic, if this becomes necessary.

With mild to moderate lameness, the question always arises as to whether horses should be lunged after diagnostic anesthesia. In my experience, several displaced fractures have developed in horses that were lunged after perineural anesthesia. Interestingly, the hind limbs were more often affected than the forelimbs. The bones most commonly affected are the proximal aspect of the proximal phalanx and the distal third metacarpal (MCIII) or third metatarsal (MTIII). Fractures of other bones after lunging are rare. The following general rules should be observed:

- Horses with acute lameness (up to 6 weeks) should not be lunged.
- The horse must be quiet and under the control of the examiner during lunging; otherwise, lunging should be discontinued.
- Trotting the horse in hand in a straight line is always safer than lunging in a circle.

Every diagnostic anesthetic injection proximal to the palmar digital nerve block increases the risk of fracture; therefore, if an incomplete fracture of the proximal phalanx is suspected, blocks should not be continued above this point.

Bandaging and Stall Rest

At the end of the diagnostic analgesia procedure, a bandage moistened with disinfectant solution should be applied to the leg for 12 to 24 hours. This is to prevent swelling associated with the injections in the distal limb. The horse should remain in a box stall for 24 hours. Under no circumstances should a horse be turned out after diagnostic analgesia. Horses should be rested for 3 days after intrasynovial analgesia.

COMPLICATIONS

Swelling often occurs for a few days in limbs that have had multiple perineural anesthetic injections. In these cases, the limb must be kept under bandages for several days. In rare cases, skin necrosis can develop after perineural analgesia.

In fractious horses, the needle may break and one part of it remain under the skin. Whenever possible, an attempt should be made to remove the part of the needle buried in the tissues.

Adjacent structures may be damaged; however, the risk of this is low if a correct approach and proper injection technique are applied.

Infection of synovial structures or cellulitis after perineural or intrasynovial anesthesia is very rare, particularly when aseptic technique has been followed. The risk of cartilage damage and joint infection or inflammation is also very low provided that intrasynovial analgesia is performed correctly. Therefore, it usually is unnecessary to advise the owner explicitly of these risks (in my opinion). Joint hemorrhage, which can occur after joint puncture, does not appear to have any long-term adverse effects, but it may dilute the local anesthetic and result in a false-negative test.[13]

APPLICATION

Perineural analgesia must be carried out in a systematic and logical manner. The aim is to be specific and exact, while avoiding an excessive number of nerve blocks. Generally, the procedure is begun distally, and progresses proximad. The more distal the nerve block, the more specific is the diagnostic anesthesia. Perineural anesthesia is usually applied medially and laterally at the specific location at the same time. In addition to perineural anesthesia, which usually desensitizes a single nerve (medially and laterally), ring blocks are frequently used to block the entire region distal to the anesthetized area.

Nomenclature

The names of the various diagnostic nerve blocks vary depending on the literature consulted. Because of this confusion, a simple classification system was chosen for the tables and figures in this chapter. *F* corresponds to forelimb and *H* to hind limb. Numbers indicate perineural injection of a nerve or nerve pair. When a ring block is used, it is referred to by the number of the nerve anesthetized.

Perineural Anesthesia in the Forelimb[14-19]

In the forelimb, a distal palmar digital nerve block is carried out, followed by a proximal palmar digital nerve block, a low four point (abaxial sesamoid and low palmar metacarpal nerve block), and a high four point (high palmar ring block, also termed proximal metacarapal nerve block).[8,9,12-19] These are followed by an ulnar nerve block and by desensitization of the medial and cutaneous nerves of the antebrachium. Each positive perineural anesthesia can be followed by a more-specific anesthesia, such as intrasynovial anesthesia or anesthesia of a specific nerve after the effect of the previous anesthetic has worn off. In some instances, it may be helpful to block only the medial or lateral palmar digital nerve. The detailed information for these nerve blocks is summarized in Table 72-1 and illustrated in Figures 72-1 through 72-3.

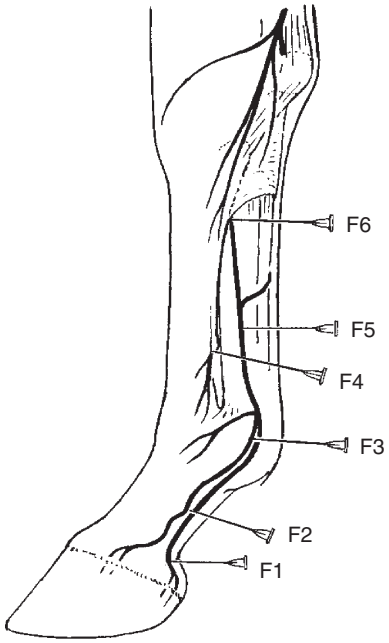

Figure 72-1. The lateral aspect of the left distal forelimb, with location of needle placement for perineural anesthesia. For an explanation of each needle position, see Table 72-1. *F1*, Distal palmar digital nerve block; *F2*, proximal palmar digital nerve block; *F3*, abaxial sesamoid block; *F4*, low metacarpal nerve block; *F5* and *F6*, high palmar nerve block.

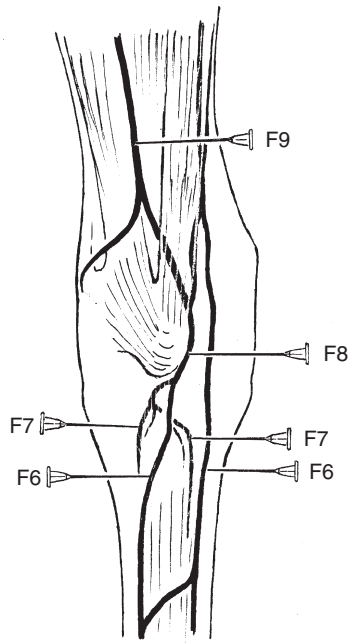

Figure 72-2. The left palmar aspect of the carpal region, with location of needle placement for perineural anesthesia. For an explanation of each needle position, see Table 72-1. *F6*, High palmar nerve block; *F7*, high metacarpal nerve block; *F8*, lateral palmar nerve block; *F9*, ulnar nerve block.

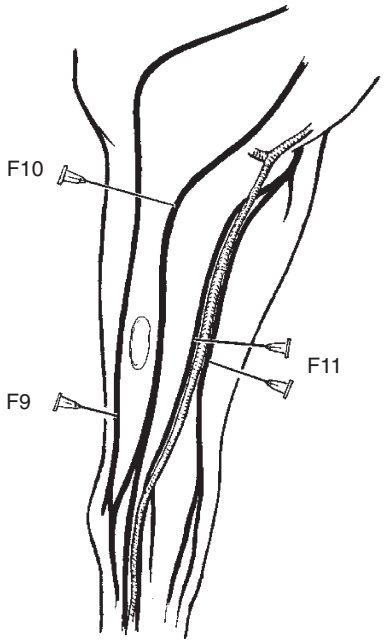

Figure 72-3. The medial aspect of the left antebrachium, with location of needle placement for perineural anesthesia. For an explanation of each needle position, see Table 72-1. *F9*, Ulnar nerve block; *F10*, median nerve block; *F11*, musculocutaneus nerve block.

Perineural Anesthesia in the Hind Limb[20,21]

Perineural anesthesia in the distal hind limb is similar to that described for the forelimb.[17] However, there are important differences in innervation, such as the dorsal metatarsal nerves, that must be taken into consideration. Safety of the examiner and horse handler is crucial, so in most cases diagnostic anesthesia is started farther proximally, provided that a lower limb problem is not suspected. In some instances, where a problem is expected distal to the metatarsophalangeal joint, a nerve block is first performed in the pastern region and then followed by a nerve or ring block proximal to the metatarsophalangeal joint (middle metatarsal ring block). These blocks are followed by a high metatarsal ring block and by fibular and tibial nerve blocks. The detailed information for these nerve blocks is summarized in Table 72-2 and illustrated in Figures 72-4 and 72-5.

Local Infiltrations

The detailed information for the different local infiltration sites is summarized in Table 72-3.

INTRASYNOVIAL ANESTHESIA

Intrasynovial Anesthesia in the Forelimb

The detailed information for the different intrasynovial injection sites in the forelimb is summarized in Table 72-4 and illustrated in Figures 72-6 through 72-9.

Intrasynovial Anesthesia in the Hind Limb

The detailed information for the different intrasynovial injection sites in the hind limb is summarized in Table 72-5 and illustrated in Figures 72-10 through 72-14.

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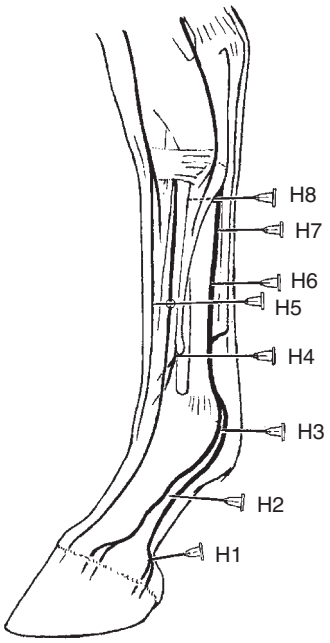

Figure 72-4. The lateral aspect of the left distal hind limb, with location of needle placement for perineural anesthesia. For an explanation of each needle position, see Table 72-2. *H1*, Low plantar digital nerve block; *H2*, high plantar digital nerve block; *H3*, abaxial sesamoid block; *H4*, low metatarsal nerve block; *H5*, mid third metatarsal fibularis nerve block; *H6* and *H7*, high plantar nerve block; *H8*, high metatarsal nerve block.

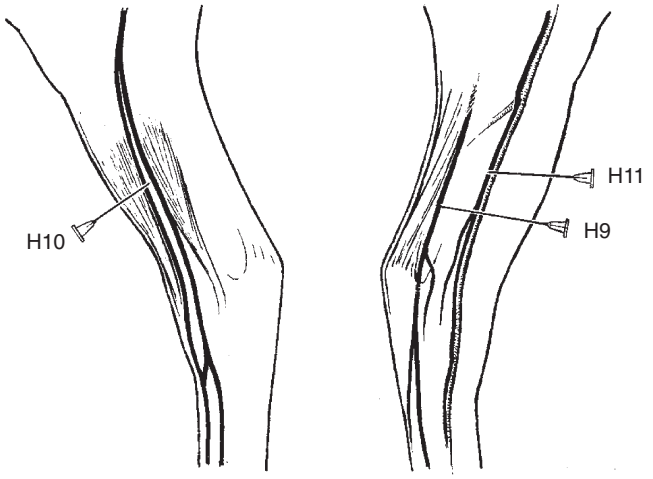

Figure 72-5. The lateral and medial aspects of the left hind limb, with location of the needle end point for perineural anesthesia. For an explanation of the needle position, see Table 72-2. *H9*, Tibial nerve block; *H10*, fibularis nerve block; *H11*, saphenous nerve block.

TABLE 72-3. Local Infiltrations

Nomenclature	Targeted Nerves or Region	Injection Site	Needle Gauge (G) and Amount of 2% Local Anesthetic	Testing the Analgesia	Desensitized Nerves or Region	Differential Diagnosis, Remarks
Analgesia of the interspinal ligament	Dorsal spinous process	The needle is inserted along the dorsal midline and directed ventrally into the interspinous space; the interspinous space can be palpated as a depression	20 G, 9 cm, 5-10 mL for interspinal space		Dorsal spinous processes, interspinal ligament	The horse must be lunged or ridden to allow judgment
Analgesia of the splint bones	Splints	The needle is inserted along the proliferative lesion between the skin and bone	20 G, 5-10 mL	Local palpation	Region around the bony proliferation	Splints, tissues distal to the infiltration

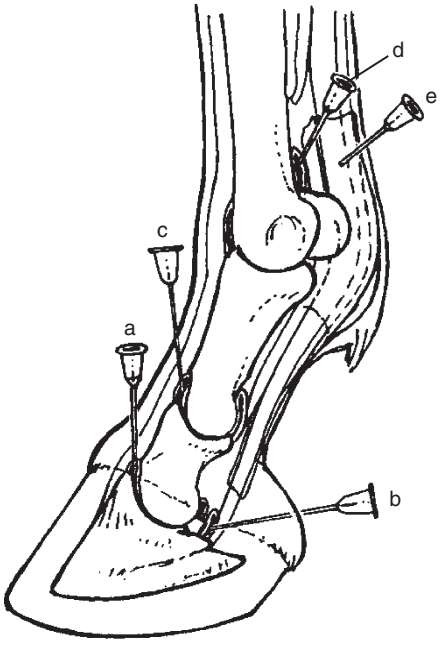

Figure 72-6. Intrasynovial injection sites in the phalangeal region. *a*, Distal interphalangeal joint; *b*, distal sesamoidean (navicular) bursa; *c*, proximal interphalangeal joint; *d*, metacarpophalangeal joint; *e*, digital flexor tendon sheath.

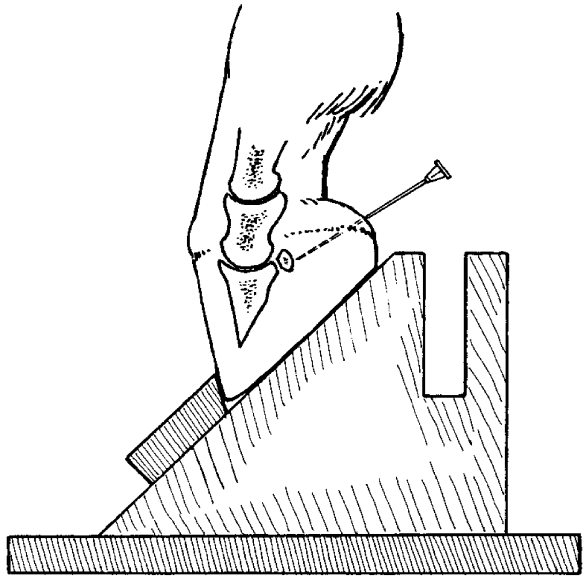

Figure 72-7. Demonstration of limb position using a foot block to facilitate injection of the distal sesamoidean (navicular) bursa.

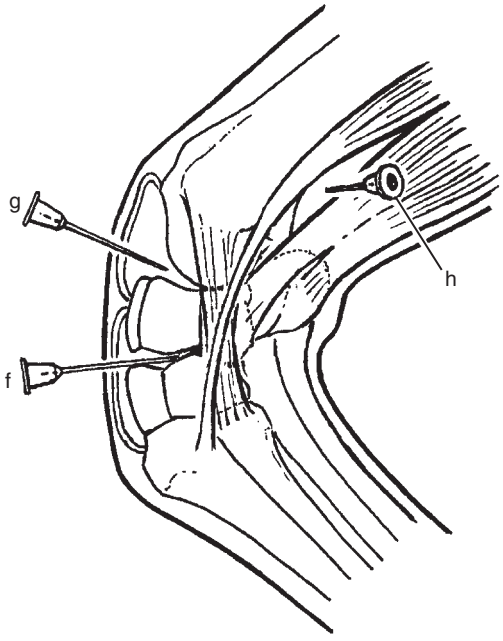

Figure 72-8. Intrasynovial injection sites in the carpal region. *f*, Middle carpal joint; *g*, antebrachiocarpal joint; *h*, carpal flexor tendon sheath.

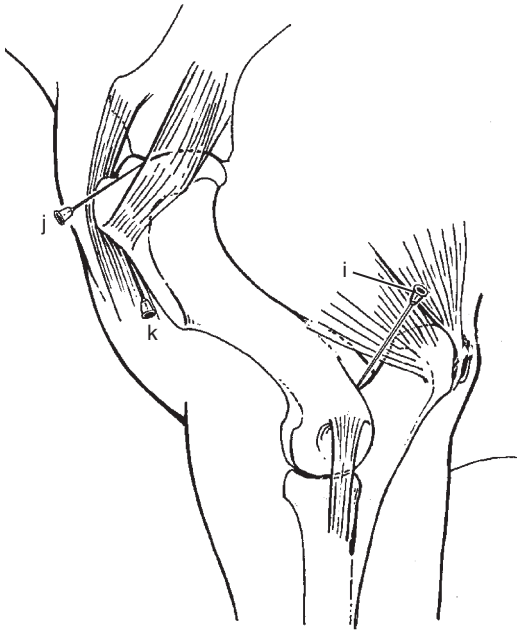

Figure 72-9. Intrasynovial injection sites in the upper forelimb. *i*, Elbow joint; *j*, shoulder joint; *k*, bicipital bursa.

TABLE 72-4. Intrasynovial Analgesia in the Forelimb

Name of the Joint, Bursa, or Tendon Sheath	Location for Injection	Alternative Approach	Technique	Needle Gauge (G) and Amount of Local Anesthetic	Onset of Effect	Important Anatomic Structures that Are Often Partly or Completely Anesthetized
Distal interphalangeal joint	1 cm dorsal to the coronary band, abaxial from, or through, the common digial extensor tendon, horizontally or slightly distad to the hoof capsule (Figure 72-6, *a*)	Lateral or palmar approach to the distal interphalangeal joint	The limb is held up and flexed or the limb may be weight bearing	20 G, 5-10 mL	5-10 min	Proximal interphalangeal joint, navicular bone and navicular bursa, insertion of the deep digital flexor tendon
Navicular bursa	Palmar midline approach (Figures 72-6, *b*, and 72-7)	Proximal palmar injection technique, lateral or medial approach	The hoof is positioned in the Hickman block and thus slightly flexed. The needle is inserted through the palmar midline, just proximal to the coronary band, and directed toward the distal sesamoid bone, which is located 1 to 2 cm distal to and halfway between the dorsal and palmar aspects of the coronary band	20 G, spinal needle, 9 cm long, 3-5 mL	5-15 min	Distal sesamoid bone, parts of the hoof caspule, insertion of the deep digital flexor tendon
Proximal interphalangeal joint	The needle is inserted 1 cm proximal to the joint space in the midline, or slightly paramedian, and directed horizontally or slightly distad into the joint space (Figure 72-6, *c*)	Proximal palmar pouch	The limb is held up and flexed, or the limb may be weight bearing	20 G, 5-10 mL	5-15 min	
Metacarpophalangeal joint	Proximopalmar or proximoplantar pouch (Figure 72-6, *d*)	Dorsal pouch, distopalmar pouch	With the limb held up and flexed, the needle is inserted immediately proximal to the condyle of MCIII between MCIII and the lateral branch of the suspensory ligamant and directed distally	20 G, 10-15 mL	10-20 min	Proximal sesamoid bones, branch of the suspensory ligament

Digital flexor tendon sheath	Outpouching of the sheath proximal to the palmar annular ligament (Figure 72-6, *e*)	Between the proximal and distal digital annular ligaments; distal to the proximal sesamoid bones, between the palmar annular ligament and the proximal digital annular ligament	With the limb held up and flexed, the needle is inserted directly into the outpouching	20 G, 10-20 mL	10-20 min	Distal and proximal interphalangeal joints, metacarpophalangeal joint, navicular bursa
Middle carpal joint	Lateral or medial to the extensor carpi radialis tendon (Figure 72-8, *f*)	Palmar pouch of the middle carpal joint	With the limb held up and flexed, the needle is inserted in a palmar direction into the joint	20 G, 10-15 mL	10-20 min	Middle carpal joint, origin of suspensory ligament, palmar metacarpal nerves
Antebrachiocarpal joint	Lateral or medial to the extensor carpi radialis tendon (Figure 72-8, *g*)	Palmarolateral pouch of the antebrachiocarpal joint	With the limb held up and flexed, the needle is inserted in a palmar direction approximately 2 cm into the joint	20 G, 10-15 mL	10-20 min	Antebrachiocarpal joint
Carpal flexor tendon sheath	Proximal aspect of the carpal sheath (Figure 72-8, *h*)	Distal aspect of the carpal sheath	The needle is inserted proximal to the accessory carpal bone between the lateral digital extensor and ulnaris lateralis tendons	20 G, 10-20 mL	10-20 min	Origin of the suspensory ligament
Elbow joint	Proximolateral aspect of the caudal pouch (Figure 72-9, *i*)	Cranial pouch	With the limb bearing weight, the needle is inserted 3 to 4 cm caudal to the lateral epicondyle and advanced 4 to 8 cm in a distal, slightly cranial and medial direction	20 G, 10-20 mL	20-40 min	
Shoulder joint	Lateral pouch of the joint (Figure 72-11, *j*)	No alternatives	The needle is inserted cranial to the infraspinatus tendon in the notch between the cranial and caudal eminences of the greater tubercle of the humerus, in a caudomedial direction (about 45 degrees) and slightly distad	20 G, spinal needle, 9 cm long, 10-20 mL	20-40 min	Bicipital bursa
Bicipital bursa	Lateral pouch of the bursa (Figure 72-11, *k*)	Proximal approach	The needle is inserted 3 to 4 cm proximal to the deltoid tuberosity, cranial to the humerus, and is directed proximad, mediad, and slightly craniad	20 G, spinal needle, 9 cm long, 10-20 mL	20-40 min	Bicipital bursa

MC, Metacarpal nerve.

TABLE 72-5. Intrasynovial Analgesia in the Hindlimb

Name of the Joint, Bursa, or Tendon Sheath	Location for Injection	Alternative Approach
Tarsometatarsal joint	Lateral approach (Figure 72-11, *l*)	
Distal intertarsal joint	Medial approach (Figure 72-10, *m*)	
Tarsocrural joint	Dorsomedial outpouching (Figure 72-10, *n*)	Dorsolateral and plantar pouches
Cuneal bursa	Medial approach (Figure 72-10, *o*)	
Calcaneal subtendinous bursa	Medial or lateral approach (Figure 72-11, *p*)	
Tarsal sheath	Medial or lateral approach (Figure 72-11, *q*)	
Femoropatellar joint	Craniomedial or craniolateral approach (Figure 72-12, *r*)	Lateral approach, subpatellar site in a flexed position
Medial compartment of the femorotibial joint	Medial approach (Figure 72-12, *s*)	Cranial approach
Lateral compartment of the femorotibial joint	Lateral approach (Figure 72-12, *t*)	Cranial approach
Coxofemoral joint	Lateral approach (Figure 72-13, *u*)	
Sacroiliac joint (Periarticular infiltration)	Medial approach; middle or caudal third of the sacroiliac joint (Figure 72-14, *v*)	Cranial third of the sacroiliac joint

MT, Metatarsal nerve.

Technique	Needle Gauge (G) and Amount of Local Anesthetic Agent	Onset of Effect	Important Anatomic Structures that Are Often Partly or Completely Anesthetized
The needle is inserted just proximal to MTIV, at the level of the palpable depression, in a craniomedial and distal direction	20 G, 3-6 mL	10-20 min	Origin of suspensory ligament, proximal aspect of MTIII, tarsometatarsal joint
The needle is inserted horizontally between the fused first and second tarsal bones and the third and central tarsal bones in a small depression just distal to the cunean tendon; this is halfway between the talus and the head of MTIV	22 G, 3-6 mL	10-20 min	Distal intertarsal joint
Dorsolateral approach: 2 to 3 mm lateral to the long digital extensor tendon 6 to 8 mm proximal from the head of the splint bones or 1 cm distal to the lateral trochlea, 70 degrees to the sagittal plane in plantaromedial direction			
Medial (or lateral) to the saphenous vein	20 G, 20-30 mL	20 min	Proximal intertarsal joint
The needle is inserted between the distal tarsal bones and the medial branch of the cranial tibial tendon at the distal edge of the tendon	22 G, 3-6 mL	20 min	
The needle is inserted proximal or distal to the tuber calcanei in the lateral or medial outpouching	20 G, 10 mL	15-30 min	
The needle is inserted in the proximal or distal outpouching of the tarsal sheath	20 G, 20 mL	15-30 min	
The needle is inserted either lateral or medial to the middle patellar ligament until the needle tip contacts the articular cartilage of the distal femur; the needle is then withdrawn slightly	20 G, 20 mL	20-60 min	Lateral and medial femorotibial joint
Alternative: Recessus subextensorius			
The needle is inserted just caudal to the medial patellar ligament, 1 to 2 cm proximal to the medial tibial plateau, perpendicular to the skin	20 G, 20 mL	20-40 min	Lateral femorotibial joint, femoropateral joint
The needle is inserted caudal to the long digital extensor tendon, 1 to 2 cm proximal to the lateral tibial plateau, horizontally and slightly cranially	20 G, 20 mL	20-40 min	Medial femorotibial joint, femoropateral joint
The spinal needle is inserted between the caudal and short cranial processes of the greater trochanter of the femur in a slightly craniomedial direction and slightly distally until the joint capsule is penetrated.	Spinal needle, 20-30 mL	20-40 min	
The needle is inserted 2 cm cranial to the opposite sacral tuberosity and directed to the midpoint of the distance between the cranial aspect of the tuber coxae and the greater trochanter	Spinal needle, 15-18 G, 20-25 cm, 20-30 mL	20-40 min	

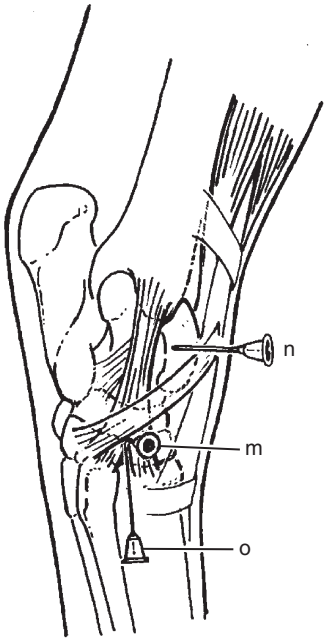

Figure 72-10. Intrasynovial injection sites in the medial tarsal region. *m*, Distal intertarsal joint (medial approach); *n*, tarsocrural joint; *o*, cunean bursa.

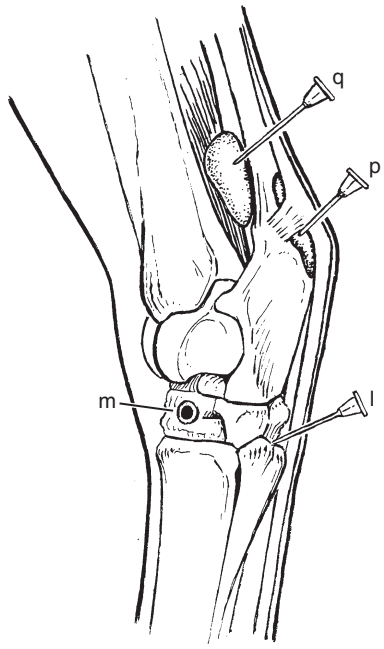

Figure 72-11. Intrasynovial injection sites in the lateral tarsal region. *l*, Tarsometatarsal joint; *m*, distal intertarsal joint (dorsolateral approach); *p*, calcaneal bursa; *q*, tendon sheath of long digital extensor (muscle).

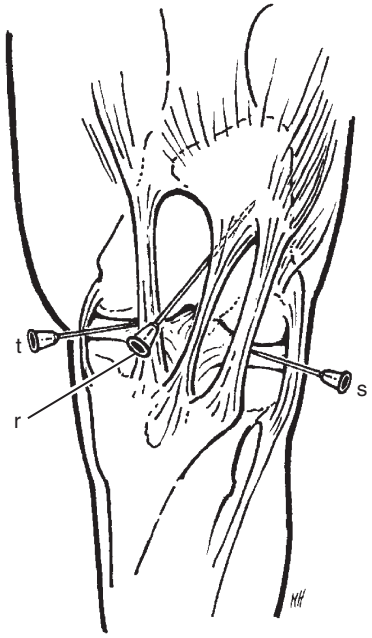

Figure 72-12. Intrasynovial injection sites in the stifle region. *r*, Femoropatellar joint; *s*, lateral femorotibial joint; *t*, medial femorotibial joint.

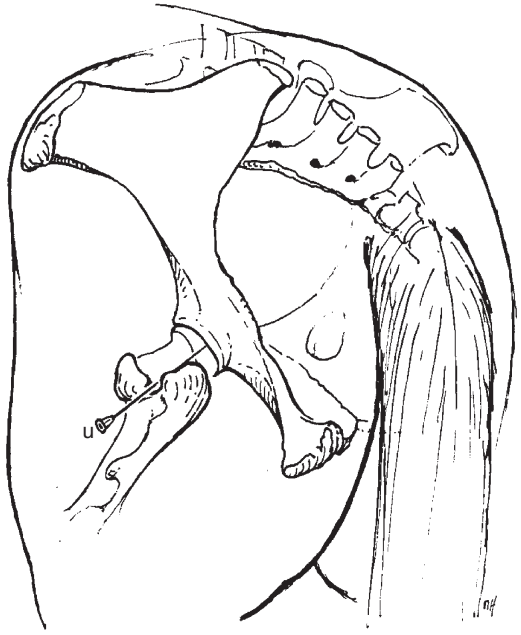

Figure 72-13. Intrasynovial injection site of the coxofemoral joint (*u*).

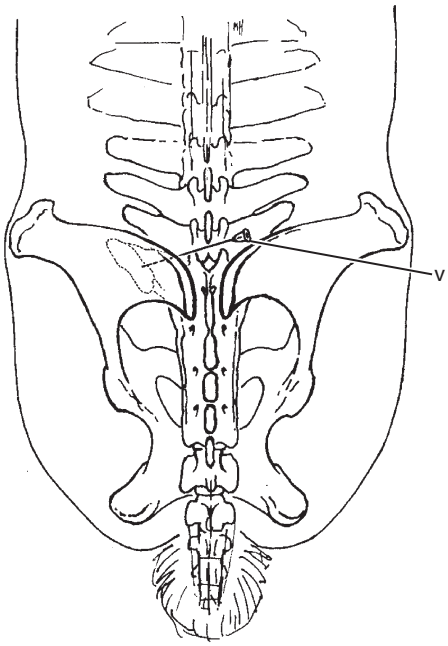

Figure 72-14. Intrasynovial injection site of the sacroiliac joint (*v*).

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CHAPTER
73

Emergency Treatment and Transportation of Equine Fracture Patients

Anton E. Fürst

In horses, the initial treatment of injuries to skin, muscles, joints, tendons, tendon sheaths, and bones greatly affects the chances for ideal healing. Especially for long-bone fractures, a successful surgical outcome is forfeited with improper initial care. Unfortunately, most fractures are exposed to substantial additional trauma during the transportation of the injured horse. In certain situations, it is necessary to use a rescue net and crane or even a helicopter to rescue a horse from a dangerous location. A large-animal rescue unit operating in Switzerland and in neighboring countries specializes in the rescue of large animals involved in automobile accidents or trapped in wells, ditches, gullies, lakes, or rivers.[1]

Rescue efforts can be conducted with the animal standing or in lateral or dorsal recumbency. Rescuing horses is a very complex procedure that must be well planned and calmly executed. A veterinarian experienced with horses should be on hand to prepare and accompany the horse during the rescue operation. Additionally, sedation or short-term anesthesia may be required in special situations. Although every rescue operation must be carried out as quickly as possible, time is of secondary importance. In emergency situations, it is imperative to remember that the horse may be in pain and that hastily improvised rescue methods will probably be inadequate and result in additional trauma. For example, narrow, flat belly bands are painful for the horse and usually fail because the animal slips out of the bands and cannot be rescued or falls out while being lifted, resulting in severe additional injury or even death.

EXAMINATION OF THE PATIENT

A thorough clinical examination is mandatory for horses with fractures.[2] Lacerations or lesions that are overlooked can affect the diagnosis and prognosis considerably. A fracture should be suspected with severe lameness and an acute onset of signs. When in doubt, the horse should be treated and handled as a fracture patient. All too often, the assessment of injuries underestimates the extent of the injury, and incomplete and nondisplaced fractures initially can be overlooked. This is particularly true in horses that have been kicked in a region where the bone lies directly beneath the skin—such areas include the scapular spine, the major tubercle of the humerus, the deltoid tuberosity, the cutaneous plane of the radius, the metacarpus, the metatarsus, the tuber coxae, the third trochanter, the sustentaculum of the talus, the calcaneus, and the tibia (Figure 73-1).[3]

TREATMENT OR EUTHANASIA

Despite great advances in veterinary orthopedic surgery and anesthesia, there are several injuries and fractures in horses that cannot be treated successfully. When the prognosis is hopeless, euthanasia should be suggested to the owner. A smooth and quiet euthanasia is of utmost importance for the owner and for potential spectators of the procedure.[4] In all cases, a veterinarian should examine the horse thoroughly before carrying out the euthanasia.

Conversely, the number of injuries and fractures that have a good prognosis with state-of-the-art treatment is increasing. Good-quality radiographs are necessary to establish an exact diagnosis and to arrive at a prognosis. When the decision has been made to treat the horse, adequate emergency treatment is the initial step, and the horse is then referred to a hospital that specializes in managing such problems.[5] It is imperative that the patient be given the opportunity to receive optimal care and treatment. However, frequently, horses with fractures are transported without adequate emergency treatment, which not only compromises or eliminates the chance of a successful surgical repair but also is extremely painful for the horse (Figure 73-2).

OPTIMAL EMERGENCY TREATMENT

Optimal emergency treatment should include the following steps: sedation and possibly anesthesia, initial wound management, stabilization of the fracture, administration of proper analgesia and anti-inflammatory medications, prophylaxis for infection, intravenous fluid therapy, and careful and safe rescue with proper transportation to a specialty clinic.

Sedation

The use of sedation is dictated by the circumstances presented. What is indicated for one horse may be hazardous for another. The use of sedatives depends on the fracture and, in particular, the type and character of the horse. In most cases, judicious use of a sedative facilitates the examination and emergency treatment of an acutely traumatized horse, especially when the horse is stressed from competing in an event or is in severe pain. The sudden inability to place weight on a limb as a result of a

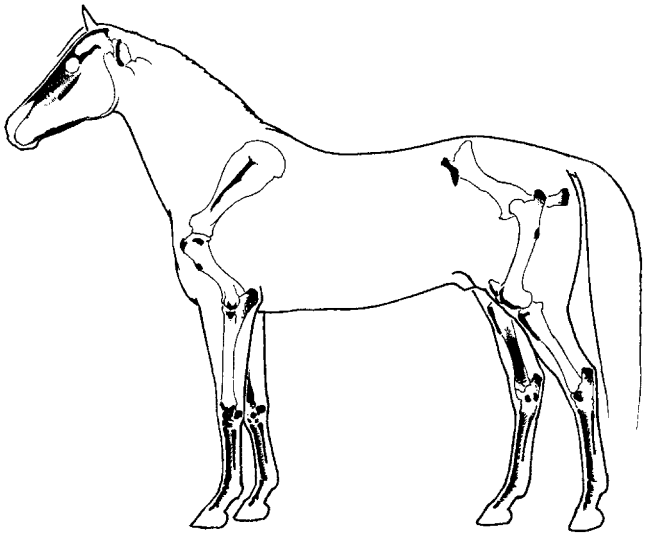

Figure 73-1. Sites on the horse (*shaded areas*) where bones are covered only by skin with no muscle protection, making them susceptible to open fractures when kicked by another horse.

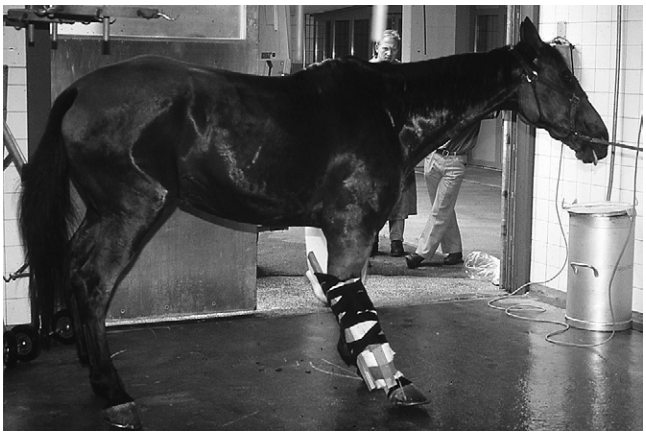

Figure 73-2. Inadequate attempt at stabilization of a radial fracture using a splint that is too short. The fracture remains unstable and the limb is now weighted by the inadequate splint bandage, which compounds the bone and soft tissue trauma. The patient is sweating profusely—a clear sign of pain.

long-bone fracture causes anxiety, which may lead to sudden violent reactions. A twitch can be useful to control a fractious or anxious horse, avoiding the ataxia that accompanies sedation.[2] Furthermore, horses do not tolerate external coaptation well, especially when the fixation extends above the carpus or tarsus. Therefore, the use of a sedative may be necessary to induce acceptance of the external coaptation device. α_2-Agonists are the drugs of choice, because they have few side effects and provide some analgesia and reliable sedation. Xylazine HCl is suitable for short procedures and detomidine HCl is used for longer sedation, which is necessary in most cases. For additional sedation and analgesia, a morphine derivative should be administered as well. Butorphanol tartrate has a wide variety of applications in horses and is an ideal drug for this scenario.

The dosage of these drugs varies according to the type of emergency. Agitated horses usually respond poorly to sedatives and require a higher dosage than normal. In contrast, a lower dose is needed when the horse's condition is compromised by shock or severe blood loss. After intravenous injection of the sedative, a minimum of 5 minutes is required to determine the effects of the sedation. This time period may seem quite long in an emergency situation, but it is prudent to wait without administering additional drugs to avoid an overdosage that could induce an unacceptable degree of ataxia or compromise the cardiovascular status of the patient. It should be remembered that cold-blooded horses require less sedative on a per-weight basis, and the dosage should not exceed that used for a 550-kg horse. Foals usually require heavy sedation to facilitate good stabilization of a fracture, and it is advantageous and often necessary to have them restrained in lateral recumbency when a splint is applied.

I favor a combination of detomidine HCl (Dormosedan) and butorphanol tartrate (Torbugesic or Morphasol). The two drugs can be combined in one syringe and given intravenously, or intramuscularly if the intravenous administration is not possible. A suggested dosage for a 400- to 500-kg horse would be 5 mg detomidine combined with 10 mg of butorphanol tartrate, intravenously. Xylazine can be used instead of detomidine. It leads to more rapid but less reliable sedation. The addition of a neuroleptic agent, such as acepromazine (0.02 mg/kg), can substantially increase the duration of sedation. However, acepromazine should not be used in stallions or physiologically severely compromised patients because of its vasodilatory effects which could compound neurogenic shock. Additional information on chemical restraint can be found in Chapter 22.

Anesthesia

For certain rescue procedures, short-term anesthesia may be necessary. Various drug combinations, such as xylazine-diazepam-ketamine or xylazine-myolaxin-ketamine, provide reliable anesthesia even in an emergency situation. (See Chapter 20 for more on anesthesia.)

Wound Management

Skin wounds must be treated with care. The hair around the wound is removed after covering the wound with water-soluble ointment. Then, a general cleaning of the area around the wound is completed with mild soap and water. The wound itself is cleaned, disinfected and covered with a water-soluble antibiotic ointment and sterile dressing, followed by a bandage. With open fractures, the bone must be cleaned also and covered with a sterile dressing.

Fracture Stabilization

Stabilization of the fractured limb in an anatomically normal position is the most important aspect of the initial treatment. This allows the patient to bear some weight without excessive damage to the fracture ends and the soft tissues. A very important additional effect of fracture stabilization is the attenuation of anxiety and pain for the patient, thereby preventing further compromise of the patient.

Goals

The goals of fracture stabilization include:

Reduction of pain and anxiety and facilitation of partial weight-bearing. Horses attempt to move a fractured limb constantly in an effort to find a stable position. Pain from this movement and the instability is extremely stressful for the fracture patient, and they often panic. Prompt and effective stabilization of the fracture by itself substantially improves the general well-being of the animal. Therefore, the fractured limb should be stabilized so that the horse is able to bear some weight on it [6-9] The concomitant reduction of pain renders the patient in better physiological condition for referral, and surgical repair can be attempted sooner.

Prevention of further compromise of the patient. Because horses cannot balance well on three limbs and repeatedly attempt to use the fractured limb, especially during transport, the continued movement of the fractured bones not only traumatizes the fragment ends, thus preventing anatomic reconstruction of the bone, but also injures the soft tissues, the integrity of which is critical for fracture healing. The skin of horses is quite thin, and the worst complication is its perforation by sharp bone fragments, resulting in contamination of the bone ends. Horses with open fractures of long bones have a very poor prognosis, and the use of modern and expensive antibiotics does not change this situation significantly. Therefore, all efforts must be made to prevent the conversion of a closed fracture to an open one.which occurs quite readily in the distal region of the limb and the medial aspects of the radius and tibia, because there is little tissue between the bone and the skin.

Immobilization of the adjacent joints. The joints above and below the fractured bone should be immobilized using some type of external coaptation, and the stabilization should extend well beyond the fracture line. In no case should the end of the coaptation device be near the fracture line, because then it acts as a lever to further displace the fracture. A cast should not end at the mid-diaphysis, and when possible it should include the hoof. These requirements severely limit the number of fractures that can be stabilized with external coaptation techniques.

Types of Stabilization

For emergency stabilization of fractures, robust materials must be used. Detailed information on bandaging and external

coaptation materials and their application can be found in Chapter 17.

ROBERT JONES BANDAGE

A Robert Jones bandage consists of many layers of cotton, each held in place and tightened by elastic gauze. Each layer is applied more tightly than the previous one. The finished Robert Jones bandage for a normal-size horse requires 10 to 15 rolls of cotton, and its diameter is about three times the diameter of the limb. This type of bandage cannot be used to stabilize a fractured limb for long without the addition of splints. When splints are added, the bandage does not have to be as thick.

SPLINTS

A variety of splints are suitable for stabilizing fractures in horses. The splints must be applied to the cranial or caudal and lateral aspects of the limb and held in place with nonelastic tape. For optimal stabilization, the splints are applied in two planes at right angles (90 degrees) to each other. Sufficient padding and good fixation are necessary to prevent slippage of the splint.

Polyvinyl chloride (PVC) splints are very stable and inexpensive, but they are difficult to mold unless heated using a propane torch. The commercially available Fraktomed splint (Fraktomedschiene), made of a polyvinyl alcohol polymer, is also suitable for immobilizing a fracture, but it is rarely available in an emergency situation. This type of splint is very rigid when cold and must be heated in steam to facilitate molding it to fit the limb. Within a few minutes, the splint hardens and becomes stable. Splints can also be improvised using metal rods, broom handles, wooden boards, and other sturdy materials. The Thomas splint is generally unsuitable for horses and should not be used to splint a fractured limb, especially in an emergency situation.

CAST

The equine cast, also referred to as a *synthetic splint*, is formed from fiberglass tape impregnated with a polyurethane resin. These materials are very well suited for immobilizing equine limbs. They are very strong, cure quickly, are easy to apply, and are very light. While the cast is being applied, the horse should stand quietly to prevent the development of microfractures and folds in the cast, which can reduce its strength and cause pressure sores. Preventing movement of the cast during application can be better achieved if the horse can be supported in a special harness. In this way, the fractured limb can be stabilized in the correct position (Figure 73-3). In some situations, especially in very unstable fractures, this may be difficult, so splints are usually the first choice for stabilization. To prevent penetration of the skin by sharp bones, a window can be made in the cast over these locations (Figure 73-4).

Figure 73-3. A horse with a fractured radius is supported in a special harness. This way the fractured radius can be stabilised in the correct position, and the cast is not subjected to movement during application, which may weaken it.

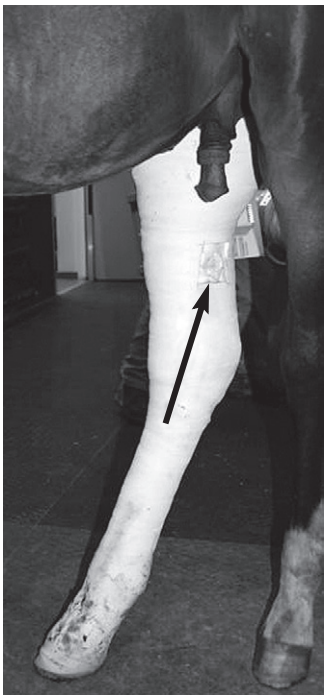

Figure 73-4. To prevent penetration of the skin by sharp ends of fractured bones, a window was made in the cast over the distal end of the proximal tibial fragment *(arrow)* in this horse.

Principles of Stabilization

PREVENTION OF SOFT TISSUE DAMAGE

Good stabilization does not inflict additional damage to the limb. Because swelling of the surrounding tissues is common after an injury, stabilization may result in pressure and friction sores as well as tissue strangulation if the splint or cast is not sufficiently padded. Therefore, the padding should be layered, with each layer being tightened with nonadhesive gauze. However, layers that are too thick (2 to 4 cm) allow movement of the bone fragments or slippage of the splint, and should be

avoided. Ideally, the thickness of each layer of the padding should be about 1 to 2 cm.

REGIONAL IMMOBILIZATION

Techniques for immobilization of equine long-bone fractures have been established.[7-9] The limbs can be divided into four distinct regions to establish principles for stabilization techniques (Figure 73-5).[8] The regions for the forelimb are (1) the hoof to the distal metacarpus, (2) the distal metacarpus to the distal radius, (3) the distal radius to the elbow joint, and (4) the elbow joint to the distal scapula. The regions for the hindlimb are (1) the hoof to the distal metatarsus, (2) the distal metatarsus to the tarsus, (3) the tarsus to the stifle, and (4) the region proximal to the stifle.

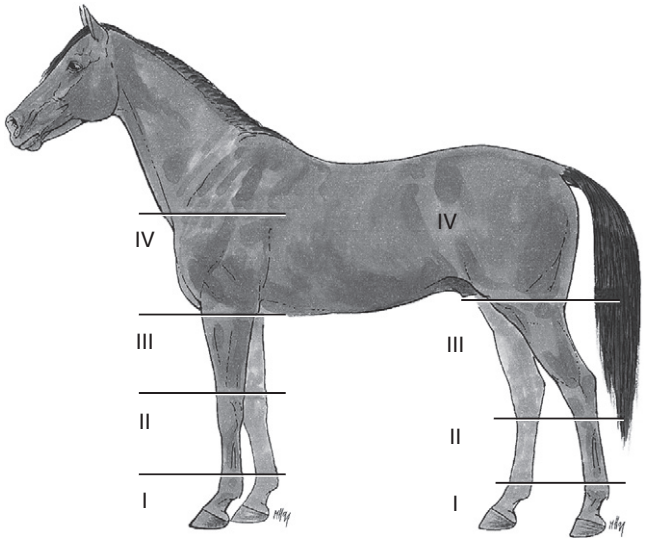

Figure 73-5. Biomechanically important divisions of the forelimb *(left)* from distal to proximal: *(1)* distal to the distal quarter of the third metacarpal (MCIII), *(2)* from the distal MCIII to the distal radius, (*3*) from the distal radius to the elbow joint, and *(4)* from the elbow joint to distal scapula. Biomechanically important divisions of the hindlimb *(right)* from distal to proximal: *(1)* distal to the distal quarter of the third metatarsal (MTIII), *(2)* from the distal MTIII to the tarsus, *(3)* from the tarsus to the stifle joint, and *(4)* proximal to the stifle joint. (From Bramlage LR: Compend Contin Educ Pract Vet 1983;5:S564.)

Region 1: Fractures of the Proximal, Middle, and Distal Phalanges and the Distal Sesamoid Bone

Incomplete and complete fractures of the proximal, middle, and distal phalanx and the distal sesamoid bone are easily stabilized with either a cast or a splint.[9] With fractures of the proximal or middle phalanx, the third metacarpal/metatarsal bone (MCIII/MTIII) and the proximal and middle phalanx must be stabilized in an almost straight line to prevent movement in the frontal plane at the fracture line. To achieve this, the heels are raised with a wedge, which also serves to facilitate the attachment of the cast distally. With the limb in this position, a cast is applied from the heels to the proximal end of the MCIII/MTIII. To obtain optimal stabilization, the entire hoof, or at least the heels, should be included in the cast. If nonmoldable splints are used instead of a cast, they are preferably fixed dorsally (see Figure 17-20).

Holding the forelimb off the ground makes application of the synthetic splint easier because the horse is less likely to move. However, this is not possible with the hindlimb because of the reciprocal apparatus, which causes flexion of all the joints when the limb is held off the ground. However, with proper sedation, most horses stand quietly for a few minutes with the injured limb lightly touching the ground and in a fairly normal position. Various other splints, such as the Kimzey (Figure 73-6) or monkey splint (Figure 73-7), can be used in breakdown injuries of the suspensory apparatus instead of a cast.

Region 2: Fractures of the Third Metacarpal, Third Metatarsal, Carpal, and Tarsal Bones

With fractures of MCIII/MTIII, a cast or splint is applied from the hoof to the elbow or stifle joint, respectively (Figures 73-8 through 73-10). Depending on the temperament of the horse

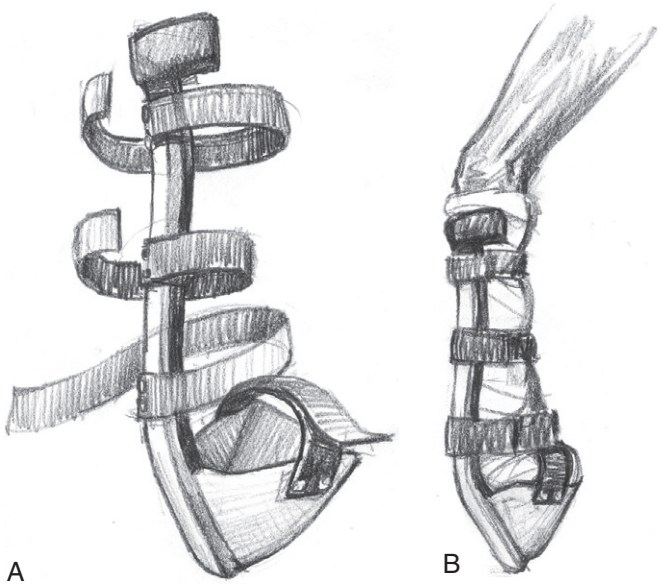

Figure 73-6. A, A Kimzey splint showing nylon straps fitted with hook-and-loop patches. **B,** The splint applied to the distal limb of a horse over a bandage. These splints are well suited for first aid for disruptions of the suspensory apparatus.

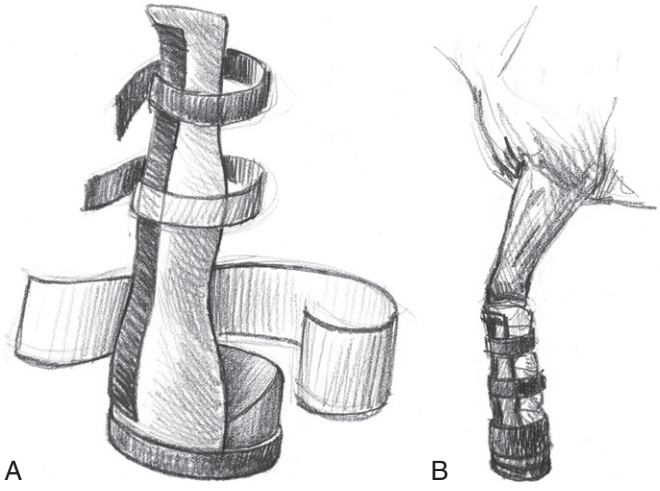

Figure 73-7. A monkey splint **(A)** applied to the distal limb of a horse **(B).** These splints align the phalanges and third metacarpal into one plane.

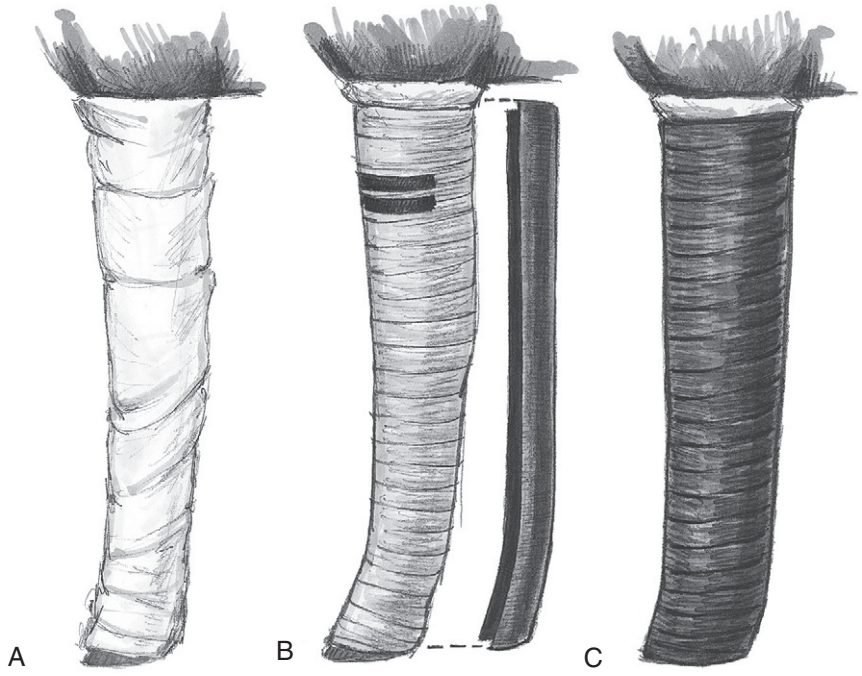

Figure 73-8. Application of a full forelimb splint. **A,** The padding is applied evenly in several layers over the entire limb, and each layer is tightened separately. **B,** A PVC-pipe splint is applied to the caudal aspect of the bandage and can be augmented with one pole medially and laterally (not shown). **C,** The splints are incorporated into the bandage with tape.

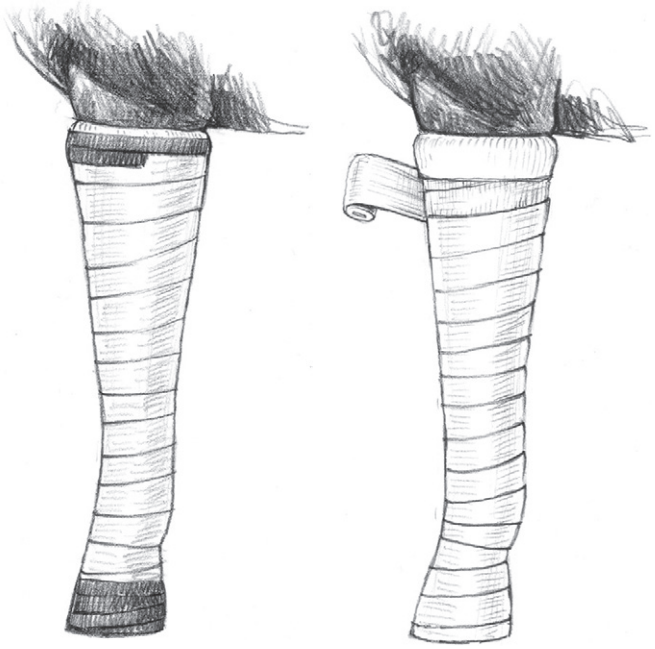

Figure 73-9. Application of a full forelimb cast using the technique described in Chapter 17 but extending over the entire forelimb up to the elbow joint. Care is taken to apply the cast material evenly along the entire limb and to fold back the stockinette over the top of the cast, incorporating it into the last layer to provide a smooth top edge.

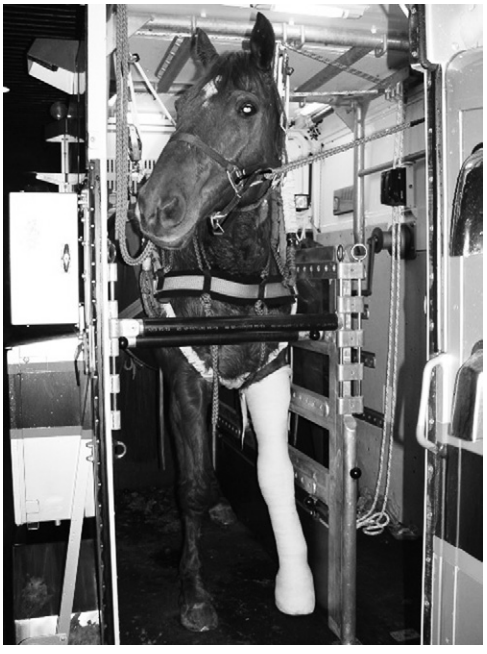

Figure 73-10. A horse with a fracture of the metacarpus, which has been stabilized with a full limb cast. The horse is loaded in an emergency trailer and is ready for the transport. The harness supports the patient during transport.

and the location of the fracture, the splint or cast may reach only the top of the calcaneus for fractures of the distal metatarsus. However, this does not produce optimal stabilization and should be looked at as an exception rather than the rule. Extending the external coaptation above the tarsus should always be the goal and should in most cases be possible with the use of adequate sedation.

Region 3A: Fractures of the Radius and Tibia

Fractures of the radius and tibia present special difficulties because the large muscle masses most often prevent fixation of the elbow and stifle joints. Furthermore, contraction of the extensor tendons, located craniolaterally on the limb, causes abduction of the limb below the fracture line, which can produce skin perforation on the medial aspect of the radius or

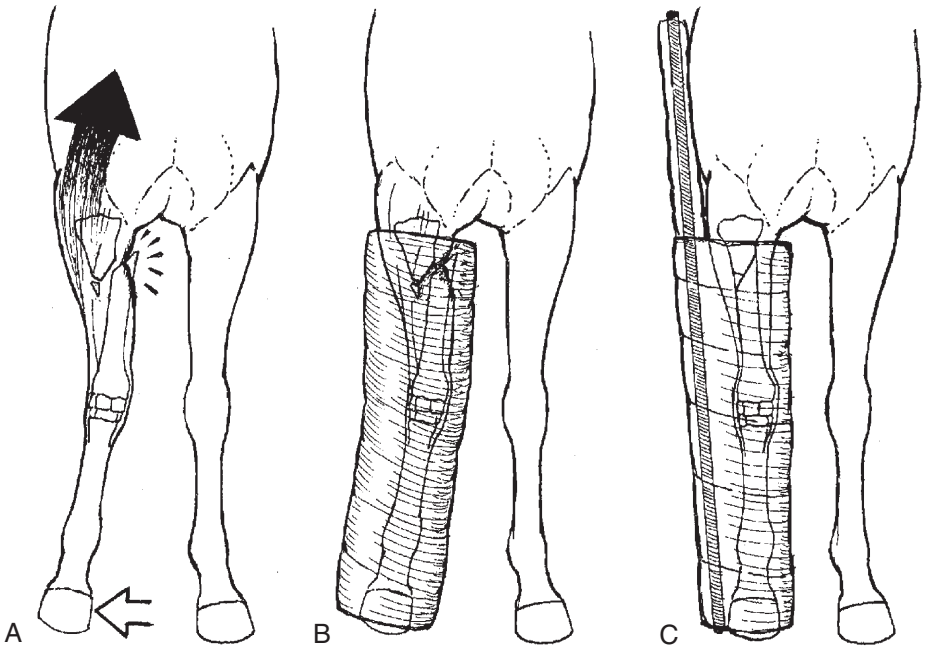

Figure 73-11. Splinting of a proximal radial fracture: **A,** All the muscles are arranged cranially, laterally, and caudally, which results in a lateral deviation of the limb when the muscles contract to provide support to the fractured limb. This may result in perforation of the skin at the medial aspect of the limb by sharp bone edges; **B,** A Robert Jones dressing or cast has been applied to the limb. It ends near the fracture site and provides only limited support. Also, it increases the leverage arm, causing more tissue damage; **C,** The incorporation of long splint or wooden plank to the lateral aspect of the bandage effectively counteracts the valgus-inducing forces and adds significant stability to the splint bandage. With such a splint, the patient may load the fractured limb.

tibia (Figure 73-11, *A*). Therefore, stabilization up to the elbow or stifle is not sufficient and will not prevent abduction. In these fractures, a stable splint or cast should first be applied up to the elbow or stifle joint (see Figure 73-11, *B*) and when possible, even higher up. This should subsequently be augmented with an additional splint applied laterally and reaching from the hoof to the point of the shoulder or hip, respectively. Additional padding is added to the splint so that it conforms to the contour of the limb (see Figure 73-11, *C*). Immobilization is difficult in the hindlimb because of the reciprocal apparatus. Flexion of the stifle, which cannot be prevented, results in flexion of the hock via the superficial digital flexor muscle and the peroneus tertius muscle. This results in large forces acting on the tibia so that dislocation of the fragments is unavoidable. Regardless, these fractures are stabilized as well as possible using this technique. In some foals, a cast can be applied proximal to the elbow and stifle joint (Figure 73-12), which improves the stabilization of a fracture of the radius and tibia. Moreover, the horse must be properly padded and stabilized in the emergency trailer (Figure 73-13) or van.

Region 3B: Fractures of the Ulna

Fractures of the ulna result in failure of the passive stay apparatus of the forelimb (triceps apparatus), which allows the horse to stand with little muscular effort for prolonged periods of time. Hence, the horse assumes a typical stance with the carpus in flexion (Figure 73-14). Therefore, the limb should be stabilized with a splint that extends the carpus so that the horse can bear weight on the limb. The splint should be applied caudally and should extend from the metacarpophalangeal region to the level of the elbow joint (Figure 73-15).

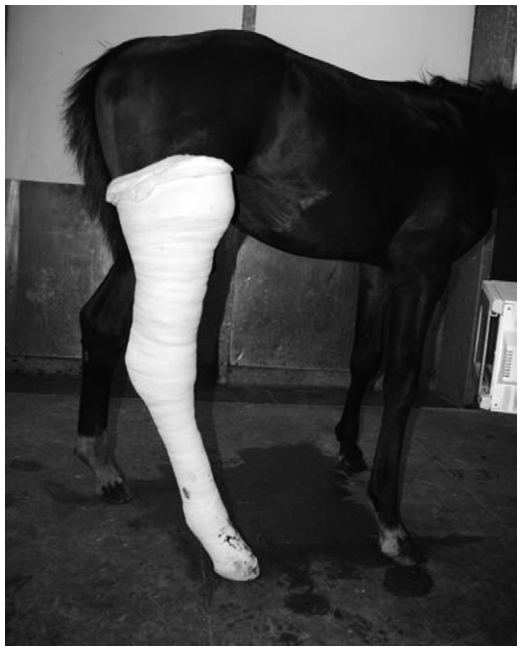

Figure 73-12. In some foals, a cast can be appplied proximal to the stifle joint, which improves the stabilization of a fracture of the tibia (region 3).

Region 4: Fractures of the Humerus and Femur

Fractures of the humerus and femur do not require a bandage or a splint because they cannot be adequately immobilized and there is enough muscle mass to protect the bone. These fractures are rarely open and a bandage would be counterproductive, because it would only add weight without providing stabilization. In some cases, fractures of the humerus or neck of the scapula can be helped with a splint that extends the carpus, similar to the situation with fractures of the ulna.

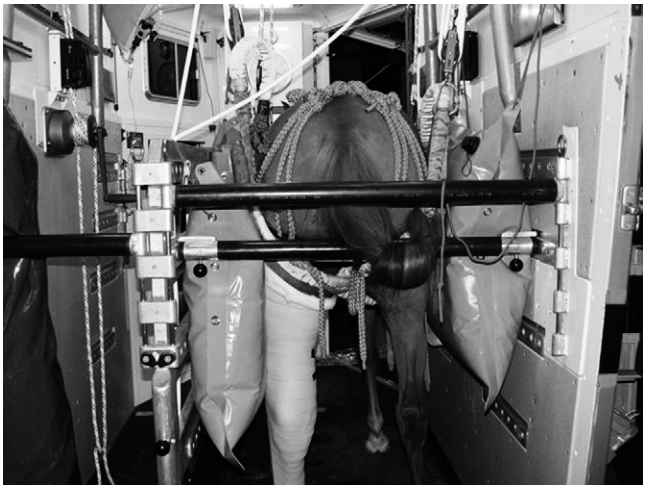

Figure 73-13. The horse with a tibial fracture is properly stabilized and padded in the emergency vehicle. Notice the dunage bags that are inflated to prevent side-to-side movement of the horse.

Analgesia and Anti-Inflammatory Medications

Systemic analgesics should be given to the horse as soon as the fracture is stabilized. Nonsteroidal anti-inflammatory drugs, such as phenylbutazone or flunixin meglumine, are the most commonly administered anti-inflammatory therapy for musculoskeletal injuries in horses. Control of inflammation is an important step in limiting the risk of thrombosis, maximizing perfusion of the limb, and preparing the limb for surgical repair. However, when fracture stabilization is inadequate, the use of a potent analgesic is contraindicated, because this leads to an overload of the injured limb with associated complications.

Antimicrobial Therapy

Immediate administration of systemic antibiotics is indicated in horses with open fractures or large wounds. Otherwise, antibiotic therapy can be delayed until the time of surgery. Antibiotics administered intravenously immediately prior to a surgical intervention achieve effective blood and tissue concentrations within approximately 20 minutes. The regimen of choice is a combination of 30,000 IU/kg of crystalline penicillin and 7 mg/kg of gentamicin sulfate, both administered intravenously. (For details on surgical site infection and antimicrobial use, see Chapter 7).

Intravenous Fluid Therapy

Fractures are rarely associated with severe hemorrhage. However, pain can induce neurologic shock (see Chapter 1), leading to substantial fluid shifts, which should be supported using intravenous fluid therapy. After an indwelling intravenous catheter

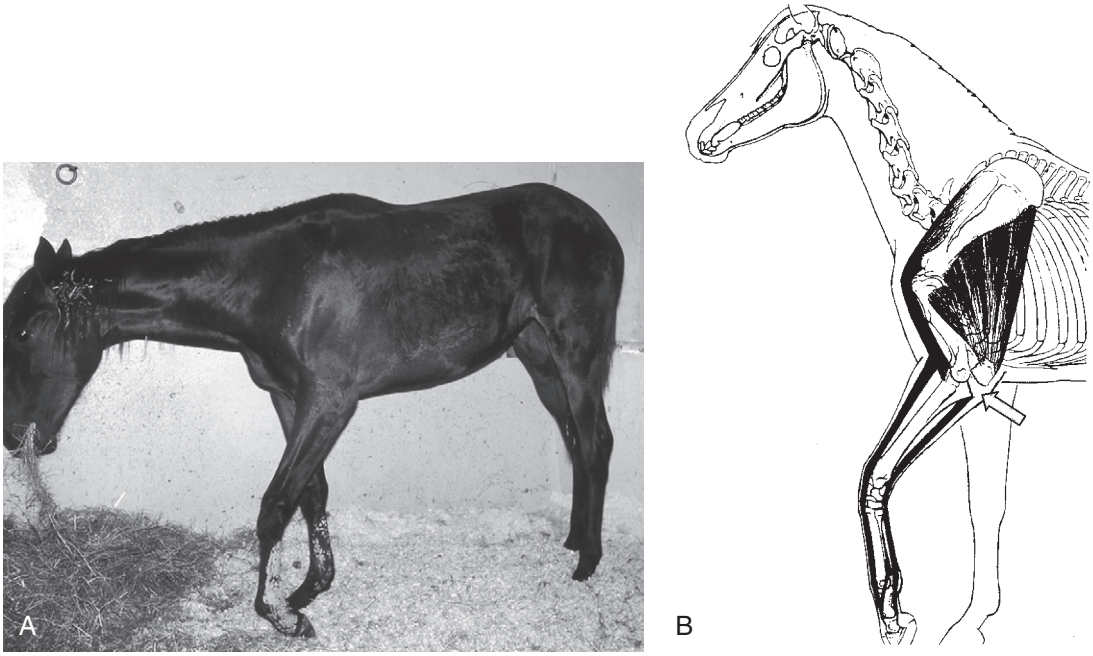

Figure 73-14. A, Typical posture of a horse with a fracture of the olecranon process of the ulna; **B,** Illustration of the forces that affect a horse's limb movement in the presence of an olecranon fracture. The carpus cannot be maintained in extension because the triceps muscle inserts only on the proximal fragment, which results in the formation of a fracture gap *(arrow)* when it contracts.

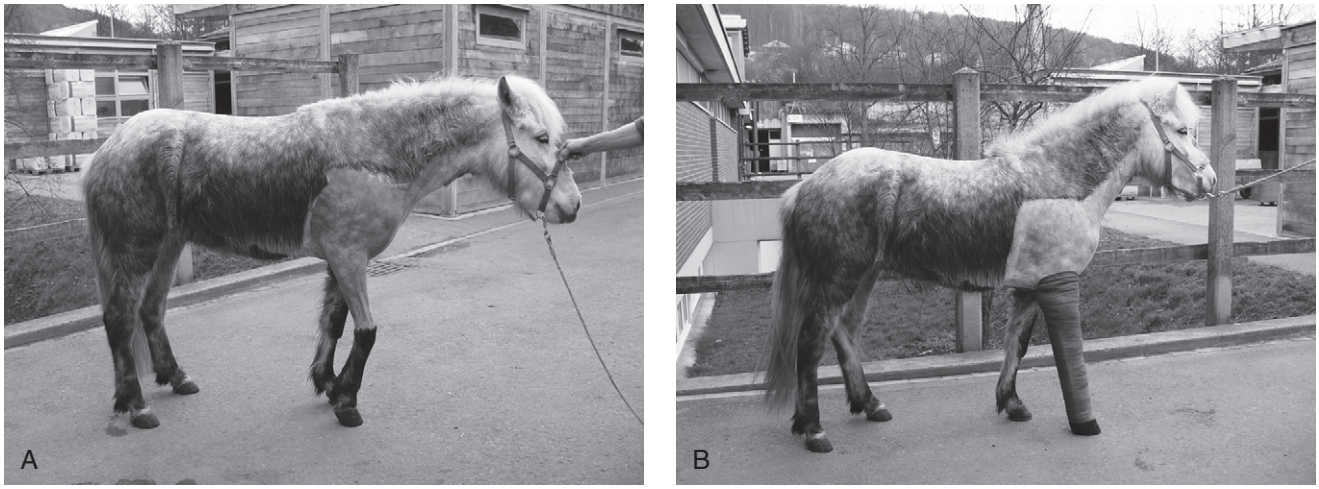

Figure 73-15. A, An Icelandic Horse with a fracture of the olecranon process of the ulna. **B,** The same horse after correct application of a caudal splint.

is placed and secured to the skin, a solution of electrolytes and glucose (up to 2% of body weight) should be administered with the drip rate set at maximum. (See Chapter 3 for details on fluid administration.) Horses out in the open should be kept warm with a horse blanket or an aluminum emergency blanket. (See Chapter 1 for details on physiologic response to trauma.)

Transportation of the Horse

The injured horse must be transported carefully and humanely, according to animal welfare legislation.[7-10] It is advantageous to have a specialized large-animal trailer or van and the support of an experienced veterinarian or assistant.[9,10] However, some controversy exists: there are those who think that this kind of transportation can be accomplished with normal horse trailers that are minimally adapted.[11] In my experience, horses with a fracture should be transported by a specialized large-animal trailer built to transport injured horses.

Equipment

The emergency trailer or van should have enough room for two standing horses or one recumbent horse.[9] Ideally, there should be enough space to handle the horse from all sides. The ceiling of the trailer should be approximately 30 cm higher than in standard horse trailers, and it should be equipped with a nonslip floor. It should be well ventilated and spacious with a good lighting system for night transport. Additionally, the vehicle should be solidly built, preferably of aluminum. The loading and unloading ramps should be long enough so that the slope is minimal, which is especially important for horses wearing a splint. The ramp must have an even, nonslip surface, and the end must be flush with the ground. The vehicle must be equipped with a winch and a strong frame to support a harness. Different sizes of suspensory harnesses are required with a minimum carrying weight of 1200 kg. Other requirements are a gurney or a mat on which a recumbent animal can be pulled into the trailer. Curtains or movable lightweight walls are helpful when the working area needs to be blocked off from the view of spectators. For safety reasons, the driver of the vehicle must be able to observe what is going on via a video camera in the trailer and able to have audio contact with the support assistants caring for the horse during the transport. The vehicle should be equipped with a heater and the floor should be well padded (e.g., with an air mattress) to minimize vibrations during the transport of a recumbent animal. The interior installations should be adjustable to accommodate horses of different breeds and sizes. The emergency trailer or van should be stocked with all the necessary medical supplies for various emergency treatments including shock therapy.

Loading Injured Horses

It is best to maneuver the vehicle as close to the injured horse as possible to avoid unnecessary walking.[9] As the steepness of the loading ramp increases, so must the interior height of the trailer (minimum height, 2.35 to 2.5 m), particularly when a forelimb is injured. Increasing the height of the nose of the trailer using the jack will lower the height of the ramp somewhat. Horses also load better when the trailer is positioned so that the horse can walk in the same direction as the barn. The horse should be acclimated to the cast or splint by walking a few steps before being loaded.

Application of a bridle over a halter is recommend for better control during loading. Two readily available lunge lines are tied to either side of the trailer and are used to direct the horse into the vehicle. For injured horses, the stall partition should be moved to one side to provide the maximal amount of space for loading. Before loading, the service door and the front-unloading door should be opened and the interior lights turned on at night to illuminate the interior as much as possible. Two assistants hold the lunge lines and move slowly toward each other, progressively narrowing the space between them. Touching the horse with the lines may help loading and is not necessarily discouraged. As soon as the horse is in the trailer, the rump bar is replaced, the ramp is closed, and the service door

and the front-unloading door are closed. The stall partition and chest bar are the last barriers to be put in place.

It has been recommended that horses with forelimb fractures be transported facing backward in an attempt to relieve the stress on the forelimbs during braking.[12] However, almost all standard European two-horse transporters do not provide enough space for the horse to comfortably stand backward; when the chest and rump bars are in place, the horse would have to keep its head either very high or turned to the side because of the ramp (the back door) in front of it. This would be very uncomfortable for the horse. Furthermore, the head and neck are important for balance but they have to be free to perform this function properly. Finally, it is very difficult if not impossible to load an injured horse backward or to turn it around after it is inside the trailer. Loading the horse via the front unloading ramp is not practical because the space is limited and the slope of the front ramp is usually steeper than that of the rear ramp. For a horse standing backward in the trailer, the injured forelimb would then be positioned at the back of the transporter between the rear axle and the ramp, where the ride is the roughest.

The horse has to balance not only during breaking but also during acceleration and in curves. Therefore, the driver must take the time to ensure a comfortable ride for the horse. An assistant in the trailer with the horse helps to calm it and relay any developing problems directly to the driver. For horses with hindlimb injuries, the driving speed must be as slow as possible, because the ride is the roughest behind the rear axle.

Transport in a Supporting Harness

A harness should be used to transport horses with severe injuries.[10,13] Horses that are transported with a harness arrive at the hospital in much better general condition than those that are not. Transportation in a harness can provide tremendous relief for an injured horse. Many horses allow the harness to support their entire weight during transport. Most horses alternately rest one limb and then the other. After some time, horses with a properly splinted limb frequently bear some weight on the fractured limb while being supported by a harness, thereby resting the healthy contralateral limb. For this type of transport, a person experienced in large-animal rescue operations should always be present in the trailer to provide optimal care for the patient. In extreme cases, a veterinarian may be required to travel with the horse as well. Most horses can be easily positioned in a harness provided that it is applied snugly and gives support to the patient from the front as well as from the back. The harness must be fitted so that it does not interfere with the horse's respiration and balance. During transportation, the bellyband is tightened several times, and the front and hind belts are adjusted until the horse is comfortable. It is imperative that everything be checked repeatedly throughout the transport so that problems can be immediately identified and corrected (Figure 73-16).

Unloading Fracture Patients

As a general rule, the horse should get off the trailer using the sound limbs first. With a forelimb injury, the horse should be backed off the transporter. With hindlimb injuries, the horse should be unloaded via the front ramp. As for loading, the trailer jack can be adjusted to provide the optimal ramp angle for unloading.

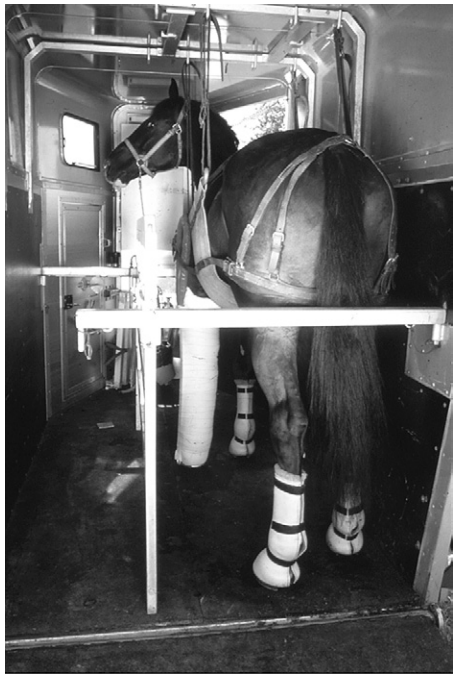

Figure 73-16. A fracture patient supported by a harness is ready to be transported to a specialty clinic.

Injured horses, particularly those with fractures, should be sedated for unloading. Most horses feel insecure when they are presented with new surroundings and when forced to use the injured limb after having been supported in a harness. This stress may result in hectic or hesitant and uncontrolled movements during unloading. Sedation dampens these reactions, and unloading is safer for both the horse and the handlers.

Transport of Recumbent Horses

As a general rule, horses should be transported standing whenever possible. Horses that are unable to stand are transported in lateral recumbency, and anesthesia may be induced for the ride to the clinic.[9] Transportation of horses under general anesthesia is rare; the anesthesia must be carried out and maintained by a veterinarian, who must travel with the horse. The duration of transportation in such cases must be as rapid as possible, perhaps assisted by a police escort, and it should not exceed 90 minutes.

A mechanical or electric winch, transport mat, insulated air mattress, head protection for the horse, and hobbles are some of the equipment needed for transportation of recumbent horses.

When the decision has been made that the horse is to be transported in lateral recumbency, an indwelling intravenous catheter is placed and intravenous fluids are started. This is the only way of ensuring rapid treatment at any time. Horses that cannot rise are sedated and those that can rise are anesthetized so they can be transported in recumbency. After induction, anesthesia must be maintained during the entire transport period. General anesthesia is usually induced and maintained using injectable drugs, some of which can be administered via a drip (see Chapter 20). The van is positioned as close to the

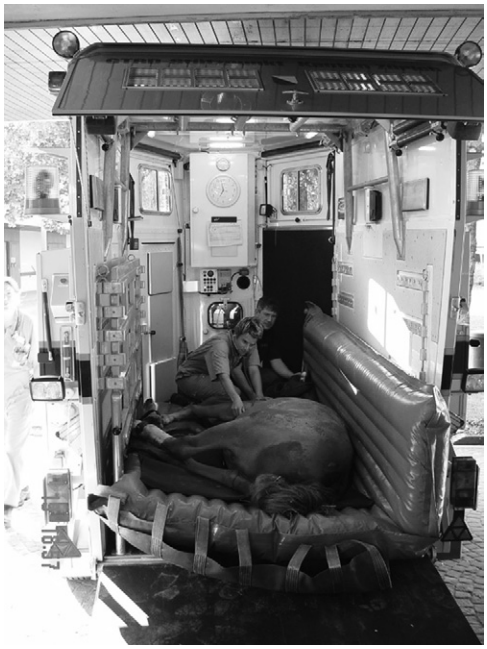

Figure 73-17. A recumbent patient was anesthetized and pulled into the trailer using a winch and rests comfortably on an air mattress, which was inflated after the horse was positioned in the trailer. Two assistants help with anesthesia.

horse as possible, and the stall partition is placed to one side so that the entire interior space can be used. The winch wire is pulled out to the desired length and the horse is placed on the rubber transport mat or another device so that it can be pulled into the van. Either a hand or electric winch can be used, but there should be a backup if the electric one fails.

For recumbent horses, leather boots are placed on the hooves to reduce the risk of injury, and hobbles are applied to all limbs. The head and especially the eyes must be well protected. This is best achieved with padded leather headgear after the halter has been removed to prevent damage to the facial nerves. The horse can be pulled into the van using a slide mat.

Recently, special air mattresses have been developed for the transport of recumbent horses; these mattresses are used by the large-animal rescue unit operating in Switzerland and Liechtenstein (Herr Ruedi Keller, Embrach).[9] Before inflation, the air mattress is placed between the horse and the transport mat, which are then pulled into the van together. The mattress is subsequently inflated using compressed air. This procedure takes about 2 minutes and ensures a comfortable ride for the patient. Horses can be transported great distances without the risk of pressure necrosis or nerve damage (Figure 73-17). Often, the amount of drugs used to sedate the horse can be reduced as well.

For long distances, a mobile anesthetic machine operated by experienced assistants may be necessary. This enables the administration of intravenous fluids and air supplemented with oxygen (approximately 2 to 12 L of oxygen per minute). Unloading the recumbent horse is straightforward and is achieved by pulling the transport mat out of the van with a winch or crane or by hand.

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CHAPTER 74

Bone Biology and Fracture Healing

Mandi J. Lopez and Mark D. Markel

The bones of the skeleton are dynamic structures that protect internal organs, provide rigid segments for muscles to generate forces of motion and locomotion, and serve as the largest reservoir of calcium and phosphate in the body.[1] Bone has an intricate, tightly regulated structure composed primarily of mineral, organic matrix, cells, and water. Although mineral composes two thirds of the dry weight of bone, the relative amounts of all components convey mechanical properties crucial to bone function.[2] In turn, maintenance of normal bone performance depends on constant structural renewal through a dynamic remodeling process. In addition to contributing to the renewal process, cells that line the internal and external surfaces

of the bone, with their secreted growth factors and interactions with the microenvironment, give bone a unique ability to repair itself without a scar following fracture.[3] Bone healing is a highly orchestrated series of biological events that depend upon the site and severity of the injury as well as fracture management. Fractures in the horse pose distinct challenges associated with size, anatomy, and locomotion that contribute to fewer successful repairs than comparable fractures in other species. Noninvasive mechanisms to assess healing quality and strength are critical to fracture management in the horse. Methods to accelerate fracture healing have been the subject of extensive research aimed at reducing complications and improving success rates in the horse. Information in this chapter includes the structure, function, and biomechanical characteristics of bone, as well as the forces that result in specific fracture configurations. The natural process of bone healing, how fracture stability influences the process, mechanisms to determine fracture healing strength, and treatments to accelerate fracture healing are also presented.

STRUCTURE AND FUNCTION OF BONE

Anatomy

Bones are divided into the major categories of long, cuboidal, and flat. In general, flat bones surround vital structures, cuboidal bones compose complex joints, and long bones make up the majority of the peripheral and appendicular skeleton. Equine long bones have a mechanical advantage compared to other species because their proportionately greater length contributes to superior power and speed. Three regions are recognized in long bones. The long, narrow, central region is the diaphysis (Figure 74-1). On each end of the long bone is a wide epiphysis that contains an articulating surface. Distribution of loads across the wide epiphyseal surfaces reduces the stresses experienced by the joints. The metaphysis serves as the transition between the diaphysis and each epiphysis. The physis, or growth plate, separates the metaphysis and epiphysis on each end. Initially, all axial bones form by ossification of a cartilaginous model, endochondral ossification, whereas membranous bones like the skull form from a fibrous precursor.[4,5] Long bone growth in length occurs at the physis via continued *endochondral ossification*. When growth ceases at skeletal maturity, the physis becomes inactive and remains as a narrow, indistinct demarcation between the epiphysis and metaphysis.

Briefly, endochondral ossification, responsible for long bone longitudinal growth, occurs as chondrocytes arranged in columns parallel to the long axis of the bones differentiate and form histologically distinct zones. The zone of *resting cartilage,* containing the least metabolically active chondrocytes, is closest to the epiphyseal end of the growth plate. In the next zone, the *zone of proliferation,* cell division occurs in a plane perpendicular to the long axis of the bone and thereby increases the length of the cell column.[6] Chondrocytes mature, become encased in extracellular matrix, and assume a round morphology in the *pre-hypertrophic* zone just prior to the *hypertrophic zone,* where chondrocytes cease dividing, increase in size, and hypertrophy. Finally, hypertrophic chondrocytes are replaced by mineralized bone and bone marrow via vascular invasion, cartilaginous matrix resorption, and recruitment of osteoblasts that deposit bone matrix in the *zone of calcification*.[7] All mammalian physeal long bone growth follows this same general pattern.

Independent of macroscopic anatomy, all skeletal segments have an outer cortex of compact (cortical) bone and an inner medulla containing bone marrow as well as spongy (cancellous) bone. About 90% of the long bone diaphysis is calcified with both cortical and cancellous bone, but the medulla also contains hematopoietic bone marrow in immature animals and yellow, fat-replete non-hematopoietic bone marrow in adults. The ratio of cortex to medulla varies with bone and function. Cortical bone thickness gradually decreases closer to the metaphyseal zone, and cuboidal bones as well as the metaphysis and epiphysis have comparatively thinner cortices. Cancellous bone is composed of three-dimensional networks of fine supporting rods of bone or trabeculae and hence is referred to as *trabecular bone.* Trabecular bone is more metabolically active and responsive to metabolic homeostasis changes than cortical bone. Cortical and trabecular bone have similar basic composition but significantly different porosities. The porosity of trabecular bone is significantly greater than cortical bone, primarily because of vascular and bone marrow intratrabecular spaces in the former.

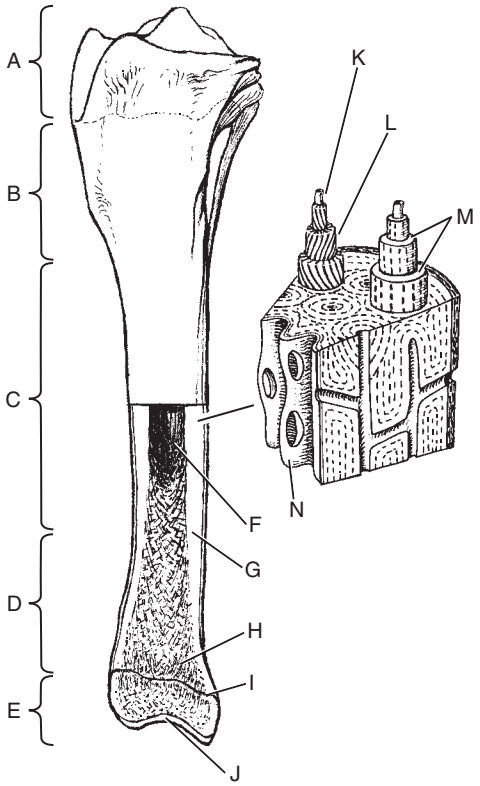

Figure 74-1. Illustration of an immature equine femur with the regions and types of bone indicated. The *inset* is a schematic of bone microstructure showing major osteonal components. *A,* Proximal epiphysis (the separate ossification center of the tibial crest is not shown); *B,* proximal metaphysis; *C,* diaphysis; *D,* distal metaphysis; *E,* distal epiphysis; *F,* medullary cavity; *G,* caudal cortex (compact bone); *H,* cancellous bone; *I,* distal physis; *J,* articular cartilage; *K,* central capillary in Haversian canal; *L,* collagen fibers (alternating their orientation in each lamella); *M,* concentric lamellae (3-7 μm) with osteocytes *(dots); N,* endosteum.

Cortical and trabecular bone are composed of osteons (see Figure 74-1). *Cortical osteons* are called Haversian systems (Figure 74-2) and *trabecular osteons* are called packets. Haversian systems have a cylindrical shape and form a branching network within cortical bone. Concentric, flat, platelike layers of bone, or lamellae, form the walls of Haversian systems. Trabecular osteons are also composed of concentric lamellae, but they are semilunar in shape. Osteons are further classified according to

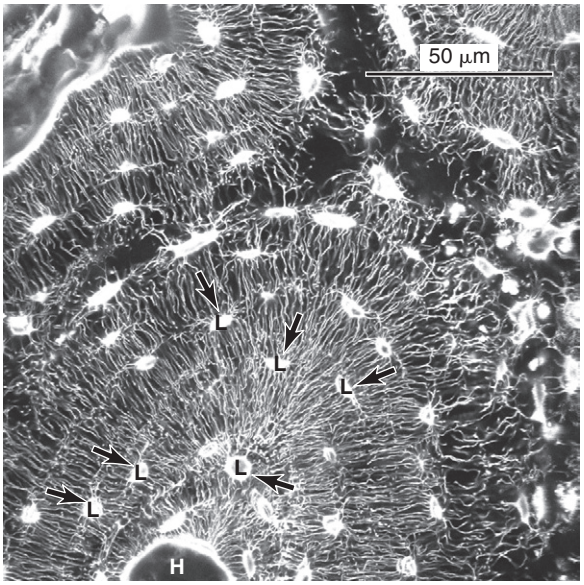

Figure 74-2. Gray-scale composite of Z-stacked confocal photomicrographs of osteocytes surrounding an osteon *(lower left)* in the third metacarpal bone in a racing Thoroughbred. A dense syncitial network of vital osteocytes *(small arrows)* and their canaliculi are demonstrated connecting adjacent lacunae *(L)* before ultimately reaching a Haversian canal *(H).* Scale bar equals 50 μm. (Courtesy Peter Muir, University of Wisconsin, Madison.)

the arrangement of the collagen fibers within their lamellae. Different collagen fiber arrangements have characteristic birefringent patterns when viewed with polarized light. The arrangement of collagen fibrils within the lamellae of osteons confers differing biomechanical characteristics that are reflected in the heterogenous mechanical properties of a whole bone.[8] The spaces between osteons, often called *cement lines,* are composed of matrix components and interstitial lamellae. The composition and protein arrangement within cement lines make them more flexible than the osteons they surround. Osteons can also be classified according to the time of formation. Primary osteons form during *appositional bone growth* (growth resulting in increased bone thickness). Secondary osteons are created throughout life when osteoblasts deposit bone at the end of bone tunnels (cutting cones) created by osteoclasts during the process of bone remodeling.

Periosteum is a thin layer of osteogenic and fibroblastic cells within a well-developed nerve and microvascular network located along the periosteal cortex of cortical bone.[9] Long bones are covered by a periosteum, except on articular surfaces or points of ligament, tendon, or joint capsular attachment. Periosteum is composed of two distinct layers. The *outer fibrous layer* of periosteum is composed of fibroblasts, collagen and elastin fibers, and a nerve and microvascular network.[10] The *inner cambium layer,* which directly contacts the bone surface, contains adult mesenchymal progenitor cells, differentiated osteogenic progenitor cells, osteoblasts, fibroblasts, microvessels, and sympathetic nerves.[11] It provides the cells for fracture healing and appositional bone growth. The periosteum is attached to the outer cortical surface by thick collagenous fibers, Sharpey's fibers. Bones widen through periosteal apposition when osteoblasts add mineralized tissue on the periosteal bone surface. Toward the bone ends, the periosteum continues directly into the perichondral ring that encircles the periphery of the growth plate.[12] Both the periosteum and the perichondrium are firmly attached to the epiphysis. The endosteum is a membranous structure covering the inner surface of cortical bone, trabecular bone, and the blood vessel canals (Volkman's canals) that contains blood vessels, osteoblasts, and osteoclasts.

Long bones have afferent and efferent vascular systems. The afferent vascular system consists primarily of nutrient arteries, proximal and distal metaphyseal arteries, and periosteal arterioles. The efferent vascular system includes the large emissary and nutrient veins that drain the medullary contents, cortical channels, and periosteal capillaries.[13] The direction of blood flow in the efferent (venous) system is primarily away from the cortex toward the medulla (centripedal) and volume is about six- to eightfold greater than the afferent (arterial) system. In mature animals, about 70% of the afferent blood flow within the diaphysis of mature bone is toward the cortex away from the medulla (centrifugal) and 30% toward the medulla (centripedal).[14] The endosteal circulation supplies the medullary areas and the inner two thirds of cortical bone, whereas the periosteal arterioles supply the outer third. Periosteal capillaries are connected to those of the underlying cortex, but under normal conditions little to no blood passes centripetally from periosteum to cortex because of the centrifugal pressure gradient across the cortical capillaries in mature animals.[15] However, because of anastomoses between periosteal and endosteal circulatory networks, blood can flow in either direction, and the relative contributions of centrifugal and centripedal blood flow in the afferent vascular system are affected by growth and injury. The primary blood supply and direction of blood flow in the afferent vascular system changes during long bone growth, and in the young animal, the periosteal contribution is much greater than at skeletal maturity.[16]

Bone is a composite material consisting of organic and inorganic components. Inorganic, organic, and water components are approximately 40%, 30%, and 25% by volume, respectively.[17] The inorganic component is mostly crystalline *hydroxyapatite*: $[Ca_3(PO_4)_2]_3Ca(OH)_2$ and 90% of the organic matrix is composed of type I collagen. Bone hydroxyapatite crystals are very small, approximately 200Å in their largest dimension. Their structure makes them more soluble than geologic hydroxyapatite crystals, allowing them to support mineral metabolism.[18] In normal bone, the mineral provides stiffness and strength, and collagen conveys ductility (the ability to plastically deform without fracture) and toughness (the ability to absorb energy).[19] *Type I collagen* is a triple helix that contains three polypeptide chains, α-chains, each approximately 1000 amino acids (Figure 74-3). Two of the three polypeptides are identical (α_1) and the third is structurally similar but genetically different (α_2). Individual α chains of type I collagen consist of repeating glycine, proline, hydroxyproline, and hydroxylysine amino acids. The polypeptide chains are cross-linked by hydrogen bonds between charged residues. They form linear molecules that are aligned parallel to each other to form collagen fibrils. Collagen fibers are composed of grouped fibrils.

The organic *extracellular matrix* is composed predominantly of collagens, noncollagenous glycoproteins, hyaluronan, and proteoglycans. In addition to type I collagen, almost 30 noncollagenous proteins have been identified in bone. The extracellular matrix provides *structural strength* to tissues to maintain the complex architecture and the shape of organs. It is a scaffold for the cells and a reservoir for growth factors and cytokines, and it modulates cell activation and turnover. In general, the extracellular matrix is a dynamic network of molecules secreted by cells that in turn regulate cell behavior by modulating their

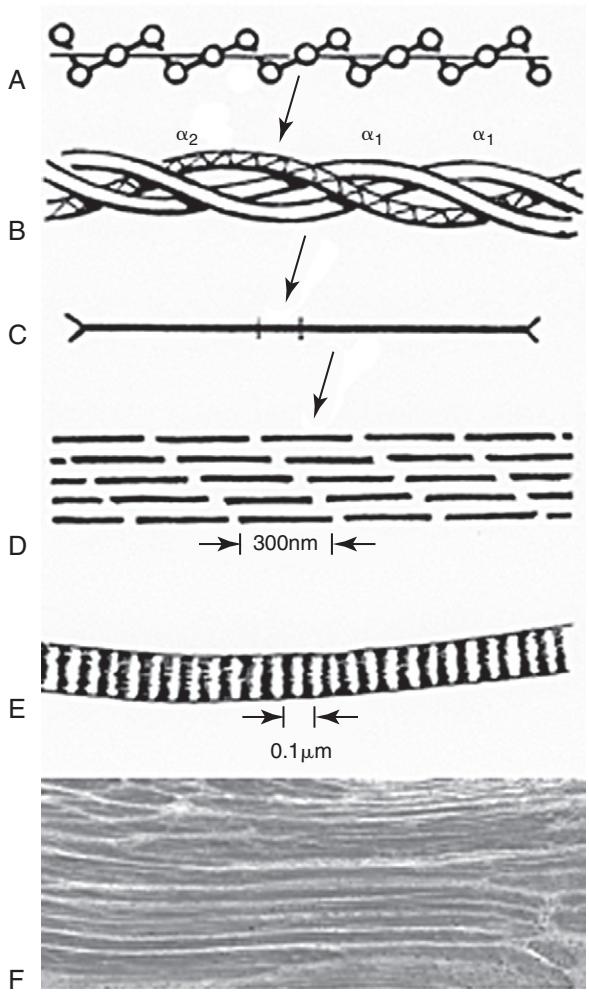

Figure 74-3. Illustration of the molecular features of type I collagen from the α chain to the collagen fiber: **A,** α Chain; **B,** Triple helix consisting of two α_1 and one α_2 chains; **C,** Tropocollagen molecules; **D,** Collagen fibril with quarter stagger arrangement; **E,** Fibril with repeated banding pattern; **F,** Collagen fibers.

proliferation and differentiation. Various cell types secrete different matrix molecules, the nature and amount of which change with age.

Cytokines (such as osteocalcin, bone sialoprotein, osteopontin, and osteonectin, among others) play important roles in bone matrix mineralization.[20] It is thought that the proteins help regulate mineral deposition by binding calcium and phosphate to control the size and number of hydroxyapatite crystals.[18] *Proteoglycans* are biological molecules composed of a specific core protein with covalently linked glycosaminoglycan (GAG) chains (see Figure 78-10). Glycosaminoglycans are linear, negatively charged polysaccharides, which can be divided into two classes of (1) *sulfated GAGs*, comprising chondroitin sulfate (CS), dermatan sulfate (DS), keratan sulfate (KS), heparin, and heparin sulfate (HS); and (2) *nonsulfated GAGs*, such as hyaluronan. Proteoglycans provide *flexibility* and *resilience* to the organic matrix.

Osteoblasts, osteocytes, and osteoclasts are the predominant cell types in bone. *Osteoblasts* are derived from self-renewing, multipotent stem cells through a multistep differentiation pathway.[21] The multipotent cells give rise to osteoprogenitor cells, which differentiate into preosteoblasts and then mature into ostoeblasts. Osteoblasts synthesize new collagenous organic matrix and regulate its mineralization by releasing small, membrane-bound matrix vesicles that concentrate calcium and phosphate and enzymatically destroy mineralization inhibitors.[22] When embedded in matrix, the osteoblasts become *osteocytes*, which have extensive filopodia, actin-based morphological structures at the periphery of cells, within canaliculi in mineralized bone. An extensive cannalicular network connects osteocytes to bone surface lining cells, osteoblasts, and other osteocytes. Connexins are proteins that maintain gap junctions between cells to allow direct communication through intercellular channels.[23] Osteocytes are linked metabolically and electrically through gap junctions, which are required for their maturation, activity, and survival. At the completion of bone formation, 50% to 70% of osteoblasts undergo apoptosis, and the remainder become osteocytes or bone-lining cells. The *bone-lining cells* regulate mineral ion influx and efflux into and out of bone extracellular fluid, and they retain the ability to redifferentiate into osteoblasts.[24] They also function in mechanosensation by transducing stress signals from bending and stretching of bone into biologic activity. Fluxes of calcium across gap junction are believed to stimulate transmission of information between osteoblasts on the bone surface and osteocytes within bone.[25]

Activated *osteoclasts* are derived from mononuclear precursor cells of the monocyte-macrophage lineage originating in bone marrow.[26] Osteoclast formation, activation, and resorption are regulated by interleukin 1, interleukin 2, colony stimulating factor, parathyroid hormone, 1,25-dihydroxyvitamin D, calcitonin, receptor activator of nuclear factor κB ligand (RANKL), and osteoprotegerin (OPG), among others.[27] However, RANKL and macrophage colony-stimulating factor (M-CSF), both produced by stromal cells and osteoblasts, are the two cytokines essential for osteoclast development. RANKL is part of the tumor necrosis factor family and is required for osteoclast formation, whereas M-CSF is necessary for differentiation of osteoclast precursors, osteoclast survival, and cytoskeletal changes for bone resorption. OPG is a membrane-bound and -secreted protein that binds RANKL to inhibit its action at the RANK receptor. Activated osteoclasts secrete hydrogen ions via H^+-ATP proton pumps and chloride channels in their cell membranes to lower the pH and mobilize bone mineral.[28] They also secrete enzymes including tartrate-resistant acid phosphatase, cathepsin K, matrix metalloproteinase 9, and gelatinase from cytoplasmic lysosomes to digest the organic matrix and form saucer-shaped Howship's lacunae on the surface of trabecular bone and Haversian canals in cortical bone.[29] Osteoclasts bind to bone matrix peptides via integrin receptors in osteoclast membranes.[30] They attach to the bone matrix via podosomes rather than by focal adhesions formed by most cells. After binding, a ruffled cell border forms when enzyme-containing vesicles fuse with the membrane. Additionally, a sealing zone forms around the periphery of the osteoclast attachment to the matrix, isolating the acidified resorption compartment.[31] Disruption of either the ruffled border or the sealing zone interferes with bone resorption.

BIOMECHANICAL DEFINITIONS

Mechanics is the study of the effect of forces on objects. The strength of a bone is determined by geometric factors like size, shape, and internal structure, as well as such material factors

as composition. Bone functions much like an elastic material under normal physiological loads with little to no change in external appearance, and this function is reflected by its mechanical properties, especially strength and stiffness. Mechanical properties can be assessed by evaluating the response of bone to externally applied forces called *loads*. When forces act on an object, they alter its shape and size, a process known as *deformation*. Mechanical studies can be divided into two broad categories, static and dynamic. Static studies evaluate bodies at rest, and dynamic studies evaluate moving bodies and can be subdivided into kinematics and kinetics. The vast majority of bone and bone-implant testing is static.

Descriptions of standardized mechanical testing fixtures and protocols are available from the American Society for Testing and Materials (ASTM), but many investigators use custom testing fixtures and protocols for the particular specimens to be evaluated. In most mechanical tests, the applied force is measured by a load cell, and changes in specimen dimensions are measured by motion of the load application system, a strain gauge or extensometer, or another real-time mechanism such as a motion-detection system. Testing is usually performed under load or displacement control, which refers to the test's *end point*. Specifically, investigators can define the end point as a predefined travel distance of the load-application system *(displacement control)* or a predefined load on the load cell *(load control)*. The primary criterion for selecting load or displacement control is whether the dependent variable is displacement or load, respectively.

It is often useful to separate the mechanical properties of the composite materials of a bone (i.e., *material properties*), from those that depend on its dimensions (i.e., *structural properties*). In general, the testing protocols and raw data may be similar for both types of testing. When testing *structural properties* of a bone, the relationship between load and displacement can be represented graphically on a load-deformation curve (Figure 74-4). The initial curved segment of the curve is the *toe region*, a low-force, high-deformation region that reflects a typical response of tissues to physiologic forces characterized by fluid motion as well as straightening and stretching of protein fibers like collagen and elastin. The following linear segment of the curve is the *elastic region* and shows the ability of the bone to resist deformation. In this region, the structure maintains the capacity to return to its original form upon removal of the load. The slope of the curve is often referred to as the *stiffness*. It is critical to understand that the stiffness refers to the composite structure or construct and depends on the shape and interaction between separate components when applicable. As loading continues through the elastic region, the *yield point* is reached, beyond which permanent deformation occurs in the plastic segment of the curve. That is, the structure will not return to its original shape when the load is removed. The amount of plastic deformation, like elastic deformation, depends on the applied load. With increasing load, the bone or construct will eventually undergo significant plastic deformation at a load and deformation defined as the *failure point*. The amount of plastic deformation corresponding to failure depends on the parameters defined in the investigation. The ultimate load is the load beyond which the structure essentially loses all capacity to withstand increasing forces. The ultimate load may be identical to or higher than the failure load. Taken together, the information on a load-deformation curve largely depends on the bone configuration and the relative orientation of the load.

Material properties may be of greatest interest in investigations surrounding properties of fracture callus or the effects of exercise or diet on bone. For material properties testing, areas of interest are typically included in specimens prepared from larger structures. The specimens are generally identical in size, shape, and microstructural orientation. As indicated earlier, the testing process may be very similar to the testing of structural properties, but the load and deformation recorded are stress and strain, respectively. Stress (σ) is the intensity of the force divided over the area that it acts upon. Common stress units include pounds per square inch (psi) and pascals (Pa) (Table 74-1).

A good series of relationships to know is megapascals [MPa] = 10^6 Pa = 1 N/mm^2 ~ 145 psi. Stress is further defined as normal or shear. *Normal stresses* occur when forces are applied perpendicular to the surface of a structure, and *shear stresses* occur when forces are applied parallel to the surface. Tension results in positive normal stress, and compression results in negative normal stress. Strain (ε) is the change in dimension divided by the

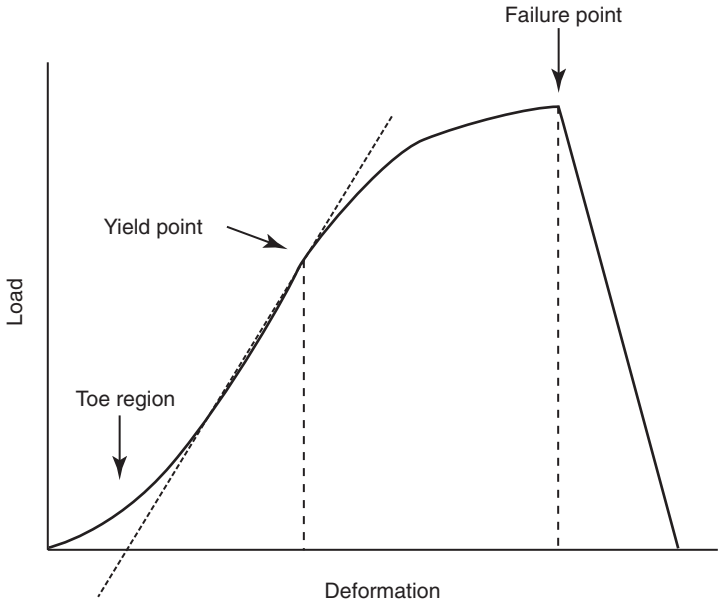

Figure 74-4. Representative load-deformation curve characteristic of a *whole bone structure* (mechanical testing of the *structural properties* of the whole bone).

TABLE 74-1. Stress and Strain Definitions, Symbols, and Units

	Stress	Strain
Definition	The intensity of the force divided over the area that it acts upon	Change in dimension divided by the original dimension (x_1-x_0/x_0)
Symbol	Sigma (σ)	Epsilon (ε)
Units	Kilopounds per square inch (ksi), kilopounds per square foot (ksf), pounds per square inch (psi), pounds per square foot (psf), tons per square foot (tsf), pascals (Pa), megapascals (MPa), kilopascals (kPa)	Normal strain is dimensionless and sometimes expressed as a percentage ($[x_1$-$x_0/x_0] \times 100$). Shear strain is often measured in radians.
Normal	Perpendicular to the surface	Perpendicular to the surface
Shear	Parallel to the surface	Parallel to the surface
Poisson's ratio (ν)	Ratio of lateral normal strain to longitudinal normal strain	Ratio of lateral normal strain to longitudinal normal strain

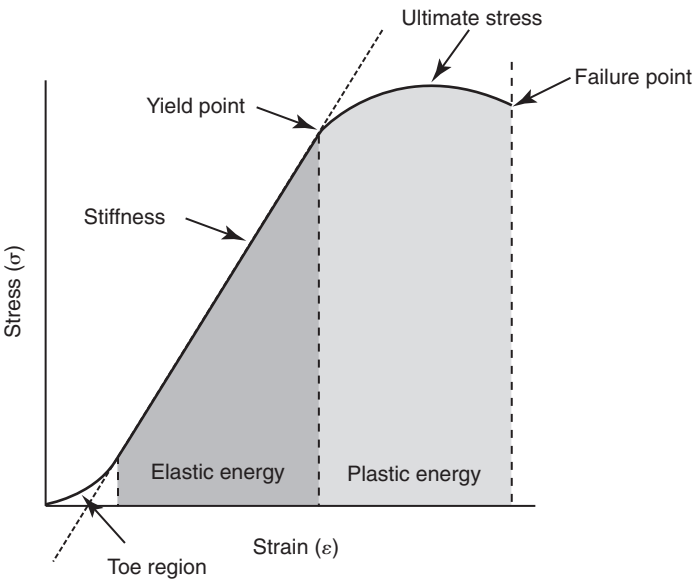

Figure 74-5. Stress-strain curve typical of a *bone sample* (mechanical testing of the *material properties* of the bone sample). (Courtesy P. Daigle, Louisiana State University, Baton Rouge)

original dimension, and the same conventions, normal and shear, apply to strain relative to changes in dimension. Strain is dimensionless, though it is sometimes expressed as a percentage. The ratio of lateral normal strain to longitudinal normal strain is called Poisson's ratio (ν). Shear strain, often measured in radians, refers to deformation in which a plane in the body is displaced parallel to itself. Quantitatively, it is the displacement of a plane relative to a second plane divided by the perpendicular distance between them, half the change of an original 90 degree angle, or the angle between original and deflected locations of the edge of a structure.

A *stress-strain curve* typically looks very similar to a load-deformation curve with stress on the ordinate versus strain on the abscissa (Figure 74-5). The slope of the elastic segment of the stress-strain curve is called *Young's Modulus* when the stress is normal and the strain is linearly proportional to the applied stress. The slope is the shear modulus when the stress is primarily shear. The yield and failure points are similar those described earlier, with the notable exceptions of the units and that they represent material versus structural properties. Additionally, the ultimate stress and strain may be different from the failure stress and strain. Toughness is the energy absorbed by the structure during the loading process. It is equivalent to the area under the curve, and is often expressed as the work of fracture. Brittleness is the opposite of toughness, which is not to be confused with strength. A structure can be tough and have a low or high fracture strength, and the same can be said for brittleness. The yield or failure energy is dissipated when the structure yields *(yield point)* and then fails *(failure point)*. When a crack starts to form, energy is released as two new surfaces are created. If the amount of energy released is less than that required to form the crack, it will stop. Otherwise, it will continue to spread and more cracks will form, consistent with catastrophic failure. The more energy stored in a bone prior to failure, the greater the comminution and/or soft tissue damage that will be caused when a fracture occurs.

The mechanical properties of bone depend on direction of the applied forces, a property called *anisotropy*. In contrast, a material that exhibits neither structural orientation nor dependency on loading orientation is said to be isotropic. *Bone is strongest in compression, weaker in shear, and weakest in tension.* Protein fiber arrangement, osteon composition, and bone mineral distribution contribute to the anisotropic properties of cortical bone. The same factors contribute to anisotropy in trabecular bone with the added component of bone porosity. Cortical bone is more brittle than trabecular bone and fails at a significantly lower strain, albeit a higher load. Trabecular bone stores more energy prior to failure compared to cortical bone

because of a higher toughness. Isolation of the contribution of each bone component to diverse loading to prevent injury and advance treatment of orthopedic injuries is often the goal of many biomechanical studies.

The deformation and strain response of bone to load depends upon many factors, including bone anatomy and composition as well as direction, rate, magnitude, frequency, and duration of applied load. During normal daily activities, bone is subjected to complex loading conditions, most of which include forces applied from numerous directions simultaneously. Replication of complex loading conditions in the laboratory is possible, but data reduction and interpretation is quite complex. Hence, loading strategies are often reduced to single-axis loading in tension, compression, bending, shear, torsion, or a combination of compression and torsion (Figure 74-6).

Equal and opposite loads are often applied to the ends of the bone for *tension* testing. Maximum stresses occur on a plane perpendicular to the applied tensile load and result in transverse fractures (Figure 74-7), and bone fails as a result of osteonal pullout. Transverse fractures from tensile forces occur in the proximal ulna, the proximal sesamoid bones, the patella, and the calcaneus. When loaded in *compression*, maximum stresses again occur in a plane perpendicular to the load as the bone shortens and widens. The fracture configuration that results from compression is about 45 degrees offset from the direction of maximum shear forces. This is largely because of the pattern of microstructural failure at the level of the osteons. Current knowledge suggests that the tough cement lines impose high resistance to microcrack formation and propogation. However, with increasing loads, the microcracks propagate along the relatively more brittle and stress-concentrating structures of the Haversian system, which results in a fracture that is about 45 degrees from the long bone axis.[32] Such fractures are probably best exemplified by dorsal metacarpal stress fractures in the horse.

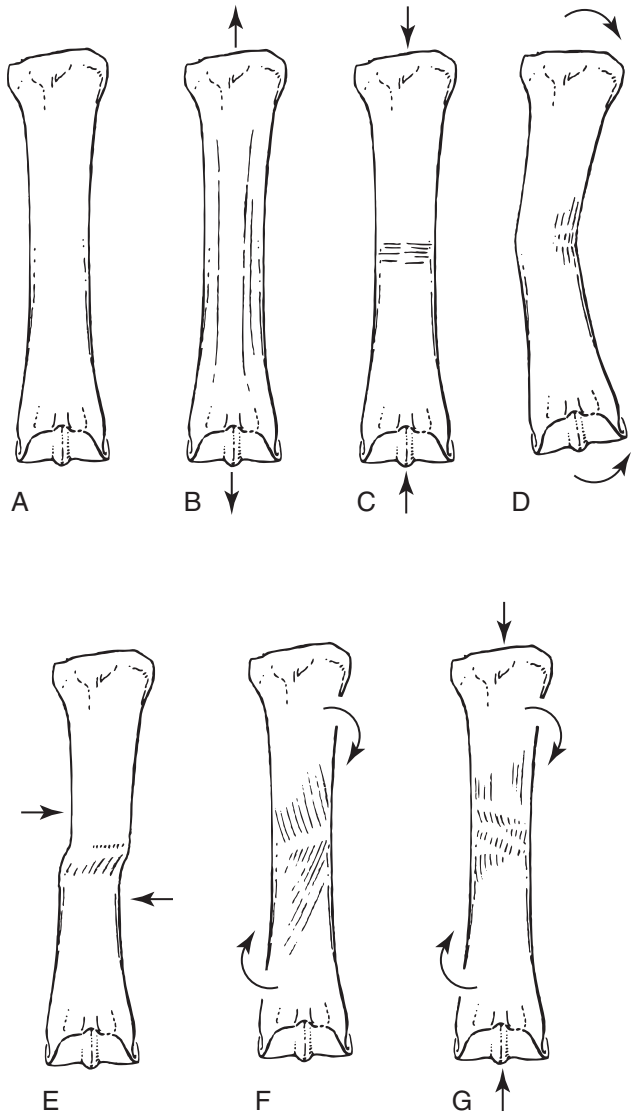

Figure 74-6. Schematic representations of bone loading conditions. **A,** No load; **B,** Tension; **C,** Compression; **D,** Bending; **E,** Shear; **F,** Torsion; **G,** Combined compression and torsion.

Torsion testing causes rotational displacement of bone ends around a central axis relative to each other and generates torque in the structure (see Figure 74-6, *C*). Shear stresses are distributed over the entire bone, and their magnitude increases proportionately with increasing distance from a neutral axis, usually located at the central axis of rotation. Specifically, the shear stresses are the product of the distance from the central axis and the torque from the applied load divided by the polar moment of inertia of the bone, which in turn depends on the bone structure. Based on beam theory, maximal shear stresses occur in planes parallel and perpendicular to the neutral axis. In turn, maximum tensile and compressive forces occur in mutually perpendicular planes where there are zero shear forces. Taking into consideration the variations attributable to bone anatomy and composition, a bone loaded in torsion generally begins to fail in shear parallel to the neutral axis of the bone and continues to fail along the tension plane, around 45 degrees offset from the neutral axis. The pattern continues along the length of the bone to produce a spiral fracture.

Bending results in a combination of tension and compression (see Figures 74-6, *D*, and 74-7, *D*). Tension predominates on one side of the neutral axis and compression occurs largely on the side directly opposite. As stated earlier, the neutral axis is a plane within the bone that does not experience any stresses, and the greater the distance from the neutral axis, the larger the tensile and compressive stresses. Three- or four-point bending is often applied experimentally to test the bending properties of bone (Figure 74-8). As indicated by the names, the differences are in the number of force application points, three or four, which are typically arranged equidistant from one another. For *three-point bending*, three forces produce two equal moments. Each moment is the product of one of the two peripheral forces and its perpendicular distance from the middle force. A typical three-point bending fracture in a horse occurs at the top of a cast or when a horse steps in a hole and the limb hits the top edge. Failure typically begins on the tensile surface of the bone and propagates along the maximum tension plane toward the compressed surface until the maximum shear force at about 45 degrees from the neutral axis is reached and exceeds the tensile forces and the fracture continues along them. The typical fracture contains a butterfly fragment on the compressive side. *Four-point bending* describes two force couples producing two equal moments. The bending moment is the same in the areas between the force couples. Four-point bending rarely results in a fracture clinically, but it is often used for bone testing. Because

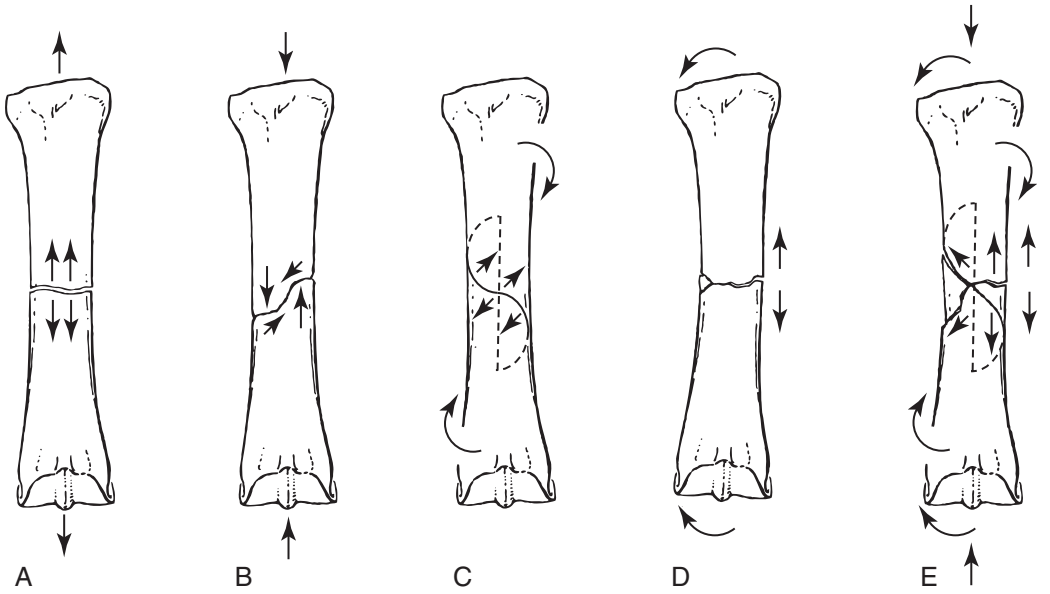

Figure 74-7. Long bone fracture configurations associated with specific force application. **A,** Transverse fractures are characteristic of pure tension. **B,** Oblique fractures occur in compression. **C,** When loaded in pure torsion, fractures initiate parallel to the bone surface and then propagate along the oblique tensile forces to give a spiral fracture configuration. **D,** In pure bending, the bone initially fails in tension and then continues along shear planes, resulting in a butterfly fragment on the compression surface. **E,** In combined compression and rotation, comminuted fractures with characteristics of both loading conditions can occur.

the bending moment is uniform between the two central force couples, bone fractures at the weakest point between the two.

Shear loading refers to application of loads applied parallel to one surface of a bone. A bone subjected to shear loading experiences shear stress and strain and deforms in an angular manner (see Figure 74-6, *E*). Shear loading should not be confused with shear stresses that are produced by the angular deformations caused by the loading mechanisms discussed earlier. Physeal fractures are examples of fractures that result from shear loads.

Bone is rarely loaded in one direction but usually experiences multiaxial loads.[33] Hence the study of *multidimensional loading* on equine bones is essential to predict, prevent, and stabilize fractures.[34] *In vivo* bone loading can be difficult to assess given typical bone anatomy and that loading is affected by a multitude of factors including gait, animal signalment, and ground surface, among others. Multiaxial loading applies the same concepts as uniaxial loading, but outcomes are assessed in two or three dimensions. Three-dimensional analysis generally provides the most realistic assessment of the complexity of bone-loading behavior. Analysis is sometimes simplified with assumptions of symmetry and/or isometry in one or more planes.

Bone anatomy contributes significantly to the structural mechanical properties of whole structures. The overall amount of bone based on cross-sectional area and volume, the relative distribution of bone throughout the structure, and the proportion of cortical versus cancellous bone all influence the structural properties. The cross-sectional area of bone contributes to stiffness and strength characteristics. When comparing two bones with identical anatomy, the bone with the greater surface area usually has higher stiffness, yield, and failure point. The *area moment of inertia* is a value that takes into account the contributions of surface area and bone distribution around the neutral axis to resist deformation with higher values corresponding to higher resistance. A *bending moment* is equal to applied force multiplied by length. Hence, longer bones tend to experience higher moments, or internal loads, from the same force given the greater distance between force-application points.

The response of bone to load depends on the rate of application of the load. This is related to the viscoelastic properties of bone. As bone deforms, it stores the applied energy as strain energy, a form of potential energy. The energy is released when the bone fractures. Largely because of its microstructure, bone is stiffer when loaded at a faster rate, and hence it stores more energy prior to failure compared to slower loading rates. Therefore, a *bone loaded rapidly fails at a higher load and releases more energy than if it is loaded slowly*. At a lower loading rate, bone is less stiff, fails at a lower load, and stores less energy prior to failure. High-energy fractures have significant communition and soft tissue injury, whereas low-energy fractures have little comminution and less damage to surrounding structures.

Fractures can occur as a result of a single incident or secondary to repeated loading cycles. Bones that fail secondary to *repeated cyclic loading* are typically viewed as failing secondary to fatigue. Under cyclic loads, the susceptibility of bone to fracture is related to its crystal structure and collagen orientation reflected by its viscoelastic properties. Cortical bone is particularly vulnerable to tensile and compressive cyclic stresses. Fatigue load under certain strain rates can cause progressive accumulation of microdamage in cortical bone. If the process continues, the bone may eventually fail through coalescence and propagation of the cracks. Although cortical bone is relatively brittle and has poor fatigue resistance *in vitro*, it is a living tissue that can remodel and repair during and after loading, as occurs in bucked shins in horses. Periosteal new bone formation near microcrack formation can arrest crack propagation by reducing the stresses encountered at the tip of the crack. In a

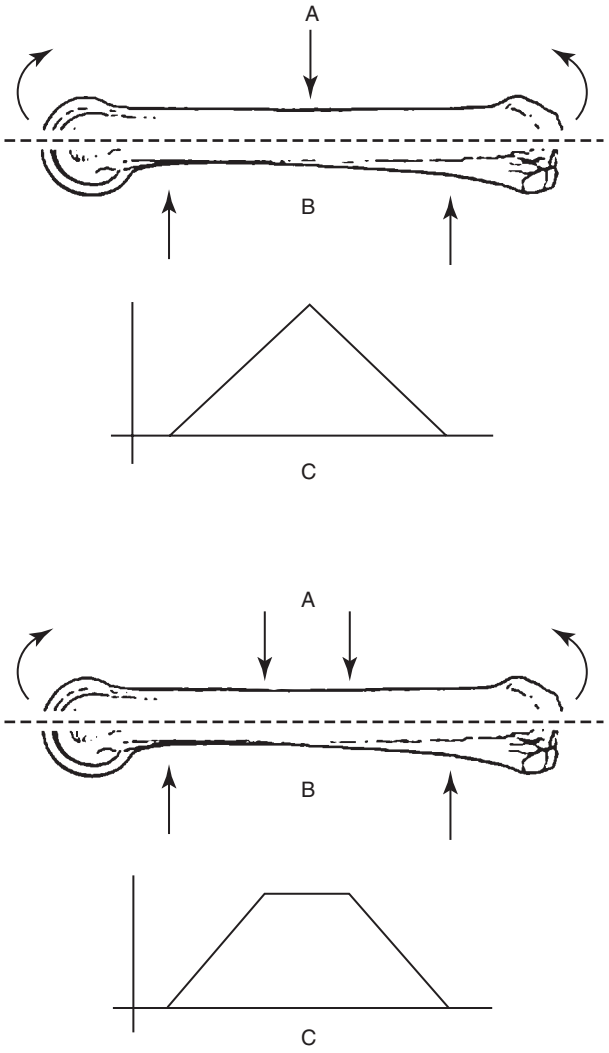

Figure 74-8. Schematic representation of three-point *(top)* and four-point *(bottom)* bending. Three-point bending consists of three forces acting on a bone to produce two equal moments. Each moment is the product of one of the two peripheral forces and its distance from the middle point of force application. Four-point bending occurs when two force couples act on a bone to produce two equal moments. The bending moment magnitude is the same between the central force couples. *A,* Compression; *B,* tension; *C,* bending moment (Nm).

fatigue test, the endurance limit of a bone is the stress level under which no fractures can develop regardless of the number of loading cycles applied. Therefore, for the repair process to be effective following fatigue-induced microcracks, relatively low loads must be present and be maintained to allow sufficient time for the repair process to occur.

BIOLOGICAL REACTION AND HEALING OF BONE

Fracture healing is a highly orchestrated series of complex biological events driven by intracellular and extracellular signaling that follows a defined temporal sequence. Molecular mechanisms that regulate embryologic skeletal development are recapitulated during the process of fracture repair.[35] Local and systemic factors including growth and differentiation factors, hormones, and cytokines interact with primordial cells recruited locally or from the circulation. Research provides significant understanding of the molecular mechanisms of the bone healing process.

Fracture healing is divided into direct (primary) and indirect (secondary). *Direct fracture healing* occurs when there is anatomic reduction of the fracture fragments by rigid internal fixation that minimizes interfragmentary strain. The process involves direct regeneration of the Haversian system between abutted fracture fragments to restore mechanical integrity. Healing occurs by direct growth of secondary osteons from one fragment to another and by intramembranous bone formation. Vascular endothelial cells and perivascular mesenchymal cells provide osteoprogenitor cells that differentiate into osteoblasts. During the process, little or no periosteal callus is formed. *Indirect fracture healing,* most common in horses, occurs through the process of endochondral bone formation when the fracture fragments are not sufficiently immobilized or approximated for direct healing (Figure 74-9).

Fracture healing can be described as a series of biological stages (inflammation, repair, and remodeling) that follow each other in sequence, with some degree of overlap.[36] Like all repair responses, fracture healing begins with induction of an immune response.[37] Cytokines, platelets, bone morphogenetic proteins (BMPs), and mesenchymal stem cells (MSCs) are key to this stage that begins immediately after fracture and lasts between 2 and 3 weeks, the shortest phase of the repair process.

During the initial *inflammatory stage,* lysosomal enzymes from disrupted osteocytes trigger the destruction of the organic matrix, and the necrotic material induces an intense inflammatory reaction. Acute-phase proteins activate proteolytic enzyme cascades. Cytokines secreted by inflammatory cells recruit MSCs and have a chemotactic effect on other inflammatory cells. A hematoma forms, and attached platelets are activated by thrombin and subendothelial collagen to release platelet-derived growth factor (PDGF) and transforming growth factor-β (TGF-β). The proteins induce MSC migration, activation, and proliferation, induce platelet aggregation, and have a chemotactic effect on inflammatory cells. At the same time, BMPs are released from bone matrix and expressed by MSCs.[38] The earliest soft callus is rich in type III collagen. The stromal cells proliferate and differentiate into chondrogenic and osteogenic lineages within the matrix template for the calcified callus. Additionally, *angiogenesis*, a prerequisite for continued regeneration, occurs during this early phase and is regulated by such factors as fibroblastic growth factor (FGF), vascular endothelial growth factor (VEGF), and angiopoietin 1 and 2.[39] The initial blood supply to the periosteal callus originates from the surrounding soft tissues. It is transient and distinct from periosteal arteries. In spite of the initial vascular proliferation, the callus is relatively hypoxic. As healing progresses, the extraosseous blood supply diminishes.

The *repair phase* follows and overlaps with the inflammatory phase when the majority of the callus consists of unmineralized matrix. The matrix subsequently mineralizes to form *woven bone*, a form of bone that has randomly distributed collagen fibrils and is deposited only during initial bone formation and fracture repair.[40] Bone formation in the callus proceeds by both intramembranous and endochondral ossification, the relative amount of each depending on the degree of immobilization of the fracture ends and physiologic loading. Endochondral ossification initially occurs adjacent to the fracture site periosteum.

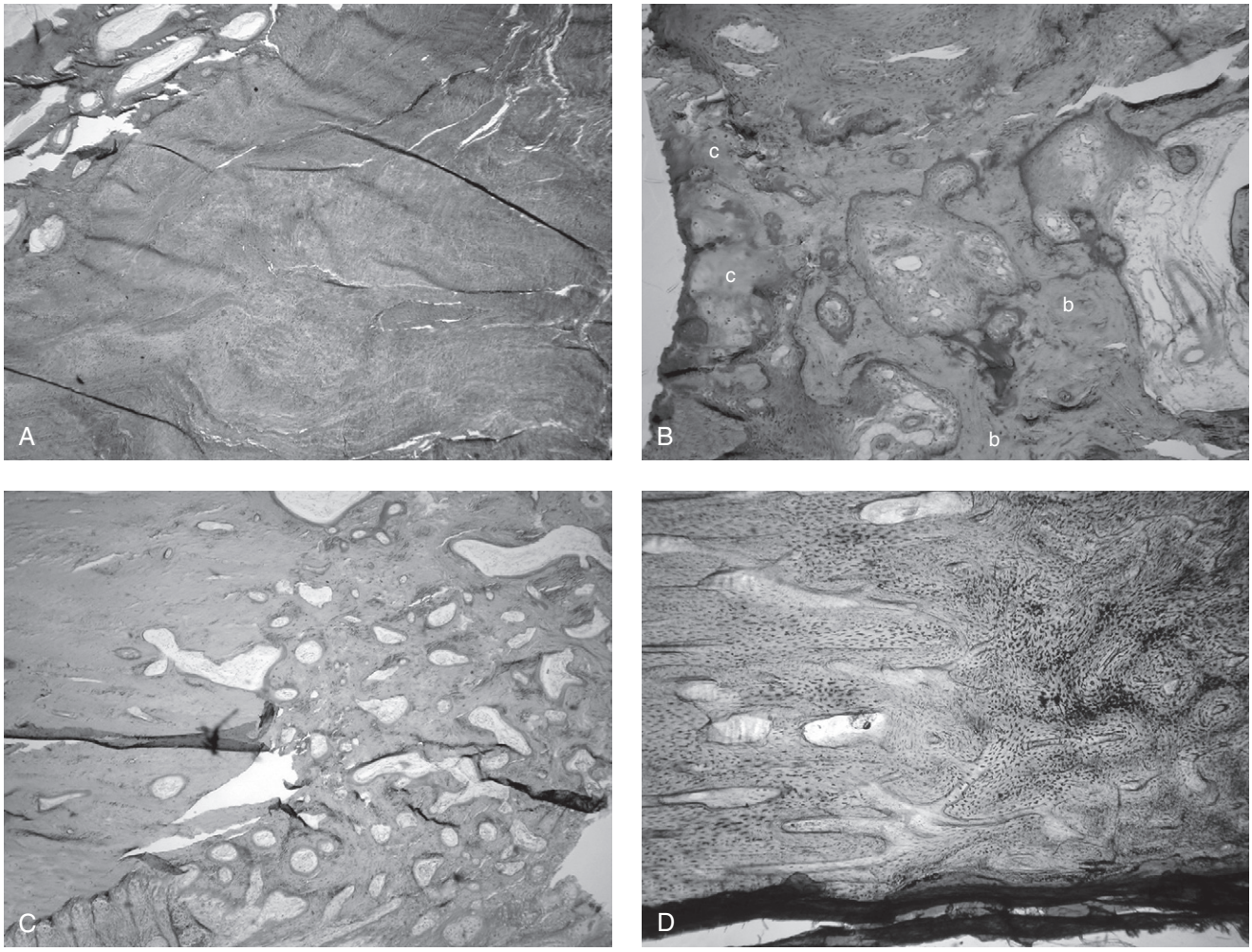

Figure 74-9. Light microscopy of 5 μm undecalcified specimens of a fracture gap (Goldner, ×20). **A,** Two weeks after fracture when the gap is filled with undifferentiated tissue. **B,** Four weeks after fracture with islands of cartilage *(c)* and woven bone formation *(b)*. **C,** Eight weeks after fracture with bone filling the gap. **D,** Twelve weeks after fracture with cortex almost entirely reconstituted, although it is still significantly more porous than normal cortical bone.

Within days after fracture, recruited MSCs begin to proliferate and subsequently differentiate into chondroblasts. The cells synthesize and secrete cartilage-specific matrix including type II collagen and proteoglycans. When mechanical stability is established, the cartilage undergoes hypertrophy and mineralization in a spatially organized manner. As vessels invade, the calcifying, hypertrophic chondrocytes are removed by chondroclasts and woven bone formation occurs by MSCs that have undergone osteogenic differentiation. Type I collagen is superimposed on the early cartilagenous matrix, and types II and III are removed. Crystals of calcium hydroxyapatite become clustered around the fibrils, eventually forming a rigid callus. Blood vessels begin to pass through the fibrous callus to unite the vasculature of the fragments. Eventually, the soft callus is replaced by mineralized callus and woven bone. Bone mineralization always begins on a solid surface that can include the external or internal surfaces of the bone ends at the fracture site and the surfaces of larger fragments. Formation of a bony bridging callus is the final step in the reparative phase of fracture healing and is considered the point of clinical union. The mineralized callus then undergoes the process of remodeling. The repair phase can take between 2 and 12 months.

The *remodeling phase* is the third and longest stage of fracture healing, when mineralized cartilage continues to be replaced by woven bone, which is remodeled into *lamellar bone*. Lamellar bone is the most abundant form of bone in most mammals. Sublayers of mineralized collagen fibers compose individual lamellar units. The sublayers are variably oriented relative to each other to create a complex, layered structure. Osteoclasts remove woven bone to create bone tunnels known as *cutting cones,* which are filled with lamellar bone around a central capillary channel by osteoblasts (Figure 74-10). Bone remodeling is largely regulated by the mechanical environment and the resultant piezoelectric charges generated in the crystalline bone. Weight-bearing stresses cause concave surfaces to become electronegative and convex surfaces to be electropositive. Osteoblastic activity is enhanced on electronegative surfaces and osteoclast activity is higher on electropositive surfaces. This activity is part of Wolff's law. The final result of the remodeling stage is regenerated bone with organic and mineral phases suited and aligned to resist physiological stresses and strains.

The ultimate goal of fracture healing is to reconstruct the original cortical bone. The cortical ends of the fracture site become avascular and necrotic immediately following fracture.

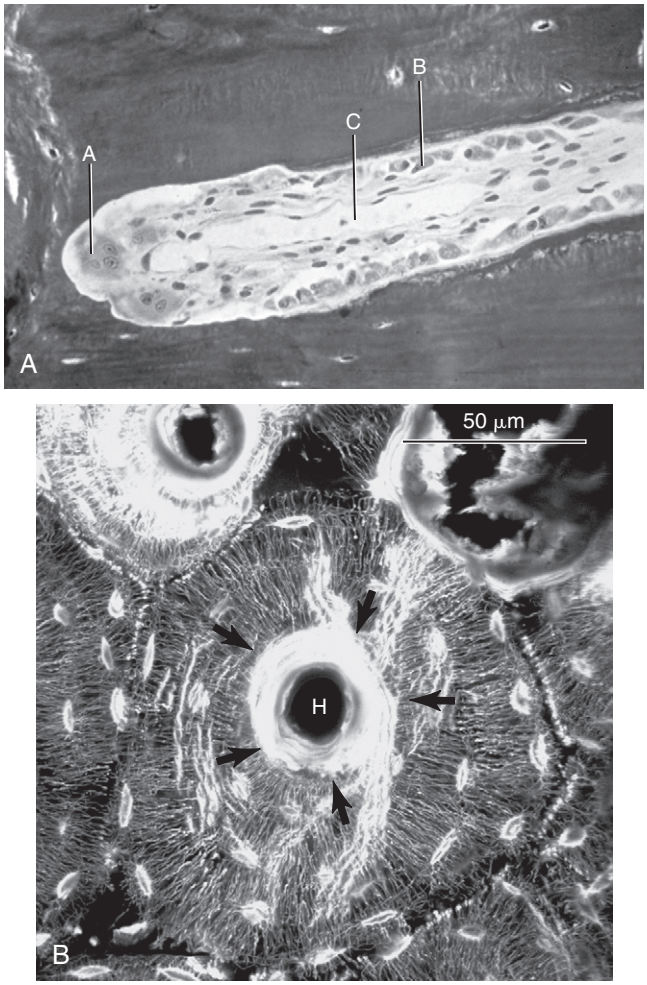

Figure 74-10. A, Photomicrograph of a longitudinal section of a bone metabolic unit (BMU) depicting osteoclasts *(A)* at the tip and the corresponding "Howship lacunae," the osteoblasts *(B)* lining the peripheri of the BMU and the centrally located blood vessel *(C)* in the Haversian canal. **B,** Gray-scale composite of Z-stacked confocal photomicrograph of a BMU in cross section depicting the central Haversian canal *(H)* surrounded by brightly fluorescing mineralized matrix *(arrows)* recently deposited in a cutting cone. Scale bar equals 50 μm. (**A,** Courtesy R. Schenk, Bern. **B,** Courtesy Peter Muir, University of Wisconsin, Madison.)

Fortunately, this vascular compromise does not prevent the avascular ends from providing important biomechanical roles by serving as supportive elements for fracture-fixation devices. Haversian remodeling of the uniting fracture serves two principal functions, including the revascularization of necrotic bone at the fracture site and the reconstitution of the interfragmentary gaps. There are three specific requirements for Haversian remodeling to occur across a fracture site. These include adequate reduction, rigid fixation, and sufficient blood supply. Haversian remodeling of the fracture typically begins in the second to third month after fracture healing. It is unknown exactly what factors initiate the dramatic increase in secondary osteon formation that occurs during fracture healing, although it has been postulated that the activation of Haversian remodeling is related to tissue damage at the fracture site.

Progression of a secondary osteon from one fracture fragment to another does not require intimate contact of fracture fragments. Even with perfect reduction and rigid internal fixation, there are incongruencies at the fracture site that result in small gaps between areas of contact. These gaps are typically filled within weeks after the fracture occurs by direct lamellar or woven new bone. Secondary osteons use this gap tissue as a scaffold to grow from one fragment to another. Although the growth and progression of secondary osteons are critical for final union, their growth typically results in a transitory reduction of cortical bone density because of the increased porosity. It is apparent that fracture healing progresses through a variety of pathways that are strongly influenced by a number of factors. Fortunately, the process tends to have a high level of tolerance and adaptability.

EVALUATION OF HEALING FOR CLINICAL JUDGMENTS

Determination of effective osseous fracture union can be a diagnostic challenge. Clinical assessment requires a comprehensive consideration of numerous factors including the initial injury, standard healing period, fracture site pain, palpable callus, lameness, and stability on manual examination.

Radiographic evaluation is the standard modality to assess fracture healing. Secondary bone healing is the most common mechanism of equine fracture healing, and radiographic changes coincide with the three phases of fracture repair described earlier. Up to a week following the initial trauma, sharp fracture margins are typically visible.[41] After the inflammatory phase is established, necrotic bone is removed by osteoclasts and phagocytes. During this period, approximately 2 to 3 weeks after fracture, *the fracture gap appears to widen* and develops an indistinct margin. During the ensuing repair phase, periosteal, endosteal, and intercortical *calluses become evident*, though they vary in radiographic visibility depending on their degree of mineralization; which, in turn, depends upon local blood supply and tissue oxygenation. Initial callus formation occurs within 5 days after trauma, but bony callus requires significant mineralization to be evident radiographically. As healing progresses, fracture lines disappear and the external callus increases in opacity relative to adjacent bone. The size and shape of an external callus is significantly influenced by motion at the fracture site, with greater motion resulting in a larger callus. In the remodeling phase, the woven bone of the callus transitions to lamellar bone. As remodeling progresses, the callus assumes the original bone configuration. Traditionally, bony union is characterized radiographically by a bony callus, obliteration of the fracture line by bony trabeculae, and cortical bone bridging of the fracture gap.[42]

Methods to improve evaluation of equine bone healing are an area of ongoing research. Nuclear scintigraphy is one modality that has been used to evaluate equine fracture healing.[43-45] It is advantageous in that it provides a clear assessment of fracture vascularity for prognostic purposes. Both computed tomography and nuclear scintigraphy are used to detect and assess equine fractures.[43,46-49] Dual x-ray absorptiometry (DEXA), magnetic resonance imaging (MRI), and ultrasonography are popular modalities to evaluate fracture healing in human and small animal patients, but they are less routinely applied to the horse.[50-52] Some fractures, such as incomplete metacarpal stress fractures, are best identified and monitored with MRI.[53] Methods to enhance traditional radiographic assessment of equine fracture healing like MRI and ultrasonography are becoming more

common with development of equine-specific equipment and methodologies. Combinations of various imaging modalities can augment fracture diagnosis and determination of healing progress. Radiography and ultrasound are used to assess some fractures and their healing, especially those that have articular components or are in bones that are difficult to isolate radiographically such as the pelvis and scapula.[54-56] A combination of complementary modalities allows optimal assessment of some fractures.

COMPLICATIONS

Equine fracture healing complications may include infection, fixation failure, delay or failure of bony union, laminitis, and, in foals, contralateral angular limb deformity. These broad generalizations simply represent some common problems associated with fracture repair. Each fracture and patient is unique and subject to individual variation and associated complications. Complications should be specifically addressed as they arise.

Perioperative antibiotic administration and improved surgical techniques and equipment have significantly reduced the incidence of nosocomial equine orthopedic infections, but severe soft tissue damage often associated with the initial injury can result in exposed bone and severe contamination. In spite of appropriate surgical débridement and antibiotic administration, compromised blood supply, swelling, edema, and necrotic tissues make an ideal environment for bacterial proliferation. Local infection can easily proceed through the cortex, marrow, and articulating surfaces of injured bones. Based on a recent retrospective study, *closed fractures* are 4.23 times more likely to remain uninfected, and these patients are 4.59 times more likely to be discharged from the hospital than those with open fractures.[57] Additionally, *closed reduction* and internal fixation is associated with a 2.5-fold reduction in rate of postoperative infection and a 5.9 times greater chance for discharge from the hospital compared to open reduction and internal fixation. Treatment of osteomyelitis following fracture stabilization requires individualized assessment and therapy. Additional information on bone infection and its treatment can be found in Chapters 7 and 85.

There are many factors associated with delay or failure of fracture union including, but not limited to, fracture and incisional infection, inadequate reduction and immobilization, and soft-tissue disruption. Long bone fractures are expected to heal within approximately 4 months in adult horses and 3 months in foals, but there is no definite timetable to define delayed union or nonunion. For delayed union and nonunions, potential systemic conditions that may inhibit normal bone formation should be ruled out. Healing progresses, but at a slower rate than normal in cases of *delayed union*. Radiographic signs may include a persistent fracture line, minimal callus, and intramedullary sclerosis. Treatment depends on the suspected cause of the inadequate healing response. Noninvasive mechanisms to stimulate the healing response have variable success. Improved stabilization, fracture dynamization, and/or surgical débridement with implantation of osteogenic substances may be employed to stimulate the healing process. *Nonunion* occurs when fracture repair ceases before the bony structure is restored. Radiographic findings may include lack of bridging bone or callus across the fracture site; sclerotic, blunt fracture edges; persistent fracture lines; and lack of progressive change toward union on serial radiographs. There are many types of nonunion including hypervascular, oligotrophic, avascular, comminuted, and defect. *Hypertrophic* nonunions, in contrast to the other types, have abundant blood supply and exuberant callus (Figure 74-11). They are typically treated with improved stabilization. The other forms of nonunion require steps to address the nonviable and/or missing tissue. Typical treatment consists of débridement and addition of graft or other substances to stimulate osteogenesis and provide a bony template for bone ingrowth as appropriate. Additionally, careful consideration of fracture stability is necessary.

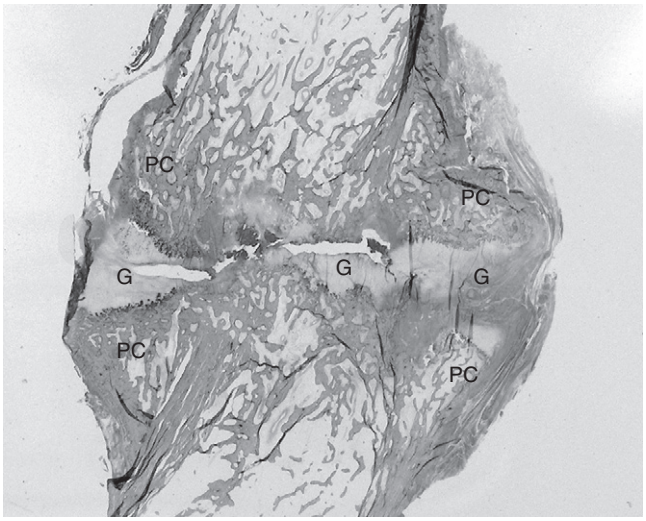

Figure 74-11. Light microscopy of an undecalcified specimen illustrating hypertrophic non-union (Goldner, ×4). Note abundant periosteal callus *(PC)* without bridging of the fracture gap *(G)*.

Stress-induced laminitis is a compounding factor in many fracture repairs. The basic cause of the condition is related to continued severe lameness after fracture fixation. The primary goal of any form of fracture stabilization is return to weight bearing on the affected limb as quickly as possible. In spite of appropriate, stable reduction and analgesic administration, continued pain in some fractured limbs results in increased weight bearing on the contralateral limb. Stress-induced laminitis generally does not have a metabolic component. It can result in rotation and even sinking of the distal phalanx as the germinal layers separate from the horny tissues within the hoof capsule. In contrast to adult horses, foals are rarely affected by laminitis, but instead can develop angular limb deformities in the uninjured limb if severe lameness persists in the fractured limb as a result of the prolonged tripod stance.

Fixation failure can occur for any number of reasons in the horse, not the least of which are forces inherent to the equine limb. Use of an equine external skeletal fixation device designed specifically for horses to bear full weight immediately in cases of severely comminuted fractures of the distal aspect of the limb fracture historically was complicated by fractures of the third metacarpus through pin holes in up to 22% of cases.[58] New device designs have significantly reduced fracture complications, but external coaptation is often applied after the device is removed.[58] Hybrid external fixators are used on occasion to stabilize fractures in foals and ponies.[59] External coaptation is not routinely used alone for fracture stabilization but is used more often to protect surgical implants in the postoperative period or to protect fractures after implant removal. Transfixation casts

are used on occasion, but they are typically reserved for foals or fractures distal to the carpus or tarsus in adult horses.[60] Cast complications, pin breakage or loosening, pin hole fracture and lysis, and pin track infections are reported problems.[61] Surgical implant complications are often associated with inadequate fixation rigidity compared to the mechanical demands placed upon the repair. Construct failure may occur when forces are experienced that exceed implant strength such as during recovery, or when the period of bone healing is longer than the implant's fatigue life. Implant loosening as a result of localized implant or bony failure can result in an unstable fracture and delayed healing. Fracture instability related to inadequate fracture fixation technique is often associated with an unsuccessful outcome.[62] Although no amount of caution can prevent all fixation-associated problems, appropriate preoperative planning, optimal fracture stabilization, and supportive postoperative care improve chances of success considerably.

DISTRACTION OSTEOGENESIS

Distraction osteogenesis or callostasis is a technique used to correct skeletal deformities. It involves forming new bone by controlled separation of stabilized bone ends on each side of a corticotomy or osteotomy.[63,64] Application of gradual traction promotes regeneration and progressive growth of bone and soft tissues at a rate that is well tolerated by the tissues, resulting in minimal trauma. To achieve bone formation, there must be adequate fixation stability, periosteal and medullary blood supply, minimal soft tissue disruption, and physiologic use of the bone.[65,66] Bone formation also depends on the appropriate distraction rate and rhythm, length of distraction and number of distraction increments every 24 hours. New bone, referred to as *regenerate bone,* forms according to a general principle called *the tension-stress effect*.[67] Specifically, early callus tissue subjected to slow steady traction is metabolically stimulated to differentiate and proliferate in the distraction gap.

The process of distraction osteogenesis is divided into three periods; latency, activation, and consolidation.[63,64] The *latency period* is the time between bone transection and the initiation of distraction, which is postulated to enhance cellular and vascular responses and improve osteogenesis. The *activation period* is characterized by bony in-growth as distraction is applied. Within 3 to 7 days after bone transection, osteoblast or osteoclast progenitor cells on the bone surface differentiate according to the mechanical and biological environment and begin to proliferate. The generally accepted distraction speed is 1 mm/day. This can be divided into two or three segments to be applied during a 24-hour period. Osteoid is laid down in parallel columns extending centrally from each bone surface.[67] Lamellar bone typically develops within the columns, but fibrous tissue can form if the fracture site is sufficiently unstable. The distraction device remains in place to serve as rigid skeletal fixation until maturation of new bone is achieved during the *consolidation period*. Metaphyseal cancellous bone heals differently than cortical bone because cancellous bone is inherently more stable.[68] Periosteal callus formation does not occur unless there is significant instability. Instead, osteoblasts deposit new bone on trabeculae, and the fracture gap fills with woven bone, bridging the trabeculae prior to cortical union. As with any fracture, distraction osteogenesis must be based on the patient's age and health, the bone involved, the osteotomy type and location, and the degree of soft tissue trauma.

Distraction osteogenesis has limited potential for equine application. Circular external fixator strength is usually inadequate for stabilization of equine long bones with the potential exception of small foals. Successful correction of a deviated nasal septum and premaxilla in a yearling filly and mandibular brachygnathia in a yearling colt has been accomplished using this technique.[69-71] With careful case selection and appropriate external or internal fixator construction, distraction osteogenesis may be a useful treatment in certain equine patients.

STIMULATION OF BONE HEALING

Enhancement of fracture healing for rapid restoration of skeletal function will greatly benefit the equine patient. Both biological and mechanical methods to accelerate the process of bone repair have been evaluated in the horse. Historically, autogenous corticocancellous bone grafts have been the gold standard to facilitate bone repair, though both autogenous and allogeneic demineralized bone matrix and autologous cortical bone have also been used.[45,72,73] A novel application of an allogeneic bone graft in the form of an implant was recently described in a report on the mechanical properties of proximal sesamoid fractures stabilized with cortical bone screws machined from allogeneic cortical bone.[74] Bone grafts may provide cells *(osteogenesis)*, induce bone formation by host cells *(osteoinduction)*, or provide a scaffold for bone formation *(osteoconduction)*. Graft quality is affected by donor condition and comorbidities. Additionally, transmission of pathogens through allogeneic grafts remains a significant concern. Successful graft incorporation requires an appropriate match of the biological graft activity, the surrounding host tissues, and the mechanical environment (for more information, see Chapter 77).

One of the most highly investigated mechanical techniques to stimulate fracture healing in the horse is application of energy in the form of pulsed ultrasound and electric and electromagnetic fields.[75-79] The premise for many of the treatments are based on Wolff's law; living bone adapts its structure in response to mechanical stress by producing electric potentials, which optimize bone growth. There is a large body of evidence demonstrating that metabolic activity and mechanical deformation in living bone generate steady direct current and time-varying electric fields, respectively.[80] Numerous basic research and clinical investigations surrounding stimulation of bone repair with energy application have been conducted across species. Though there are anecdotal reports of treatment success in the horse, clinical efficacy has yet to be clearly established.[75,81,82]

Technological advances are evident in recent research surrounding equine fracture treatment. The three native benefits of bone grafts—osteogenesis, osteoinduction, and osteoconduction—have yet to be fully replicated by a single product. However, research has been focused on the ability to confer all or some of the same benefits by other means. Biodegradable bone cement has been used experimentally to adhere equine bone fragments to parent bone and support fracture healing.[83] Administration of BMPs, other growth factors, and cytokines to fractures via biocompatible carriers, adenoviral vectors, and genetically engineered cells have shown promising results to accelerate equine fracture repair in controlled studies (Figure 74-12).[84-88] Adult mesenchymal stromal cells exhibit osteogenesis *in vitro* and have significant promise to enhance current equine fracture repair strategies. The number of osteoprogenitor

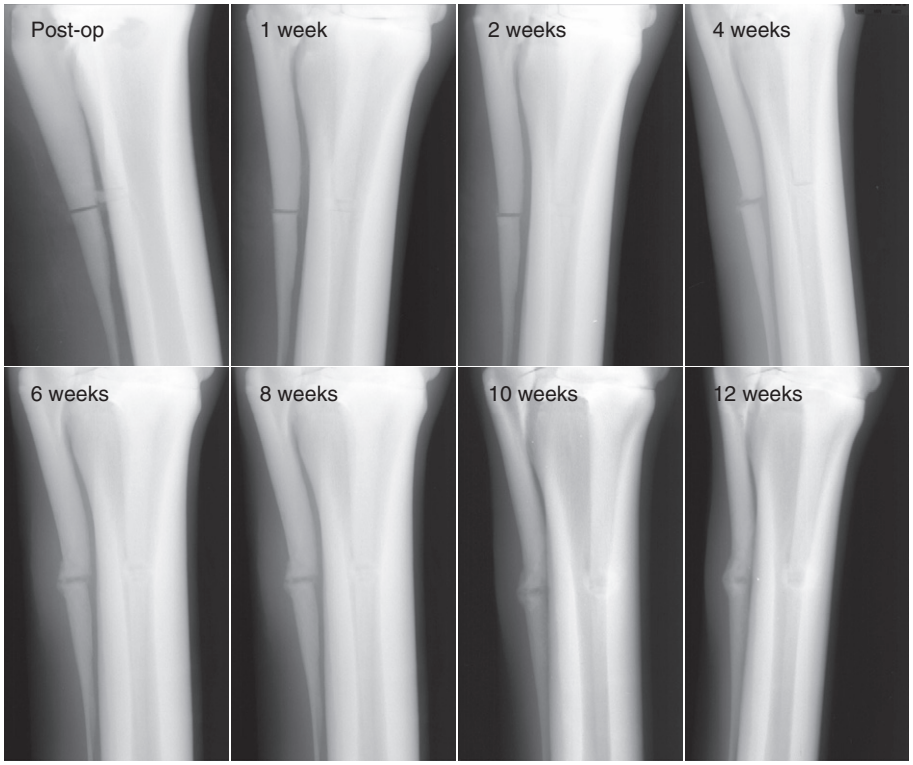

Figure 74-12. Radiographic images of fourth metatarsal osteotomy from immediately postoperative to 12 weeks after surgery following treatment of the gap with rhBMP-2 combined with calcium phosphate cement, demonstrating progression toward union by 10 weeks.[86]

cells varies among donor sites, and the number and multipotentiality of MSCs declines with age. Hence, use of culture-expanded or otherwise purified and concentrated equine MSC populations will likely be necessary to obtain sufficient cells with appropriate osteogenic potential. A number of reports have evaluated the *in vitro* potential of progenitor cells from various equine tissue sources to differentiate toward the osteogenic cell lineages,[89-93] but their *in vivo* use to enhance bone repair following culture expansion or other means of purification is not well established. Continued efforts to optimize fracture diagnosis and stabilization and to enhance the natural repair process will undoubtedly contribute to a better quality of life for equine companions.

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CHAPTER
75 Metallic Instruments and Implants

John A. Disegi

METALLIC INSTRUMENT MATERIALS

Stainless Steel

The types of stainless steel that are used for instruments may be classified according to their microstructure, magnetic attraction, corrosion resistance, and hardness.[1] A general comparison of commonly used stainless instrument materials is shown in Table 75-1.

The term *martensitic* describes the type of microstructure associated with 400 series stainless steel, which is highly magnetic and has moderate corrosion resistance. The martensitic compositions contain 12% to 18% chromium, a medium to high carbon content, and other minor elements. Common types of martensitic alloys of the 400 series of stainless steel include 420A, 431, and 440B; each grade has an equivalent Deutsches Institut für Normung (DIN) designation. The 400 series nomenclature refers to various well-defined alloys, which include compositional trends related to carbon and chromium content. For instance, the suffix letters A, B, or C define an increasing carbon range for a given alloy (420C > 420B > 420A) and the chromium range increases as the three digit 4XX number increases (440 > 431 > 420). The alloys are fabricated into various instruments in the soft condition and are heat-treated to develop full mechanical properties. Heat treating[2] is a hardening process that transforms the microstructure with temperatures in the range of 930° to 1150° C, followed by a controlled quench in air or a liquid. The hardened alloy is then tempered at an intermediate temperature of 150° to 370° C to develop the final optimal properties.[2] For a given alloy, the hardness typically decreases as the tempering temperature increases. Martensitic alloys are very hard and resistant to wear, which makes them ideal for cutting instruments (such as drills, taps, countersinks, reamers, chisels, and bone-cutting forceps) and for use in noncutting tools (such as screwdrivers and wrenches).

Precipitation hardenable (PH) stainless steels contain substantial amounts of chromium, nickel, and copper, plus controlled levels of secondary elements.[2,3] The PH compositions are usually fabricated in the annealed (partially heat-treated) condition, and the heat treatment is finished in a one-step aging treatment that promotes hardening as a result of precipitation of a secondary strengthening phase within the martensitic matrix structure. Various age-hardened techniques (e.g., H900 and H950) are used, and the final hardness is inversely related to the age hardening temperature. PH stainless steels are used for a variety of noncutting instruments that require a moderate hardness level. The PH grades do not contain a high carbon content, so edge retention and wear resistance are inferior when compared with the 400 series martensitic compositions. A commonly used PH-grade alloy contains 17% chromium plus 4% nickel (17-4PH) and is identified as Type 630.

The term *austenitic* describes the type of microstructure associated with 300 series stainless steel, which may be slightly magnetic and has excellent corrosion resistance. The austenitic compositions[3] usually contain 16% to 18% chromium and 8%

TABLE 75-1. Classification of Stainless Steel Instrument Materials

Microstructure	Magnetic Attraction	Corrosion Resistance	Hardness	Common Types
Martensitic	High	Moderate	High	400 series
Precipitation hardenable	High	Moderate	High	PH grades
Austenitic	Low	High	Moderate	300 series

TABLE 75-2. Comparison of Common Stainless Instrument Alloys

Classification	Type	DIN	Condition	Hardness (HRC)
Martensitic	420A	1.4021	Hardened + tempered	47-50
	431	1.4057	Hardened + tempered	41-46
	440B	1.4112	Hardened + tempered	53-56
Precipitation hardenable	630	1.4542	H900	44
	XM-25	—	H900	43
Austenitic	302	1.4300	Highly cold-worked	45
	304L	1.4306	Cold work 50%	33
	316	1.4401	Cold work 50%	37

DIN, Deutsches Institut für Normung; *HRC,* hardness Rockwell C.

to 10% nickel. The low carbon grades, such as 304L (*L* indicates low carbon), meet a compositional requirement of a maximum of 0.03% carbon. Other major elements may be added to improve the microstructure stability and corrosion properties. The 300 series stainless steels may be strengthened by cold working, but they do not have outstanding cutting or wear properties because of the low carbon content. The austenitic alloys, except for 316 and 316L, which contain 2% to 3% molybdenum, also become slightly magnetic as the amount of cold-work increases. Some of the austenitic stainless steels can be cold-worked to a very high tensile strength and may be used for some noncutting applications including drill guides, clamps, hollow sleeves, springs, and washers. Table 75-2 compares some typical stainless steel instrument materials.[1]

TABLE 75-3. Typical Tensile Properties for Aluminum and Aluminum Alloys

Alloy, Temper	Ultimate Tensile Strength* (MPa)	0.2% Yield Strength† (MPa)	Elongation‡ (%)
1100-O	90	35	45
2024-T4	470	325	19
5052-H32	227	193	18
6061-T6	310	276	25

*Ultimate tensile strength: Failure stress (maximum load ÷ cross-sectional area).
†Yield strength: Stress at start of permanent deformation (at 0.2% stress to strain offset deviation).
‡Elongation: A measure of ductility (amount of total extension under load).

Aluminum

Aluminum is available in various purities. An aluminum composition frequently used for bone plate or rod templates is known as *Grade 1100*. This grade of aluminum meets a minimum aluminum content of 99% and is supplied in the soft annealed condition (O temper). Aluminum templates are low in strength, highly ductile (malleable), nonmagnetic, and lightweight. They may be color-anodized to complement color-coded instrument systems. The anodized color produced on aluminum is a result of an organic dye that is infiltrated into the aluminum oxide surface.[4] The surface is then chemically sealed, and other additives may be incorporated into the coating. Additional details regarding surface treatments for aluminum are covered later.

Aluminum Alloys

Aluminum alloys have more strength and less ductility when compared with pure aluminum. The alloys are grouped according to 2xxx through 8xxx identities, depending on the major alloying elements that are present. Aluminum alloy compositions that are frequently used for instruments include 2024, 5052, and 6061. The alloys are usually produced at a designated temper, which covers the heat-treating and/or other finishing operations that define the supplied metallurgic condition. A 5052-H32 designation indicates a 5052 alloy with a H32 temper (strain hardened/stabilized/quarter hard). Another common designation is 6061-T6; the T6 temper refers to solution–heat-treated plus artificially aged condition. Aluminum alloys are used for depth gages, intramedullary (IM) nail-insertion instruments, hollow external-fixation rings, graphic case modules, and screw racks. Table 75-3 shows typical tensile properties for annealed pure aluminum and aluminum alloys with designated tempers.[5]

Aluminum alloys are nonmagnetic and lightweight. Machined or forged aluminum alloy instruments may be given specialized anodizing treatments to provide modified surface characteristics. The anodizing process for aluminum consists of detergent cleaning, rinsing, electrolytic anodizing, dyeing (optional), rinsing, and sealing. Conventional anodized aluminum films may be clear (not dyed) or may be produced in a variety of colors that meet standardized dye color charts. Conventional anodizing increases the corrosion resistance and surface hardness. Another specialized anodizing treatment is known as *hard anodizing* or *hard coat*. Hard anodizing provides an increased surface hardness of around 60 to 65 HRC (Hardness Rockwell C) for improved wear resistance.[4] Hard-anodized films may also be produced that contain a polytetrafluroethylene polymer impregnated into the aluminum oxide film or the polymer may codeposited during formation of the aluminum oxide coating.[6] In both instances, the polymer plus hard coat provides reduced frictional properties and improved wear and galling resistance. *Galling* refers to the adhesive wear that occurs when two metals rub together at high points on their mating surfaces. Chemical cleaning solutions containing chlorine, iodine, or certain metal salts may attack the anodized coating. Aluminum contact with strong alkaline cleaners must be avoided to prevent aggressive chemical attack.

Other

Titanium alloys, such as Ti-6Al-4V or Ti-6Al-7Nb, occasionally are used for noncutting instrument applications. The

nonmagnetic alloys have high strength and low weight, and they may be anodized for color-coded systems. External fixation components have been designed to take advantage of these properties. Cobalt-base alloys have a high modulus of elasticity, which is beneficial for small-diameter guide wires and aiming instruments that require high stiffness.

METALLIC IMPLANT MATERIALS

Commonly used metallic implant materials for fracture fixation include stainless steel, unalloyed titanium (also known as *commercially pure titanium*), and titanium alloys. Some cobalt-base alloys are also used for specialty applications. Standardization of implant materials is covered by the International Organization for Standardization (ISO) and the American Society for Testing and Materials (ASTM). Trade names vary from each supplier, but all implant materials must meet industry requirements for composition, microstructure, mechanical properties, and corrosion resistance.

Stainless Steel

ISO 5832-1 or ASTM F 138 (bar and wire)/ASTM F 139 (sheet and strip) stainless steel is used extensively in the orthopedic implant industry. The iron-based stainless composition is known as *wrought 18% chromium-14% nickel-2.5% molybdenum implant alloy* and it contains minor additions of residual elements.[7] This material is also referred to as *implant quality 316L stainless steel* in the United States. The alloy is composed of chromium to provide corrosion resistance, nickel for microstructural stability, and molybdenum for improved resistance to pitting and crevice corrosion. The alloy contains a low carbon content (maximum, 0.03% carbon) for improved resistance to intergranular corrosion.[8] Special melting techniques ensure that the implant alloy has a low amount of nonmetallic inclusions, no secondary magnetic phases, and elevated chromium and molybdenum levels. The material must meet a compositional index defined by the equation: $\%Cr + 3.3 \times \%Mo \geq 26$ to ensure adequate *in vivo* resistance to pitting and crevice corrosion.[8] For multicomponent devices, such as plates and screws, it is not advisable to mix stainless steel and titanium implants because an accelerated form of corrosion, known as *galvanic corrosion*, can occur.[7] Implant stainless steel may be used in the annealed or soft condition for reconstruction plates that are highly contoured or for cerclage wire that may be subjected to a large degree of twisting and torsional deformation.

The alloy may also be supplied in the cold-worked or moderate-strength condition to resist the stress loading encountered by bone plates and bone screws. *Cold-working* refers to a metalworking operation that permanently deforms the material at room temperature to increase the strength, usually by reducing the cross-sectional area by drawing or rolling. Small-diameter products (such as Kirschner wires, Schanz screws, and Steinmann pins) may be fabricated from extra-hard or highly cold-worked material with a very high tensile strength (greater than 1350 MPa) for improved bending resistance.[9] Table 75-4 shows relative tensile property requirements of stainless steel bar used for these applications according to ASTM F 138 specification.

The excellent corrosion resistance of stainless steel is primarily a result of a chromium oxide film, known as the *passive layer*, which is present on the surface. Chemical passivation in nitric acid is a commonly used method of surface finishing for stainless steel implants.[10] Immersion in 20 to 45 volume-percent nitric acid passivation solution removes surface contaminants, such as cutting tool transfer films, oxide from heat treatment, imbedded particles, and burned-in lubricants. The passivation process restores maximum corrosion resistance but does not affect part dimensions.

Electropolishing is another surface treatment that consists of applying an electric current to an implant immersed in a specially formulated chemical solution under specified conditions of time and voltage.[11] The treatment removes a microscopic amount of metal, decreases the surface roughness of the implant, provides a low coefficient of friction, improves corrosion resistance, and creates a chemically passivated surface (Figure 75-1). Repeated steam autoclaving will not disrupt the passive film or alter the mechanical properties of stainless steel implants. For applications requiring improved fatigue strength, some implants undergo *shot-peening* before electropolishing. The implant surface is subjected to high-velocity impaction by metallic or ceramic particles under well-defined conditions. Shot-peening produces a roughened surface with increased residual compressive stress for enhanced fatigue life.[12] Implant quality stainless steel is completely nonmagnetic in all conditions, and implants will not exhibit torsional movement or displacement during MR scans. Long and thin implants like Kirschner wires and Steinmann pins with specific length-to-diameter ratios may show an RF temperature rise because of ½ wavelength resonance heating

TABLE 75-4. ASTM F 138 Minimum Tensile Properties for 316L Stainless Steel Bar

Condition	Ultimate Tensile Strength* (MPa)	0.2% Yield Strength* (MPa)	Elongation* (%)
Annealed	490	190	40
Cold-worked	860	690	12
Extra-hard	1350	—	—

*Ultimate tensile strength: Failure stress (maximum load ÷ cross-sectional area).
ASTM, American Society for Testing and Materials.

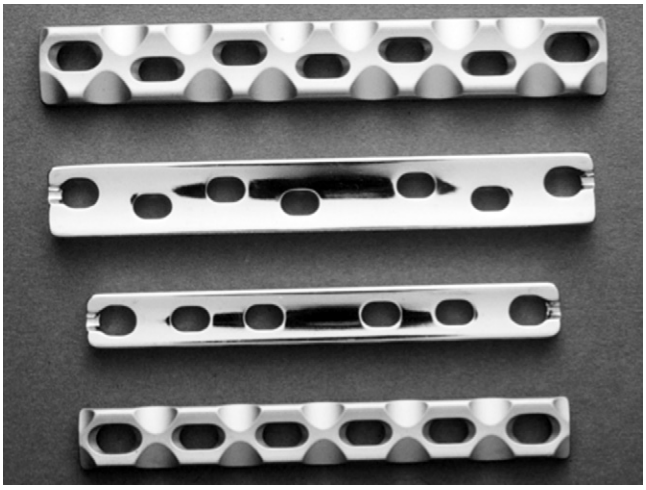

Figure 75-1. Bone plates showing their contact surfaces. The middle two stainless steel plates are electropolished, whereas the top and bottom titanium plates have an anodized surface.

when tested in an MR scanner according to ASTM F 2182.[13] Signal artifacts may obscure complete MR visualization in the vicinity of the stainless implant. The size of the artifact induced by an implant in a specific MR environment has a complex relationship to implant size, shape, and composition.

The 15% nickel content may provoke a *metal sensitivity reaction* and is responsible for about 90% of the metal allergies that are clinically observed in people.[14] Metal sensitivity reactions in animals are not well documented, but the best animal species for predicting human metal sensitivity is the guinea pig when evaluated according to intradermal, topical, or repeated dermal patch-testing methods.

Titanium

Commercially pure (CP) titanium, also known as *unalloyed titanium*, is available in five implant compositions designated Ti Grade 1 ELI, 1, 2, 3, and 4, according to ISO and ASTM implant industry standards.[15,16] The extra-low interstitial (ELI) grade has the lowest content of nitrogen, carbon, iron, and oxygen. Each grade in the annealed condition has a different combination of tensile strength and ductility. The strength increases and the ductility decreases as the grade changes from the lowest designation (grade 1 ELI) to the highest designation (grade 4). Table 75-5 shows the minimum tensile properties for CP titanium bar according to ISO 5832-2. Cold-working may be used to increase the strength of titanium that is designated Grade 4B in ISO 5832-2, but a minimum 10% elongation must be met. CP titanium has better biocompatibility than stainless steel and typically contains less than 0.05% nickel.[17] Unalloyed titanium implants will not cause metal allergy reactions and are recommended for humans in situations where metal sensitivity is preoperatively verified or where 316 L stainless steel implants have provoked an allergic response.[18]

Titanium also exhibits unique biocompatibility properties, which include soft tissue and bone adhesion to the implant surface.[19] A major advantage of tissue integration at the surface has been the possibility of less bacterial colonization and reduced infection.[20] Tissue adjacent to pure titanium implants becomes well vascularized, with less tendency toward capsule formation. The major disadvantage associated with titanium implants is the possibility that implant removal may be difficult, especially for well-osseointegrated implants. The pitting and crevice corrosion resistance of titanium is superior to stainless steel. Titanium implant materials also have a lower density, have a lower modulus of elasticity, and provide significantly less MR artifact when compared to stainless steel.[21] The density of titanium is 57% of the density of stainless steel and represents a weight reduction of nearly 50% when implants of similar dimensions are compared.

The *modulus of elasticity* is a constant physical property that describes the stress per unit strain in the elastic region. A material with a high modulus of elasticity will transfer less stress from the implant to the bone. This may produce a condition known as *stress shielding*, which is not ideal because bone must be adequately stressed to consolidate properly during the bone-healing stage.[22] The modulus of elasticity of titanium is about 55% of stainless steel, and the low modulus is desirable because of increased stress transfer. The modulus consideration is less important for implants with a small cross-sectional area.

Titanium implants may have a special *anodized surface* finish that increases the thickness of the protective titanium oxide passive film. The anodizing treatment for titanium is different from the electrolytic anodizing treatment used for aluminum. The titanium implants are immersed in a chemical solution and a known electrical voltage is applied for a specified time. Visible light diffraction within the oxide film creates a distinct color, which depends on the thickness of the oxide film.[23] No pigments or organic coloring agents are present in the anodized titanium film. The titanium anodizing process is capable of producing a variety of colors that permit the design of color-coded implant systems (see Figure 75-1). Multiple steam sterilization cycles will not significantly change the appearance of anodized titanium implants. However, fingerprint contamination from skin contact should be avoided when handling the implants between autoclave cycles.[18] Gloved handling of anodized titanium implants prevents the discoloration of isolated areas during steam autoclaving.

Titanium Alloys

Titanium-6 aluminum-4 vanadium alloys, which have approximately 6% aluminum and 4% vanadium, are available in two compositions that are identified as Ti-6Al-4V or Ti-6Al-4V ELI. The ELI designation denotes lower oxygen content and lower hydrogen content. Another titanium alloy widely used as an implant material contains titanium, 6% aluminum, and 7% niobium; it is identified as Ti-6Al-7Nb. Unique characteristics of titanium alloys include higher tensile strength capability, lower ductility, similar modulus of elasticity, and equivalent density when compared to CP titanium. Important physical properties[18] of CP titanium, titanium alloy, and stainless steel are compiled in Table 75-6. Data on anodic polarization, pitting, and stress corrosion cracking indicate that Ti-6Al-7Nb is an extremely corrosion-resistant alloy.[24] Unalloyed titanium and titanium alloys have well-documented notch sensitivity

TABLE 75-5. ISO 5832-2 Minimum Tensile Properties for Commerically Pure Titanium

Grade	Condition	Ultimate Tensile Strength* (MPa)	0.2% Yield Strength* (MPa)	Elongation* (%)
1 ELI	Annealed	200	140	30
1	Annealed	240	170	24
2	Annealed	345	275	20
3	Annealed	450	380	18
4A	Annealed	550	483	15
4B	Cold-worked	680	520	10

*Ultimate tensile strength: Failure stress (maximum load ÷ cross-sectional area).
ELI, Extra-low interstitial.

TABLE 75-6. Physical Properties of Metallic Implant Materials

Material	ISO Standards	ASTM Standards	Density (g/cc)	Modulus of Elasticity (GPa)
316L stainless	5832-1	F 138	7.95	186
		F 139	7.95	186
Ti Grade 1 to 4	5832-2	F 67	4.51	103
Ti-6Al-7Nb	5832-11	F 1295	4.52	105
Ti-6Al-4V ELI	5832-3	F 136	4.43	114
Ti-15Mo	—	F 2066	4.96	78

ASTM, American Society for Testing and Materials; *ELI*, extra-low interstitial.

properties. *Notch sensitivity* is a term that describes the relative effect that local irregularities or stress raisers have on mechanical properties.[25] The notch sensitivity resistance of implant-quality stainless steel is similar to unalloyed titanium and is somewhat better than conventional titanium alloys. Titanium-base biomaterials are not classified as notch-sensitive materials on the basis of notch tensile data that have been published.[26] Some newer β-titanium alloys, such as Ti-15Mo, actually have improved notch sensitivity properties when compared to stainless steel. Implant design and manufacturing methods can influence the notch sensitivity resistance, and clinical factors, such as surgical technique and handling, must also be considered.

Titanium alloys may be color anodized in the same manner as CP titanium. The major difference is that the anodized film is an oxide mixture composed of thermodynamically stable oxides (i.e., TiO_2 + Al_2O_3+ Nb_2O_5).[27] Retrieval analysis of human hip joint prostheses concluded that Ti-6Al-7Nb alloy is extremely biocompatible as evidenced by osseous integration at the implant surface.[28]

Cobalt-Base Alloys

Cobalt-base alloys are used primarily for prosthetic implants, such as total hips, total knees, and total disc replacement. Co-28Cr-6Mo is the predominant alloy that is fabricated for joint replacement applications. The nickel content of this alloy is typically less than 0.5%, and nickel sensitivity reactions have not emerged as a clinical problem. This alloy may be *hot forged* into complex shapes, the tensile strength can exceed 1172 MPa,[29] and the wear resistance is outstanding. Overall, corrosion resistance is considered to be superior to stainless steel but inferior to titanium.

Other implantable cobalt alloys have been used for specialty trauma products, such as orthopedic cable, Kirschner wire, and implantable distractor components. Unique properties of cobalt-base alloys include a high modulus of elasticity, which may be 25% greater than stainless steel. Cold-working increases the strength of cobalt-base alloys, and thermal aging heat treatments can significantly increase the yield strength and ultimate tensile strength. Certain cobalt base alloys contain greater than 10% nickel, which may provoke a nickel allergy reaction.[30,31] The excellent corrosion resistance of the cobalt-base alloys is predominantly the result of the chromium content, which typically exceeds 19%. Chromium and cobalt are considered metal-sensitizing agents but the clinical incidence of sensitivity reactions are relatively rare when compared with nickel.[14]

METALLIC IMPLANT FAILURE

Fatigue

Fatigue is a phenomenon leading to fracture under repeated or fluctuating stresses having a maximum value less than the ultimate tensile strength of the material.[32] The fatigue loads are well below the level that normally would be required to cause fracture within a single load cycle. The fatigue cycle is the time interval during which the stress is regularly repeated. One common measure of the fatigue properties is known as the *endurance limit*, which is the maximum stress below which a material can presumably endure an infinite number of stress cycles.[3]

Many material variables influence fatigue test results, such as grain size, processing history, surface finish, and degree of cold-work. Testing variables include type of alternating load, specimen geometry, frequency, and test environment.

A common method of expressing the fatigue properties of a material is known as the S-N curve. The *S-N curve* is a best-fit plot of the individual test values, where *S* refers to the applied cyclic stress and *N* is the number of cycles required to fracture the test specimen or to achieve runout. *Runout* is defined as the maximum cyclic stress that does not create fatigue fracture at a predetermined number of cycles (usually 10^6 cycles or greater). Various types of fatigue tests are configured to evaluate either multiple numbers of a single implant, multicomponent devices, or constructs. Artificial bone materials are usually selected for construct fatigue testing to minimize the mechanical property variability associated with human and animal bones.

Methodology related to *bending fatigue testing* of various types of bone plates is documented in ASTM F 382.[33] A bone plate is placed in a four-point bending fixture and oriented in a manner that mimics clinical bridging of a fracture site. The bone plate is subjected to a constant frequency cyclic load in four-point bending. The test method establishes a uniform four-point bending fatigue test to characterize and compare the fatigue performance of different plate designs. Bending fatigue curves for 3.5 mm DCP and 3.5 mm limited-contact dynamic compression plate (LC-DCP) tested in air at a frequency of 5 Hz with runout defined as 10^6 cycles are shown in Figure 75-2. The bone plate fatigue curves reveal that the DCP has increased resistance to maximum compressive fatigue loads when compared to LC-DCP in the low load–high cycle region (greater than 10^5 cycles). Maximum runout load for the DCP (n = 4) was 323 N, whereas the maximum runout load for the LC-DCP (n = 5) occurred at 268 N.

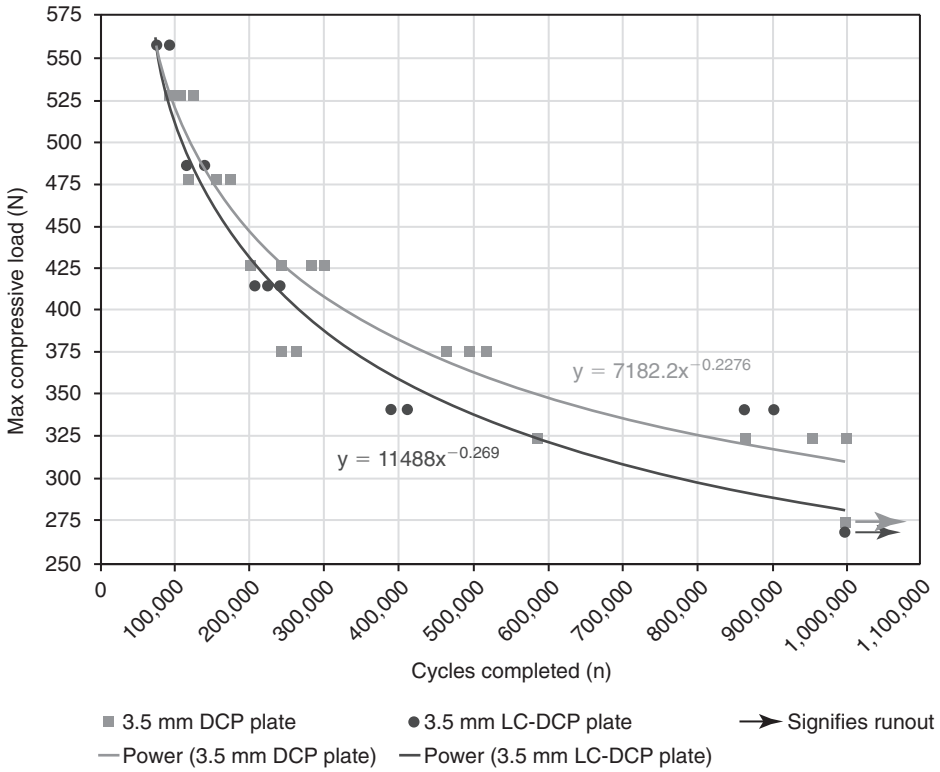

Figure 75-2. S-N four-point bending fatigue curve for 3.5 mm dynamic compression plate (DCP) and 3.5 mm limited-contact dynamic compression plate (LC-DCP).

The endurance limit for 316L stainless steel and CP titanium increases as the percentage of cold-work increases.[7,34] Cross-sectional area is a factor because only small to moderate-size cross sections can be obtained in the highly cold-worked condition. Many fracture-fixation implants require a certain balance between strength and ductility (i.e., ability to contour a bone plate) so that specifying the highest attainable tensile strength is not always desirable. Titanium alloys exhibit a greater 0.2% yield strength when compared with CP titanium, and this factor is primarily responsible for the superior fatigue properties of titanium alloys when compared with CP titanium.

The majority of well-designed metallic fracture fixation and reconstruction implants will not fracture during clinical use, but fatigue failures represent a failure mode that may be encountered. Long bone and spinal implants, especially, may be subjected to relatively high *in vivo* loads after implantation. All load-sharing implants have a finite fatigue life that may be compromised when delayed healing or persistent nonunion are clinically verified. This relationship is often referred to as the race between bone healing and fatigue failure.[35]

Failure Analysis

Failure analysis of retrieved implants is performed by implant device manufacturers to gain a better understanding of the factors responsible for device failure. Clinically retrieved implants are cleaned and disinfected before transport in approved biohazard packages. It is especially important to cover or isolate the fractured implant specimens to preserve the features of the original fracture surfaces. In certain instances, permission to perform destructive testing must be obtained before any failure analysis investigation can be initiated. Light microscopy examination at low-power magnification is used to survey the region of interest. Pitting, crevice corrosion, wear tracks, fretting corrosion,* or other unusual macro-features may be observed during light microscopy examination. Nondestructive portable x-ray fluorescence analysis or conventional destructive analytical techniques can be used to verify the material composition. Metallographic examination may be performed on specimens that are cut adjacent to a fracture surface. Samples are mounted in plastic media, polished, and etched to reveal grain size, secondary phases,† microstructural anomalies, and surface discontinuities. Accurate Knoop or Vickers microhardness measurements on mounted samples may be used to obtain approximate tensile strength values from conversion charts.

Fracture surface examination with the *scanning electron microscope (SEM)* is the primary method for detecting and identifying device failure modes. This is especially true for fatigue failures, which account for the majority of fracture-fixation

*Fretting corrosion is an accelerated form of corrosion that can occur when the protective passive film is mechanically disrupted in a corrosive environment. The relative motion of the underside of a bone screw head with the contact surface of a bone plate is a typical example.

†Implant quality 316 L stainless steel is composed of a single-phase austenitic microstructure, and secondary phases such as delta ferrite, chi phase, and sigma phase must be absent. Delta ferrite is a magnetic phase that is undesirable because of MR scanning considerations, molybdenum-rich chi phase decreases the corrosion resistance of the surrounding austenitic matrix structure, and sigma phase may contribute to embrittlement.

implant failures that are investigated. SEM analysis is generally considered a nondestructive test, but specific geometric features may require cutting a retrieved implant without disturbing the fracture surface so the implant can be inserted into the vacuum chamber. A retrieved 4.5 mm 12-hole broad DCP that fractured during implantation is shown (Figure 75-3). Low-amplitude motion between the underside of the screw head and the bearing surface of the screw hole created a fretting corrosion pattern because of passive film disruption at the screw-plate interface. The amount of fretting corrosion was not excessive and did not appear to be the primary cause of failure. The fracture surface next to the right side of a screw hole is highlighted in the SEM image (Figure 75-4). Multiple slip bands, which are the group of parallel lines observed under an optical microscope that signify plastic deformation due to crystal structure displacement in a definite crystallographic direction, were present near the screw hole, and this suggested that significant plastic deformation occurred before failure. Fatigue beach marks, which appear as concentric clamshell striations, were present and confirmed that fatigue fracture was the primary failure mode. Analysis of the fatigue striation pattern revealed the origin of the crack initiation. The shear lip (the narrow, slanting ridge along the edge of an implant fracture surface) in the upper-right area coincided with the location of the final overload fracture site.

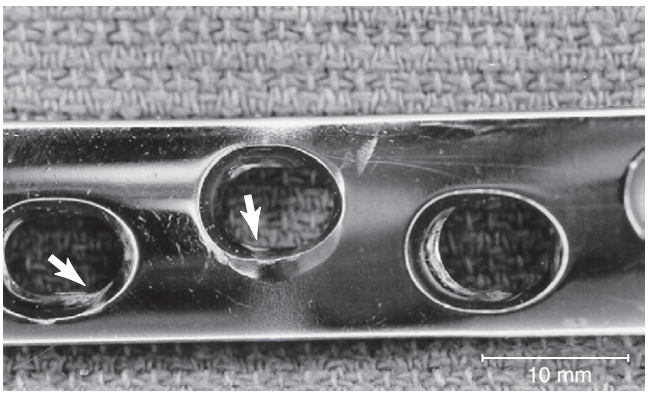

Figure 75-3. Evidence of fretting corrosion around some of the screw holes *(arrows).*

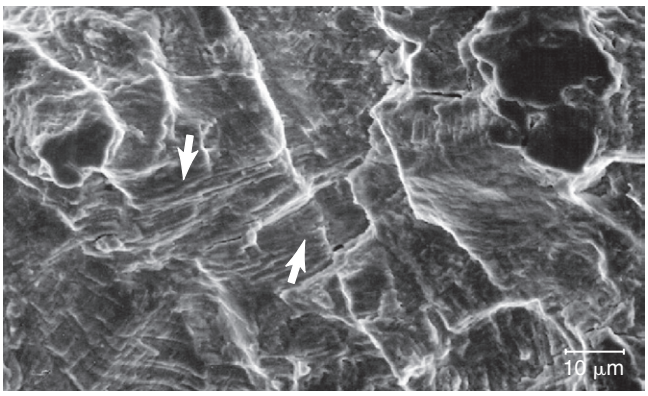

Figure 75-4. The scanning electron microscope shows fatigue striations *(arrows),* which are parallel lines commonly observed on fracture surfaces that are transverse to the direction of local crack propagation.

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CHAPTER 76

Principles of Fracture Treatment

Jörg A. Auer

Fractures are diagnosed in practically every bone of the horse and encountered at all ages. Fractures vary in clinical presentation and significance, ranging from exercise-induced fractures causing only relatively minor lameness, such as chip fractures of the carpus, to fractures causing a non–weight-bearing lameness, such as a transverse failure of the third metacarpal bone (MCIII). The management of intra-articular chip fractures and osteochondrosis lesions is discussed in Chapters 80 and 88, respectively. In this chapter, the nonsurgical and surgical principles of major fracture treatment are presented.

Fracture treatment in the horse follows the same basic guidelines developed for humans[1] and small animals.[2,3] Many techniques can be derived from them, but some principles in the treatment of equine long bone fractures are unique.[4,5] These differences are discussed in detail, with emphasis given to surgical fracture treatment, including external coaptation and internal fixation.

NONSURGICAL MANAGEMENT

Some fractures heal sufficiently with nonsurgical management to allow the animal to return to an athletic career. Nonsurgical management techniques include stall rest and external coaptation.

Stall Rest

Frequently, horses are admitted in a "fracture-lame" state without a visible or palpable fracture. According to the anamnesis, these patients can be found on pasture with the non–weight-bearing lameness, or they do not return to the stable in the evening with the other horses. The physical examination may reveal a small wound over a vestigial metacarpal or metatarsal bone, the radius, or the tibia. In the case of splint bone fracture, radiography may reveal a fracture or even multiple fractures. In selected cases, these fractures are amenable to nonsurgical management with a bandage alone. Additional information on the treatment of these fractures is found in Chapter 93.

Other fractures amenable to management by stall rest include fractures of the deltoid tubercle, nonarticular patellar fractures, and fractures of the scapular spine. However, for the vast majority of fractures, nonsurgical management is not the treatment of choice and should not be advocated.

External Coaptation

For a detailed description of the indications and applications of external coaptation devices, such as fiberglass casts and splints, please review Chapter 17. In this chapter, external coaptation is discussed as it pertains to fracture treatment.

Splints

The indications for limb splints as a sole means of fracture treatment are limited. More commonly, they are used as a form of emergency fixation (see Chapter 73). This type of external coaptation may be employed as treatment modality in fissure fractures of the diaphysis of the radius and tibia or as adjunct treatment to internal fixation of a fracture, either during the immediate postoperative period or as an intermediary step after removal of a cast.

In acute fissure fractures of the radius and tibia, radiography may not show a fracture. Affected animals are usually reluctant to bear any weight on the limb. Initial management should include not only applying a splint bandage but also preventing the animal from lying down. It is frequently during the process of lying down or getting up that these fissure fractures evolve into complete fractures. Tying the horse with a short rope and providing a filled hay net to allow them to eat is an effective management method. Nonsteroidal anti-inflammatory agents are indicated to provide some comfort. However, the dosages should not be so high that they abolish the lameness completely. A complementary or independent management option involves applying a sling or rescue net (Figure 76-1).[5] Usually the animals tolerate these slings very well. They allow the patient to rest their intact limbs for some time by lowering their abdomen into the sling. It is important that the sling be installed

Figure 76-1. An adult horse suffering from a fissure fracture of the tibia, supported by a rescue net. The net, which is applied relatively snugly to allow the horse to apply some weight to it, is tolerated very well.

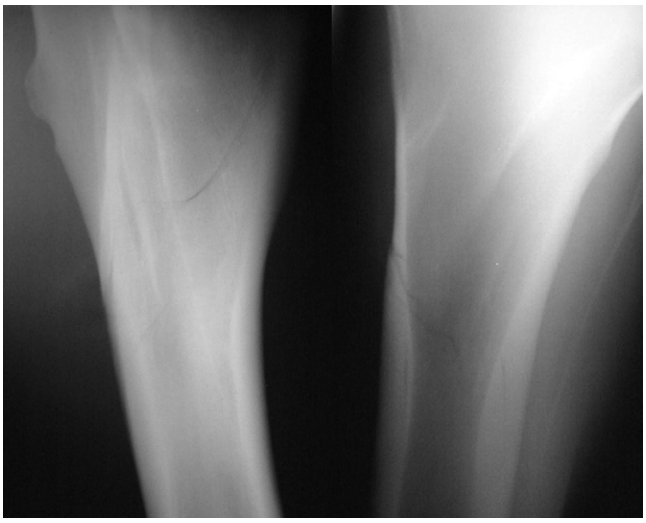


Figure 76-2. Craniocaudal *(left)* and lateromedial *(right)* radiographic views of a fissure fracture in the proximal metaphysis of the tibia of an adult horse.

such that the body has to be lowered only a few centimeters to allow it to rest in the sling. If the body has to be lowered too much, complete fractures of the bone may still occur. The Anderson Sling developed in California has also proved to work very well as a recovery system and for prolonged support of horses during their postfracture fixation period.[6]

If initially no fracture can be found on radiographs, additional images should be taken a few days later. Usually, at this point, fractures may be seen radiographically (Figure 76-2). Depending on the configuration of the fissure fracture and the width of the fracture gap, the management may be modified. When the fissure lines are small with a barely visible gap, no bandages are needed, but the animal is maintained in the sling for 2 to 3 weeks. If the fracture line is long and associated with a significant gap, the splint bandage should be maintained.

Splint bandages should be changed every 3 to 4 days, and in hot and humid climates they should be changed more frequently. Caution should be used during the process because bearing weight on the limb without the splint or bandage could have deleterious effects on fracture healing. Caution: Fissure fractures penetrating a joint should be treated surgically.

Casts

External coaptation using fiberglass cast material as the primary treatment technique of a fracture may initially be considered as a conservative, less expensive type of treatment. However, soft tissue problems may require frequent cast changes, usually carried out under general anesthesia, which frequently increase the costs until they exceed that of a state-of-the-art internal fixation performed upon admission. Additionally, the advantages of early return to function, achieved through internal fixation, are lost. Cast materials selected for fracture treatment should be fiberglass because it allows the skin and the limb underneath the cast to breathe. Also, fiberglass weighs less, so the animal is more comfortable.

Casts should be palpated daily and evaluated for hot areas. Any odor from the cast should be investigated, and weight-bearing on the limb should be evaluated. Sudden changes in weight-bearing patterns, edema above the proximal cast end, and a foul odor or wet spots on the cast are signals of skin damage and possible necrosis underneath the cast. The same is true when hot areas develop in the fetlock region or at the dorsal aspect of the cast in the region of the proximal MCIII or third metatarsus (MTIII). All of these signs signal the need for cast removal.

The first cast should be changed after 3 to 4 days, because during this time initial swelling has subsided, resulting in a loose cast that is ineffective in stabilizing the initial fracture and may even cause some additional damage to the soft tissues. Casts applied to foals should be changed at 10- to 14-day intervals and eliminated as soon as possible. In adult animals, longer intervals are tolerated. If the condition of the skin beneath the cast and the weight-bearing patterns permit, up to 5 or 6 weeks may be allowed before the cast is changed. Longer intervals reduce costs and, in most cases, are followed by better results. Some horses have thinner and more sensitive skin, which is more likely to be traumatized by a cast; in these cases, shorter intervals between cast changes may be required.

Complications

Nonsurgical management of fractures is associated with various complications (see Chapter 17). When internal fixation is not applied, the fracture fragments are not stable. The resulting callus formation often impinges on soft tissue structures or tendons and may prevent future athletic use. However, bone remodeling after fracture healing may reduce such a callus, eliminating impingement on the soft tissue structures.

Skin trauma from casts and splints can be severe enough to endanger the outcome of fracture healing. It is important that the skin remain dry and healthy. Development of cast sores should be prevented whenever possible. Occasionally skin damage occurs at the time of injury or during the transport to the referral clinic, the latter as a result of inappropriate first aid. In thoses cases, special care has to be taken during cast application. If pressure sores do develop, casts must be changed

frequently and padded so that the pressure on the skin is redistributed.

If infection develops underneath the cast, swelling causes increased pressure within the tissues. Because the skin cannot expand beyond the inner limitations of the cast, a compartment syndrome develops, resulting rapidly in tissue necrosis. Additionally, drainage from the limb accumulates within the cast, and the skin is damaged by enzymes. Hosing down the fiberglass cast with copious amounts of water on a daily basis prolongs its usefulness and postpones a necessary change, but an infected limb should be maintained under a cast only if no alternative exists.

During the time that the limb is maintained in the cast, the joints are unable to move and the articular cartilage is poorly nourished. This results in loss of proteoglycans and subsequent degeneration of the cartilage. Additionally, the soft tissue structures surrounding the joint are not flexed and stretched, which causes them to become weak and inelastic. When prolonged external coaptation is used, these pathologic changes are exacerbated and are referred to as *cast disease.*

If a foot is maintained under a cast while a limb fracture is allowed to heal, it is prevented from expanding during weight-bearing and the structures underneath the hoof wall constrict. After the cast is removed and the limb is loaded once again, the foot expands, which causes pain for several days.

Flexing of the joints after a prolonged fixation in one position causes pain and an initial unwillingness to bear weight. This, however, is overcome after a few days in most cases and can be facilitated with anti-inflammatory drugs.

SURGICAL MANAGEMENT

Equine bone reacts to trauma and fracture with active new bone formation and subsequent remodeling. Many osteons are mobilized to facilitate remodeling of the cortex. However, fracture healing in the horse occurs at a slower pace than in most other animals, especially ruminants, small animals, and humans.[7] Therefore any adjunct treatment that benefits bone healing is advantageous.

External Fixation

External fixation using intraosseous or transosseous pins and clamps is common in humans[8] and small animals but less so in the horse.[8] This type of fixation is employed frequently as emergency treatment in open fractures or in severely comminuted fractures when anatomic reduction, reconstruction, and internal fixation of the fracture are not possible. External fixation techniques can be applied using three types of constructs: transfixation-pin casts, external fixators, or external skeletal fixation devices.

Transfixation-Pin Casting

Transfixation-pin casting was popularized around 1990.[9] This efficient and relatively easily applied treatment method is indicated for comminuted fractures of the phalanges, the distal MCIII/MTIII, and breakdown injuries of the metacarpophalangeal joint.[10] A retrospective study involving more than 50 horses and ponies treated by means of transfixation-pin casting showed that comminuted fractures had better results (86% healed) than simple fractures (23% healed).[9] It is assumed that the micromotion between the fragments after fixation was distributed between more fragments in the comminuted fracture and therefore was decreased compared with simple transverse fracture, where distribution was not possible, leading to reduced strains and stresses exerted upon the bone fragments and the granulation tissue bridging them initially.

Under aseptic conditions, two or three cross pins that are 4 to 6 mm in diameter are introduced in the metaphyseal region of the bone. The use of positive-profile pins (IMEX) is preferred.[10] A 30-degree divergence of the pins in the frontal plane results in a stronger fixation and lower risk for postoperative fracture.[11,12] The pins should be separated by 2 to 4 cm. A stab incision is made down to the bone and, using tissue protection, the predetermined-size hole is prepared. An effective method of heat control is to initially drill a smaller hole, followed by stepwise enlargement through larger drill bits. Simultaneous flushing with approximately 500 mL of cold sterile saline solution per drill hole, applied with a bulb syringe, is effective in reducing friction and thereby heat production. The saline lubricates the drill bit because of its special construction. Production of heat is associated with bone necrosis around the pin and its resultant loosening.

A pin with a diameter that is 0.1 mm larger than the prepared hole (radial preload of 0.1 mm) provides the best pin holding strength with the least weakening of the bone surrounding the implant.[1] If positive-threaded pins are used, appropriate threads have to be cut in the predrilled hole before pin insertion. Newer pin generations are equipped with a self-tapping device at the beginning of the threaded part. Care has to be taken to engage the threaded pin portion with both cortices. Protruding portions of the pins are cut off at a length of 3 to 5 cm from the limb, which allows their incorporation into the cast.

After applying a double layer of stockinet, followed by a double layer of resin-impregnated foam padding (3M Corporation), a 5-mm layer of fiberglass cast is applied as described in Chapter 17. The ends of the pins can be covered by hoof acrylic and incorporated in an additional layer of fiberglass cast tape (Figure 76-3). An alternative approach involves applying dowels over the pin ends, fastened with set screws on the pins, just adjacent to the layer of casting tape. These dowels are subsequently covered by an additional solid layer of cast material. These two methods prevent migration of the pins after loosening, because they are fixed within the cast. It was shown in an *in vitro* study that the fixation of the pin within the cast plays a minor role.[13] The fiberglass cast material appears to be the major determinant of axial stability. However, because it was an *in vitro* study, the long-term effect of such a fixation could not be evaluated. The originally proclaimed beneficial effect of incorporating the horizontal pins in a U-bar fastened around the distal limb[9] has been abandoned because it could not be shown that it was more effective in supporting the loads.[14] A combination of transfixation-pin casting and strategic lag screw placement across major fracture fragments to ensure anatomic reduction of intra-articular fractures may speed up fracture healing.

Centrally threaded, positive-profile transfixation pins have greater resistance to axial extraction in the diaphysis than in the metaphysis of equine MCIII bone *in vitro*.[15] Again, this observation resulted from an *in vitro* study and may lead to the conclusion that it is advantageous to introduce the pins into the diaphyseal region of the long bone. However, my experience is that diaphyseal pins lead to more complications, which include

ring sequestrum formation and subsequent pathologic fractures (see later under "Complications").

A sleeve pin cast representing a modification of the external transfixation device[16] has been developed. It consists of smooth pins with negative threads on either side over which tapered sleeves are applied and fixed with nuts aginst the bone; the pins were then incorporated in fiberglass cast material and tested in a servo-hydraulic material testing machine (MTS Bionix 858) (Figure 76-4).[17] The results showed that the mean load to failure for the tapered-sleeve transfixation-pin cast was significantly greater than that for a standard transfixation pin cast. This device has not been tested clinically yet.

Recently a novel *pin-sleeve cast* (PSC) transfixation system was introduced.[18] The system consists of a 45-mm long hollow cylinder of 8.2 mm outer diameter and 1 mm wall thickness that is implanted into the bone proximal and distal to the fracture (Figure 76-5, *A*). The inside surface of the cylinder contains two circular supports measuring 1.0 mm in width and 0.5 mm in thickness; they are mounted 5 mm from either end. A 5-mm diameter pin of 120 mm length with a smooth central portion is introduced through the cylinder, contacting it only at the circular supports (Figure 76-5, *B*). Each end of the pin contains metric screw threads of 15 mm length. The pin is incorporated

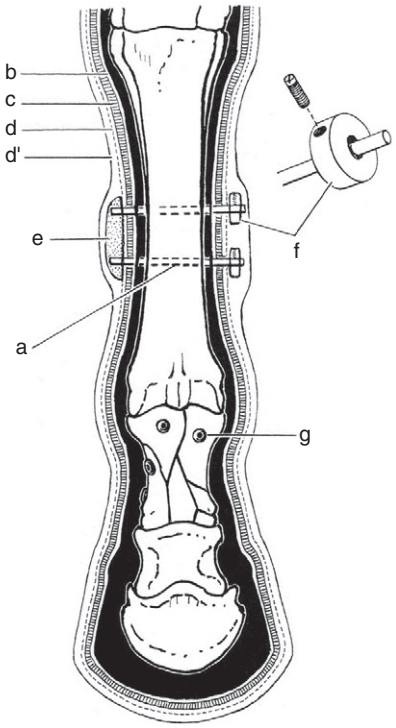


Figure 76-3. Transfixation cast for the treatment of a comminuted fracture of the proximal phalanx, partially managed by strategically placed lag screws. *a,* Parallel inserted Steinmann pins (they differ in the orientation within the frontal plane); *b,* stockinet; *c,* custom foam; *d,* initial, and *d',* second layer of fiberglass cast; *e,* hoof acrylic covering the Steinmann pin ends on the left side; *f,* custom-made dowels with set screw stabilizing the ends of the Steinmann pins on the right side; *g,* cortex screws placed in lag fashion to reduce fracture fragments.

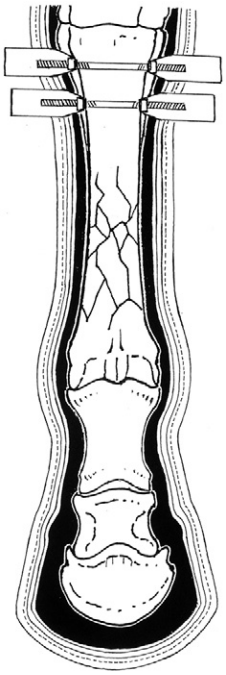


Figure 76-4. Schematic representation of a pin-sleeve cast. The sleeves (the same as shown in Figure 76-8) are mounted over a smooth pin containing negative threads at both ends. With the help of nuts, the sleeves are fixed to the bone and subsequently incorporated in a standard transfixation cast.

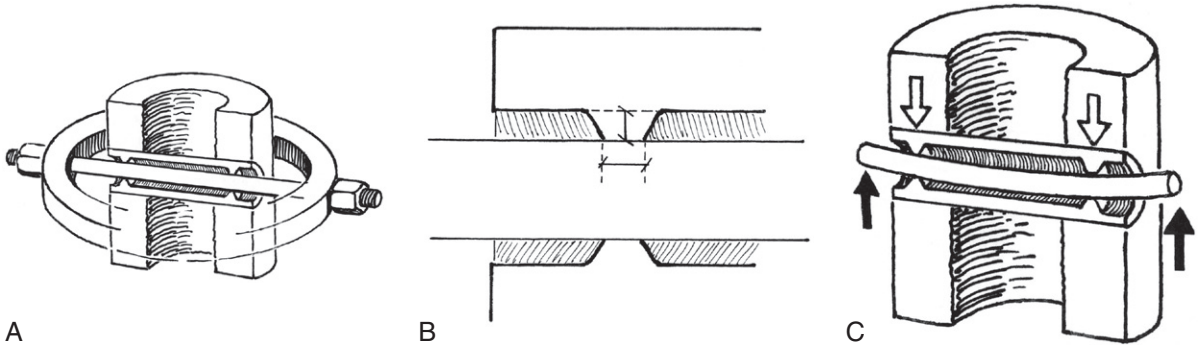


Figure 76-5. Schematic representation of a novel pin-sleeve cast (PSC) transfixation system. **A,** Representation of the sleeve inserted into the bone and the central smooth pin tightened to the ring fixator (the pin is under tension). **B,** Close-up of the contact support of the pin within the intraosseous sleeve. **C,** During weight-bearing the pin is slightly bent without contacting the sleeve and surrounding bone, leading to minimal strain at the bone–sleeve interface.

(before introduction across the bone) into a 15-mm-wide hollow ring of 70- or 90-mm inside diameter through 5-mm holes. Using metric nuts, the pin is tightened to the outside of the circular ring around the limb, and in doing so an axial preload can be applied similar to an Ilizarov ring fixator. The rings are subsequently incorporated into a fiberglass cast. *In vitro* testing using calf metacarpal bone and an MTS testing machine (see earlier) and a 25 kN load cell (Type U-10M, 25kN, 250Nm) revealed that application of a pin preload significantly reduced strain measurement compared to the transfixation pin cast, which may prevent or retard pin loosening.[19] Because the pin only has contact with the two support rings, the pin can bend somewhat without exerting additional strain on the bone (Figure 76-5, *C*). Given that pin loosening is the result of cyclic strain at the pin–bone interface over time (at the periosteal side at the proximal aspect and at the endosteal side at the distal aspect), the significant strain reduction at the cylinder–bone interface should be an improvement.[19] Clinical tests will tell if the theoretical advantages can be transformed into empirical advantages.

Advantages of transfixation-pin casting include no or only minimal load on the fracture site and minimal distraction and movement between the fragments. Also, the tissues are spared additional trauma and further disruption of the blood supply. All the disadvantages of external coaptation, such as the development of cast disease, osteoporosis, contracted feet, and tendon laxity, apply to this type of treatment. Pin tract infection with ring sequestrum formation can occur and is preceded by a sudden onset of lameness. This usually occurs in heavy horses (greater than 500 kg of body weight) after approximately 2 weeks and warrants immediate removal of the proximal transfixation pin. Another pin can be inserted at a different location to prolong unloading of the fracture, or, alternatively, the limb can be placed in a new cast without the most proximal pin. A new pin can be expected to form a similar sequestrum within the next 2 weeks, and the procedure can be repeated. If three pins are inserted initially, transfixation may be maintained for 6 to 8 weeks before simple casting is applied. At that time, a full-limb cast is applied to prevent rotation of the MCIII/MTIII. (Rotation can occur in a half-limb cast and may lead to a fracture across a pin tract.)

External Skeletal Fixator

Application of an external skeletal fixator allows immediate, although subnormal, weight-bearing on the limb. The Steinmann pins and Schanz screws (Steinmann pins with a threaded end) used with this fixation device cause minimal additional trauma to the injured soft tissue. Because in most cases no external coaptation is used, there is easy access to an open wound, which facilitates débridement and management. Application of an external fixator also allows additional correction and fracture alignment after the initial operation, whereas a fracture treated with internal fixation can be adjusted only through an additional surgery.[20] However, this technique has not been very successful in equine patients.

An external fixator uses transversely inserted (cross) Steinmann pins or Schanz screws proximal and distal to the fracture site. These pins are firmly connected to external rods through special clamps, which may be applied in many configurations.[8] An *in vitro* study showed that the best results are achieved with an elaborate three-dimensional design, including three Steinmann pins proximal and distal to the osteotomy, inserted at different angles in the frontal plane and obliquely from proximal to distal, and connected to four external rods.[20] Such a configuration is expensive and very heavy and therefore not practical, especially for horses.

The *Seldrill Schanz screw* improves the bone-holding properties of Schanz screws. The design features a pin with a diameter that is 0.1 mm greater than the hole drilled for it (larger radial preloads result in microfractures and deformation of the bone surrounding the pin, with subsequent loosening).[1] The Seldrill Schanz screw is manufactured of pure titanium and stainless steel and has a self-drilling, self-tapping tip.[21] This implant is inserted through a stab incision without predrilling the bone or pretapping the hole, even in hard equine bone. The Seldrill Schanz screw contains a relatively thick core and thin threads and includes a portion with a built-in radial preload of 0.1 mm, immediately adjacent to the self-drilling, self-tapping tip. Because the sharp tip should not exit the opposite cortex, this implant is to be used in half splints (type I: a construct that contains one or two sidebars on one side of the bone), where the external tube or rod is located only on one side of the bone. Additionally, this device can be used in three-dimensional (type III: a construct that contains sidebars at three sides of the bone that are interconnected externally) configurations, where a half splint is connected to a full, bilateral splint (type II: a construct that contains a sidebar medially and laterally), which uses nonthreaded pins.

A modification of an external fixator, the Pinless External Fixator (Synthes Inc.), is available for selected fractures in large animals.[22] This device is manufactured in three sizes and configurations of clamps, which are applied over a bone without completely penetrating the cortex (Figure 76-6). The clamps are fastened through a connection rod, which is attached to an external fixator tube or carbon rod. This device is not rigid enough to support weight bearing of a large animal with a fractured limb, but it is effective in stabilizing mandible fractures in cattle and horses (see Chapter 102) and fractures of the tail in the horse. *In vivo* studies showed that the clamping force is maintained over several weeks while inducing only minor bony changes where the clamps contact the bone.[23] Animals tolerate the device well. The advantage of this type of external fixator is the minimal damage to bone and tooth roots.

The use of *circular external skeletal fixators* (CESFs) has become routine practice in small animal surgery for the management of developmental, traumatic, and degenerative orthopedic problems.[24,25] The advantages of this system include immediate weight bearing, excellent mechanical properties, ability to stabilize short segments, ability to adjust the frames after their application, and preservation of joint mobility.[26] The use of CESFs in large animals is mainly limited to cattle.[24,27] Sporadic case reports are published where a CESF was applied to a long bone fracture in a horse (Figure 76-7).[24] It is not very likely that this fracture fixation system will become very popular in horses in the future.

External Skeletal Fixation Device

The external skeletal fixation device (ESFD) was developed for horses with severely comminuted fractures of the phalanges, fractures of the distal MCIII/MTIII, and breakdown injuries of the metacarpophalangeal joint. The ESFD uses two or three transfixation pins in the intact bone proximal to the fracture,

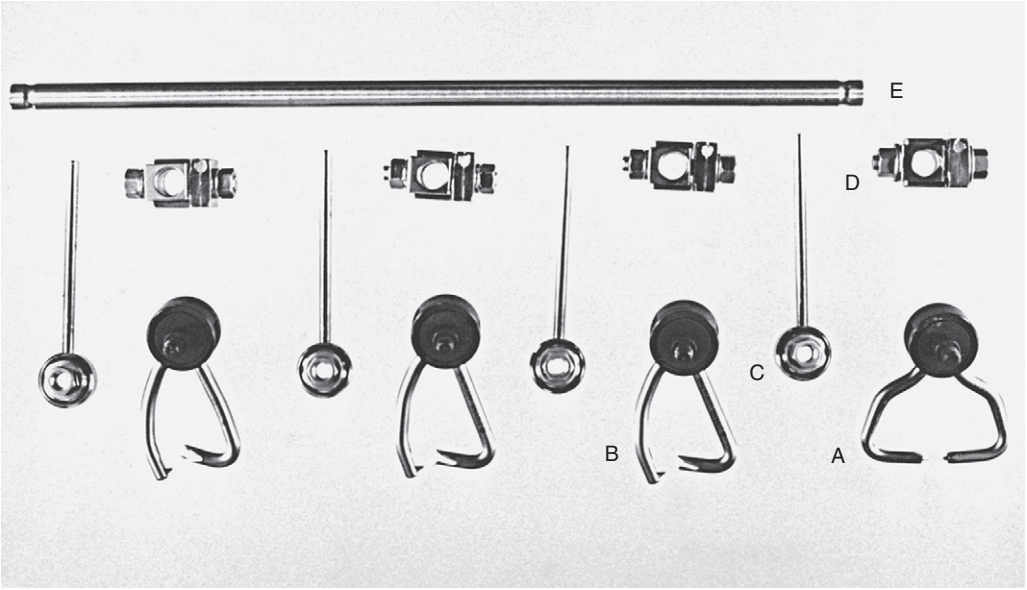


Figure 76-6. One symmetric *(A)* and three asymmetric *(B)* titanium pinless fixator clamps are shown with connecting rods *(C),* clamps *(D),* and longitudinal rod *(E).* This configuration is used to treat mandibular fractures.

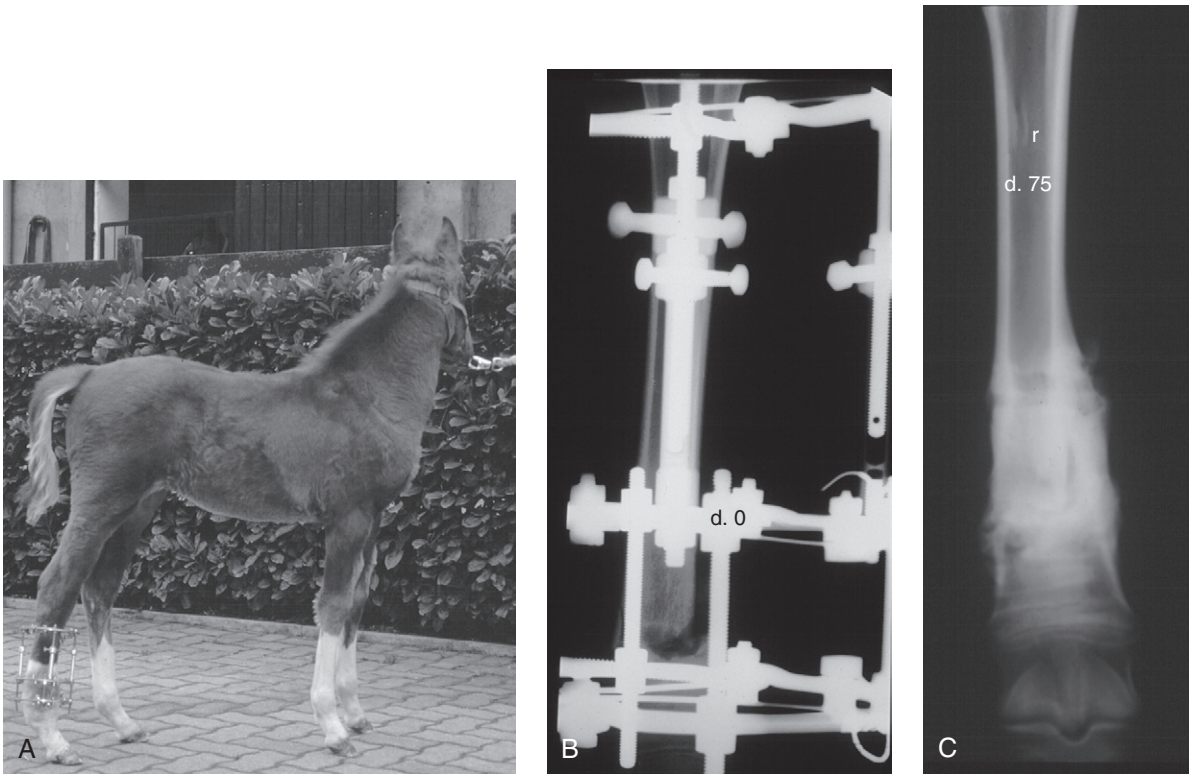


Figure 76-7. Application of a circular external skeletal fixator to distal MTIII fracture in an Arabian foal. **A,** The foal wearing the circular fixator. **B,** Radiographic view of the device in place immediately postoperatively. **C,** The healed fracture 75 days later. (Courtesy A. Ferretti, Legnano, Italy.)

and sidebars and a base plate. Weight-bearing forces are transmitted via the pins and sidebars around the fracture to the ground, allowing the animal to immediately bear full weight without loading the fracture.[16,28-30] The original report described the device and 15 cases, only four of which survived long-term.[28] Through a meticulous study of the design, an effective ESFD transfixation system has been developed and is commercially available.[16] Early complications included fractures through the pin tract while the device was worn or during recovery after implant removal. These complications were almost completely abolished with the latest generation ESFD.[30] Removal of the device on the sedated, standing animal eliminates some

problems encountered after device removal, but the risk of fracture while wearing the device continues.[30]

Up to 90% of the stresses generated at the bone–pin interface contribute directly to pin bending, resulting in uneven stress distribution, with peak stresses concentrated at the outer bone cortex.[31] The tapered-sleeve design was developed to reduce transcortical pin bending with weight-bearing. Large-diameter, tapered sleeves are applied over the transfixation pins (biaxially loaded in tension and shear) and are incorporated in a stronger, lighter frame (Figure 76-8).[32] *In vitro* tests applying cyclic loading to this ESFD showed that significant increases in stiffness, reduced bending, and increases in load-to-failure-of-bone could been achieved with the tapered-sleeve design.[32] Since bone failure occurs at a finite strain level, it appeared that the larger loads to failure indicated lower strains in the bone at the working stress level. To facilitate adjustments in tranfixation pin placement and their incorporation into the sidebar, a modular sidebar was developed consisting of several elements that could be assembled to the length needed for a specific fixation. The four elements fit on top of each other and are not separated by more than 10 mm. The modular components are connected using 4-mm-thick rubber tubing and sealed with duct tape. Once in place, the hollow connecting bar construct is filled with polymer that also enters the connecting elements.[18]

The ESFD was compared in an *in vitro* study to a transfixation-pin cast and a modular sidebar construct (see later).[33] The solid ESFD has a greater stiffness, higher yield and failure load, and a lower yield and failure displacement than the transfixation-pin cast and the pin-sleeve cast. Mean cycles to failure for the transfixation-pin cast was 2996 ± 657 at a load of 16,000 N, and that for the solid ESF was 6560 ± 90 cycles at a load of 25,000 N. These results are encouraging and the new fixation device may prove to be a great alternative to transfixation-pin fixation.

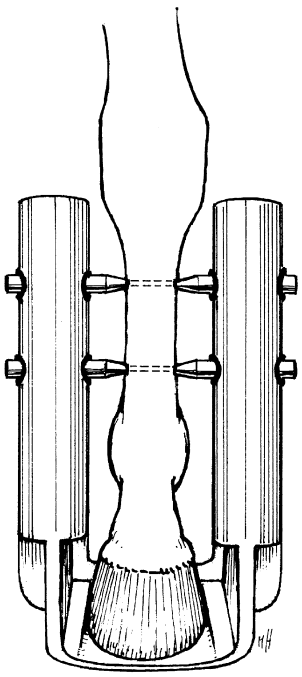


Figure 76-8. Graphic representation of the external skeletal fixation device design.[32] Two tapered sleeves are mounted and tightened on transosseous pins, which are then incorporated into the U-shaped apparatus, providing additional stability.

Aftercare

Application of an external fixator or ESFD allows easy wound management of open fractures. Débridement should be performed under aseptic conditions, and every measure should be taken to prevent additional contamination. Broad-spectrum antibiotic coverage is indicated in any horse treated with external fixation during the entire time the device is in place. The skin around the cross pins should be cleaned daily with alcohol-soaked swabs and dried before reapplying the bandage. Internal fixation of the fracture may be considered once the infection has subsided and healthy granulation tissue has formed. At that time, the external fixation device is removed.

Any sudden changes in weight-bearing patterns are indications for close scrutiny of the fracture and the fixation device. Radiographs should be taken at the onset of any complication and repeated in routine fashion at 2- to 4-week intervals without complications. Once fracture healing has occurred, the device is removed. The abrupt change in stability caused by removing the external fixator can be minimized through partial destabilization of the device. This is best achieved by moving the vertical connecting bars farther away from the limb. An alternative approach involves the strategic removal of one or two pins at 2- to 3-week intervals. After a few weeks in this configuration, the device is removed.

Complications

Loosening of the pins, infected pin tracts, and fracture through a pin hole are the main complications of external fixation in horses.[14] Loosening of cross pins is the most frequent complication of external fixation.[8,34] Weight-bearing on the affected limb causes osteolysis around the pins, followed by infection of the pin tract and subsequent loosening.[34] Once a pin loosens, pain develops. A loose pin should be removed immediately, because it no longer serves a useful function, and infection around it will not subside as long as the pin remains *in situ*. Curetting and flushing of the tract facilitates cessation of draining within a few days. If drainage persists, the skin should be reopened, followed by an additional curettage of the pin tract. Removal of a loose pin destabilizes the fixation. Depending on the degree of fracture healing, it is important that pins be removed one at a time. Premature removal of the implants results in total instability.

Removal of a loose transfixation pin is performed on the standing, sedated horse. If external coaptation was used, cast material immediately adjacent to the pin is removed and the pin is pulled out. The tract is flushed and the hole is filled with a surgical sponge soaked in an antiseptic solution and fixed in place with tape. If the pin is fixed within the cast through a dowel or a hoof acrylic pad (see Figure 76-3), a complete cast change under general anesthesia is necessary.

Osteomyelitis may develop as an extension of pin tract infection or at the open ends of the fracture. Treatment of orthopedic infections is discussed in Chapter 85. When osteomyelitis is rampant and uncontrollable, euthanasia may be the only alternative.

Soft tissue swelling occurs frequently in animals wearing an external fixator. This is a normal reaction to the implants, local

insult, trauma, and controlled infection. Therefore adequate distance between the skin and the vertical bars and clamps has to be allowed to accommodate the swelling. This will prevent skin necrosis near contact areas of the clamps.

Pathologic fracture is a common problem encountered in horses treated with the external skeletal fixation device or external fixator.[30,35,36] Frequently, a doughnut-shaped cylinder of bone is walled off around the pin (Figure 76-9). This creates a relatively large defect in the bone and significantly reduces its strength. The bone within the fixator becomes weakened by osteoporosis (disuse atrophy depending on the duration of fixation), and bearing weight on the limb can result in failure through one of the pin tracts (see Figure 76-9). If the animal is not euthanized at once after failure, the fracture must be treated immediately by either internal fixation or a full-limb cast. Changing the arrangement of the Steinmann pins as well as the threads may limit the recurrence of this complication.[35]

Internal Fixation

At the turn of the twentieth century, compression in rigid fixation was recognized as an important component of rapid fracture healing. Rigid internal fixation became an important step in reaching the goal of early return to full function for the fractured limb and the patient.[37,38] The devastating effects of prolonged external coaptation—cast disease—were prevented when this goal was attained. Early return to function allows movement of the joints, associated nourishment of the articular cartilage, and prevention of proteoglycan loss. Additionally, disuse osteoporosis is prevented, and the soft tissues surrounding the fractured bone are maintained in physiologic condition.

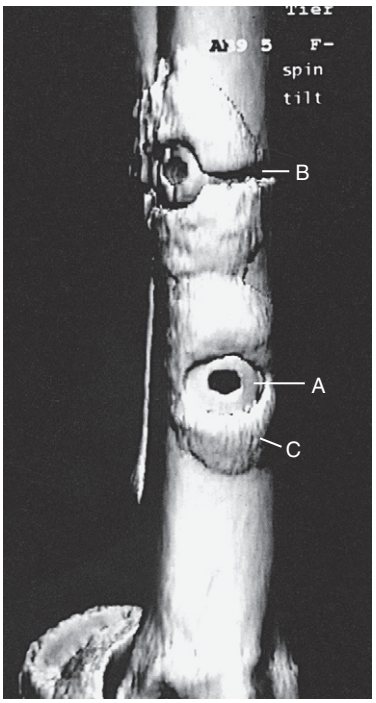


Figure 76-9. Three-dimensional reconstruction of a computed tomographic study of the third metacarpal showing ring sequestra *(A)* as a result of transfixation pinning, and a pathologic fracture *(B)* through the proximal pin tract or ring sequestrum. Periosteal new bone formation *(C)* surrounds the ring sequestra.

Early return to function is achieved through anatomic reduction of the fracture and stable internal fixation.[1]

Internal fixation is achieved through opening of the skin. This may occur through a stab incision, as with intramedullary pinning, transcutaneous interlocking, and minimally invasive plating techniques, or through opening the skin over a greater distance, followed by separation of the soft tissues surrounding the fractured bones when bone plates are applied.

Principles

Fracture fragments fixed to each other under compression heal without callus formation.[7] Precise anatomic reduction of the fracture is of paramount importance for this type of bone healing (primary union). It is also critical when the articular surface is involved, because if the reconstruction is not nearly perfect, osteoarthritis will develop (Figure 76-10).

In the last 50 years, tremendous progress has been made in the art of equine fracture treatment. The greatest influence on this achievement can be ascribed to the AO Foundation. Founded in 1958 by four Swiss surgeons, the AO (Arbeitsgesellschaft für Osteosynthesefragen) quickly developed into a worldwide organization.[1] In 1984, the AO Foundation was established, to which all the rights for royalty income were bestowed. Recently the three manufacturers that supplied surgeons with a large variety of sophisticated, high-quality implants (Mathys AG, Bettlach, Switzerland; Stratec Medical, Oberdorf, Switzerland [formerly Institut Strauman, Waldenburg]; and Synthes [USA], Paoli, PA) have merged to form a single worldwide company, Synthes, with its headquarters in Solothurn, Switzerland. For every implant sold, Synthes pays a certain amount of royalties to the AO Foundation to support teaching and the development of new instruments—a genuine approach to the improvement of fracture fixation. Techniques developed for human patients were quickly adopted by veterinarians, and many are applied in daily practice with good success.[1-4]

After the merger of the three companies, all patents were sold to Synthes, and with the interest from the investments, most of the activities of the AO Foundation can be offset. Additional income is generated through other sources. In 2008, the veterinary specialty within the AO Foundation, AOVET, was accepted

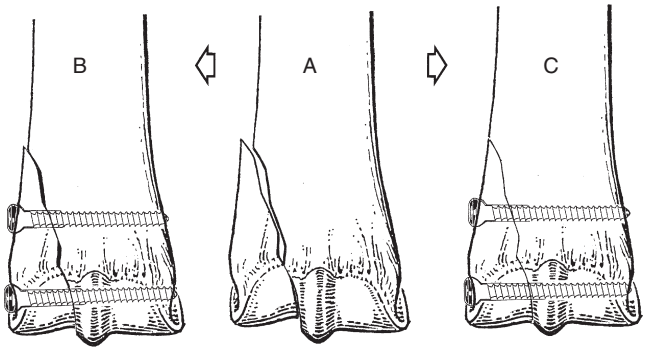


Figure 76-10. *A,* A displaced articular fracture of the lateral condyle of the distal third metacarpal. *B,* Inadequate reduction was achieved after interfragmentary compression via two cortex screws applied in lag technique. The articular surface is not congruent, which would lead to osteoarthritis. *C,* Adequate anatomic reduction was achieved before screw fixation, reestablishing the articular surface and normal bone-to-bone contact.

as a fully funded specialty next to to AOTRAUMA, AOSPINE, and AOCMF (cranio-maxillo-facial). The goal of AOVET is to establish itself as the world leader in the treatment of musculoskeletal disorders of animals, including fractures.

The implants, their function, and their application as discussed in this chapter are mainly those developed by the AO group. Included are screws, plates, pins, wires, and specially designed plates and nails. Only the instruments and implants used in equine fracture treatment are discussed.

Approach to and Manipulation of Bone

Before surgically approaching the bone, a careful diagnostic imaging study should be conducted that includes multiple radiographic views from a variety of angles. Ultrasonography, scintigraphy, and computed tomographic scans may aid standard radiography in the selection of the locations where implants are applied and the direction of their insertion. Potential interference of interfragmentary screws and plates with soft tissue structures must be considered. In articular fractures, reconstruction of the joint should be the deciding factor in the decision of how to approach the fracture and apply the implants. For some fractures, surgery should not be attempted. For example, a comminuted fracture of the radius with substantial defects in the caudal cortex has no chance to heal because continuous cycling of the implants eventually leads to implant failure. It is prudent to conduct a detailed discussion with the owners of equine patients about the chances for a successful surgical outcome, the potential complications, and the costs of the particular fracture repair before surgery. Availability of all implants and instruments needed at the time of surgery is an absolute prerequisite for a successful result. The approach to the bone should be carried out rapidly and carefully and with respect for Halsted's principles of good surgical technique. Special attention should be paid to the blood supply, avoiding severance of major blood vessels. The periosteum should be maintained with the underlying bone whenever possible. Periosteum is stripped off the bone only immediately under selected implants, such as dynamic compression plates (DCPs). More recently developed implants, such as the limited contact dynamic compression plate (LC-DCP), the point contact fixator (PC-Fix), and the locking compression plate (LCP), are applied over the periosteum (see later). Massive dissection should be avoided, because it facilitates the accumulation of blood and serum. Planning the approach relative to the application of selected implants is of great importance for a successful outcome of the surgery. For example, the approach to the MCIII is made through longitudinal splitting of the common or lateral digital extensor tendon, which facilitates secure closure of the soft tissues and skin over the implants after the fracture is repaired.[39,40]

In human and small animal osteosynthesis, biological fracture fixation has become very popular.[41] This technique abandons the dogma of anatomic reconstruction and accepts that proper axial and rotational alignment of the bone, despite incomplete reconstruction, followed by fixation of the fracture with strategically placed implants is preferred. Longer plates are used, providing better leverage conditions. Screws are inserted through the biomechanically most important holes, but not all holes in a plate are filled with screws. If possible, the plate is prebent (to conform to the shape of the contralateral intact bone), slid along the fractured bone through a small incision, and fixed with screws implanted through stab incisions. Such minimal fixation cannot be used successfully in horses. However, the principle of applying the implant by a minimally invasive technique is applicable to the horse, even if the implant has to be modified to meet the demands placed by the horse's size.

Instruments

Basic instruments used for fracture treatment include a variable-speed air or electric drill with forward and reverse gears. To facilitate mechanical preparation of the threads in the bone, switching between forward and reverse should be easy. Drill bits and guides of different types and sizes are needed (Table 76-1). Drill guides allow application of concentric pressure, and they stabilize the drill bit, which prevents breakage. The bit is also less likely to slip off the bone surface, especially when it is obliquely applied. The guides protect tissues around the hole from frictional trauma generated by the drill bit and the tap. It has been shown that drilling at maximal speed (about 90 psi of air pressure) results in less heat production than drilling at low speeds.[3] The drill bits should be sharp and should be exchanged frequently, because dull drill bits create heat in the hard and dense equine bone. This is especially true in adult animals. Continuous application of saline solution throughout the drilling procedure is important to reduce heat production.[3] The drill bits are designed to allow penetration of the fluid along their outside perimeter, which facilitates lubrication and reduction of friction. However, not enough water can be flushed into the drill hole to effectively cool the drill bit. Frequent cleansing of the drill bit to remove the swath material is the single most important factor in reducing heat production. During drilling, axial pressure is applied to the drill bit without bending it. Bending causes the drill bit to become dull too rapidly because of interference with the drill guide. More importantly, the hole will be of a larger diameter than intended. Adequate but not excessive pressure should be applied. The instruments used for screw insertion are discussed later, in the paragraphs about the lag technique. Other instruments such as various drill guides, special reduction forceps, bone clamps, rongeurs, curets, osteotomes, and a mallet (for more information, see Chapter 11) aid in bone handling as well as in maintaining compression during fracture reduction and the insertion of the desired implants.[27]

The newly developed Equine Large Fragment Set consists of several trays: the instrument set containing the different instruments (Figure 76-11), the plate set containing the different plates selected by the veterinarian, a screw set containing cortex screws, and one screw set for locking head screws.

Implants

SCREWS

Various types of screws serving different functions were developed by the AO together with Synthes (see Table 76-1). The parts of a screw include the head, shaft, core, and thread. Its attributes include pitch, shaft length, thread length, and total screw length.

Screw types

Cortex screws have a 0.7-mm thread width, and a thread length that depends on the screw length (see Table 76-1). A cortex screw does not contain a shaft portion and is referred to as a *fully threaded screw.* These types of screws are the most frequently

TABLE 76-1. Veterinary Large Animal Screw, Drill Bits, and Tap Chart

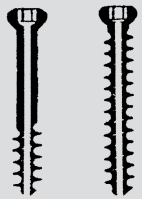
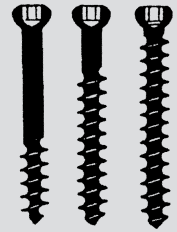
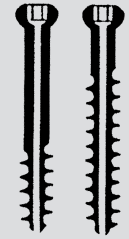
Screw Name	3.5 mm Cortex	4.5 mm Cortex	4.5 mm Shaft	4.5 mm Cannulated	3.5 mm Locking	4.0 mm Locking	5.0 mm Locking	5.5 mm Cortex	6.5 mm Cancellous	7.3 mm Cannulated
Screw Ø	3.5	4.5	4.5	4.5	3.5	4	5	5.5	6.5	7.3
Glide hole Ø	3.5	4.5	4.5	4.5 None	None	None	None	5.5	4.5	7.3
Thread hole Ø	2.5	3.2	3.2	3.2	2.8	3.2	4.3	4	3.2	5
Tap Ø	3.5	4.5	4.5	4.5	None	None	None	5.5	6.5	7.3 Optional
Screw shape										
Cannulation guide pin	—	—	—	150 mm long /1.6 mm Ø	—	—	—	—	—	300 mm long /2.8 mm Ø
Type thread	Cortical	Cortical	Cortical	Cancellous	Cortical narrow	Cortical narrow	Cortical narrow	Cortical	Cancellous	Cancellous
Pitch	1.25	1.75	1.75	1.75	0.8	1	1	2	2.75	2.75
Screw head diameter	6	8	8	6.5	5	6.6	6.6	8	8	8.2
Special head design	—	—	—	—	Conical threaded	Conical threaded	Conical threaded	—	—	—
Thread length	Fully threaded	Fully threaded	Variable	⅓ of length/ Fully threaded	Fully threaded	Fully threaded	Fully threaded	Fully threaded	16 mm/32 mm/ Fully threaded	16 mm/32 mm/ Fully threaded
Shaft diameter	—	—	4.5	3.1	—	—	—	—	4.5	4.8
Core diameter	2.4	3.0	3.0	2.7	2.9	3.4	4.4	3.8	3.0	4.5
Self-tapping	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No	No	Yes
Self-drilling	No	No	No	Yes	Available (Europe only)	Available (Europe only)	Available (Europe only)	No	No	Yes
Drive	Small (2.5 mm) hex	Large (3.5 mm) hex	Large (3.5 mm) hex	Large (3.5 mm) hex	T15 stardrive	T25 stardrive	T25 stardrive	Large (3.5 mm) hex	Large (3.5 mm) hex	Large (4.0 mm) hex

Figure 76-11. Synthes Large Fragment Set: Instruments. The set contains all the drill bits, taps, drill guides, screwdrivers (hexagonal- and stardrive), T-handle, countersink, depth gauge, push-pull device, tension device, socket wrench, and torque limiting device that are needed to insert screws (in lag fashion) into bone as well as through, DCPs, LC-DCPs and LCPs. The instruments are arranged in 3 trays that fit on top of each other into main tray. Pictographs facilitate correct positioning of each instrument into the trays. (Courtesy Synthes Vet, West Chester, PA.)

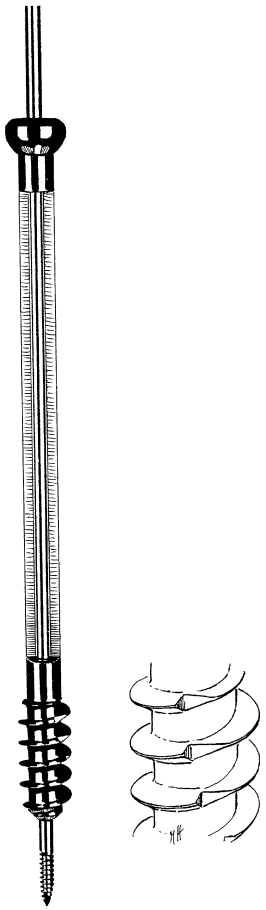


Figure 76-12. Schematic drawing of 7.3-mm cannulated screw with the guide pin inserted and half of the shaft removed. Insert: the reverse-cutting design of threads, which facilitates screw removal after healing of the fracture. (From Nixon AJ: Equine Fracture Repair. Saunders, Philadelphia, 1996.)

applied in equine osteosynthesis. The *shaft screw* (see Table 76-1) is an exception and therefore a special cortex screw. The shaft screw contains a shaft portion of the same diameter as the outside diameter of the threads.[42] The threads have the same geometry as the cortex screws. Shaft screws are available in various shaft and thread lengths. Because of the smooth shaft portion, these screws are ideally suited for lag screw fixation, especially through LC-DCPs, where up to 40 degrees of angulation relative to the long axis of the plate can be achieved.

Cancellous screws have a wider thread diameter than the cortex screws and they have a different pitch (see Table 76-1). This screw is designed to improve holding power in soft cancellous bone, but it is only rarely used in equine internal fixation.

Cannulated screws contain a central canal for a guide wire (see Table 76-1).[43] The design resembles the cancellous screw, because it has a thinner shaft and a wider thread portion. The 7.3-mm cannulated screw contains a self-drilling and self-tapping tip, as well as a reverse cutting device at the back end of the threads (Figure 76-12).[44] An *in vitro* study using equine cadaveric femurs revealed that the 6.5-mm cancellous and the 7.3-mm cannulated screws vary in insertion properties (the 7.3-mm cannulated screw requires significantly greater insertion torques), but they have similar pullout properties in the mid, proximal, and distal metaphyses of foal femurs.[45] Both screw types have greater holding power at the mid-diaphyseal location than at metaphyseal locations. Because of the overall similar holding power of 6.5-mm cancellous and 7.3-mm cannulated screws, it is unlikely that increasing the screw diameter beyond 6.5 mm will provide increased holding power in foal bones. The use of the 7.3-mm cannulated screw should be considered for foal femoral fracture repair when greater accuracy is needed or when bone threads for the 6.5-mm cancellous screw have been stripped.[45]

Self-tapping cortex screws contain the same thread-cutting device at the tip as the tap, obviating one step of the standard screw insertion technique (see Table 76-1).[46] These screws are popular in human surgery and are gaining more acceptance in equine surgery.[47] An *in vitro* study revealed that the mechanical properties of regular and self-tapping 4.5-mm cortex screws are similar with regard to pullout strength from the adult equine MCIII and that the self-tapping cortex screws require less than half the total insertion time required by standard screws.[47] Interestingly, bone failure and bone comminution during the pullout tests were more commonly associated with self-tapping screws.[48]

Locking head screws were introduced with the less-invasive stabilization system (LISS) and subsequently also applied in the locking compression plates by Synthes (Figure 76-13). The conical shape of the PC-Fix screw (which is no longer manufactured) served as a basis for the new design.[49] The screw head was modified with a threaded profile, which complemented the one in the LISS plate hole. This design provided a stable angular

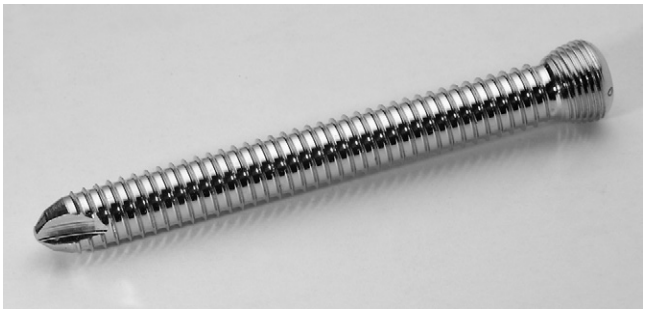


Figure 76-13. A self-tapping locking-head screw. Note the threads manufactured into the conical screw head. These interlock with complementary threads in the plate. The self-tapping ends are visible at the tip of the screw.

fixation of the screw–plate (fixator) junction: the screw head is self-centering in the hole, and it keeps the screw from backing out of the LISS and LCP fixator (see later). The pitch of the threads at the screw head is identical to that of the threads on the shaft. Because of the larger diameter of the screw head, the pitch seems larger than on the shaft. However, the threads on the screw head catch after turning only 180 degrees instead of 360 degrees in the shaft. This facilitates faster fixation of the screw head into the plate and reduces the development of plate deformation through tightening of the screw. It is important to remember that this screw must be at 90 degrees relative to the long axis of the plate.

The screw was also adapted to the unique mechanical demands of an internal fixator. The core diameter of the screw was enlarged to resist the increased bending moments and higher shear forces induced by a fixator.[47] This, plus the threaded screw–plate interface, allows the use of unicortical screw fixation in the diaphysis. The stability of unicortical fixation with locking head screws was established in a cadaveric biomechanical study.[50]

Unicortical screw fixation, in turn, allows the application of self-drilling or self-tapping screws, which was made possible by reducing the thread pitch and adding drill and tap sections to the screw tip (see Table 76-1).[39] These design changes have additional benefits: screw length determination is no longer needed because all diaphyseal screws can be the same length; therefore screw lengths are not needed in increments of 2 mm, which results in a smaller inventory; predrilling and tapping are no longer needed, and the thread profile cut into the bone is more precise (because each screw is used once and therefore the drill and tap parts are sharp), which results in a better anchorage of the screw in the bone.[49]

In the horse, self-drilling and self-tapping screws are rarely used because of their high price, and screws are usually inserted bicortically. Therefore most of the advantages just listed do not apply to this species. However, self-tapping screws are used in the horse in bicortical applications.

Currently, locking head screws for the variable axis plates are available. As the name says, these screws do *not* need to be introduced at a right angle relative to the long axis of the plate. This is possible because of the slightly curved surface along the axis of the screw head and the special configuration of the plate hole (see later).

Other screws include the Herbert screw (Zimmer Orthopedics), an example of a self-contained compression screw.[51] This screw is fully threaded and contains threads not only over its entire length but also on the head. The head is wider than the rest of the screw and can be completely buried in the bone. It has been used in condylar fractures of MCIII/MTIII.

Recently, a cannulated, tapered, variable-pitch, self-compressing screw was developed (Acutrack Equine Screw). This screw is 45 mm long, and it has a diameter of 6.5 mm at its base that tapers to a diameter of 5.0 mm at its apex. Because of the tapered shape, no glide hole is needed. The screw is manufactured of titanium. Biomedical studies comparing Acutrack to 4.5-mm AO cortex screws inserted in lag fashion revealed that the screws had similar biomedical shear properties.[52] The self-compressing action of the Acutrack Plus screw generated 65% of the compression pressure and 44% of the compressive force achieved with the 4.5-mm AO cortex screw.[53] The overall pushout strength was higher in the Acutrack Plus screw.[53] Simulated midbody fractures of the medial proximal sesamoid bone repaired with Acutrack self-compressing screws compared with 4.5-mm AO cortex screws showed mechanically comparable strengths.[54] Both constructs were mechanically inferior to intact proximal sesamoid bones.[54] A recent study compared compression pressures of the Synthes cortex screw and the Acutrack Plus screw in simulated equine MCIII lateral condylar fractures of varying fragment thicknesses. The results revealed that significantly lower compressive forces were achieved with the Acutrak Plus screws.[55]

Sizes

The size of a screw is determined by the outside diameter of the threads. The standard screws for the horse are the 4.5- and 5.5-mm cortex screws. Pertinent data on each screw type as well as drill sizes needed are summarized in Table 76-1. The 5.5-mm screw was developed for compact equine bone. This screw has advantages over the 4.5-mm screw when used in adult horses.[56-58] The 6.5-mm cancellous screw is available in three configurations: a 16-mm thread length, a 32-mm thread length, and fully threaded. Since the introduction of the 5.5-mm cortex screw, the 6.5-mm cancellous screw has diminished in importance, because the 5.5-mm screw can also be inserted when a 4.5-mm hole has been stripped. A stripped 5.5-mm hole can still be engaged by a 6.5-mm cancellous screw. Cannulated screws are manufactured in 3.5-, 4.5-, 7.0-, and 7.3-mm diameters, but only the latter two sizes are interesting for the equine surgeon, and they can be used if a 6.5-mm cancellous screw hole is stripped.

The 3.5-mm cortex screw is used to achieve interfragmentary compression of certain fractures, such as third carpal bone slab fractures. It is also applicable for anatomic reduction of long bone fractures and interfragmentary compression of the fragments. The screw has such a small head that it can be completely buried in bone, which allows plating over the screw.[30] This is a great advantage, especially if two plates have to be applied.

The recently developed 5.0-mm locking-head screws are the strongest screws available for equine fracture treatment, because of their large core diameter (4.3 mm) (see Figure 76-13). The thread width is much smaller than standard cortex screws, but these screws are tightened into the thread hole within the combi-hole of the plate, facilitating solid fixation. These screws can be applied only through the LISS and the LCPs. They are available as nontapping; self-tapping; and self-drilling, self-tapping screws. Only the first two types are of interest for equine

surgeons. The self-drilling, self-tapping screw is very expensive, and because of its design it is impossible to predetermine the depth of the screw hole. Also, in equine fracture treatment it is important to achieve screw purchase in both cortices. If a self-drilling, self-tapping screw was inserted, the sharp self-drilling part of the screw would protrude out of the trans-cortex, which could produce soft tissue damage.

The 4.0-mm locking head screw can be used with the 4.5/5.0 mm LCPs. They have the same head design as the 4.3-mm locking head screw but a thinner screw design. These screws are rarely used in equine osteosynthesis.

The 3.5-mm locking head screws have a core diameter of 2.9 mm and are therefore stronger than the cortex screw of the same size. To ameliorate the excessive rigidity of the locking plates experienced in human surgery, a dynamic locking head screw (DLS) was recently developed. This screw type was tested in recent *in vivo* studies in sheep and resulted in additional callus formation around the osteotomy site.[59,60] The DLSs are not justified for equine applications because they are expensive and the same problems are not encountered in the horse as in people.

Additional smaller screws (2.7 mm, 2.4 mm, 1.5 mm, etc.) are available as regular cortex screws or in some instances as locking head screws. These implants may be applied with the corresponding plates for the repair of skull fractures (see Chapter 102).

Most screws are available either with a hexagonal or a stardrive hole in the screw head, into which the corresponding screwdriver is inserted to power the screw into the bone.

The preparation of the screw surface plays an important role in the holding power of the implant.[56] Special surface preparation of stainless steel and titanium screws showed superior holding characteristics over the plain stainless steel and titanium screws.[61]

Functions

Screws can be used as lag screws, position screws, and plate screws.[62] There is a difference between a partially threaded cancellous or cannulated screw, which automatically produces a lag effect, and a cortex or shaft screw used in lag fashion.[3] The cancellous and cannulated screws are inserted so that all of the threads pass the fracture line. Thus tightening of the screw provides interfragmentary compression (Figure 76-14, *A*).[1-4] If the threads of a cancellous screw bridge the fracture gap, no compression can be achieved (see Figure 76-14, *B*). The same holds true for a cortex screw placed as a position screw (i.e., with no lag effect): its threads engage bone on both sides of a fracture, and because no glide hole was prepared in the near cortex, no interfragmentary compression is achieved. A plate screw lags the plate to the bone.[1,2] The lag technique may, however, also be applied through a plate hole. In this case, the screws are inserted through the plate and they cross the fracture line.[1-4]

The *lag technique* is used to insert cortex screws so that they act in lag fashion. This technique is not necessary when using partially threaded screws (such as cancellous or cannulated screws) as lag screws when applied as discussed in later paragraphs. The cis-cortex or near cortex is drilled with a drill bit having the same diameter as the outside thread diameter of the screw (Figure 76-15, *A*). This is referred to as overdrilling. Therefore, at insertion of the screw, the threads do not engage bone in that cortex but glide through, so this portion of the hole is called the *glide hole*. The outside diameter of the insert drill sleeve is the same as that of the glide hole, and the inside diameter is the same as that of the smaller drill bit, which has a diameter identical to that of the core of the screw. Insertion of this drill sleeve into the glide hole ensures concentric drilling of the trans-cortex and subsequent accurate reduction of the fracture (see Figure 76-15, *B*). The hole drilled through this sleeve across the trans-cortex or far cortex is referred to as the *thread hole*.

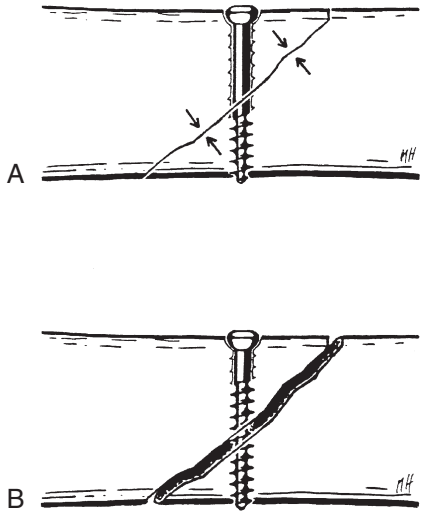


Figure 76-14. Lag screw technique. **A,** A cancellous screw of the correct thread length. All threads are located past the fracture plane, allowing interfragmentary compression. **B,** Selection of a cancellous screw with too long a thread length. Threads are located on both sides of the fracture plane, preventing interfragmentary compression.

To allow a greater contact area between the screw head and the bone, a depression is created at the near cortex using the countersink (see Figure 76-15, *C*). This decreases stress concentration at the screw head–bone interface. It is important to use the countersink in a 360-degree motion rather than in a to-and-fro motion; otherwise, an imperfect indentation is cut, preventing proper seating of the head. Countersinking is especially important in screws inserted at nonorthogonal angles relative to the surface of the bone. Care is taken to insert the nozzle at the tip of the countersink axially into the glide hole and to remove the bone making contact with the instrument. If this is not done, tightening of the screw results in stress accumulation at the screw head–bone junction, and bending of the screw head will result.

The depth gauge is then used to determine the total length of the screw, including the head (see Figure 76-15, *D*). Therefore, the length of the screw is measured to include the head. The depth gauge has a small hook at the end of its thin shaft that is inserted through the thread hole. By slightly tilting the instrument to one side, the hook catches the opposite cortex, and by sliding the movable portion toward the countersink depression, the exact length of screw is determined. In human and small animal osteosynthesis, 2 mm is added to the determined length to ensure that the tapered tip of the screw that does not engage the precut threads in the trans-cortex is positioned outside the bone. In equine bone, however, with the relatively thick cortices, the tapered tip is usually maintained within the bone, resulting in a loss of holding power of approximately 2 mm of screw. Because adequate bone stock is present

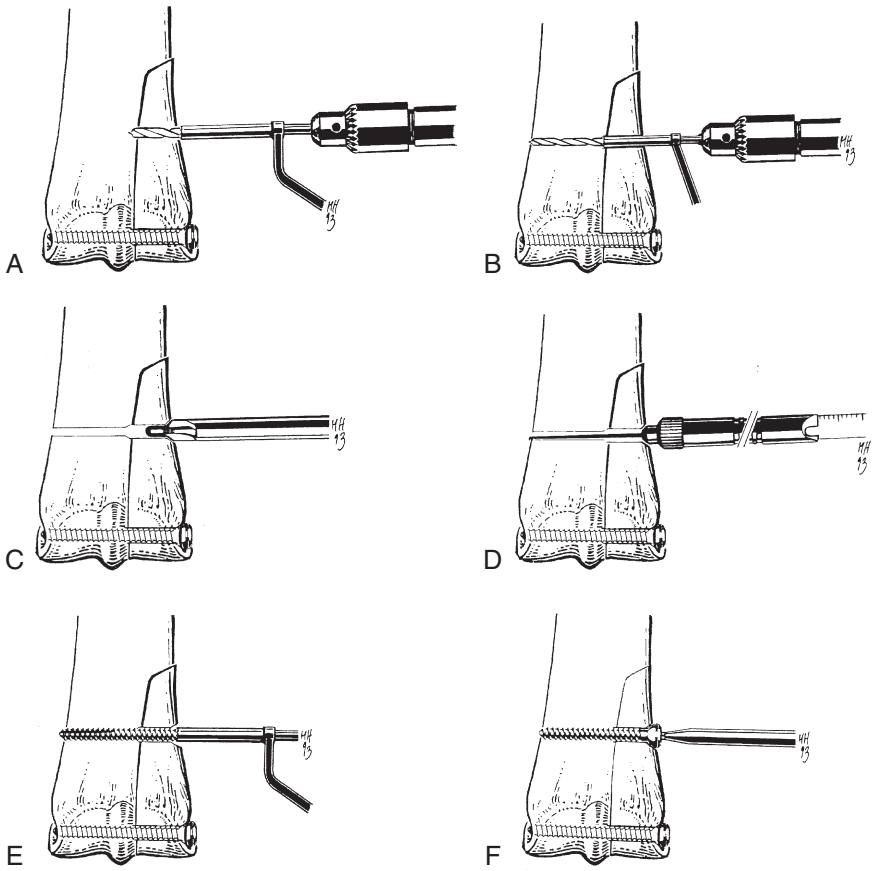


Figure 76-15. Lag technique, shown on a lateral condylar fracture of the distal third metacarpal. **A,** The cis-cortex is overdrilled. **B,** The insert drill bit is placed into the glide hole and advanced past the fracture plane, and the concentric thread hole is drilled across the trans-cortex. **C,** A depression for the screw head is prepared with the countersink. **D,** The required length of the screw is determined with the depth gauge. **E,** The threads are cut into the thread hole with the tap. **F,** The screw of predetermined length is inserted and solidly tightened with the hexagonal-tipped screwdriver. (From Nixon AJ: Equine Fracture Repair. Saunders, Philadelphia, 1996.)

to ensure secure fixation of the screw in the prepared hole, the screw that is used is either the exact length determined by the depth gauge or is 1 to 2 mm shorter.

Using the tap sleeve to protect the soft tissues as well as to help guide the tap, the tap is inserted into the glide hole and the threads are cut into the thread hole (see Figure 76-15, *E*). The threads are cut by advancing the tap three half-turns clockwise, followed by one half-turn counterclockwise. The counterclockwise action allows transport of the swath material into the flutes of the tap and ensures precise cutting of the threads without interference of the swath material cut from previous threads. An experienced surgeon may tap the thread hole with the air drill (power tapping) to speed up the procedure. This is especially advantageous when many screws are to be inserted. It requires experience, however, or serious complications may arise, such as stripping of the thread holes, cross threading of the tap, and instrument breakage. If a self-tapping screw is used, this step is not necessary. The small AO air drill and the Colibri air drill (small battery-powered drill) are not powerful enough to insert a self-tapping screw without applying a to-and-fro "power-tapping" technique. The Synthes ComPact Air Drive II, however, has one third more power and easily inserts self-tapping screws without to-and-fro movement.

Once the hole has been tapped, it is flushed to clean out swath debris and to lubricate it. A screw of the predetermined length is inserted, using the hexagonal tipped or stardrive screwdriver (see Figure 76-15, *F*) either by hand or with an air drill. Final tightening is always carried out by hand. Care is taken to avoid excessive force, which may result in failure of the screw head, or in stripping of the threads cut into the thread hole.[47] This is not a common problem in dense equine bone.

The shaft screw is inserted by applying the identical lag technique. However, care has to be taken that the glide hole is made slightly longer than the screw shaft. If the shaft is longer than the glide hole, no interfragmentary compression is achieved.

In the *lag screw technique*, a lag screw (either a partially threaded cancellous screw or a cannulated screw) is inserted after using a drill bit of only one size across the entire bone. Threads are cut along the total length of the hole with the cancellous tap, and the lag screw is inserted. The threads in the cis-cortex should not be engaged by the screw threads but only those of the trans-cortex, allowing achievement of interfragmentary compression.[1-4] Because the hardness of equine bone makes screw insertion difficult, it may be advisable to enlarge the cis-cortex with a 4.5-mm-diameter drill bit after first drilling the entire hole with the 3.6-mm drill bit.

Insertion of cannulated screws employs the same technique. However, the initial step involves placement of a guide wire in the desired location.[44] A special drill sleeve allows insertion of parallel screws close together. It is advisable to predrill equine cortical bone with a small drill bit before inserting the guide wire to prevent bending it. All instruments are cannulated to accept the guide wire. The size of the drill bit depends on the size of the screw to be implanted and the size of the guide wire. Once the guide wire is in place, its correct position and depth is ensured through radiography. If necessary, adjustments are made at this time. The measuring device is then placed over the portion protruding out of the bone. The length of guide wire

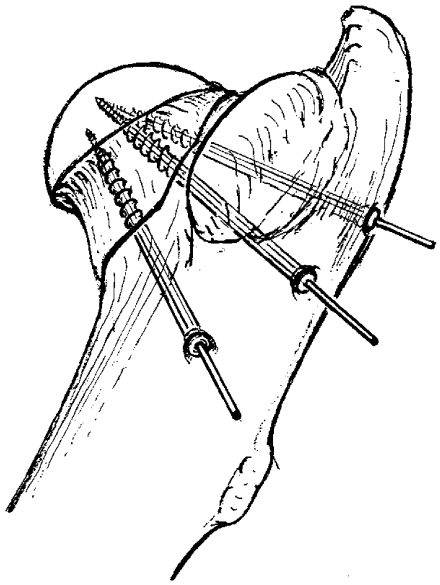


Figure 76-16. A capital femoral fracture is repaired with three cannulated screws. After the screws are inserted over a guide pin and tightened, the guide pins (which penetrated farther than the screws) are removed.

located in the bone is determined, and this determines the length of screw required. It is advisable to select a screw 3 to 5 mm shorter than the length of the guide wire inserted within the bone, to ensure secure seating of the wire throughout the implantation procedure. Subsequently, the cannulated drill bit is placed over the guide wire and the hole of predetermined length is prepared. The hole is tapped and finally the selected screw is inserted and firmly tightened (Figure 76-16). At the end, the guide wire is removed.

The 7.3-mm cannulated screw has a self-drilling and self-tapping tip. Therefore a screw of predetermined length is inserted without drilling a thread hole. Because of the initial insertion of the guide pin and the ability to select a screw of correct length, the danger of implanting a screw that is too long and protrudes from the opposite side of the bone is negligible. In equine bone, insertion occurs in the same manner as tapping, meaning that the screw is advanced three half-turns, followed by a half-turn in the opposite direction. Care has to be taken with power insertion.[39]

The *position screw* is used to maintain two pieces of bone at a certain distance apart and to prevent interfragmentary compression. This is achieved by drilling a hole of only one size (thread hole) across both cortices, followed by tapping. Only fully threaded cortex screws may be inserted as position screws. Because the threads catch in the cis- and trans-cortex, interfragmentary compression is prevented. No lag effect is achieved. It is advisable to apply a washer onto the bone surface to distribute the forces applied by the screw, because it is not possible to use the countersink. Its nozzle doesn't fit into the drill hole without overdrilling it for 13 mm (½ inch). In some instances the latter is not possible because it would result in a lag effect.

The *plate screw* is inserted by the technique described for the position screw. Any type of screw may be used in this manner. With the plate screw, the plate hole serves as a glide hole and allows compression of the plate onto the underlying bone, providing friction and stability.

Figure 76-17. Removal device for stripped 3.5-mm screw heads. An intact hexagonal hole in the screw head is shown. It is smaller than the tip of the screw. However, once the hole in the screw head is stripped, the tip of the device (whose threads are oriented in the direction opposite to those in the screw) will fit.

Screw removal

Cortex and locking-head screws are easily removed because of their fully threaded design. Similarly, shaft screws are easy to remove, because the shaft completely fills the glide hole. However, after a fracture has healed, a cancellous screw may be impossible to remove from hard equine bone, because during fracture healing, the precut threads in the cis-cortex fill in with solid bone. Removal of the screw requires the threads to cut their own way through the cis-cortex, a task for which they are not designed. This frequently results in the screw breaking, usually at the head–shaft or the shaft–thread junction. Therefore partially threaded cancellous screws should not be used when implant removal may be necessary at a later stage. Smaller cannulated screws have the same problem as cancellous screws. The 7.3-mm cannulated screw, however, contains a reverse-cutting edge at the caudal end of the threads, which facilitates recutting of the bone threads during screw removal (see Figure 76-13).[43]

Occasionally, the hexagonal indentation in the screw head is stripped during screw removal. This occurs if the screwdriver is improperly inserted in the hexagonal hole. Alternatively, if the hole is partially filled with tissue, the screwdriver cannot be inserted completely. Subsequent application of extraction force (counterclockwise motion on the screwdriver) may strip the hole within the screw head. This problem is mainly encountered with the hexagonal tipped 3.5-mm screw head and not in the stardrive head because of its smaller screw head. A special screw retrieval instrument has been designed for such situations (Figure 76-17). A shaft with a conical, threaded tip is inserted into the hexagonal indentation of the screw head. The threads of the tip have a reversed orientation compared with the screw threads. Therefore when the cone is tightened in the screw head with a counterclockwise motion, an extraction force is applied to the screw, allowing it to be easily removed. These screw-retrieval devices are available for all sizes of screws.

When a screw head is broken off, a special hollow drill bit is available to remove the bone surrounding the screw. Because it rotates counterclockwise, the threads on the inside of the hollow drill bit interlock with the screw, and advancing the drill

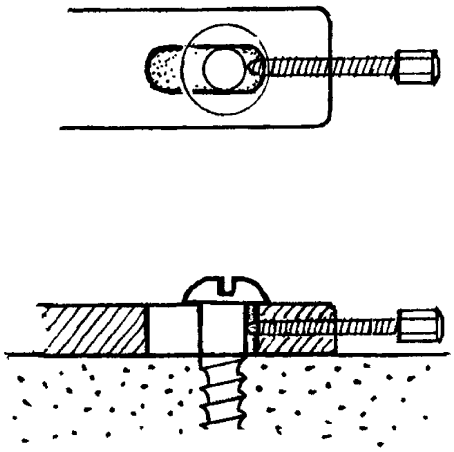


Figure 76-18. The Danis plate, developed in 1947. The plate screw is inserted into an oblong hole, and by tightening a smaller screw placed parallel to the long axis of the plate, the initial screw is displaced, providing axial compression of the fracture.

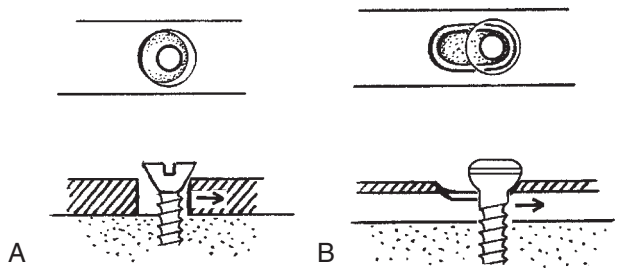


Figure 76-19. The Bagby plate, developed in 1958. **A,** A screw with a slanted screw head is inserted under load conditions into a larger plate hole, and through tightening of the screw, the axial compression is achieved across the fracture, similar to the dynamic compression plate principle shown in **B.**

bit removes the broken screw. For screws of all sizes that have broken off, a special screw retrieval set has been developed.

PLATES

The first plates to contain an axial compression device were developed by Danis in 1947.[63] They consisted of a plate with an oblong hole on one end. At the head of the plate, a compression screw could be introduced, which pushed the screw placed through the oblong plate hole toward the fracture line and in doing so provided axial compression to the fractured bone ends (Figure 76-18). Ten years later, Bagby introduced an impacting bone plate.[64] The heads of the screws he designed had a conical underside. If the screw was inserted eccentrically into the plate hole, the conical underside made contact with the edge of the plate hole. By tightening the screw, the bone into which the screw was implanted was displaced toward the fracture site and thus induced axial compression (Figure 76-19). The first plates developed by the AO in 1958 contained round holes (Figure 76-20, *A*).[63] Axial compression was applied with the help of a tension device (see later). Plate hole designs are shown in Figure 76-20 and described in the following paragraphs. The specific data on the different plates used routinely in equine osteosynthesis are summarized in Table 76-2.

The dynamic compression plate (DCP) was considered the basic plate in equine fracture treatment for a long time. Recently there has been a move away from this plate toward limited-contact dynamic compression plates (LC-DCPs) and locking compression plates (LCPs) (see later). Therefore, the detailed steps of plate application and discussion of the general principles of plate application in the horse are discussed under LC-DCP.

Dynamic compression plate

The 4.5-mm Dynamic Compression Plate is available in two plate widths. The narrow plate has holes arranged in a straight line, and the broad plate has holes offset to the left and right of the midline. The 3.5-mm broad plate, developed mainly for small animals, is manufactured from the same plate stock as the 4.5-mm narrow DCP. However, because of the smaller size of the plate holes, this basic plate is stronger than the narrow

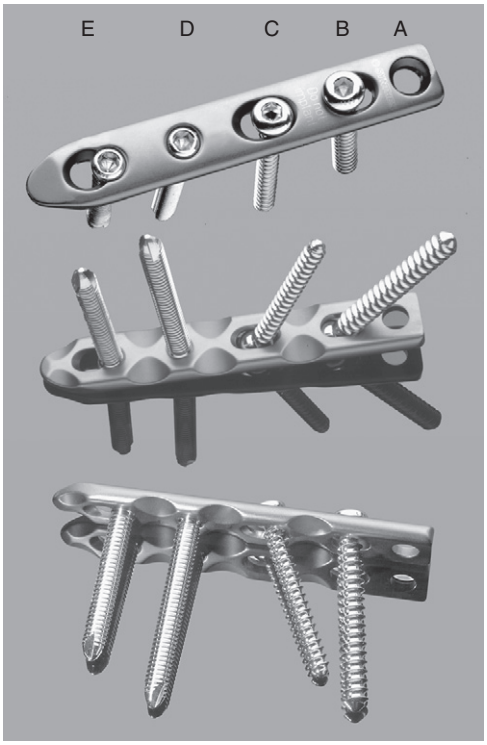


Figure 76-20. The plate holes designed for Synthes plates. *A,* The initially developed round hole. *B,* The dynamic compression plate (DCP) hole, which allows compression from one side. The screws can be angulated axially up to 25 degrees. *C,* The dynamic compression unit hole, which allows compression from either side and is used in the limited compression (LC)-DCP. The screws can be angulated axially up to 40 degrees. The plates have undercuts. *D,* Locking head plate holes used in the less-invasive stabilization system (LISS) plate, allowing only orthogonal insertion of the screws. The plates have undercuts. *E,* The combi-hole used in the locking compression plate allows the insertion of locking head screws—as shown here—and standard screws. The plates have undercuts and a pointed end that allows minimally invasive insertion through a small incision.

4.5-mm DCP and therefore may also be applied in foals.[65] The holes in a DCP are designed to achieve dynamic compression with tightening of screws inserted in the "loading" position. The holes are machined according to the sliding spherical principle with an incline or slope pointing downward towards the central portion of the plate (see Figure 76-19, *B*).[66] When a screw is inserted in the load position (offset 1 mm from the

TABLE 76-2. Standard and Special Plates Used in Large Animals

Name	LC-DCP 3.5	LC-DCP 3.5 Broad	LC-DCP 4.5	LC-DCP 4.5 Broad	DCP 3.5 Broad (Notes 1, 2)	DCP 4.5 Broad	DCS Plate	DHS Plate	LCP 3.5	LCP 3.5 Broad (Note 4)	LCP 4.5 Narrow	LCP 4.5 Broad	Equine LCP 5.5 Broad	One-Third Tubular Plate
Plate type	Standard	Standard	Standard	Standard	Special (note 3)	Special (note 3)	Special (note 3)	Special (note 3)	Special (note 3)	Standard	Special (note 3)	Special (note 3)	Special	Special
Plate cross-section														
Width (mm)	11	13.5	13.5	17.5	12	16	16	19	11	13.5	13.5	17.5	17.5	9
Thickness (mm)	3.3	4.2	4.2	5.2	3.6	4.8	5.4	5.8	3.4	4.2	4.2	5.2	6	1
Length (mm)	28 (2 holes) to 288 (22 holes)	94 (7 holes) to 289 (22 holes)	34 (2 holes) to 394 (22 holes)	106 (6 holes) to 394 (22 holes)	86 (7 holes) to 266 (22 holes)	103 (6 holes) to 359 (22 holes)	114 (6 holes) to 370 (22 holes)	See Table 76-3 below	27 (2 holes) to 287 (22 holes)	94 (7 holes) to 289 (22 holes)	66 (3 holes) to 287 (16 holes) (note 5)	116 (6 holes) to 440 (24 holes)	188 (10 holes) to 440 (24 holes)	28 (2 holes) to 148 (12 holes)
Plate angle	Straight	Straight	Straight	Straight	Straight	Straight	95°	(130° ALSO) 135°, (140°, 145°, 150°)	Straight	Straight	Straight	Straight	Straight	Straight
Angled portion	—	—	—	—	—	—	Barrel 25 mm long	Barrel 25 and 38 mm long	—	—	—	—	—	—
Screw size (mm)	3.5, 4.0	3.5, 4.0	4.5, 5.5, (6.5)	4.5, 5.5, (6.5)	3.5, 4.0	4.5, 5.5, (6.5)	4.5, 5.5, (6.5)	4.5, 5.5, (6.5)	3.5, 4.0 3.5 LS	3.5, 4.0 3.5 LS	4.5, 5.5, (6.5) 5.0 LS	4.5, 5.5, (6.5) 4.0 / 5.0 LS	4.5, 5.5, (6.5) 4.0 / 5.0 LS	3.5, 3.5 LS
Hole arrangement	Straight	Staggered	Straight	Staggered	Straight	Staggered	Staggered	Staggered	Straight	Straight	Straight	Staggered	Staggered	Straight
Hole spacing	13	13	18	18	12	16	16	16	13	13	18	18	18	12
Hole design	DCU	DCU	DCU	DCU	DCP	DCP	2 round, rest DCP	DCP	Combi-hole	Combi-hole	Combi-hole	Combi-hole	Combi-hole	Oval, round w/ collar
Plate mid-section	No	No	No	No	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Hole spacing in plate midsection	—	—	—	—	16	25	—	—	LO-LO 9 DCU-DCU 15	LO-LO 9 DCU-DCU 15	LO-LO 13 DCU-DCU 20	LO-LO 13 DCU-DCU 20	LO-LO 13 DCU-DCU 20	16

DCP, Dynamic compression plate; *DCS,* dynamic condylar screw; *DCU,* dynamic compression unit; *DHS,* dynamic hip screw; *LC-DCP,* limited-contact dynamic compression plate; *LCP,* locking compression plate; *LO,* locking.

TABLE 76-3. DHS Plate Lengths

Barrel Length	Barrel Angle	Plate Length
38 mm barrel	130°	46 (2 hole) to 238 (14 hole)
	135°	46 (2 hole) to 333 (20 hole)
	140°	46 (2 hole) to 270 (16 hole)
	145°	46 (2 hole) to 270 (16 hole)
	150°	46 (2 hole) to 333 (20 hole)
25 mm barrel	130°	46 (2 hole) to 110 (6 hole)
	135°	46 (2 hole) to 110 (6 hole)
	140°	46 (2 hole) to 110 (6 hole)
	145°	46 (2 hole) to 110 (6 hole)
	150°	46 (2 hole) to 110 (6 hole)

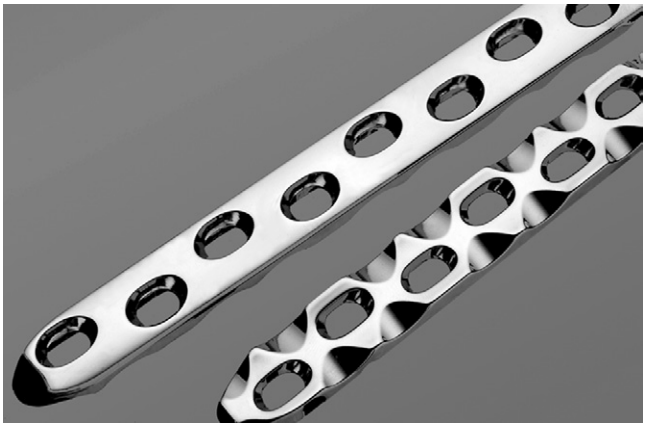


Figure 76-21. Limited-contact dynamic compression plate. The ends are pointed, the screw holes are arranged in two slightly offset rows *(top side of plate shown above)* evenly distributed along the plate, and the underside of the plate *(shown below)* contains undercuts.

center of the drill guide), the screw head contacts the plate at the top of the incline. During tightening, the screw head moves down the slope until it comes to rest at the bottom of the incline, just about in the center of the oval screw hole. Because the screw is introduced into the bone, screw movement toward the fracture line results in compression of the fractured bone ends. The center of the plate should be located over the fracture site, and this offset drilling can be carried out on either side of the fracture plane. Two screws on either side of the fracture can be used in the load position; using the plate holes alone will provide a maximum of 4 mm compression. Before tightening the second screw, the first screw on the same side of the fracture plane has to be loosened to achieve the additional 1 mm compression. Following tightening, the loosened screw is tightened again. Additional compression requires the external tension device.

Limited-contact dynamic compression plate

The DCP, up to now the workhorse for equine fracture treatment, was for a while replaced by the LC-DCP, especially in the United States. Studies in human medicine showed that the DCP caused osteoporosis under the plate, although this is not encountered in equine surgery (see later).[1] This led to the development of biologically improved plates.

In the conventional DCP, the plate holes provided the least resistance to failure. This problem was somewhat offset in equine fracture treatment by inserting screws through all plate holes. By designing a plate that contained at each cross-section along its entire length the same amount of metal, an implant of uniform bending stiffness was developed.[62] To achieve this, half-moon–shaped pieces of metal were removed from the underside of the plate. This resulted in limited contact between bone and the plate, which led to the name of the plate: LC-DCP (Figure 76-21). The limited contact surface was welcomed in human surgery to fight the problem of osteoporosis developing under the plate.[62] Extensive tests comparing the LC-DCP with the conventional DCP revealed that the LC-DCP had an equal bending stiffness and a 50% increase in the continuity of the bending stiffness.[62] This reduces local stress concentration near fracture gaps. Additionally, the blood supply of the bone under the plate was significantly improved.[67] Early mechanical tests conducted in the AO Research Institute in Davos revealed that the design of the LC-DCP provided increased resistance to cycling failure compared with the DCP.[65] The undercuts of the plate allow the development of some callus bridges over the fracture gap, which led to a significant increase in stability, despite the fact the these bridges are small. The trapezoidal cross section allows the formation of shorter but stronger bone lamellae on either side. Also, it prevents the bone from growing over the plate. The dynamic compression unit (DCU) hole design in the LC-DCP, which allows axial compression to be applied from either side of the hole, replaced the conventional DCP hole design (see Figure 76-20). This allows the distribution of the plate holes evenly along the entire plate and obviated the need for a center in the plate. The DCU hole is also undercut at each end to allow the insertion of screws up to a 40-degree angle relative to the orthogonal direction. In the DCP hole, only a 25-degree angulation can be achieved.[64]

There are two plate widths; the narrow plate has holes arranged in a straight line, and the broad plate has alternating offset holes.

The DCU holes are specially designed to allow dynamic compression as the screw is tightened. The holes are arranged according to the spherical gliding principle, with an incline, or slope, toward the center portion of the plate (Figure 76-22).[65,66] This offset drilling can be carried out on either side of each hole and therefore on either side of the fracture line.[65] The application of the LC-DCP requires the use of the special LC-DCP double drill guide identified by its undercuts on the handle, identical to the ones under the plate itself (Figures 76-23 to 76-26). (Note: the DCP drill guide should not be used with the LC-DCP.) An alternative to the LC-DCP drill guide is the *universal drill guide*, which contains a spring-loaded tip (see Figure 76-23). Pressing down on the drill guide places it near the center of the hole (see Figure 76-26, *B*). Placing the spring-loaded tip on the far end of the DCU hole relative to the fracture line (without pressing down on the drill guide) allows a 1-mm compression of the fracture line (see Figure 76-25, *A*).

The technique of application is as follows. The fracture is reduced (Figure 76-27, *A*), and it is maintained in that configuration initially with pointed reduction forceps until one or two interfragmentary cortex screws, 3.5 or 4.5 mm in diameter, can be applied in lag fashion (see Figure 76-27, *B*). The plate is then contoured, overbent at the fracture site, and applied to the bone (see Figure 76-27, *C*). The first screw hole is drilled toward one end of the plate in neutral position (green LC-DCP drill guide

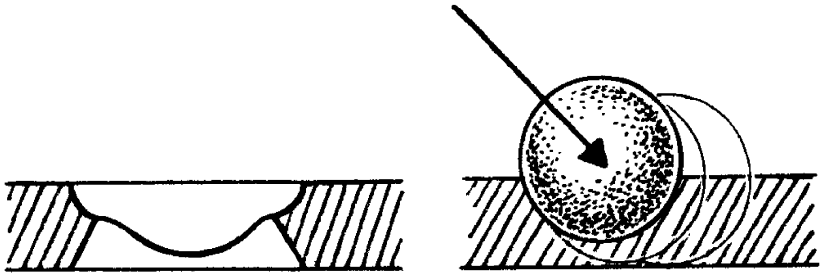


Figure 76-22. Design of the dynamic compression unit (DCU). Both sides of the hole are shaped like an inclined cylinder. Like a ball, the screw head slides down the incline. Because the screw head is fixed to the bone via the shaft, it can move only vertically relative to the bone. The horizontal movement of the head, as it impacts against the angled side of the hole, results in movement of the bone fragment relative to the plate and leads to compression of the fracture. With the DCU, compression can be achieved on either side, obviating the need for the plate to have a center, as in the dynamic compression plate.

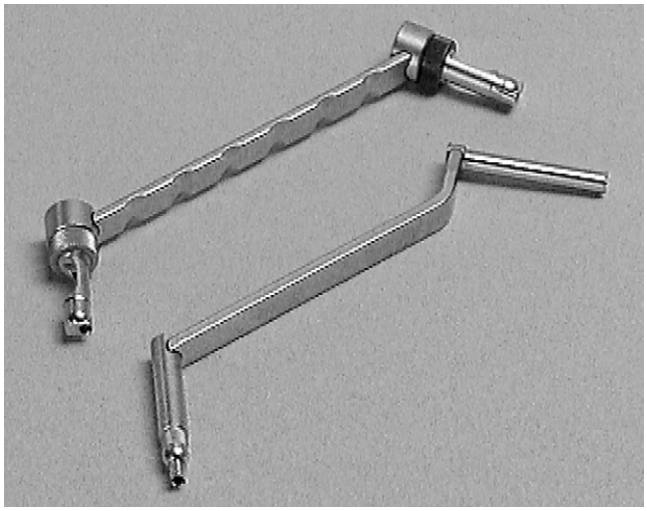


Figure 76-23. The limited-contact dynamic compression plate (LC-DCP) double drill guide *(top)* contains undercuts like those of the corresponding plate to distinguish it from the DCP double drill guide. The dark ring (green) represents the neutral guide, the light ring (yellow) the load guide. The universal drill guide *(bottom)* contains a spring-loaded guide for the thread hole and a larger guide for the glide hole. The universal drill guide is available for the different screw sizes.

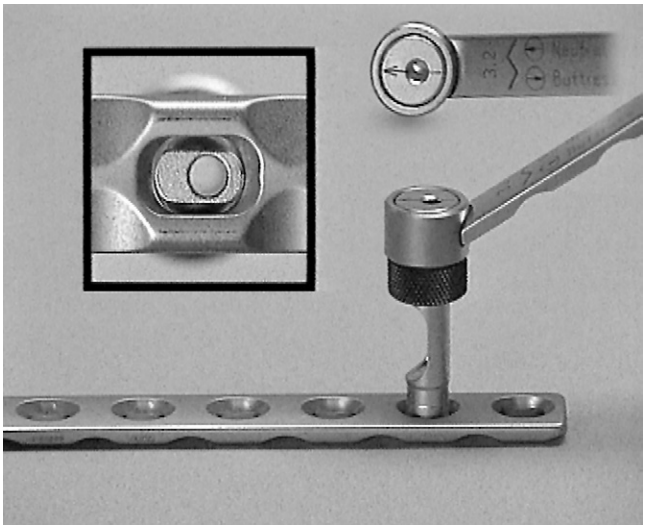


Figure 76-24. The neutral LC-DCP drill guide inserted into a dynamic compression unit (DCU) hole *(bottom).* From below the plate, it can be seen that there is a gap between the right end of the DCU hole and the drill guide hole *(top left).* From the top, the arrow pointing toward the fracture line is visible *(top right).* In the DCP neutral guide, there is no arrow.

or pressed-down universal drill guide). The screw is inserted but not completely tightened. This allows the plate to be pulled into a loaded position. The same can be achieved by drilling the initial hole through the load (yellow) LC-DCP guide or the universal drill guide placed at the far end of the hole, and maintaining the plate in the same position (see Figure 76-27, *D*). The hole for the second screw is drilled on the other side of the fracture line through a plate hole near the other end, using the load drill guide (see Figure 76-27, *E*), if additional compression is needed. Care is taken to ensure correct plate position before drilling the second hole. The hole is prepared for the screw, which is subsequently inserted. Interfragmentary compression is achieved through alternate tightening of the two screws (see Figure 76-27, *F*). More screws may be applied in the loaded position on either side of the fracture. A maximum of two screws can be placed under load conditions on either side of the fracture line. Therefore a maximal compression of 4 mm can be achieved. Before the second loaded screw is completely tightened, the first one on the same side has to be slightly loosened. This allows the additional compression to be applied. As

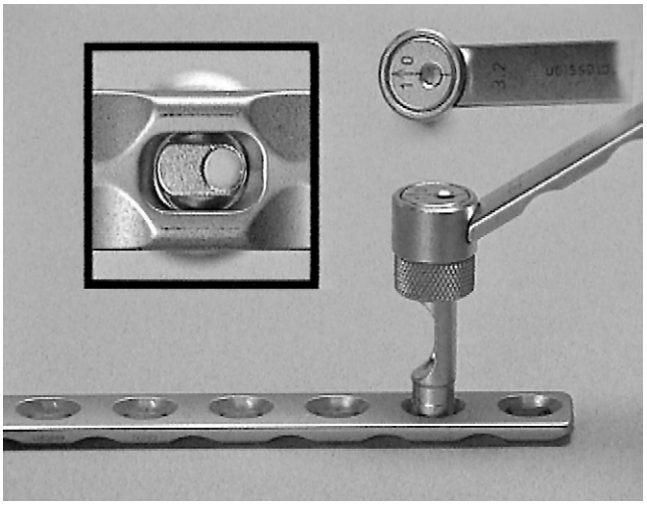


Figure 76-25. The load LC-DCP drill guide inserted into a dynamic compression unit (DCU) hole *(bottom).* From below the plate, it can be seen that there is no gap between the right end of the DCU hole and the drill guide hole *(top left).* From the top, the arrow pointing toward the fracture line is visible *(top right).*

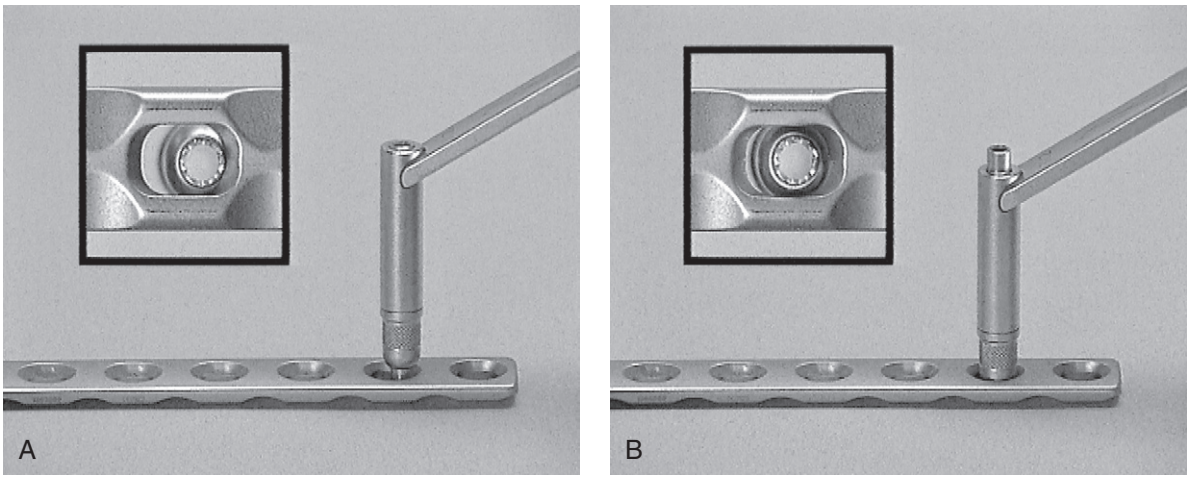


Figure 76-26. A, The universal drill guide placed into a dynamic compression unit (DCU) hole without applying pressure onto the guide. Viewed from below, the hole in the guide can be seen very close to the right end of the DCU hole. **B,** The universal drill guide placed into a DCU hole under pressure. The spring-loaded part of the guide is sticking out on top. From below, a gap can be seen between the hole in the guide and the right end of the DCU hole.

mentioned before, without this loosening, no additional compression is achieved and the two screws that are "compressed" toward each other are stressed.

The remaining screws are implanted in neutral position. Any screw placed through a plate across a fracture line is introduced using the lag technique (see Figure 76-27, *G*). All the screws are finally tightened (see Figure 76-27, *H*). A cortex screw inserted through a plate in lag fashion perpendicularly across a fracture may not achieve the desired effect, because the threads in the glide hole cut into the cortex and prevent any gliding.[65] To correct this undesirable effect, the shaft screw was developed. The shaft, which fills out the glide hole completely, does not cut into the cortex.

Any cortex or shaft screw can be inserted under load (at the far end of the oblong plate hole) or in neutral position (at the center) of each plate hole. Under load, 1-mm centripetal displacement or compression of the fracture gap is implemented. In neutral position, 0.1-mm compression is achieved. If double plating is applied, only two screws are placed under load in the second plate, which in most cases is arranged 90 degrees to the first plate.

Plates are contoured with the help of a plate-bending press to fit the surface of the bone. A perfectly contoured plate, however, compresses only the cortex immediately under the plate, whereas the opposite cortex remains decompressed (Figure 76-28, *A*). By slightly overbending the plates at the fracture site (see Figure 76-28, *B*), compression is achieved along the entire circumference of the bone (see Figure 76-28, *C*).[1-3]

Axial interfragmentary compression under a plate may also be implemented with the help of a tension device (Figure 76-29). The plate is applied to the bone with several screws in neutral position on one end of the fracture. The tension device is hooked into last hole on the other end of the plate and attached to the underlying bone through a unicortically applied 4.5-mm cortex screw. With the help of a wrench, the tension device is tightened, which pulls the plate toward the tension device and thus applies compression to the fracture site. Once adequate compression is applied, screws are inserted on the other side of the fracture through the plate in neutral position and tightened. The tension device is subsequently removed and the remaining screws are inserted in empty plate holes.

An *in vitro* study comparing the broad 4.5-mm DCP with the 4.5-mm LC-DCP revealed that the LC-DCP provided increased stability in static overload testing; however, it was significantly weaker in cyclic fatigue testing, which contradicted an earlier study that was not performed on cadaveric equine bone.[68] The results of another *in vitro* study conducted by the same group comparing the 4.5-mm LC-DCP with the 5.5-mm LC-DCP in the same model used above are interesting.[69] It showed that the 5.5-mm LC-DCP was superior in resisting static overload forces in palmarodorsal four-point bending. There was no significant difference in resisting static overload in torsion, but the 5.5-mm LC-DCP offered significantly less stability in cyclic fatigue loading. The 5.5-mm LC-DCP was previewed as the equine plate for fracture fixation and arthrodesis of the metacarpophalangeal joint. Because of the rapid gain in popularity of locking plates, the 5.5-mm LC-DCP was abandoned and replaced with a 5.5-mm LCP (see later).

The stability of the fixation is derived from friction between the implants and the bone. A technique called *plate luting* has been developed to obtain 100% plate–bone contact by applying bone cement (methyl methacrylate) between the plate and the bone.[70] This is achieved after all the screws of the plate are inserted. All the screws are then loosened, the plate is lifted off the bone, the soft bone cement is placed underneath it, and the screws of the plate are retightened, preferably with the power drill. Entrance of bone cement into the fracture line must be prevented because it retards or prevents bony union in that area. Once the screws are tightened, the soft cement fills the oblong plate holes around the screw heads and provides additional support, making the fixation extremely rigid.[70-72] When only the oblong plate holes are filled with bone cement, a similar but lesser increase in strain protection occurs.[72] Excess bone cement is rapidly and carefully removed. Plate luting is especially useful on bones with anatomically complex surfaces that make contouring of the plate difficult.[72] The addition of gentamicin into the bone cement facilitates long-term release of this antibiotic and provides effective protection against postoperative infections (see Chapter 85). Plate luting is not used in humans and

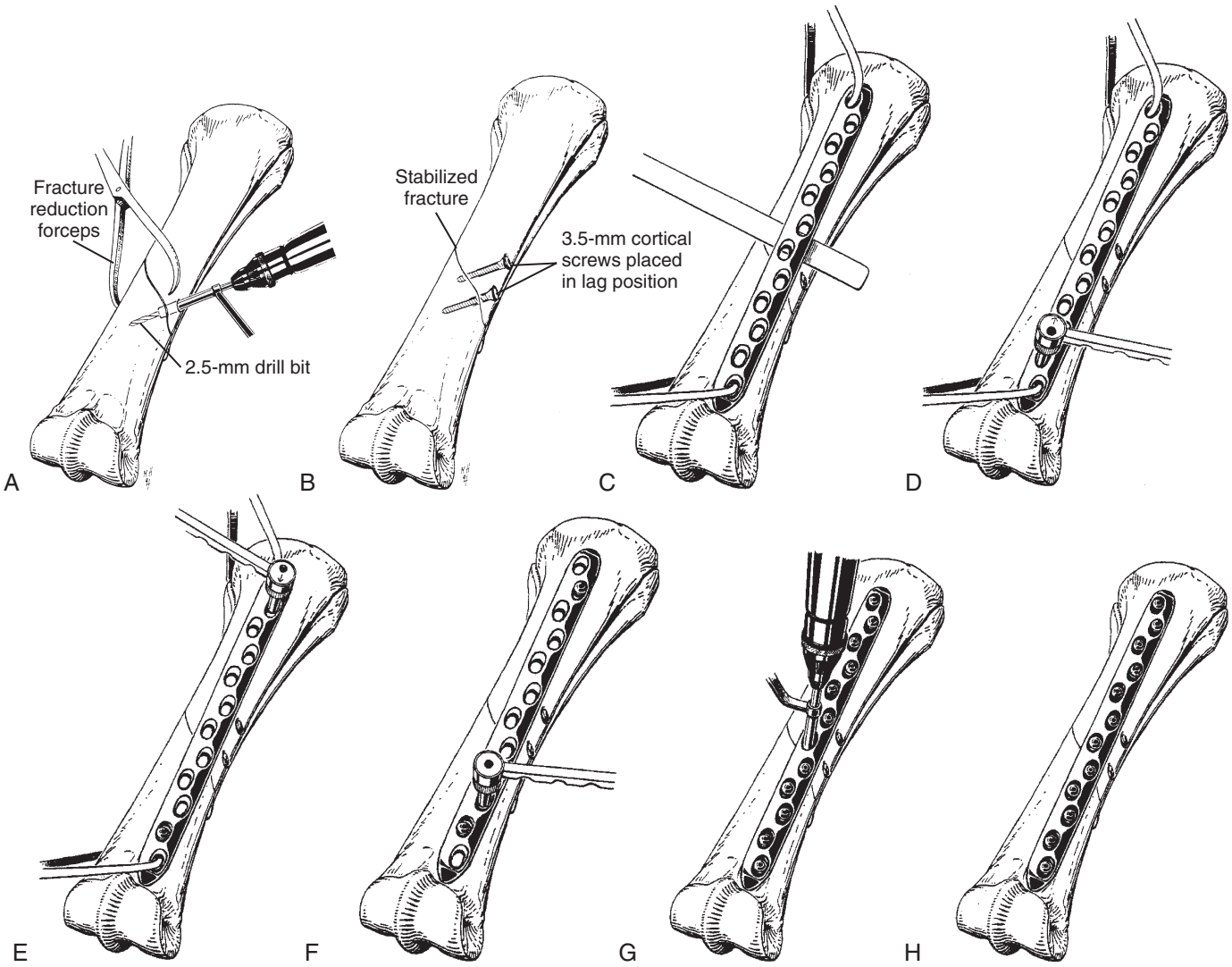


Figure 76-27. Repair of a simple oblique fracture of the third metacarpal with two cortex screws applied in lag technique combined with a broad LC-DCP as a neutralization plate. **A,** The large pointed reduction forceps maintains alignment of the fractured bone during implantation of the two 3.5-mm cortex screws. **B,** The two screws are implanted and the reduction forceps is removed. **C,** A 10-hole broad LC-DCP is applied to the dorsolateral aspect of the bone, distal from the two interfragmentary 3.5-mm cortex screws. The plate was overbent at the fracture site, allowing introduction of an aluminum template between the bone and the plate. **D,** A thread hole is drilled across the bone through the second most distal plate hole with the help of the yellow load drill sleeve. **E,** The screw is inserted but not completely tightened, followed by preparation of an identical hole at the opposite end of the plate. **F,** The second screw is inserted and both are alternately tightened, placing the fracture under axial compression. The remaining screw holes are prepared through the green neutral drill guide. **G,** A cortex screw is implanted in lag fashion across the fracture plane. **H,** All the screws are tightened. (From Nixon AJ: Equine Fracture Repair. Saunders, Philadelphia, 1996.)

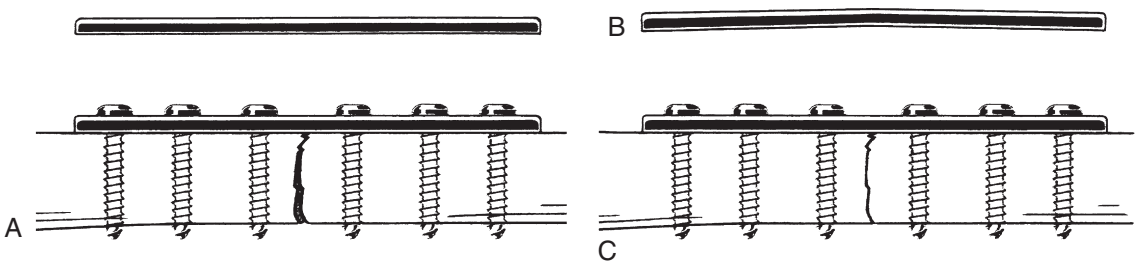


Figure 76-28. Application of a plate onto a bone. **A,** If a plate is perfectly contoured to the surface of the bone, a narrow gap develops at the fracture site opposite the plate after insertion and tightening of the screws. **B,** To overcome this problem, the plate is overbent (prestressed) about 1 mm, right at the fracture site. **C,** When the screws are reinserted, the entire circumference of the fracture is under compression.

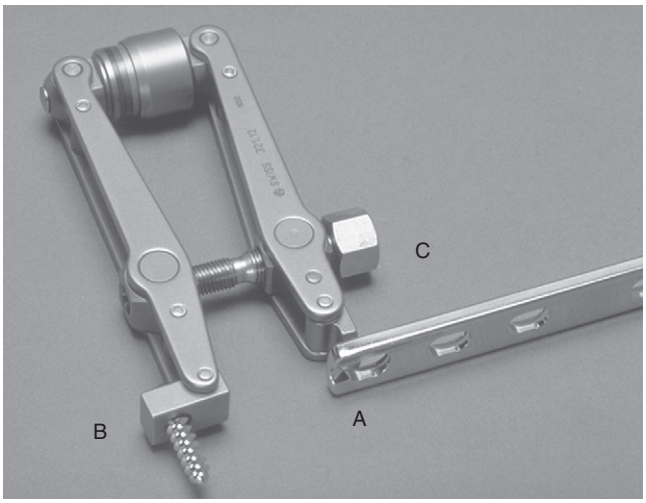


Figure 76-29. *A,* The tension device is hooked in the last plate hole. *B,* The device is attached to the bone with a short screw. *C,* Twisting of the hexagonal screw head pulls the plate toward the left side and applies axial compression to the underlying fracture.

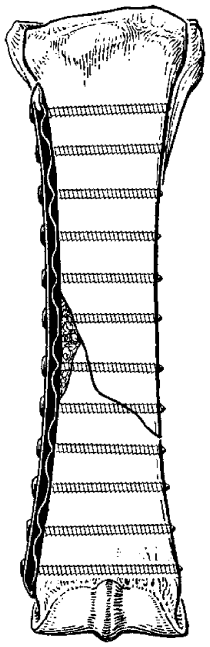


Figure 76-30. Comminuted midshaft fracture of the third metacarpal showing a bone defect. The defect is filled with cancellous bone graft, and a broad 12-hole LC-DCP is applied over the defect in buttress function. For better visualization, the dorsally applied LC-DCP, routinely used in a clinical case, is not shown.

small animals because vascular necrosis of the bone develops under the plate, resulting in pathologic fractures after implant removal. This complication has not been reported in horses. With the use of locking plates, the importance of plate luting has decreased because the locking head screws effectively prevent micromovement of the screw heads within the oblong plate holes (see later).

Screws should be inserted perpendicular to the surface of the bone. If a second plate is used, it should be positioned to allow the screw holes to be located between the screws of the other plate.[39,73,74] This reduces the likelihood of inadvertent contact between the screws of the two plates. Every hole in a plate should be filled with a screw.[39] Should a hole traverse a fracture line, the lag technique should be applied by overdrilling of the cis-cortex, and the screw should be directed so that it engages the opposite cortex next to the fracture line. Where no support can be achieved in a cortex, bone cement may be placed and the screw implanted. After the cement hardens, the screw will be solidly fixed.

Application of 4.5-mm screws through a 4.5-mm plate allows 40 degrees of longitudinal angulation and 7 degrees of lateral angulation.[65] Application of a 5.5-mm screw through the same hole allows only about 25 degrees of longitudinal angulation. The plates applied for fixation of a long bone fracture ideally extend over the entire length of the bone.[4] Shorter plates must be staggered to ensure plate coverage of the total length of the bone. The most distal end of the proximal fragment in an oblique long bone fracture should be wedged between a plate and the opposing distal fragment. Therefore the configuration of a fracture dictates to a certain extent the location of the plates to be applied (not just the tension side of the bone; see later).[39] Implants should be applied at a distance from severely bruised skin or areas of frank skin defects.

Plate functions include compression, neutralization, tension band, and buttressing. The design of a plate does not dictate its function, because the same plate may be used for different functions, and a plate may serve more than one function at a time. The compression and neutralization functions are the most frequently applied. When a plate serves in *compression* function, two screws on either side of the fracture line are placed under a load.[3] A plate serves a *neutralization* function after anatomic reconstruction and interfragmentary compression of a simple or comminuted fracture is accomplished by several screws placed in lag fashion.[1-4,39] The various diverging shear, bending, and rotational torque forces exerted by these screws on the surrounding bone are neutralized by one or two plates. Such plates effectively bridge the proximal and distal aspects of the bone and protect the fixed fracture. The screws are inserted in the neutral position. Application of neutralization plates has to be planned ahead of time to avoid interfering with the reduction screws or eliminating options for their placement. Most plates applied satisfy both compression and neutralization functions.

A plate applied in the *tension band function* transforms the tensile forces applied to the fractured bone underneath into compressive forces. The classic example of such a plate is one that is applied to fix an olecranon fracture. Because these plates are subjected mainly to tensile forces, they may be smaller than the plates used in compression or neutralization functions.

Cortical bone defects that persist after anatomic reconstruction of the fracture cause instability of the repair because of stress concentration. Such areas need to be protected and bridged by an implant, maintaining length by preventing collapse of the fixation (Figure 76-30). A plate applied in such a fashion is called a *buttress plate.* It is advisable to fill the defect in the bone with a cancellous bone graft or bone replacement material. Any screw placed through the plate in the region of the defect should engage the trans-cortex. All the other principles for plate fixation are applied.

One-third tubular plate

One-third tubular plates are very thin and, depending on the size of the hole, may be applied with a 3.5- or a 4.5-mm screw (see Table 76-2). The 3.5-mm plate is applied using standard technique in fracture treatment of proximal MCII, MCIV, MTII, and MTIV fractures in adult horses and of nondisplaced ulnar fractures in very young foals.

T-plates

T-plates are available for 4.5- and 5.5-mm screws as well as in a smaller version for the 3.5-mm screws. These plates are suited to areas where tension is applied without bending and sufficient space is not available for the application of a straight, regular plate. T-plates have been used for arthrodeses of the tarsometatarsal and distal intertarsal joints. The popularity of these plates has drastically decreased during the last few years.

Dynamic condylar screw and dynamic hip screw system

Dynamic condylar screw (DCS) and dynamic hip screw (DHS) plates are implant systems that were developed on the principles of the angled blade plate (ABP).[75] The set contains the instruments, screws, and selected plates. These plates consist of a long lag screw with a 12.5-mm (½-inch) thread width, a 25-mm (1-inch) thread length, and an 8-mm shaft diameter. The shaft is flattened on two opposing sides to prevent rotation of the screw after it is introduced into the barrel of the plate. The DCS or DHS is inserted at a predetermined angle (95 degrees for the DCS plate and 135 degrees [standard] for the DHS plate) (see Table 76-2). The most important step in the application of the DCS and DHS plate systems is the correct insertion of the 2.5-mm guide pin. Drill guides aid in the placement of the guide pin, which is best verified intraoperatively with an image intensifier. (Intraoperative radiographs, despite being more time consuming, are satisfactory as well.) Predrilling of the cortex with a 2.5-mm drill bit facilitates insertion of the guide pin. Care has to be taken that all of the four points on the base of the drill guide under the handle are in contact with the bone during drilling, even if the drill guide tip does not make contact with the underlying bone (Figure 76-31, *A*). Tilting of the drill guide with its tip down to bone results in false orientation of the DCS, which prevents the plate from making contact with the bone. This requires complicated adjustments of the plate angulations to make the plate contact the bone and prolongs the surgical procedure. If the plate–barrel junction is not in direct contact with the bone, the gap may be bridged with polymethyl methacrylate (PMMA). Once the guide pin is correctly placed, the subsequent steps are easily accomplished because all instruments are hollow and accept the guide pin.

The measuring device is placed over the guide pin, and its depth in the bone is determined (see Figure 76-31, *A*). The triple reamer placed over the guide pin allows simultaneous drilling of the core hole, the portion for the plate barrel, and the beveled plate–barrel interface (see Figure 76-31, *B*). The reamer is assembled and adjusted to a length measuring 5 mm less than the pin portion located within the bone. This ensures persistence of the pin in its position throughout the screw insertion procedure. After preparing the screw hole, the threads are cut using routine technique (see Figure 76-31, *C*), and the screw of appropriate length is introduced, followed by application of the plate (see Figure 76-31, *D*). Once the shaft of the screw and the barrel are aligned, the barrel slides easily over the shaft and the plate position can be adjusted before impacting it onto the bone (see Figure 76-31, *E*). The barrel has the same inside diameter as the screw shaft cross section (8 mm and flattened at two opposite sides). The DCS plate has a barrel length of 25 mm, whereas the standard DHS has a barrel length of 38 mm (1½ inches). A special version of the DHS plate has a 25-mm barrel length. After their implantation, the lag screw and the plate are joined with a connecting screw, making the two components work as one unit (see Figure 76-31, *F*). Tightening of the connecting screw creates interfragmentary compression, provided the lag screw has passed the fracture line.

The DHS plate is the strongest plate available from Synthes.[73] The DCS and DHS plates are versatile, rapidly implanted, and a real asset to large animal surgery, especially when treating long bone fractures in adult horses.[76,77] The DCS system is useful in metaphyseal fractures of the MCIII or MTIII (Figure 76-32), the proximal radius, and even the femur. The DHS may be applied for arthrodesis of the metacarpophalangeal joint and in selected femoral fractures.[75] Combined with 5.5-mm screws, these plates produce extremely strong fixations.

Recently, the DCS/DHS plates were upgraded and equipped with combi-holes to allow insertion of locking head screws (see later, under "Locking Compression Plate").

Point contact fixator

The point contact fixator (PC-Fix) plate, which is no longer manufactured, is mentioned here for historical purposes, because it represented the first type of internal fixator developed by Synthes.[77-79] It was manufactured from pure titanium, and it had round, conical screw holes, evenly distributed along the plate, and a specially designed underside that represents a further development of the LC-DCP. One row of points is arranged along either side of the plate, with the points being located between two plate holes. Between two points, the plate is undercut in an arcuate pattern, similar to the LC-DCP but to a greater extent. The short unicortical screws contained a conical head that locked in the plate hole while being tightened, forming a solid unit between plate and screws, without applying any load onto the screw threads (Figure 76-33). The first clinical tests conducted with the PC-Fix were done on large animals, mainly cattle (Figure 76-34).[80] The subsequently developed PC-Fix II was manufactured only as a 3.5-mm implant system. It had a slightly altered plate design and was applied with self-tapping screws.[81] A special plate-bending device was developed to prevent altering of the plate holes during bending. Development of this system led to the next generation of titanium implants: the LISS, which is described next.

Less-invasive stabilization system

The less-invasive stabilization system (LISS) consists of forged titanium plates, manufactured for the bones and sides of the bones (lateral versus medial aspect) to which it will be applied. The plates cannot be bent and are applied to the bone with self-cutting, self-tapping titanium screws of predetermined length. The shape of the plate is predetermined and forged accordingly.[49,82] A guide system for transcutaneous insertion of the screws is mounted on the plate head (Figure 76-35, *A*). This bar also facilitates insertion of the plate through a small surgical incision at one end of the bone and subsequent advancement of the plate along the periosteum, therefore bridging the approximately reduced fracture. Once in place, the last plate hole is approached through a stab incision and connected

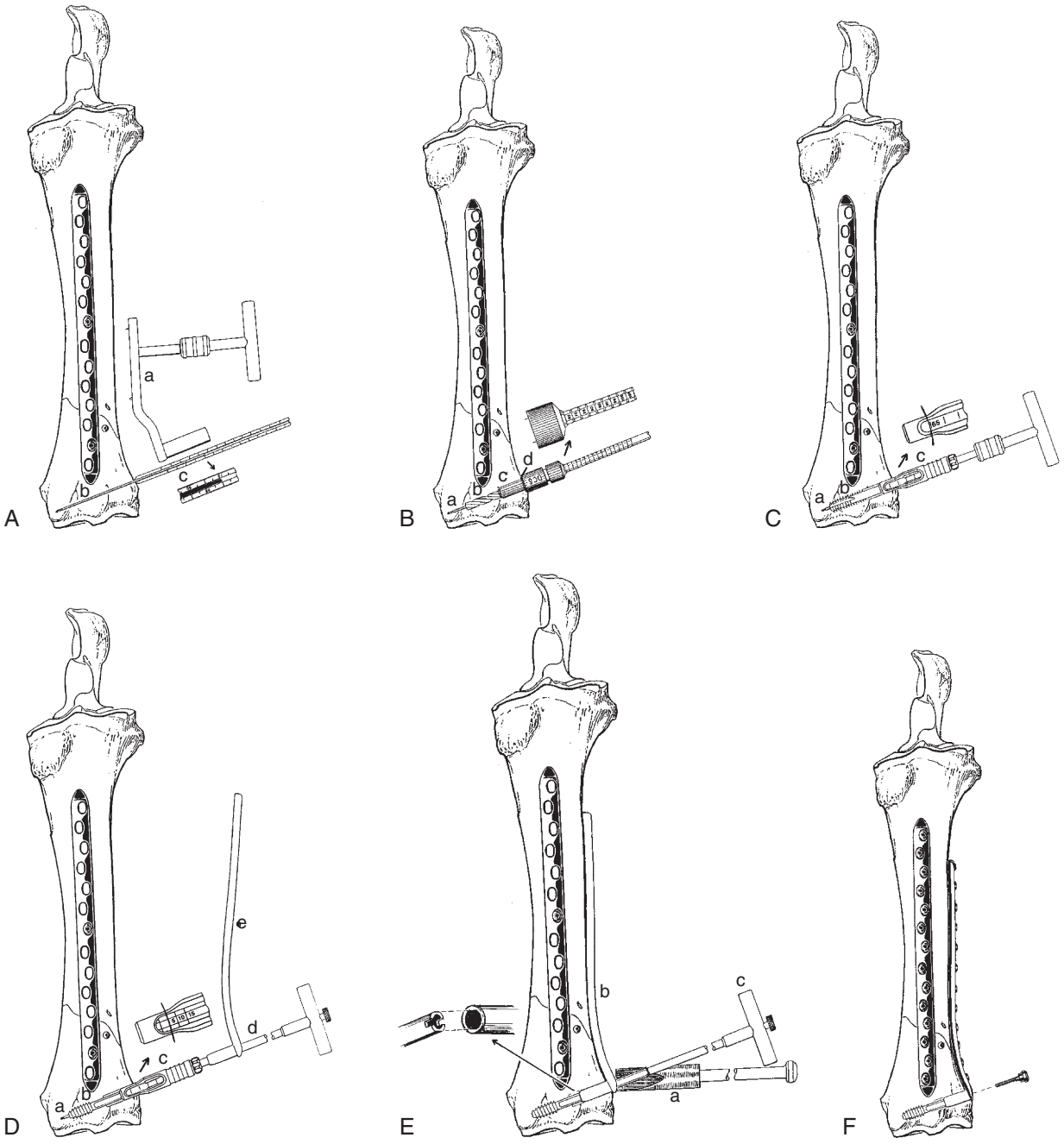


Figure 76-31. Application of an LC-DCP and a dynamic condylar screw (DCS) plate to a radius fracture. **A,** The fracture is initially repaired with two interfragmentary 3.5-mm cortex screws, applied in lag fashion. Subsequently, a 14-hole LC-DCP is applied to the cranial bone surface (tension side) under compression. The guide pin *(b)* is applied through the special drill guide *(a).* The measuring device *(c)* applied over the guide pin allows determination of the length of the pin inserted in the bone (70 mm). **B,** The DCS triple reamer is assembled and set for the 65-mm drilling depth, which is 5 mm less than the pin length in the bone and ensures maintenance of the pin during DCS screw insertion. The triple reamer is placed over the guide pin *(a)* and the shaft hole for the DCS screw *(b),* the barrel hole for the plate *(c),* and the barrel–plate junction *(d)* are prepared. **C,** The DCS centering sleeve *(c)* is placed over the tap *(b),* which is subsequently placed over the guide pin *(a).* After inserting the centering sleeve into the barrel hole, the tap is advanced to the desired depth (65 mm). **D,** The DCS coupling screw *(d)* is placed through the T-handle and the DCS plate *(e)* is selected (12-hole) and connected to the DCS screw *(b)* of the desired length (60 mm). The centering sleeve *(c)* is applied over the coupling screw. After placing the assembly over the guide pin, the screw is inserted to a depth of 65 mm, which is marked on the centering sleeve as 5 mm. **E,** After tightening the screw and adjusting the horizontal bar of the T-handle parallel to the long axis of the bone, the DCS plate is seated over the shaft of the DCS screw with the help of the DCS impactor *(a)* and a mallet (not shown). Orientation of the instruments and implants is important, because the DCS screw *(left insert)* and the plate barrel *(right insert)* contain complementary parallel contours, which have to be aligned to allow sliding of the barrel over the screw shaft. **F,** The DCS compression screw is inserted through the plate barrel, inserted into the back end of the DCS screw, and tightened. This unites the three components (DCS screw, DCS plate, and connecting screw) into one unit. Insertion of all remaining screws and tightening of them completes the procedure.

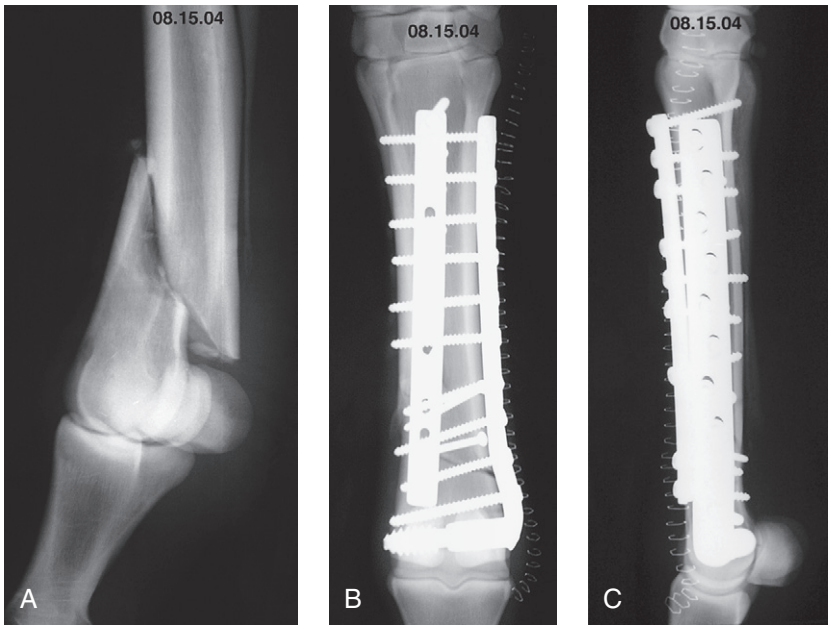


Figure 76-32. Preoperative **(A)** and postoperative **(B** and **C)** radiographs of a metaphyseal of the third matacarpal fracture in an adult Icelandic pony. The fracture was repaired with a laterally applied DCS plate and a shorter dorsal DCP.

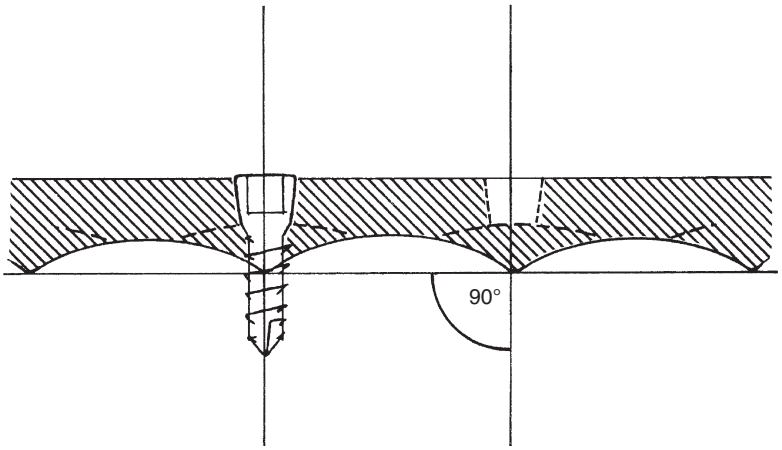


Figure 76-33. Side view of PC-Fix. The arcs under the plate are easily recognized and lead to the points in contact with the bone. The unicortical screws are implanted perpendicular to the long axis of the plate, where the conical screw heads lock within the plate holes. This is an internal fixator. The plate is applied over the periosteum.

through the last hole in the aiming device with a drill sleeve, which is threaded into the plate. This establishes a solid frame, which maintains its angles during screw application and ensures that the screws are inserted orthogonal to the long axis of the plate so that they may be threaded into the plate holes. The remaining screws are inserted transcutaneously through stab incisions. An anatomic plate–bone interface is not important, because the screw heads interlock with the round, threaded plate holes, establishing a solid internal fixator. If the correct implant is selected, implantation of the system is efficient. This system is especially well suited to foals. A successful fixation has also been accomplished in a tibial fracture in a calf (see Figure 76-35, *B* and *C*).[83]

Locking compression plate

The locking compression plate (LCP) is an implant system that combines the two treatment methods in one implant: compression plating and internal fixation.[84] The LCP was developed to include the axial loading capabilities of the DCP and LC-DCP, the decreased plate–bone contact of the LC-DCP, and the rigidity and stiffness of the LISS, where locking screws were first used.[82,84] The goals were met by designing a combi-hole where either a standard screw or a locking screw can be inserted. It is not necessary to only apply locking head screws.[85] An *in vitro* study comparing the application of two LCPs at right angles to each other with identical constructs using DCPs, LC-DCPs, and the clamp-rod internal fixators (CRIFs) in four-point bending showed that implanting two locking screws on either side of an oblique saw-cut across the artificial bone composite (Canevasit) provided significantly increased stiffness to the construct.[86] Because the strength of a screw depends mainly on the core diameter and not thread width, the thicker core of the locking screws and the thin threads make the screw several times stronger than the conventional cortex screws.[79]

The LCP uses a combi-hole, which is a combination of a DCU and a LISS hole (see Figure 76-20). The surgeon may select the type of screw to be inserted at any given place—either a 4.5-mm or a 5.5-mm cortex screw (or even a cancellous screw) at an axial angle up to 40 degrees, or a 4.0-mm or 5.0-mm locking head screw with a thick core and thin threads and

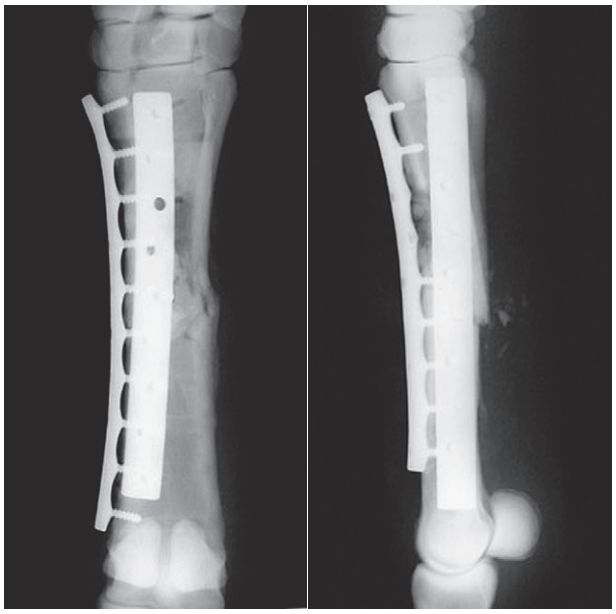


Figure 76-34. Two PC-Fix plates are applied at right angles to each other in a comminuted and open third metacarpal fracture in a horse. Note the different configurations of the plates and the unicortical screws. With time, the empty spaces under the plate filled with bone.

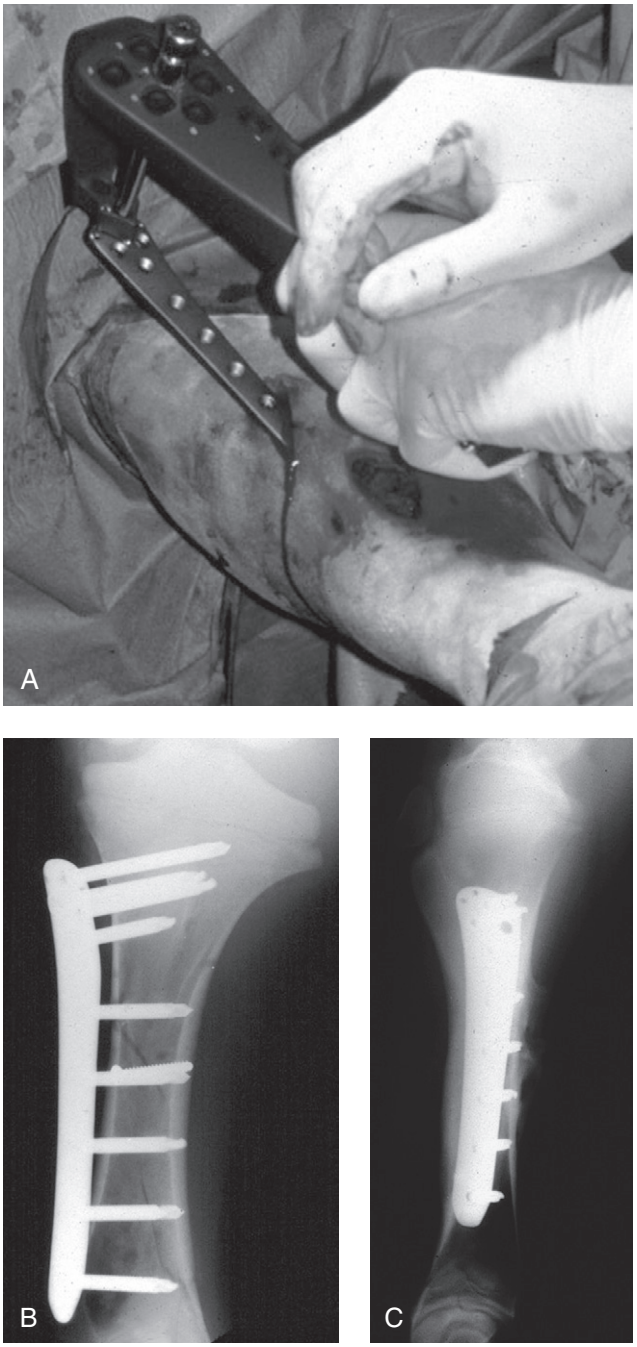


Figure 76-35. A, A less-invasive stabilization system (LISS) is inserted through a small proximal incision to repair a tibial fracture. **B** and **C,** Postoperative radiographs show the plate attached to the bone, but only the screws make contact with the bone. The fracture healed without problems.

additional threads at the screw head (Figure 76-36). The locking head screw, however, has to be inserted orthogonal to the long side of the plate. The original plate contained two beveled ends to allow insertion through a small incision and sliding of the plate along the periosteum of the fractured bone. Because the locking part of the combi-hole is positioned toward the center, insertion of a locking screw next to an articular surface left the bevelled tip extended over the joint. This led the veterinary specialists to modify the plate. Together with Synthes, the plate was redesigned with a pointed, bevelled tip at one end and a rounded edge at the other end. The plate has a slightly oval stacked hole, allowing insertion of either a locking head screw (the hole contains threads) or a cortex screw, the latter of which could be slightly angled away from the joint (Figure 76-37). A special tissue spreader has been developed to prepare the future plate bed. The thin beveled end separates the soft tissues from the periosteum. After the plate is contoured with the help of the intact contralateral bone, it can be fixed to the bone via a minimally invasive procedure. It is inserted through a small incision at one end of the bone, and the screws are then placed transcutaneously through stab incisions.

By substituting cortex screws through some holes, costs can be significantly decreased without jeopardizing the stability and stiffness of the construct. Without application of the push-pull device (Figure 76-38) or standard cortex screws, both of which press the plate onto the surface of the bone, a gap of 2 mm will be present between the plate and the bone after its application. Therefore a basic decision has to be made at the onset of any LCP application regarding whether the plate has to be in close contact with the bone or not. In horses, it is desirable to have close contact between the bone and the plate to increase friction, which further stabilizes the construct.

Once the fracture is anatomically reduced and stabilized by cortex screws placed in lag fashion, the first plate is positioned with the plate holder, and the push-pull device is inserted at a slight angle through the DCU portion of the combi-hole (Figure 76-39, *A*). By turning the piston clockwise, the plate can be pressed onto the bone surface. At the same time, it temporarily fixes the plate to the bone. A second such device can also be applied through the stacked combi-hole on the other end of the plate, if desired. Next, all the strategic cortex screws are inserted and tightened to facilitate axial compression (if deemed necessary) and solid bone–plate contact at both ends of the bone

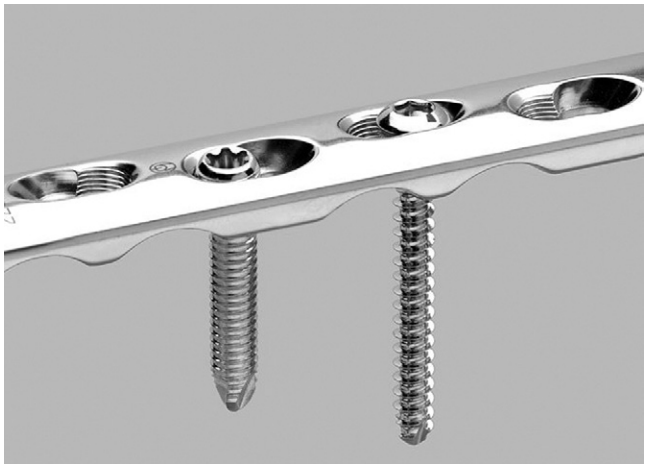


Figure 76-36. A narrow LCP shown with a 4.5-mm cortex screw and hexagonal drive *(right)* as well as a 5.0-mm self-tapping locking screw with a stardrive *(left)*. (Courtesy Synthes Vet, West Chester, PA.)

Figure 76-37. A 10-hole veterinary LCP. The plate has one rounded end containing a stacked combi-hole through which either a locking head or a cortex screw can be inserted. The other end has a tapered and pointed tip that facilitates minimally invasive plate insertion. Note that the DCU parts of the combi-holes are arranged on both sides of the center of the plate toward the ends, whereas the threaded parts of the combi-holes point toward the center. (Courtesy Synthes Vet, West Chester, PA.)

(see Figure 76-39, *B*). After removal of the push-pull device and the plate holder, the second plate is applied at a 90-degree angle to the long axis of the first plate, using the same technique (see Figure 76-39, *C*). Next the locations where the locking screws will be implanted are selected to avoid contact with interfragmentary screws and the cortex screws of the other plate. Planning how screws are to be inserted is very important, even more so when LCPs are applied, because the locking screws *must* be inserted perpendicular to the plate. Screw position is different if a locking screw or a cortex screw is used through a combi-hole, which represents additional complexity when planning the surgery. Further complications arise if an original DCS containing mainly DCP holes and an LCP are combined, because the hole lengths and their spacing along the two plates differs. As mentioned before, the DCS is now available with combi-holes. Once locking screws are applied, the plate is solidly fixed in its position.

The LCP drill guide is carefully twisted into the threaded part of the combi-hole at a right angle to the long axis of the plate. To facilitate perpendicular insertion and subsequent solid engagement of the threads in the plate hole, the drill guide is placed onto the combi-hole and then twisted backward until a click is heard, which happens when the drill guide slips from the upper thread onto the one just below it. Then the drill guide is twisted forward to engage the threads of the combi-hole. When the drill guide is solidly seated, its position relative to the plate is evaluated once again, ensuring its perpendicular orientation. All the LCP drill guides provided in the set can be fixed

Figure 76-38. The push-pull device, which presses the LCP into close contact with the bone, resulting in increased stability of the construct. (Courtesy Synthes Vet, West Chester, PA.)

to the plates to speed up the procedure. Once their position perpendicular to the long axis of the plate and parallel to each other is confirmed, all the holes are drilled (see Figure 76-39, *D*). The drill guides are removed, and the screw lengths are determined using the depth gauge. The 4-N torque-limiting device is attached to the power drill followed by the insertion of the power attachment of the stardrive. By pressing the screwdriver into the stardrive indentation of the LCP screw in the rack, the correct screw is selected and inserted into the predrilled hole using a power-tapping technique. The screw is fully inserted until the torque-limiting device is idling in the plate hole, meaning that the 4-N insertion force has been reached. This precautionary step was initially introduced in human surgery to prevent cold welding between the titanium plate and screws. Because stainless steel is predominantly used in equine surgery, the danger of cold welding is circumvented. Nevertheless the use of the torque-limiting device is encouraged. Equine bone is hard, and if long screws are inserted, the 4 N threshold may be reached before the screw head threads are completely engaged in the plate. It is therefore prudent and good technique to complete final tightening with the hand screwdriver (see Figure 76-39, *E*).

Once all locking screws in the first plate are implanted, the same procedure is repeated in the second plate (see Figure 76-39, *F*). Any empty plate holes in both plates are filled by applying a cortex screw through the DCU portion of the combi-hole at the angle necessary to prevent contact with screws in the other plate.

Because the threads of the screw head and the threads in the plate are solidly intertwined, they form a unit. This prevents the screw head from moving within the plate hole if certain strains are applied, which significantly increases the stiffness of the construct. All the locking screws applied to a plate feel very solid, because the threads in the screw head engage the corresponding threads in the plate. This does **not** mean that the screw is solidly inserted in the bone underneath the plate. This is a new experience for the surgeon and must always be kept in mind. Under certain circumstances, it may be difficult to twist the drill guide perpendicularly into the threaded portion of the combi-hole, because major muscle bellies may be in the way. In these occasions, separate stab incisions through the muscle bellies are necessary to access the combi-hole at a right angle. The drill guides can be extended by threading one on top of the other, and in doing so, attain correct engagement in the plate and correct position for drilling.

The LCP has, in a short period of time, established itself as the preferred plate of equine fracture fixation despite its higher costs, mainly caused by the screws.[87] A recent study comparing 4.5-mm LCPs with 4.5-mm LC-DCPs confirmed the superior strength and stiffness of the LCP.[88]

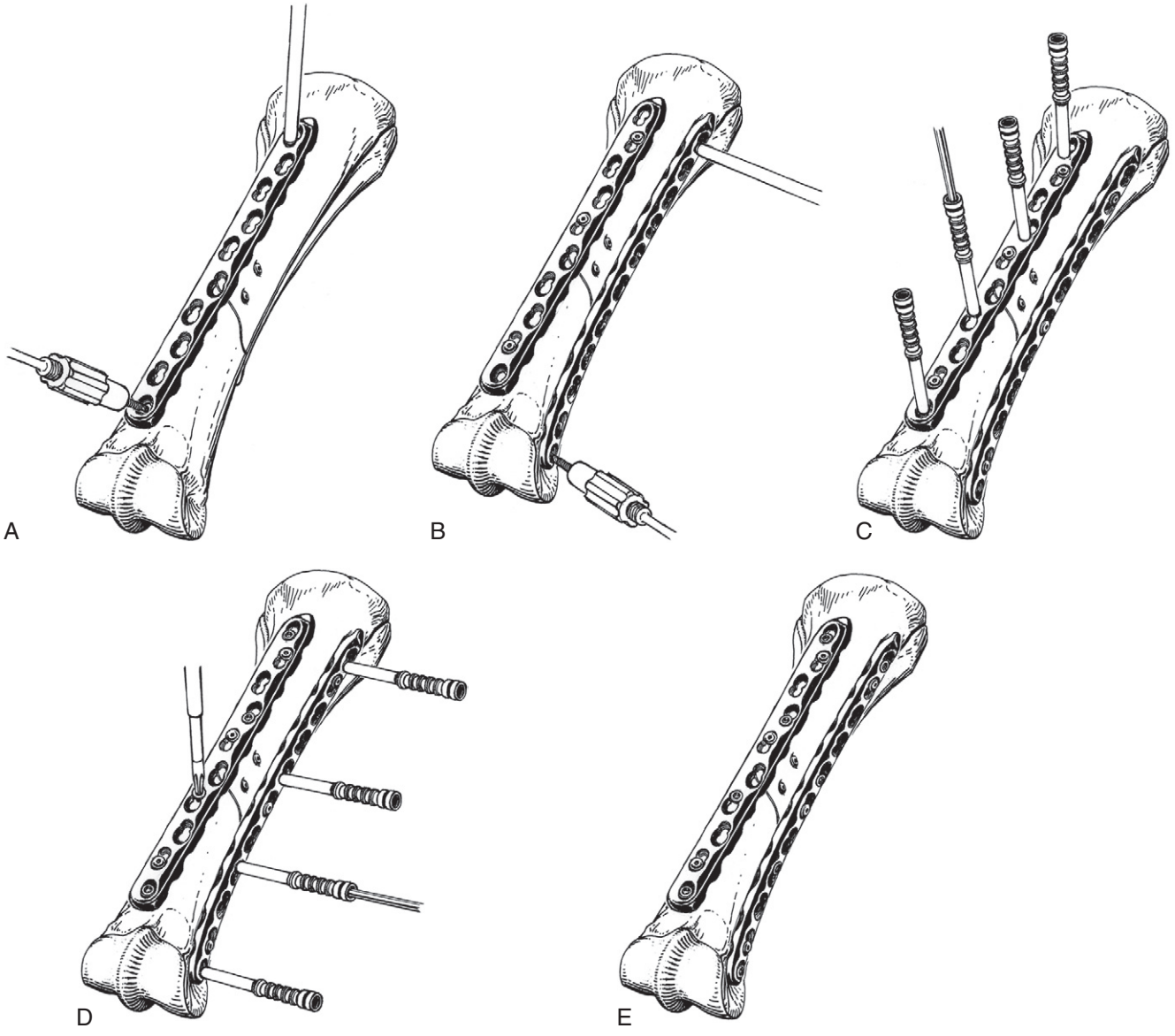


Figure 76-39. Application of two LCPs to an oblique midshaft third metacarpal fracture. **A,** The fracture is reduced and stabilized by the two 3.5-mm cortex screws applied in lag fashion. A 10-hole broad veterinary LCP is applied to the bone with the plate holder and temporarily fixed in place with the push-pull device. By turning the piston clockwise *(arrow),* the plate is pressed onto the bone surface. **B,** To facilitate good plate–bone contact along the entire plate, cortex screws are implanted and tightened using plate screw technique at both ends and in the center near the fracture. Once in place, the lateral 11-hole narrow veterinary LCP is applied to the bone using the same technique. Note that the plate can be applied farther distad on the lateral aspect of the bone than on the dorsal aspect. **C,** Next the holes where locking screws are to be inserted are selected and the drill guide is twisted into the threaded portion of the combi-hole. Because the plate is solidly fixed to the bone, all four drill guides provided in the set are applied, followed by drilling all four holes. **D,** The locking head screws are inserted and tightened. The four drill sleeves for the locking head screws are then placed into selected plate holes, making sure that screws can be placed perpendicularly without interfering with previously inserted implants. **E,** All the remaining plate holes are filled with cortex screws inserted using the plate screw technique. Where indicated, lag technique is applied to increase interfragmentary compression.

Because of the rapidly increasing popularity of locking plates, a 5.5-mm LCP especially designed for equine fracture repair has been developed. Again, this plate was tested in an *in vitro* study against the 4.5-mm LCP.[89] The 5.5-mm LCP was superior in resisting static overload in palmarodorsal four-point bending and cyclic fatigue testing. These results were superior to those achieved with the 5.5-mm LC-DCP. Taken together, these findings have established the 5.5-mm LCP as the premier equine plate for long bone fracture fixation and arthrodesis of the metacarpo- or metatarsophalangeal joint.

The human femoral LCP is ideal for lateral application to the equine radius. This bone has a slight craniocaudal curvature when viewd from the side. It is therefore not possible to apply a straight plate to its lateral aspect and span the entire bone. Either the middle holes are behind the bone or the proximal holes are in front of the bone. The human femoral LCP has a slight bend that matches the equine radius perfectly. The ideal combination is a 5.5-mm equine LCP applied cranially and a human femoral LCP applied laterally (Figure 76-40). The implants are available in stainless steel in all sizes (see Table 76-2).

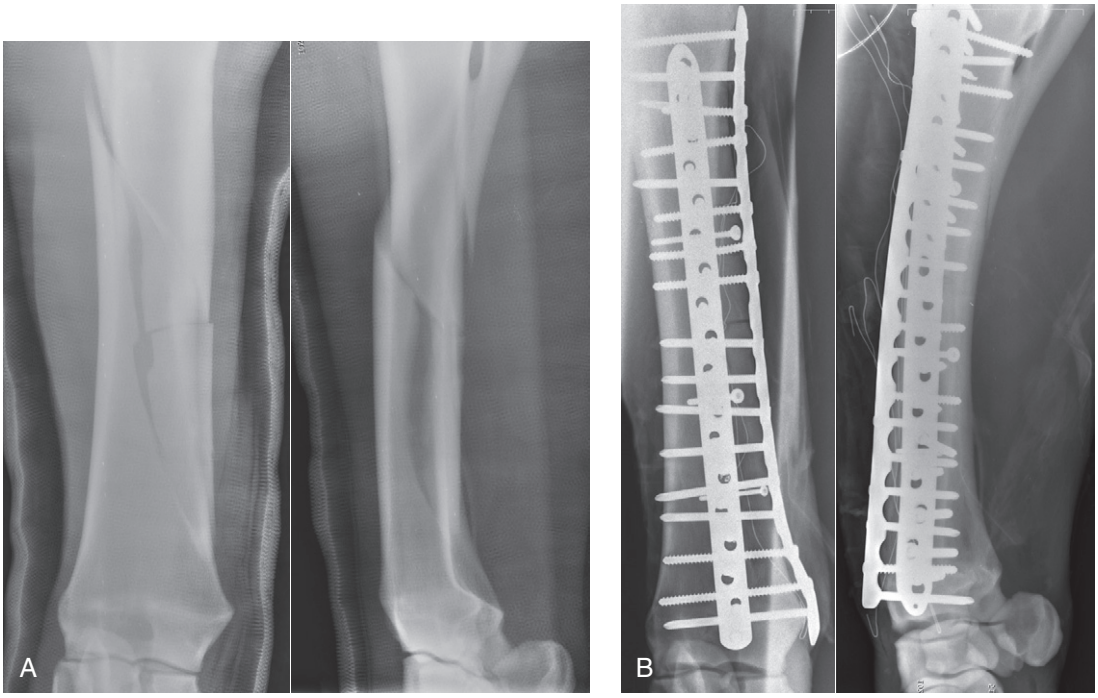


Figure 76-40. Preoperative **(A)** and postoperative **(B)** radiographs of an oblique, spiral radial fracture. The fracture was repaired with a 17-hole 5.5 mm veterinary LCP cranially and a 17-hole 4.5-mm human femoral LCP (a slightly axially bent plate) laterally.

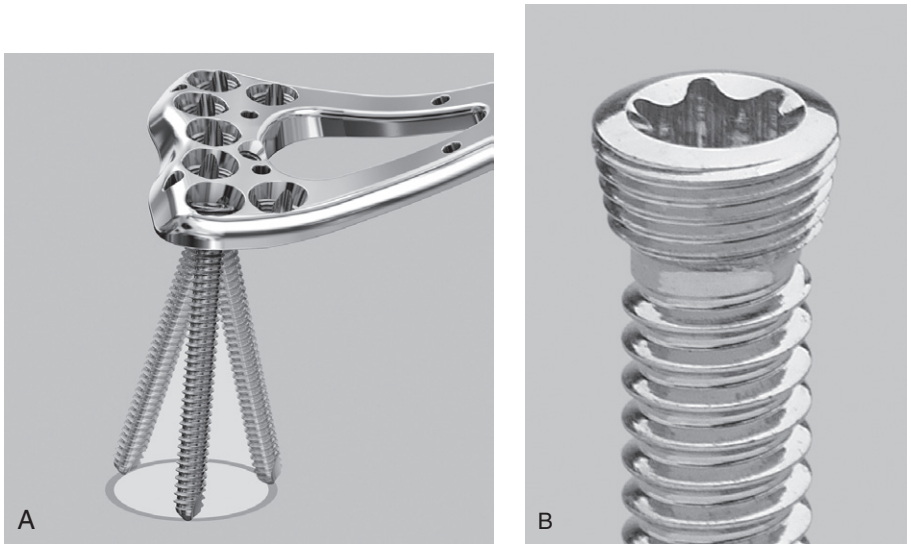


Figure 76-41. A, The top portion of a variable angle LCP (VA-LCP) demonstrating the angles a screw can be inserted through the holes. **B,** Close-up side view of the screw head, depicting its threads on the rounded head and the stardrive design for the screw driver. (© by Synthes Inc, West Chester, PA.)

Because of the disadvantages posed by the need for locking head screws to be inserted perpendicular to the long axis of the plate, attempts were made to develop a locking mechanism that allows the insertion of locking head screws at various angles. Thus the variable angle LCP (VA-LCP) was created. Screws can be angled anywhere within a 30-degree cone around the central axis of the plate hole (Figure 76-41, *A*). The plate hole has a cloverleaf shape (see Figure 76-41, *B*) containing four threaded ridges, where the specially constructed screw head can interlock with the plate. The head of the variable angle locking screw is rounded to facilitate various angles within the locking hole (see Figure 76-41, *C*). A special double drill guide has been developed that facilitates drilling of the variable angle screw holes on one side and fixed angle drilling on the other (Figure 76-42). The nozzle of the drill guide inserts coaxially into the central hole. In contrast to standard drill guides, which support the drill bit along its entire length, the funnel shape of the angle screw hole guide allows the drill bit to glide along the guide wall at a 30-degree angle in any direction selected (Figure 76-43). The first application of these holes was implemented in the 2.4-mm Variable Angle LCP Distal Radius System (Synthes Inc.) for humans. The plate is anatomically contoured to the volar aspect of the distal radius and is designated to address both simple and complicated fractures. If this plate hole design

Figure 76-42. Double drill guide: left for fixed-angle drilling, right for off-axis drilling. (© by Synthes Inc, West Chester, PA.)

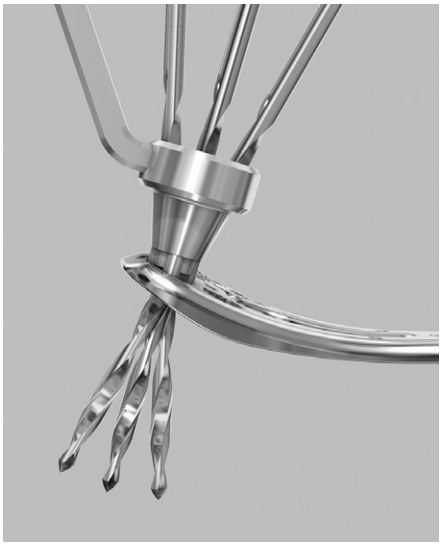


Figure 76-43. The off-axis drill guide is coaxially inserted into a plate hole. With the drill bit leaning onto the side-wall of the drill guide, total angles of 30 degrees can be achieved through the same plate hole in all directions. (© by Synthes Inc, West Chester, PA.)

is considered an improvement in human surgery, it will hopefully be applied to other locking plates.

The LCP is the ideal plate for the veterinary surgeon. It can be applied in the same manner as a DCP with only cortex screws. Because the plate possesses the same type of undercuts as the LC-DCP, it can be used as such. In other words, the LCP fulfills all the desired demands and by purchasing only LCPs the veterinarian can reduce the armamentarium considerably. The price of the LCP is slightly higher than the DCP and LC-DCP and therefore costs are not a problem. So all the veterinary surgeon needs to decide is whether to apply cortex screws, locking head screws, or a combination thereof.

Addendum

Thorough knowledge of the basic principles of internal fixation and familiarity with the instruments and numerous implants are prerequisites for successful surgery. Everyone interested in treating long bone fractures should complete a basic and advanced AOVET course on internal fixation.

CERCLAGE WIRE

Cerclage wire is used frequently in humans and small animals.[90,91] Tension band fixations with pins and wire are often carried out in dogs and cats. Cerclage wires are also applied around oblique long bone fractures in small animals. Also, this type of fixation has been used with small Steinmann pins to temporarily stabilize comminuted long bone fractures before plate and lag screw application.[3] Application of cerclage wire in equine long bone factures has not been successful because of a lack of stability and breakdown of the fixation. However, cerclage wires may be applied in a few situations. Proximal sesamoid fractures have been successfully treated with cerclage wires,[92] but this technique was recently abandoned because of unsatisfactory long-term results.

One frequent application of cerclage wire is growth retardation surgery, even though the wire is not applied in cerclage fashion (see Chapter 86). Cerclage wires have been used to treat nondisplaced ulnar fractures in foals.[93,94] The wires are passed through holes placed in the frontal plane across the proximal and distal fragments of the ulna. A small loop created before entering the hole in the distal fragment allows even tightening on both sides of the bone after the wire is applied in figure-of-eight fashion, and the ends are twisted together. This type of fixation results in reduced trauma to the ulna and radius, and it prevents inadvertent fixation of the ulna to the radius, which can occur when using a plate in a young foal. (This may induce subluxation of the cubital joint.) A total of three or four figure-of-eight wires are applied.

Cerclage wire is used in arthrodesis of the fetlock joint.[95] In comminuted fractures of the proximal sesamoid bones associated with complete breakdown of the suspensory apparatus, a tension band is inserted in figure-of-eight fashion through the palmar/plantar aspect of the metacarpophalangeal joint, before applying the dorsal plate (see Chapter 81).[95]

One frequent application of cerclage wires is their use as a tension band to manage mandibular and maxillar fractures (see Chapter 102).

Wire tightening is carried out with utmost prudence. Initially, the wires are loosely twisted by hand. Then, with a pair of flat pliers, the two ends are grabbed, pulled at a right angle away from the bone, and, while decreasing the pulling force, evenly twisted around each other. Care must be taken not to twist one wire end around the straight end of the other wire (this type of fixation slips off under tension). Overtightening may result in wire breakage and breakdown of the fixation.

CABLES

Two types of cables have been introduced into orthopedic surgery as treatment modality for specific fractures. Ultrahigh-molecular-weight polyethylene (UHMWPE) cable has been tested in an *in vitro* model for the repair of proximal sesamoid bone fractures and compared with the commonly used stainless steel cerclage wire (SSCW).[96] The ultimate tensile strength of UHMWPE cable constructs was 34% greater than that of SSCW constructs. Fatigue strength was 2 to 20 times greater for UHMWPE cable constructs than for SSCW constructs. Separation of fragments was 153% less for limbs repaired by the cable construct compared with those repaired by the transfixed cerclage technique.[96] These cables may also be beneficial in the use of fetlock breakdown injuries as a palmar figure-of-eight tension band.

Another type of cable consists of multiple woven stainless steel, titanium alloy, or cobalt chromium alloy strands. It is available as 1.0- and 1.7-mm-diameter cables, consisting of a central bundle of 19 strands surrounded by eight outer bundles of seven strands each. It is designed to be used with all Synthes stainless steel and titanium plates. Specially designed positioning pins are used in empty plate holes, and the cable is threaded through an oblong hole in the pin. Once the cable is tightened,

the pin cannot move because it is pressed into the plate hole and therefore confined. A special tensioning device is used to tighten the cable before it is crimped with a cable crimper. Care should be taken to not exceed 50 kg of tension. Applying tension at levels higher than 50 kg may cause the cable to cut through soft or osteopenic bone (which is not a problem in horses). This product is used mainly in human orthopedic surgery in the management of periprosthetic fractures in elderly adults, where other internal fixation devices are not successful.[97] Additionally, it is used as a tension band in the management of patellar fractures and olecranon osteotomies. Indications for these cables in horses are similar to those for the UHMWPE cables. Recently these implants were adopted for mandibular fractures (see Chapter 102).

PINS

Steinmann pins

Steinmann pins are rarely used in fracture treatment in horses, mainly because they do not provide stability.[97] They are used as transfixation pins in conjunction with external coaptation (see earlier discussion).

Intramedullary application of Steinmann pins is used only in humeral fractures in foals.[98] Multiple pins are introduced parallel to each other to fill the entire medullary space at its isthmus at the distal third of the bone. This "stacked-pin" method is presently the treatment of choice in these fractures. For additional stability, these pins may be encircled by cerclage wires and placed into the medullary cavity, possibly through a cortical defect or a drill hole. The wires are subsequently tightened. In cases with a cortical defect, the cerclage may also be applied intramedullarly. The advantage of the stacked-pin method is an increase in rotational and bending stability. A single pin provides no rotational stability. Collapse of the fracture along the single pin is a frequent complication.

Application of multiple pins across a capital femoral physeal fracture has been advocated and has met with some success. Steinmann pins have also been used in the treatment of olecranon fractures in the very young foal in combination with a tension band made of multiple cerclage wires.[96]

Rush pins

The Rush pin method of fracture treatment was popular before bone plating was introduced. Fracture fixation using these devices is an art.[99] The slightly prebent pins are introduced obliquely into the distal fragment and advanced toward the opposite cortex. The tip, which is flattened on one side, slides off the opposite cortex and is redirected toward the cis-cortex. The pin length has to allow the tips to engage in the cis-cortex both proximally and distally to the fracture, providing four-point contact. Usually two pins are introduced, one from each side of the bone. When performed correctly, rotational stability is achieved with a minimum of implants and surgical trauma. The Rush pin fixation technique is not applicable to comminuted or open fractures.

NAILS

Intramedullary nails have a place in equine long bone fracture repair, but the ideal implant has not been developed despite recent efforts.[10,43] Initially, intramedullary nails manufactured for human application were tried in equine fractures with mixed success.[100] For example, in two reports, a solid titanium nail was inserted through the middle carpal joint after removing the articular cartilage of the middle carpal and carpometacarpal joints.[101,102] The joints were fused to facilitate solid fixation. Transfixation was achieved with 4.5-mm screws inserted through the proximal aspect of the nail. Although good results were achieved, the fact that the joints had to be fused to facilitate healing of MCIII was undesirable. These fractures can heal with plating techniques without fusing a joint.

A system of intramedullary interlocking nails (IINs) has been developed for equine humeral and femoral fractures (Figure 76-44).[103-106] Comparison of this method of fracture repair to fracture plating has met with mixed results in experimental studies. A cadaveric *in vitro* biomechanical study on immature equine femurs revealed that a diaphyseal osteotomy fixed with two DCPs at 90 degrees to each other provided superior strength and stiffness compared with an IIN and a construct of an IIN and cranially applied DCP.[104] In another *in vitro* study on osteotomized tibias, a construct composed of a 16-mm stainless steel nail with a wall thickness of 4 mm and four 8-mm interlocking screws was compared with a human unreamed femoral interlocking nail (UFN) and to tibias treated by means of double plating.[107] The interlocking nails achieved similar loads until failure, but the plates demonstrated higher yield loads. *In vitro* cadaveric studies tested several interlocking nail configurations in MCIII and femurs in foals and in MCIII with and without a gap in adult horses.[108-110] All constructs were weaker than the intact bone, and the parallel alignment of the holes for the interlocking screws were stronger than the offset screws.[109] One study compared IIN with a combination of an IIN and a DCP and with two DCPs in a 1-cm gap model in foals, showing the double-plating construct to be closest to the intact bone in most aspects, followed by the combination and the IIN alone.[107]

Several application principles have been advocated in foals for the presently used equine IIN.[111] The location and configuration of the fracture significantly affects the ultimate outcome of the repair. If possible, three interlocking screws should be inserted on either side of the fracture. Fractures near the epiphysis are less readily stabilized with an IIN, and the epiphyseal segment is at an increased risk for secondary fracture through the interlocking screw holes. In these instances, some type of supplemental fixation is desirable to decrease the potential of catastrophic failure of the fixation. In long oblique fractures, the IIN should be positioned to allow one or two interlocking screws placed in lag fashion across the fracture plane, if possible. In a nail–plate construct, the plate is applied 90 degrees to the interlocking screws. Whenever possible, bicortical screws are inserted through the plate.

A lateral approach is used to expose the fractured humerus and femur. Before reduction, the fracture is débrided and the medullary cavity of the distal fragment is reamed. Reaming is accomplished with rigid reamers of increasing size to arrive at an ultimate hole diameter of 13 mm. This procedure destroys the intramedullary blood supply and slows healing. Unreamed intramedullary interlocking nails have not been successfully applied in living horses. Additional exposure is usually necessary to provide access to the proximal end of the bone for normograde reaming of the proximal segment. After both fragments are reamed, the fracture is reduced and held in that position with bone clamps. A nail of appropriate length is chosen, and with the targeting jig attached, the nail is inserted into the reamed medullary canal. Washers are used to prevent the conical heads of the cortex screws from penetrating the cortical bone.

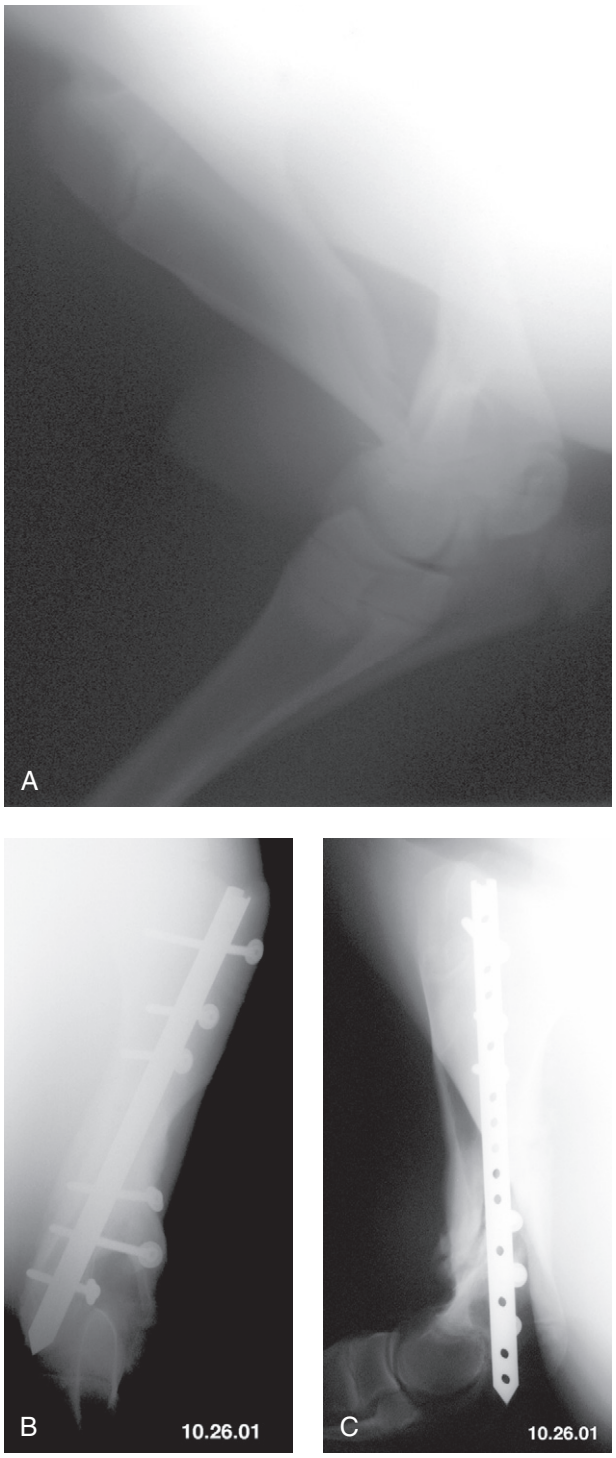


Figure 76-44. This long oblique humeral fracture **(A)** was treated with an intramedullary interlocking nail. To provide a greater screw–bone contact area, washers were used. The craniocaudal **(B)** and lateromedial **(C)** 2-month follow-up radiographs show progressive bone healing in the fracture gap. Bone length is maintained. (Courtesy J. P. Watkins, Texas A&M University.)

A successful result was achieved with an IIN in 5 of 10 foals with humerus fractures; the foals ranged in weight from 136 to 204 kg. They attained athletic soundness and performed their intended purpose without complications. Three out of three neonates with femoral fractures healed after IIN fixation. Four out of six older foals with short oblique femur fractures also were successfully treated in this manner. In these foals, a DCP was applied to the cranial aspect of the bone in addition to an IIN.

AFTERCARE

After fracture fixation with any of the internal fixation devices and techniques described here, overlying soft tissues and skin are closed in routine fashion. The use of of continuous suture patterns is advocated to reduce surgery time. Depending on the type of fixation and the technique of recovery from anesthesia, application of some type of external coaptation may be considered, because the animal has to be able to get up and place weight on the limb immediately after surgery. If a pool recovery is implemented, the limb and skin incision is in most cases protected only by a plastic adhesive sheet (Ioban 2) after applying cyanoacrylate superglue to the skin incision. This sheet is covered with elastic adhesive tape (Elasticon), which is exchanged for a regular bandage or splint bandage after successful recovery from anesthesia (see Chapter 21).

External coaptation may consist of a fiberglass cast or a heavy splint bandage (see Chapter 17). Depending on the type of fixation, external coaptation is maintained for a few days to weeks. A fiberglass cast can be applied over a bandaged limb, and after the cast has cured, it can be split in half along the frontal plane. These "half shells" are reapplied using nonelastic adhesive tape. Such coaptation allows evaluation and, if necessary, wound management of the limb underneath. The unaffected contralateral limb should be protected and supported by a pressure bandage. In young foals, too much support is to be avoided to prevent temporary weakness of the flexor tendons. It is important to keep the patient comfortable with the help of moderate amounts of anti-inflammatory and analgesic drugs. These drugs should be used judiciously to prevent toxic reactions and to allow some residual amount of pain, so the patient will protect the injured limb. If a non–weight-bearing lameness lasts for a prolonged period, other problems develop in the healthy limbs, especially in foals. Therefore close observation of the patient is important until the animal starts to increase weight-bearing on the fractured limb.

Application of a frog pad in adult horses may prevent the development of laminitis in the contralateral foot. Aside from postoperative infection (see Chapter 85), laminitis is the major complication that occurs after fracture repair. Again, administration of anti-inflammatory drugs in moderate amounts may prevent this problem. As early as possible, the animals should be allowed to walk. Although controlled exercise is advocated, free pasture exercise is discouraged. This is especially important if a weight-bearing fixation was performed and the animal is not placed in an external coaptation device. Patients with casts should not be exercised at all.

Implant Removal

Implants are foreign bodies and may have to be removed, particularly in young athletes. Therefore, implant removal depends on the use of the horse, the type of fracture treated, the type of implants employed, and the potential development of complications, including postoperative infection. Lag screws are not removed in horses unless they produce pain or bone reactions. The practice of removing lag screws in young racehorses has gained popularity. Frequently the reason for implant removal is

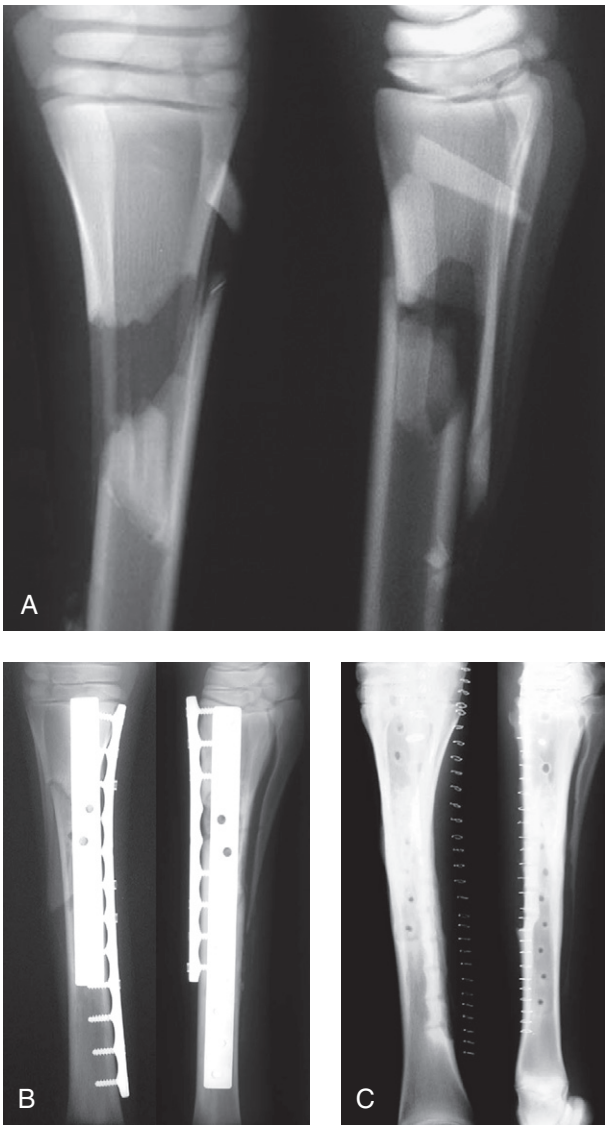


Figure 76-45. A, A 2-month-old Thoroughbred colt was admitted with a comminuted fracture of the third metatarsal. **B,** The fracture was treated with two PC-Fix plates applied over the periosteum, which prevented adequate visualization and resulted in suboptimal fracture reduction. An infection developed, which was managed with parenteral broad-spectrum antibiotics, daily flushing of the surgical site, and local antibiotic application. Within a month, the infection had resolved. Bacterial cultures taken at the time of implant removal revealed no growth. **C,** The healed fracture after implant removal. The foal developed into a successful racehorse.

a request by the owner. Cerclage wire used for fracture fixation does not need to be removed unless the wire breaks, as it does frequently in the treatment of transverse fractures of the proximal sesamoid bones.[92]

Plates applied to long bones of horses should be removed in most cases. This is especially important in foals and if the animal enters or resumes an athletic career. Implants applied to the femur or olecranon and for arthrodeses purposes are left in place. This presupposes that no problems are encountered with the implants.

The time to remove implants after the fracture has healed depends on the age of the animal, the type of fracture treated, and the implants used. In a condylar or stress fracture of MCIII/MTIII, screws may be removed about 2 months postoperatively. Plate removal in foals may be carried out at an average of 4 to 6 months after fracture treatment. Staggered removal of the implants is advocated if two plates were applied, because it reduces the risk of refracturing the bone through one of the screw holes. (Filling the empty screw holes in the bone with a bone graft has been recommended in humans.[79])

An important reason for implant removal is infection surrounding the implants. An infection, once established around implants, persists and does not resolve until after the implants have been removed, even in the presence of broad-spectrum antimicrobials.[38] Fractures can heal in the presence of infection if rigid internal fixation is maintained. However, it is frequently a race between loosening of the implants caused by the infection, and healing of the fracture. Once an infected fracture is healed, the implants are removed to allow resolution of the infection. If the implants are removed too early, before adequate healing of the fracture has occurred, refracture of the bone is likely. In one case, however, where titanium implants were applied to a comminuted open fracture of the proximal MTIII in a Thoroughbred foal, a postoperative infection resolved completely before implant removal (Figure 76-45).

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CHAPTER 77

Bone Grafts and Bone Replacements

Jörg A. Auer, Brigitte von Rechenberg, Marc Bohner, and Margarethe Hofmann-Amtenbrink

Biological bone grafts are distinguished from synthetic bone replacements because they come from different sources and require a variety of pretreatments.[1,2] However, the mechanism by which both are integrated into host bone is similar. All grafts are eventually replaced with host tissue by a process called *creeping substitution*.[1,3] Mesenchymal stem cells or osteoprogenitor cells and capillary sprouts from either adjacent bone marrow, periosteum, endosteum, or surrounding soft tissues grow into the porous structure of the grafts. There, stimulated by various local substances, they differentiate into osteoblasts, producing new mineralized osteoid that is deposited either on the bone matrix or on the synthetic ceramic structure. Osteoclasts

precursors are recruited from the vascular system, and they differentiate to facilitate physiologic remodeling of the graft into mature lamellar bone.[1,3,4] Pretreatment of allogeneic or xenogeneic bone grafts is necessary to reduce antigenicity that could lead to graft rejection.[1]

TYPES AND DEFINITIONS

A distinction is made between *fresh biological bone grafts* and preserved ones. A bone graft may be transferred immediately from the donor to the implant site, in which case it is a fresh graft. *Preserved grafts* include specially prepared pieces of dead bone—for example, decalcified, freeze-dried, irradiated, or sterilized bone. In a third category are *synthetic bone replacements*, which consist of ceramics such as tricalcium phosphate (TCP) or hydroxyapatite (HA), but also polymers and composites.[5]

Bone grafts can be classified according to their origin. A graft harvested from one site and applied to another in the same individual is an *autograft*. An *allograft* refers to tissue removed from one individual and given to a genetically different individual in the same species. Tissue transferred between two members of different species is a *xenograft*. If the transferred tissues are applied in an anatomically similar location, the graft is an *orthotopic* graft; if the implantation site is dissimilar, the graft is referred to as *heterotopic*. Bone grafts may be composed of purely cancellous or cortical bone, or they may be composed of a combination—for example, a corticocancellous graft or, in combination with articular cartilage, an osteochondral graft. In equine orthopedic surgery, fresh bone grafts are always autografts. The most frequently used fresh grafts are autogenous cancellous bone grafts, but cortical chips taken from the fracture site, free cortical grafts, and vascularized cortical bone grafts may be used. Free cortical grafts depend on the ingrowth of host vessels for their nutrition, whereas vascularized cortical bone grafts are harvested with their blood vessels to be anastomosed to the local blood supply.

Preserved grafts are pretreated with either freezing alone, freeze-drying, radiation, sterilization, or complete or surface decalcification.[1,6] The process of pretreatment kills viable bone cells, reducing antigenicity considerably, and allows safe and sterile storage of the bone grafts. These grafts are kept in bone banks, which makes them easily available on request.[6,7] Their ease of accessibility prevents the need for a second surgery to harvest the autografts and thus decreases surgery time.

Bone grafts are used most often to facilitate healing after long bone fracture repair, arthrodesis, and comminuted phalangeal fractures in the horse, and autogenous cancellous bone grafts are the most frequently applied type.[8] Therefore the major emphasis in this chapter is on this technique. Bone banking is discussed for the sake of completeness. In humans and small animals, a variety of bone grafts are used, most of which are not readily applicable to the horse.

BONE GRAFTS

Jörg A. Auer, Brigitte von Rechenberg

Bone is the second most commonly implanted material in the human body, after blood transfusion, with an estimated 600,000 grafts performed annually. Although the market for synthetic bone replacements (bone graft substitutes) is more than $1 billion, the sales of bone grafts is still more than half that amount.[9] Reports of autologous bone grafting date back to the ancient Egyptians, yet the modern scientific study of grafting began in the early nineteenth century. Since then, the indications, methodology, and science of bone grafts in patients with nonunion fractures and bone loss have been established and refined, and new methods of harvesting and treatment are being developed and implemented.[9]

Functions

Depending on the type of bone grafts, their main function is osteoinduction or osteoconduction.[1] *Osteoinduction* refers to the local triggering of osteogenesis (new bone formation), whereas in *osteoconduction*, the matrix of the graft acts as a scaffold into which mesenchymal cells grow.[6] In fresh autogenous grafts, osteogenesis occurs partially as the result of the activity of viable osteoblasts aligned on the surface of the living bone graft. Therefore the greater the surface is, the more living cells are available and the greater is the osteogenic activity that is observed. However, up to 90% of living cells die after bone graft transfer, with only the surface cells surviving. Therefore the greater the number of surface cells transferred, the larger is the number of osteogenic cells that survive in the graft bed. As a result of this, loosely arranged cancellous bone grafts are the most desirable in the horse. The number of surviving cells is also influenced by the handling of a graft during surgery, where dehydration and compacting of the graft are to be avoided.[6]

Some systemic influences and local factors, such as local and systemic cytokines, inflammatory mediators, and growth factors, are very important for the mechanism of osteoinduction.[10-12] Cytokines are polypeptide molecules capable of signal transduction in cells through specific surface cell receptors. Inflammatory mediators elicit changes in cell metabolism through activating either cyclic guanosine monophosphate (cGMP) or cyclic adenosine monophosphate (cAMP).[11,13] Cytokines, such as interleukins (ILs) and tumor necrosis factors (TNFs), and inflammatory mediators, such as nitric oxide and prostaglandin E_2, may be produced either by local mesenchymal, fibrous, or vascular tissue cells. On the one hand, these signals attract and recruit undifferentiated mesenchymal cells from the environment to the local area, which differentiate into osteoblasts; on the other hand, they also induce bone biodegradation, facilitating bone graft replacement (creeping substitution). Through the locally invading cells and ongoing bone matrix biodegradation, a myriad of local growth factors are released that allow the mesenchymal cells to proliferate and differentiate. Through various cell generations, the graft undergoes enchondral ossification into woven bone. This bone is replaced later with mature lamellar bone. Growth factors (GFs) involved in fracture healing and graft incorporation are platelet-derived GF (PDGF), fibroblast GF (FGF), insulin-like GF (IGF), transforming GF (TGF), and the bone morphogenetic proteins (BMPs), including osteogenic proteins (OPs).[10] The BMPs and OPs belong to the TGF-β superfamily.[12] Various BMPs are now produced synthetically by recombinant processes with molecular biology technologies and are used to promote graft incorporation.

Research has detected differences in activity of bone marrow cells of different bones and between cortical and cancellous bone.[14] *In vitro* explant cultures of cortical and cancellous osteogenic cells revealed that on average, cortical bone sites yielded significantly greater activity than did cancellous bone sites. Between cortical bone sites, there was no significant difference in osseous activity. Among cancellous sites, the radial cancellous

bone yielded significantly more activity than did the tibial cancellous bone. Among appendicular skeletal sites, tibial metaphyseal bone yielded significantly less activity than did all other long bone sites. This study detected evidence of heterogeneity of equine osteogenic cell populations at various skeletal sites. Further characterization of the dissimilarities is warranted to determine the potential role heterogeneity plays in differential rates of fracture healing between skeletal sites.[14] Differences were also detected in a human study, where iliac and tibial bone grafts were compared in their efficacy in ankle fusion.[15] This histologic investigation revealed that iliac bone grafts contained active hematopoietic marrow, whereas quiescent medullary fat predominated in tibial grafts.

Support, another important function of bone grafts, is mainly derived from corticocancellous or osteochondral bone grafts. These grafts may be transferred as fresh autogenous, vascularized grafts or as allogenous grafts. With allogenous cortical grafts, there are fewer surviving cells than with cancellous bone grafts, and nutrition depends on the formation of new haversian and Volkmann canals and canaliculi, which means it is a slow remodeling process that may take longer than 1 year, depending on the size of the graft.[16] This type of bone grafting is not very important in fracture repair in horses.

The successful incorporation of a bone graft depends on the interplay of the following six parameters[6,7]: (1) the host bed, (2) the viability of the bone graft, (3) the volume of bone to be grafted, (4) the growth factor activity of the host bed, (5) the metabolic activity index, and (6) the homostructural function of the bone graft.

The condition and location of the *host bed* determines the response to osteoprogenitor cells and perivascular connective tissue. Different bones exhibit different fracture healing rates, depending on local blood flow and bone marrow activity. The more viable and uncompromised the host bed, the better the acceptance of the bone graft.

The better the *viability of the bone graft* is, the better the acceptance of the graft. Therefore, cancellous and vascularized corticocancellous or cortical bone grafts may have the greatest chance of acceptance.

The larger the *volume of bone to be grafted,* the longer it takes to become completely incorporated into and remodeled by the host tissue. The longer the time needed for complete incorporation, the greater is the likelihood for development of complications. In large defects, there is a greater need for incorporation of a bone graft with a large surface, such as a cancellous graft. Such a graft demands absolutely rigid internal fixation until structural rigidity is achieved.

Growth factor activity of the host bed and bone graft induces proliferation of perivascular connective tissue, facilitating osteogenesis. Augmentation of the bone graft can be achieved by GFs through recruitment of additional cell populations as well.

The *metabolic activity index* (MAI), which is composed of parameters such as heart rate, blood flow, basic metabolic rate, respiratory rate, and body temperature, is correlated with the capacity to incorporate bone grafts, repair fractures, and respond to local growth factors.[7] The MAI of humans is 1.0, which is close to the MAI of the dog (1.5). The MAI of the horse has not yet been determined. The closer the MAI of two species is, the better is the correlation of the factors influencing bone healing and the more meaningful are comparative studies conducted between them.

The *homostructural function* of a bone graft, such as "spacer" function or support function, together with the fixation technique, influences the incorporation of the graft considerably. It may take years until it is completely incorporated or rejected.[7]

Bone grafts in diffent forms, with or without augmentations, have been studied experimentally or applied in clinical studies with mixed results. The following are some examples. Bilateral augmentations of the sinus floor in six adult sheep with mesenchymal stem cell–augmented bovine bone mineral on one side and autogenous bone in the other side revealed better results on the augmented side.[17] Composite graft material consisting of allogenic cancellous bone combined with autologous red marrow was used in the treatment of fresh traumatic bone defects and chronic nonunions of 38 human patients with good results.[18] The authors judged this combined graft as being safe and effective. In another clinical study reporting on 68 patients suffering from nonunion fractures, autologous bone grafts (47 patients) were compared to homologous grafts (21 patients).[19] No statistically significant differences in the effectiveness and time to bone union were found between recipients of autogenous versus homogenous grafts. One group of researchers added platelet-rich plasma (PRP) to autologous cancellous bone grafts and compared the effects to autologous bone graft alone in an experimental study in minipigs.[20] The results in the PRP group were superior to the control group, even in the semiquantitative assessment of the osseous bridging in both observed areas of the defect. Within the limits of the present study it could be demonstrated that PRP combined with autologous cancellous graft leads to a significantly better bone regeneration compared to isolated application of autologous cancellous graft in an *in vivo* critical size defect on load-bearing long bones of minipigs.[g] From these studies, it can be anticipated that further progress in grafting techniques, especially augmented autologous and homologous grafts, can be expected in the near future.[20]

Surgical Techniques

General Guidelines

It is advisable to use a separate surgical team for harvesting the cancellous bone graft and to plan it so that it can be immediately embedded into the recipient site by the surgeons repairing the fracture.[21] If there is any time lag between harvesting and implantation, the graft should be stored in blood-soaked sponges. It has been shown that exposure to air or to saline-soaked sponges decreases cell survival.[21] Additionally, exposure of the bone graft to antibiotics before and during harvesting is detrimental to survival of the cells.[22]

The bone graft should be lightly packed into the host site (Figure 77-1). Excessive packing should be avoided to allow the host tissues and fluids ingress to nourish the graft. The graft should be exposed as much as possible to bone marrow and endostium of the parent portion of the bone.[1] It is of paramount importance to ensure rigid internal fixation of the fracture in the presence of a bone graft to allow adequate ingrowth of a healthy vasculature. Otherwise, the cells in the bone graft may not survive, as a result of the lack of oxygen, compounded by mechanical damage.[21] Absolute asepsis is a prerequisite for successful application of a bone graft.

Locations

The principal donor sites for cancellous bone grafts in the horse include the tuber coxae (Figure 77-2, *A*), the sternum

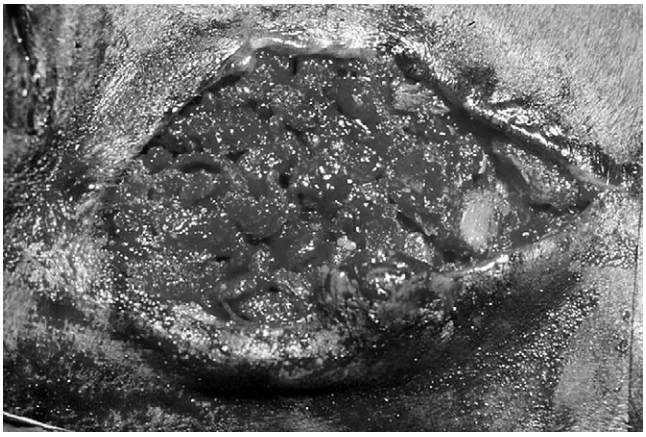


Figure 77-1. A cancellous bone graft placed in a bone defect of the skull.

(Figure 77-2, *B*), and the medial aspect of the tibia (Figure 77-2, *C*). All these sites are easily accessible, and large amounts of (viable) cancellous bone may be harvested.[23] There is little danger of a fracture through a cortex portal in the proximal tibia, provided the holes are appropriately located.[24] Cancellous bone samples from these conventional donor sites were evaluated for the presence of osteoprogenitor cells and compared to samples taken from the fourth coccygeal vertebra and tibial periosteum.[25] Sternal and tibial bone yielded viable osteogenic cells from 25% and 50% of horses, respectively, whereas yields from tuber coxae, coccygeal vertebra, and periosteum were 75%, 100%, and 100%, respectively. Tuber coxae and tibial periosteum had significantly greater numbers of osteoprogenitors compared with the fourth coccygeal vertebra.[25] Although one study proposed the proximal humerus as an additional graft site, it is not recommended as a viable option.[26]

TUBER COXAE

A straight 3-cm (1-inch) skin incision should be made over the tuber coxae, through the fat pad and subcutaneous tissues to the bone (see Figure 77-2, *A*).[27] Using a 5.5-mm drill bit passed through the 5.5-mm drill guide, a hole is prepared across the thin cortex. With the help of a curet, the cancellous bone located within the ilium is harvested in the quantity needed. Care is taken to avoid breaking through the thin inner cortex of the ilium, which could cause complications. If needed, additional holes may be drilled adjacent to the first one at a different angle.

A bone graft–harvesting drill (Figure 77-3) has been introduced. With this device, 4-mm-diameter cancellous bone plugs can be harvested through a stab incision (Figure 77-4). These plugs are an ideal size when implanted into drill holes of the same diameter during arthrodesis of the distal intertarsal joint (see Chapters 81 and 97).

The fat and subcutaneous tissues are closed over the bone in a simple-continuous pattern; an interrupted tension-relieving pattern is used in the skin. In selected cases, the skin may be stapled. A stent bandage, consisting of a gauze sponge rolled up tightly and placed in a longitudinal direction, is sutured over the incision. This bandage protects the skin edges and reduces movement in this region, facilitating skin healing. It might be advisable to cover this area with additional padding, especially during the recovery phase and in horses with a tendency to rest in a recumbent position.

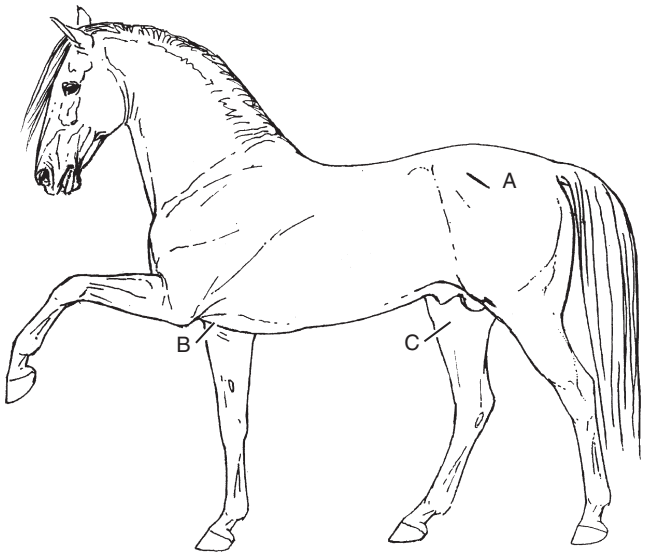


Figure 77-2. Skin incision sites used for harvesting cancellous bone for autologous bone grafting purposes. *A,* Tuber coxae, the most frequently used site. *B,* The sternum, ideal when the horse is positioned in dorsal recumbency. *C,* The proximal and medial aspect of the tibia.

Figure 77-3. The bone graft harvesting drill with 4.0-mm-inside-diameter hollow drill bits is shown being applied to the tuber coxae of an anesthetized horse.

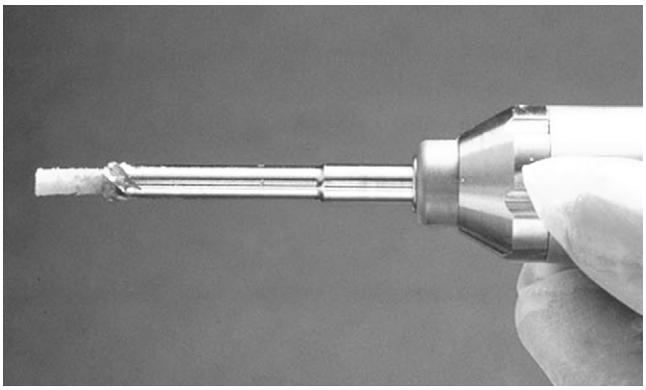


Figure 77-4. A 4.0-mm-diameter cancellous bone plug is shown protruding from the hollow drill bit.

STERNUM

The sternum is an especially advantageous donor site, because, when the animal is placed in dorsal recumbency, access is easy for harvesting (see Figure 77-2, *B*).[28] The sternum may also be approached with the animal in lateral recumbency; however, this is somewhat more cumbersome and the surgeon may have to perform the surgery standing in an undesirable position. Fracture treatment in a forelimb using internal fixation augmented by an autograft of cancellous bone from the sternum would be difficult to perform with two surgical teams working simultaneously, because of the proximity of the two surgical incisions.

A straight incision is made on the midline until the sternum is encountered. The hyaline cartilage is split longitudinally with the scalpel, and with the help of a periosteal elevator, the sternebrae are identified. The cancellous bone graft is harvested from each sternebra that is isolated. Care is taken not to incorporate hyaline cartilage within the bone chips. If more cancellous bone is needed than is contained within one of the sternebrae, the next nucleus may also be excavated and used as a graft. There are six or seven usable nuclei within the sternum, allowing an adequate amount of high-quality cancellous bone graft to be harvested for any type of fracture.

Closure consists of an initial simple-continuous layer along the border of the cartilage, through which the two cartilage flaps are united, followed by routine closure of the subcutaneous tissues and skin. It may be advisable to install a suction or Penrose drain for 24 hours. Additionally, the skin incision should be protected with a stent bandage, because otherwise this region is very difficult to protect.

PROXIMAL TIBIA

A straight incision is made over the medial aspect of the proximal tibia through skin, subcutaneous tissues, and periosteum (see Figure 77-2, *C*).[24] Using a 5.5-mm drill bit, the cortex is penetrated in one or two places, depending on the amount of cancellous bone graft needed. In one reported technique, two holes were drilled next to each other so that they became one hole 8 to 9 mm (¼ to ⅓ inch) long.[25] This allowed access of a rather large curet, and a large amount of high quality cancellous bone graft could be harvested. Closure of the incision is routine. Again, a stent bandage is applied over the incision to stabilize the skin edges.

PROXIMAL HUMERUS

The quantity of cancellous bone collected from the proximal humerus with traditional harvesting instruments is comparable to that collected from other sites in horses. The procedure is associated with minimal postoperative incisional complications or lameness. However, because one horse suffered a catastrophic humerus fracture following graft harvesting from this site, further research is required to assess the effects of this procedure on humeral breaking strength. On the basis of the risk of catastrophic fracture, this technique cannot be recommended for use in clinical cases, especially if an unassisted recovery from general anesthesia is planned.[26]

BONE REPLACEMENTS

Healing of fractures with concomitant bone loss is still one of the most challenging features in trauma surgery in all species, but especially in horses. Because of their slow bone metabolism, and therefore slow callus formation and fragment bridging, the mechanical load is resting mainly on the metallic implants for the first 2 months. Hence, promotion of fracture healing using synthetic bone replacements alone or in combination with active bone-forming agents *(biomimetics)* is an attractive alternative to the gold standard of autologous cancellous or cortical bone grafts (see earlier). The main reasons to use bone replacements instead of autografts are to reduce donor bed morbidity, including pain, and to decrease surgery and anesthesia time for the patient. With the increasing role of nanotechnology, polymers, and molecular biology in modern medicine, bone replacements using a combination of three-dimensional (3-D) scaffolds or matrices in combination with biomimetics may in the future well exceed the results expected with traditional bone-grafting procedures.

Bone replacements based on natural materials (allografts or demineralized bone matrices [DBMs]) have been applied in human and animal surgery with good success. DBMs, previously produced as a human product, are now available commercially in the equine form (COLLOS E).[29] Earlier products (e.g., Bio-Oss) consisted of bovine (demineralized) bone sources with the potential for causing weak allergenic effects, although pretreatment with hydrochloric acid was supposed to destroy antigenic matrix properties.[30]

Bone banks are common in human medicine for use of allografts, especially to replace joint structures,[31] but never became routine in equine surgery because of the risk of infections, slow incorporation of full (cortical) grafts, and the overall low turnover for such a product in clinical practice.

Many new synthetic bone replacements are on the market that either replace autologous bone grafts alone (ceramics, bone cements) or that are used in combination with other biomaterials (e.g., hydrogels, polymers), biomimetics (e.g., growth factors), and/or primary mesenchymal stem cells. Although these products are available, they are still rarely used in equine patients in a clinical setting and are more often applied in research, mostly as cartilage replacement in combination with microfracturing procedures[32] or osteochondral grafting.[30,33,34] Additionally, bone cements[35] and composites[36] have been successfully used to treat subchondral cystic lesions in young horses. However, none of these materials have been used to supplement fracture repair in the horse. On the other hand, this may change in the future, and therefore possible materials or composites to be used in horses are outlined in the following sections.

Ceramic Bone Substitutes

Marc Bohner

Chemistry

There are two main classes of ceramic bone substitutes (CBSs): *calcium sulfates (CaSs)* and *calcium phosphates (CaPs)*. Both families consist of several chemical compounds representing more than a dozen compositions (Table 77-1).

The first CBS that was used *in vivo* was α-CaS-hemihydrate (β-$CaSO_4 \bullet \frac{1}{2}H_2O$; also known as "plaster of Paris").[37] Addition of water to this material elicits an exothermic reaction, with the end products being a set form of gypsum ($CaSO_4 \bullet 2H_2O$) and heat.[38,39] Besides β-$CaSO_4 \bullet \frac{1}{2}H_2O$ and gypsum, there is another clinically relevant CaS, which is α-$CaSO_4 \bullet \frac{1}{2}H_2O$. α-$CaSO_4 \bullet \frac{1}{2}H_2O$ and β-$CaSO_4 \bullet \frac{1}{2}H_2O$ have the same crystallographic structure and composition, but the former compound is produced in an aqueous solution, whereas the latter

TABLE 77-1. Main Ceramic Bone Substitutes

Name	Formula	Ca/P	Mineral Name	Symbol
Monocalcium phosphate monohydrate	$Ca(H_2PO_4)_2•H_2O$	0.50	*	MCPM
Dicalcium phosphate	$CaHPO_4$	1.00	Monetite	DCP
Dicalcium phosphate dihydrate	$CaHPO_4•2H_2O$	1.00	Brushite	DCPD
Octocalcium phosphate	$Ca_8H_2(PO_4)_6•5H_2O$	1.33	*	OCP
Precipitated hydroxyapatite ("tricalcium phosphate")	$Ca_{10-x}(HPO_4)_x(PO_4)_{6-x}(OH)_{2-x}$	1.50-1.67	*	PHA
Amorphous calcium phosphate	$Ca_3(PO_4)_2•nH_2O$ n = 3-4.5; 15%-20% H_2O	1.50	*	ACP
Monocalcium phosphate	$Ca(H_2PO_4)_2$	0.50	*	MCP
α-Tricalcium phosphate	$α\text{-}Ca_3(PO_4)_2$	1.50	*	α-TCP
β-Tricalcium phosphate	$β\text{-}Ca_3(PO_4)_2$	1.50	*	β-TCP
Sintered hydroxyapatite	$Ca_5(PO_4)_3OH$	1.67	Hydroxyapatite	HA
Oxyapatite	$Ca_{10}(PO_4)_6O$	1.67	*	OXA
Tetracalcium phosphate	$Ca_4(PO_4)_2O$	2.00	Hilgenstockite	TetCP
Calcium sulfate	$CaSO_4$	—	*	CS
Calcium sulfate hemihydrate a	$CaSO_4 • ½H_2O$	—	*	a-CSH
Calcium sulfate hemihydrate b	$CaSO_4 • ½H_2O$	—	Bassanite	b-CSH
Calcium sulfate dihydrate	$CaSO_4•2H_2O$	—	Gypsum	CSD

Note: The first six calcium phosphate compounds precipitate at room temperature in aqueous systems. The last six calcium phosphate compounds are obtained by thermal decomposition or thermal synthesis.
*Not known or not indicated.

compound is obtained in dry conditions.[39] This provides $α\text{-}CaSO_4 • ½H_2O$ crystals with a more compact shape, which is of relevance when trying to produce strong and dense gypsum cements or gypsum-based drug carriers with a slow release rate.

Hydroxyapatite ($Ca_5[PO_4]_3OH$), β-tricalcium phosphate ($β\text{-}Ca_3[PO_4]_2$), and their composites (commonly called biphasic calcium phosphate) are the most common CaP bone substitutes. However, apatites can be synthesized with various structures and compositions. Moreover, many other CaPs, such as dicalcium phosphate dihydrate and octocalcium phosphate, are available and have been used *in vivo*.

To simplify, there are presently two classes of CaP: low-temperature and high-temperature CaPs. Low-temperature CaPs are obtained at room temperature, either through a setting (i.e., hardening) or a conversion reaction. These CaPs can be found *in vivo* and typically have a small average crystal size and a large specific surface area (up to 200 m^2/g for apatites). High-temperature CaPs are generally obtained by sintering reactions, which are above 700° C to 800° C. Because high-temperature CaPs are easier to synthesize than low-temperature CaPs, most commercial products are obtained via sintering reactions, an example of which is chronOS, the AO standard. However, low-temperature CaPs, such as dicalcium phosphate dihydrate (= brushite)[40] or precipitated apatite,[41] are likely to become more important in the future, because these compounds are very similar in physical structure to the CaPs present in the body.

Hydraulic cements set or harden via dissolution-precipitation reactions in an aqueous solution. For example, $α\text{-}CaSO_4 • ½H_2O$ and $β\text{-}CaSO_4 • ½H_2O$ dissolve in water, then gypsum crystals nucleate and grow. If the powder-to-liquid ratio is large enough (typically more than 2 g/mL), gypsum crystals grow close enough to entangle and hence provide mechanical stability to the resulting hardened compound. The setting reaction is generally exothermic, but the rate of heat release is too low to cause biocompatibility problems. Hardened cement blocks are nanoporous or microporous, and their porosity is typically on the order of 40% to 50% of the volume.

Whereas plaster of Paris (also a CaP compound) has been used for over 100 years, the discovery of CaP cements is recent.[42] The first products were introduced in the mid 1990s. Interestingly, many compositions have been proposed, but the end product of the reaction consists generally of brushite (e.g., chronOS Inject) or an apatite (e.g., Norian SRS). As a result, one talks about brushite or apatite cements. Because of their higher solubility, brushite cements tend to biodegrade much faster than apatite cements (see later).

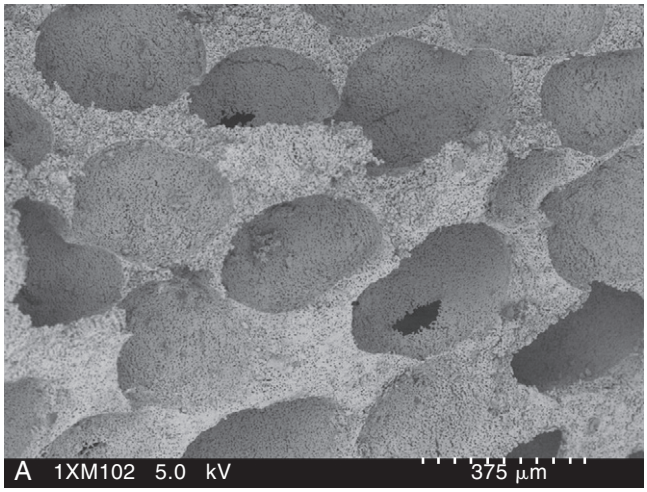
Porosity

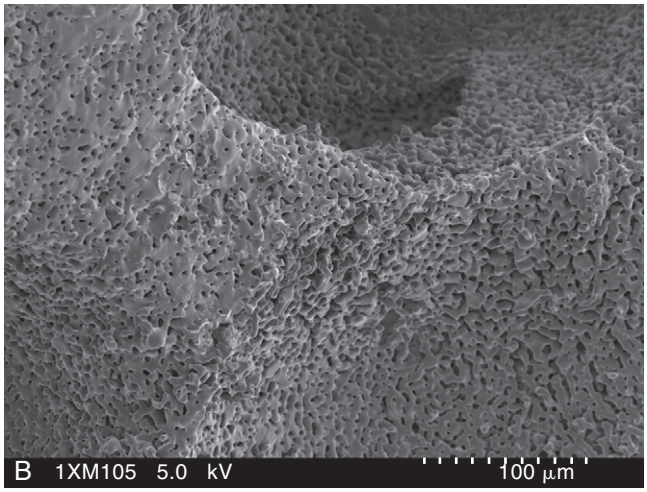
The porosity strongly influences the mechanical and biological properties of CBSs. Generally, two types of pores are discussed: micropores and macropores (Figure 77-5). Micropores typically have a diameter in the size range of 0.1 to 10 μm. Micropores have been considered to promote ceramic biodegradation,[43-46] but the scientific data are very scarce. It is, therefore, not possible to define an optimum micropore size or volume fraction. However, micropores are essential to prevent crack propagation[47] and hence should be a standard feature of CBSs. A practical advantage for the clinician is that a microporous CBS can be shaped with a blade. Presently, only a few products contain a significant fraction of micropores, one of them being chronOS.

Macropores typically have a diameter in the size range of 50 to 2000 μm. Macropores enable blood vessel and cell ingrowth; hence they promote bone ingrowth and have short biodegradation times.

Mechanical Properties

Compressive strengths as high as 150 megapascals (MPa) (i.e., almost as high as bone) have been reported for some CBSs. Unfortunately, CBSs are ceramics and therefore they are inherently brittle. As a result, tensile strengths are typically 5 to 20 times lower than compressive strengths, and shear properties are very poor. Therefore CBSs can break at very small loads (much





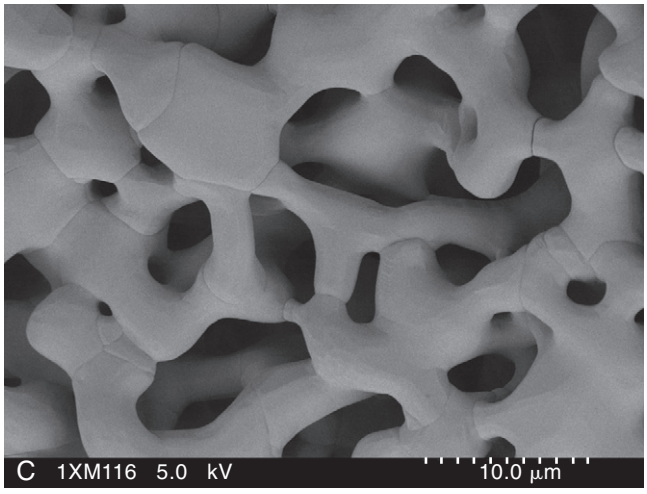


Figure 77-5. Microporosity and macroporosity in a β-TCP bone substitute as seen by scanning electron microscopy. **A,** Overall view of a macroporous bone substitute. **B,** Closer view of the macroporosity. The micropores are visible in the walls of the macropores. **C,** Microporosity. (Size of the bar: 500, 100, and 10 µm.)

lower than the listed average mechanical strength) and hence should be used in combination with internal or external fixation.

Many factors such as porosity, chemistry, and crystal and grain sizes influence the mechanical properties of CBS. Typically, an increase of porosity decreases the mechanical properties of a material exponentially. Moreover, and as previously mentioned, micropores decrease the brittleness of a CBS.[47] Furthermore, the least soluble CBSs, such as hydroxyapetite, tend to have the best mechanical properties. Finally, a small crystal or grain size favors better mechanical properties.

Forms

CBSs are available in various forms: granules, macroporous blocks, hydraulic cements, and putties (pastes). Each of these forms has specific advantages and disadvantages. *Granules* can be used to fill in any defect, but this procedure is often cumbersome and granules may migrate. Bone formation and ceramic biodegradation are both optimal because blood vessels and cells can invade the space between the granules. *Blocks*, such as cylinders, wedges, and prisms, are difficult to place in complicated defect geometries. However, blocks are mechanically stable and often have an optimal porous structure facilitating fast blood vessel and cell ingrowth. *Hydraulic cements* are generally injectable. As a result, cements are easy to apply into any defect and can be shaped. Moreover, cements harden with time, hence providing a stable (but not load-bearing) defect site. However, commercial cements are not (yet) macroporous. Therefore biodegradation occurs slowly, layer by layer.

The latest form of CBS is the putty, which is a thick paste.[48] The nature of putties varies from one producer to another. Some putties are simultaneously a hydraulic cement, and hence harden shortly after mixing. But traditionally, putties consist of a mixture of a gel and granules and do not harden. The advantage of puttiescompared to granules is that it is easier to apply a putty than granules. Moreover, the granules in the putty do not move out of the defect as easily as granules alone.

Biological Behavior

The mechanisms of biodegradation of CBSs vary widely depending on their solubility. CaS dissolves *in vivo* because body fluids are undersaturated in CaS. As a result, a change of blood supply or sample volume is expected to modify the dissolution time. A large gap occurs between implant and bone.[49] Typically, the amount and quality of the bone formed in the defect filled with CaS is poor: it consists of very narrow and small trabeculae, which tend to biodegrade with time. On the other hand, traditional CaPs such as β-tricalcium phosphate, hydroxyapatite, and their composites are insoluble in body fluid (e.g., serum).[50] This low solubility leads to an osteoclast-mediated biodegradation,[51] the rate of which depends on the composition. Whereas β-TCP is typically biodegraded within a year,[51] hydroxyapatite can be considered to be inert.[52] However, for all traditional CaPs, a direct apposition of bone on the ceramic surface is observed. Other CaPs, such as dicalcium phosphate dihydrate, have an intermediate solubility and hence an intermediate biodegradation rate (Figure 77-6). In that case, biodegradation mostly occurs via dissolution or the action of macrophages.[53] Moreover, often a small gap between bone and implant develops. This gap is typically filled with osteoid tissue.

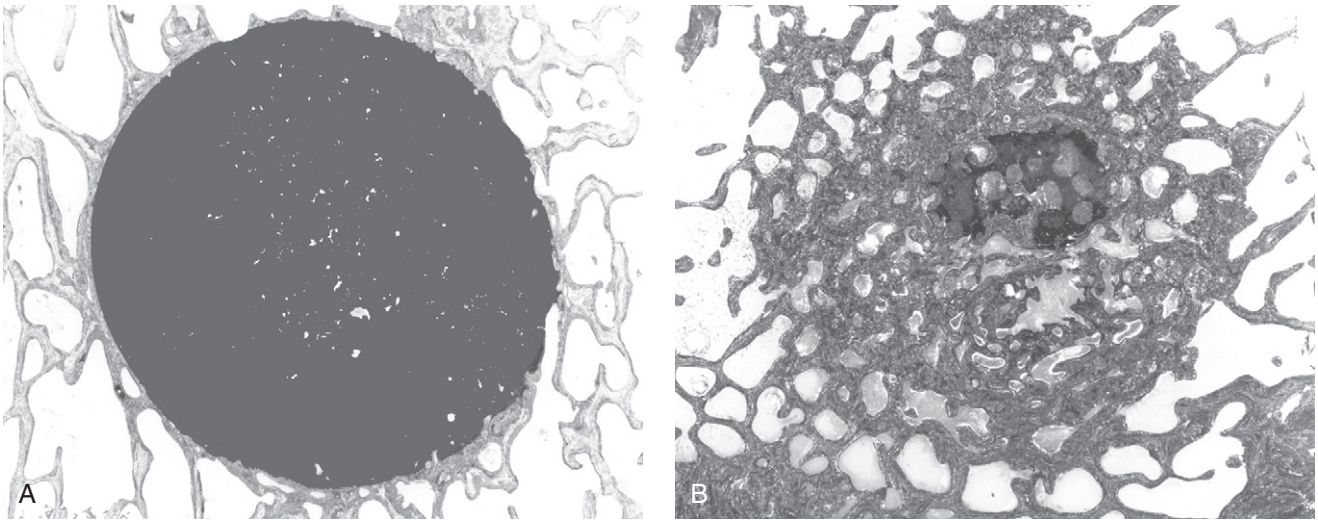


Figure 77-6. General appearance of an 8-mm cylindrical defect filled with **(A)** apatite and **(B)** brushite cement after 6 months of implantation in a sheep.[53] **A,** Only very small amounts of bone can be seen within the apatite cement (lighter areas within the cement). **B,** Brushite cement is almost completely biodegraded and replaced by new bone.

Apatite cements are slowly biodegraded (see Figure 77-6),[51,53,54] whereas hydroxyapetite is practically nonbiodegradable.[52] This apparently peculiar difference is caused by a variation of crystal size: apatites obtained from hydraulic cements are nanosized, whereas sintered hydroxyapetite is microsized. Actually, apatite cements have a biodegradation rate similar to that of β-TCP.

Many studies have been conducted to determine an optimal macropore size for bone ingrowth and biodegradation rate.[55-63] However, the results are not conclusive for two main reasons: it is difficult to synthesize ceramics with perfectly controlled geometries, and most *in vivo* studies have considered only a few geometries (e.g., one or two) at a few implantation times (e.g., one or two). Nevertheless, it was shown that a pore diameter in the range of 100 to 1000 μm is adequate,[55-63] macropores should be interconnected, and the size of the interconnections should be larger than about 50 μm.[62] A study applied a theoretical approach to determine an adequate pore structure to minimize the biodegradation time; it revealed that a pore diameter in the range of 200 to 800 μm is optimal but that this depends on the size of the bone substitute, with larger volumes requiring larger pores.[64]

Polymers

Margarethe Hofmann

Classes of Polymers

Polymer-based biomaterials were developed to replace metals or natural polymers because their properties could be tailored according to the requirements of the patient, for example, specific mechanical, chemical, or cell biology–related properties. These materials had to be adapted from the industrial application for which they were developed to fulfill the requirements in medicine. They have to be biocompatible, which means that they must be nontoxic, nonantigenic, and noncarcinogenic,[65] and they must have "the ability of a material to perform with an appropriate host response in a specific application."[66]

Polymers have a structure that in its simplest form is a linear chain. To these chains, branches can be added and combined into networks. Such polymers can be classified as:

- Natural or biopolymers, which are produced by living organisms. This group includes cellulose, starch, chitin, proteins, peptides, and DNA- and RNA-based materials. The monomeric units of the polymers are built of sugars, amino acids, and/or nucleotides. These natural polymers are all biodegradable.
- Synthetic polymers are produced by polymerization, a process by which small molecules called monomers are bonded together covalently. The synthetic polymers can be classified into three main categories: thermoplastics, thermosets, and elastomers. Synthetic polymers can be biodegradable, biodegradable-like, or nonbiodegradable.

When going back in the history of orthopedic implants, the polymeric biomaterials can be grouped into three generations.

FIRST-GENERATION

First-generation polymers include nonbiodegradable materials like silicone rubber, polyethylene (PE), acrylic resins, polyurethanes (PUs), polypropylene (PP), and polymethyl methacrylate (PMMA). One of the most prominent materials is bone cement based on PMMA and is intended for use in arthroplastic procedures of the hip, knee, and other joints when polymeric or metallic prosthetic implants are fixed to living bone. PMMA has excellent properties as a fixation medium for implants and is still in clinical use with a good rate of success. In equine clinics, it is used for plate luting and as antibiotic-impregnated beads that are prophylactically applied near the bone plates for local infection control. PMMA has significant drawbacks because of its toxic degradation products, which may provoke fat embolism. In addition, heat is produced during polymerization, which may cause necrosis in the tissues surrounding the implant. The continuous improvement in the synthesis of PMMA bone cements has altered their microstructure and mechanical properties. Nevertheless, the U.S. Food and Drug

Administration (FDA) reclassified the material in 2002 from a class III to a class II (special controls) device.[67] Today PMMA is also used in vertebroplasty and kyphoplasty where the vertebral body is augmented to increase stability.[68]

Other polymers of the first generation are of the family of PEs, to which belong ultrahigh-molecular-weight polyethylene (UHMWPE), a thermoplastic PE with extremely long chains and molecular weights of some million daltons. Such long chains are of interest in applications exposed to high load bearing, such as the liner of acetabular cups in total hip arthroplasties, in the tibial insert and patellar component in total knee arthroplasties, and as a spacer in intervertebral artificial disc replacement. This material exhibits special physical properties, such as high abrasion resistance and low friction coefficient. Despite these properties, wear is still a problem and needs to be improved. The high impact strength and the excellent toughness combined with a low density make this material a good candidate, relative to metals, for use in implants. Easy fabrication and desirable biological properties (good biocompatibility and biostability) make this material a good selection for surgeons.[68]

Another material that has been increasingly used in orthopedic, trauma, and spinal applications since its commercialization in the 1980s is the group of polyaryletherketones (PAEKs), which can be reinforced with fibers like carbon or glass to improve stiffness and long-term stability.[68] The group of PAEKs (including the most important representatives used in spine and orthopedic applications consisting of poly[aryl-ether-ether-ketone] [PEEK] and poly[aryl-ether-ketone-ether-ketone-ketone] [PEKEKK]) represents high-temperature thermoplastic polymers consisting of an aromatic backbone molecular chain interconnected by ketone and functional ether groups. PEEK-OPTIMA, carbon fiber reinforced PEEK-OPTIMA compounds, and composites produced by Victrex meet the criteria for FDA Master Files.[69]

SECOND AND THIRD GENERATION

The polymers in these two generations are variably biodegradable. Their interaction with the biological environment and their degradation properties define the crucial factors for their use, such as in tissue engineering. The important group of polymers called bioelastomers is part of this new generation of polymers having the following characteristics: good biocompatibility with surrounding tissues, glass transition temperatures* (Tg) lower than body temperature (35° C to 40° C), and good elastic properties (e.g., the ability to return to at least 1.25-fold the original length after 1 minute of release if stretched to 1.5-fold its original length for 1 minute maintaining the stretch stress in the range of 0.1 to 20 MPa). These bioelastomers can be classified into two categories: (1) elastomers suitable for long-term physiological contact or implantation and (2) biodegradable elastomers for a determined time of physiological contact or implantation.[70]

A perfect biodegradable implant can be described as follows: "Once implanted, it should maintain its mechanical properties until it is no longer needed and then be absorbed and excreted by the body, leaving no trace."[71] The ideal polymer for such an application needs to fulfill the following criteria[72]: (1) the polymer does not evoke an inflammatory or toxic response disproportionate to its beneficial effect; (2) it is metabolized in the body after fulfilling its purpose, leaving no trace; (3) it is easily processed into the final product form; (4) it has an acceptable shelf life; and (5) it is easily sterilized. Polymers fulfilling these demands can be used to manufacture scaffolds, which can be loaded with specific drugs or growth factors and implanted into the target tissue. The initial time, duration, and quantity of drug released from the scaffolds can be tailored, and once the drug is released, the scaffold disintegrates and does not have to be removed through a second surgical intervention. Examples of constructs that could be used in fracture repair are a scaffold impregnated with osteoblasts, chondrocytes, mesenchymal stem cells, various tissue and growth factors to improve bone defect healing, and/or antibiotics to control or prevent infection. Such constructs may help to reduce or even eliminate problems of donor site scarcity, immune rejection, and pathogen transfer.[73,74] The scaffold also could be applied in the clinical fields of tissue replacement and tissue function enhancement. In the latter, the form and structure of the polymeric scaffold would resemble the structure and form of the tissue or organ that it replaces. Examples include scaffolds used as artificial skin, artificial pericardium, or periosteum that would be manufactured as a flat microporous membrane. Tubular scaffolds could be used for blood vessels or nerve regeneration, and solid block scaffolds for bone substitutes and repair of articular cartilage would be contoured to the proper shape in surgery.[75]

Degradable polymers used in load-bearing orthopedic implants are of interest as skeletal fixation devices. Ideally they would only stay in the body to fulfill a temporary function and prevent the necessity of retrieval surgeries. The advantage compared to metallic devices is that these implants would lose their mechanical properties gradually during degradation, whereas in the best-case scenario, the healing tissue would gain strength, so that at the end of the healing process the implant would be fully degraded and the damaged tissue would be completely remodeled. To achieve this mechanical balance, the stiffness (E-modulus) of the implant would need to be comparable to that of the tissue it substitutes, such as bone. Another advantage of polymeric implants is their radiopacity, which would allow radiographic assessment of bone healing without implant superimposition.

Polylactides and their combinations have been chosen as scaffolds because of their excellent biocompatibility and their relatively high mechanical strength. However, they have a relatively poor long-term performance under prolonged mechanical loading because these implants, produced by poly(L/DL-lactides) (PLDLLAs), develop severe plastic deformation after about 6 months. This behavior may be related to the intrinsic properties of amorphous polylactides rather than degradation of the polymer.[76]

Polyurethane (PU) elastomers belong to another very important and popular group of polymers. They were developed more than 50 years ago as biostable implant materials. PUs show a greater versatility in physical and mechanical properties than other polymers. They can be tailored, which also influences their biological properties, like biodegradation and biocompatibility. The incorporation of soft components may enhance degradation, which depends on the crystallinity or amorphous behavior of the soft components. Hydrolysis is likely the main degradation pathway.

*"The liquid-glass transition (or glass transition for short) is the reversible transition in amorphous materials (or in amorphous regions within semicrystalline materials) from a hard and relatively brittle state into a molten or rubber-like state. An amorphous solid that exhibits a glass transition is called a glass."[71a]

TABLE 77-2. Important Biodegradable Polymers of Synthetic and Natural Origin

Abbreviation	Polymer	Properties
PGA	Poly(glycolide)	Biodegradable, thermoplastic polymer, and simplest linear aliphatic polyester
PLA	Poly(lactide) acid	Has softer amorphous regions and can exist in two different isomeric configurations: poly-L-lactic acid (PLLA) or poly-DL-lactic acid (PDLLA)
LPLA; also called PLLA	Poly(L-lactide)	Biodegradable, thermoplastic aliphatic polyester derived from renewable resources such as corn, starch, or sugarcane
DLPLA; also called PDLLA	Poly(DL-lactide)	Amorphous poly-DL-lactide (PDLLA) by polymerization of a racemic mixture of L- and D-lactides Used for sutures, stents, dialysis media, drug delivery devices, and tissue engineering
LDLPLA	Poly(DL-lactide-*co*-L-lactide)	Poly(D,L-lactide) homopolymers polymerized either from meso-lactide or from equimolar (racemic) combinations of D-lactide and L-lactide They are considered to be amorphous and typically possess nominal mole fractions that equal or exceed 50% lactide.[134]
DLPLG	Poly(DL-lactide-*co*-glycolide)	Poly(D,L-lactide-coglycolide) copolymers are copolymerized from a combination of glycolide and either meso-lactide or racemic quantities of D-lactide and L-lactide (amorphous and mole fractions similar to LDLPLA).
LPLG	Poly(L-lactide-*co*-glycolide)	See DLPLG.
PGA-TMC	Poly(glycolide-*co*-trimethylene carbonate)	Biodegradable, thermoplastic polymer and simplest linear aliphatic polyester; it can be prepared starting from glycolic acid by means of polycondensation or ring-opening polymerization
PDO or PDS	Poly(dioxanone)	Colorless, crystalline, biodegradable synthetic polymer
PCL	Poly(e-caprolactone)	Biodegradable polyester with low melting point; degrades by hydrolysis of its ester linkages in physiological conditions
PHB	Polyhydroxybutyrate	Polyhydroxyalkanoate (PHA) produced by microorganisms; water insoluble and relatively resistant to hydrolytic degradation
POE	Polyorthoester	Bone filler material
PHEMA	Poly(2-hydroxyethyl-methacrylate)	Shows physical properties similar to natural gel-like extracellular matrices; used with hyaluronic acid (HA) for orthopedic application
—	Chitosan	Prepared from chitin by partial deacetylation; is nontoxic, biocompatible, and biodegradable[135]

Because of their special mechanical properties, biodegradable elastomers can be used in the repair or replacement of tissues of a similar structure (tissue engineering). As mentioned earlier, this group of materials can sustain and recover from multiple deformations without irritating the surrounding tissue.[70]

Biodegradable polymers of both synthetic and natural origin, most of which appear in Table 77-2, are used for surgical sutures, orthopedic implants, plastic surgery, drug delivery, cardiovascular applications, and tissue engineering. In orthopedics, many different polymers and combination products with polymers using hydroxyapetite, fibers, or growth factors are used to fill osteochondral defects, to facilitate bone regeneration of mandibular defects, and to fill cartilage defects. Screws are manufactured from polylactides (PLAs) or by combining polyglycolic acid (PGA) and variations of PLAs. Meniscus staples are produced from polyglycolide or polyglycolic acid (PGLA), a copolymer of PLA and PGA, and pins and rods are made out of PGLA, a combination of poly-L-lactide (PLLA) with poly-DL-lactide (PDLLA) and PLA. Bone plates and meshes can be manufactured out of PLLA-PDLLA, PGA-PLLA, and PLLA-PDLLA; the combination is also used for spinal cages.[77]

Mechanism of Polymer Degradation

There are different names to describe the degradation of a polymer during its lifetime, including bioabsorbable, biodegradable, and bioresorbable, all of which mean that the material degrades over a certain time through hydrolysis into fragments and water. If this process does not provoke any harm or negative side effects (inflammation or toxic response) to the patient, the material is biocompatible. Hydrolysis can be influenced by several factors, including (1) the production process, which influences the microstructure, and (2) the sterilization techniques, which strongly influence the surface of the material or implant.

The initial aim of polymeric implant development was to replace metallic implants. Therefore materials were selected that resist degradation within the human body. However, the polymers did not always meet the requirement of clinicians; some materials failed because it was difficult to achieve sufficient inertness and because problems with cracks, oxidation, or degradation occurred.[78,79] This balance between degradation of synthetic or biopolymers and the regeneration of new animal or human tissue without any negative side effects is one of the big challenges in polymer development. Thus it is critical to understand the mechanisms of polymer biodegradation in relation to the surrounding tissue.

For biodegradable elastomers, the *rate of degradation* is one of the most important properties. All biodegradable polymers contain hydrolyzable bonds. For this reason, the most important mechanism of degradation for tissue engineered scaffolds is chemical degradation through hydrolysis or enzyme-catalyzed hydrolysis.

Passive hydrolysis is the determining mechanism of degradation for many bioelastomers. The chemical bonds determine the rate of hydrolysis, but other factors may influence the type, time, and intensity of degradation. These factors include the crystallinity and hydrophilicity of polymers, the polymers' composition, and the pH and temperature of the surrounding tissue. Anhydrides and orthoesters are the most hydrolyzable components, followed by esters and amides. The degradation rate is mostly determined by the time dependent molecular weight and is much higher for hydrophilic polymers than for hydrophobic ones.[70]

The molecular weight of the polymer begins to decrease immediately (PGA, PLA) or a few weeks (PLLA) after placement in an aqueous medium. PGA biodegrades rapidly and loses its strength within 1 month. In both the semicrystalline structured PLA and PGA, degradation occurs in two phases: (1) the *hydrolysis phase*, in which water molecules inserted into the long macromolecule cleave long chains into shorter ones, and (2) the *metabolic phase*, where macrophages phagocytize the small polymer fragments. The liver eventually metabolizes the breakdown products of glycolic and lactic acids.[80]

During the hydrolysis phase, acid degradation products may be liberated and released into the surrounding tissues, provoking inflammatory cellular reactions involving macrophages and giant cells. After the polymer has been depleted, these cell reactions decrease, and the space occupied by the former implant is filled with connective tissue or bone. Although the mechanism of degradation is often well known, it is difficult to predict the rate of degradation and the reaction with the surrounding tissue. Depending on the size, the material, and the target tissue, the time it takes to completely degrade can vary between days and years.[81]

Outlook

Vast improvements have been made in the materials recently developed, including improved biocompatibility, better adaptation of mechanical properties compared to the surrounding tissue, and new functions (smart or intelligent materials). Materials scientists and biologists have worked together to develop polymeric materials that respond better to cell–matrix interactions and improve repair, regeneration, and recovery of a tissue to its functional state. Designing new biomaterials is a combination of modeling, materials science, biotechnology, advanced cell biology, and eventually animal testing. It becomes increasingly important to expand our knowledge about cellular signaling processes and to learn more about how biomaterials affect bioadhesion. Soon, the surface and interfaces of biomaterials will have the potential to deliver complex molecules such as drugs, genes, or mesenchymal stem cells to stimulate the biological environment. Polymeric nanoparticles alone or applied to surfaces may serve as a source for such molecules, and smart gels will respond to temperature changes and pH, triggering the release of molecules. Therefore implants will not only serve as replacements of tissue but also may become valuable components in the treatment of diseases.[82]

Because implants do fail (implant loosening), modifying the implant design to mimic the surface of bone (cell–biomaterial interaction) may improve osseous integration and overcome such problems in orthopedic and dental applications. There is increasing interest in placing an osteoconductive protein in an osteoconductive carrier medium to facilitate timed-release delivery and/or to provide a material scaffold for bone formation.[83]

Gels

Brigitte von Rechenberg

Hydrogels consist of a network of polymer chains that have the capacity to hold water (up to 99% volume) with their hydrophilic groups and therefore remain flexible, similar to tissue.[84] Their 3-D structure mimics the *in vivo* tissue environment and enhances the interaction between cells and the customized macromolecules of the gels. Because chemical cross-linking of hydrogels disrupts the delicate structure of impregnated pharmaceuticals, growth factors, or cells, they have been replaced by gels that spontaneously assemble under physiologic conditions.[85] However, swelling of hydrogels after polymerization may be an advantage if a "press fit" of implants is attempted and micromotion must be prevented.[86] The porous structure of hydrogels may be changed by cross-linking density, which may influence cell behavior or survival[87] as well as degradation and overall biocompatibility, such as inflammatory reactions within the environment.[88]

Natural hydrogels are based on fibrin,[89] which allows the incorporation of bioactive substances via heparin-binding delivery systems in a 3-D matrix.[90] Synthetic hydrogels such as agarose or alginate are often used for *in vitro* systems to study cell interactions and proliferation, such as with chondrocytes,[91] but new technology has resulted in the synthesis of mechanically stronger materials based on polyethylene glycols. Therefore smart hydrogels, either protein or DNA based and hybrid hydrogels,[92] have a promising future for medical use in the surgical patient.[93,94] A combination of natural and synthetic hydrogels resulted in successful repair of tibial defects in rabbits,[95] and a synthetic polymer alone was successful to bridge a tibia defect in sheep.[96]

Hydrogels are normally degraded through hydrolysis, and depending on their composition, their degradation can be fast or slow. Modern chemistry facilitates controlled degradation as well as incorporation and release of biomimetic substances.[97] Histologically, hydrogel remnants commonly can be visualized in the cytoplasm of macrophages.

In the equine literature, the use of hydrogels is reported for research in cartilage cell cultures[98] and wound healing.[99] Reports of hydrogels used in fracture or bone defect healing as the sole substitute are absent.

Composite Materials

Composite materials, often abbreviated to "composites," are engineered or naturally occurring materials made from two or more constituent materials with significantly different physical or chemical properties (that remain separate and distinct at the macroscopic or microscopic scale within the finished structure). Modern polymer technology makes it possible to synthesize biocompatible, mostly porous structures that can be further enriched with appropriate coatings and gelling fluids to improve cell adhesion, proliferation, and differentiation. Often, advantage is taken of the different constituent mechanical properties, such that two-layer grafts are created, such as in osteochondral grafts.[100,101] As with all biomaterials, biocompatibility of composites depends not only on the finished product but also on the byproducts generated during degradation, and in the case of composites it depends on the interaction of the different constituents.

Biomimetics

Brigitte von Rechenberg

The term *biomimetics* originally referred to the "study of the structure and function of biological systems as models for the design and engineering of materials and machines," whereas the roots come from the Greek words *bios* (meaning "life") and *mimesis* (meaning "to imitate"). Although *bionics* and *biognosis* are often also used as synonyms, the term *biomimetics* is preferred when "chemical reactions, in nature, involve biological macromolecules (e.g., enzymes or nucleic acids) whose chemistry can be replicated using much smaller molecules *in vitro*." Today, the term is used broadly in biotechnology to designate molecules that are known to enhance new tissue formation.

Biomimetics are normally applied locally as proteins, peptides, or plasmids, although they are seldom used alone but rather in combination with scaffolds or other biomaterials as carriers. If these molecules are applied alone, elimination is normally so fast that their biomimetic effect is ablated. The combinations allow a constant and often protracted release of the molecules; therefore, effective concentrations are maintained over an ensured time period. Most of the biomimetics are dose and time dependent, and therefore their use has to be carefully orchestrated in the desired environment.

Growth factors were the first molecules detected to enhance tissue formation *in vitro* and *in vivo*. Among those, bone morphogenetic protein played a major role in the history of bone research leading to the so-called bone induction principle[102] and the discovery of its related molecule, bone morphogenetic protein, which is now commonly referred to as BMP.[103] Today up to 20 different BMP molecules are known, of which BMP-2 through BMP-7 were later found to be part of the transforming growth factor-β (TGF-β) superfamily. Though BMP-2 and BMP-4 were shown to be important molecules for bone induction, BMP-7 (or osteogenin [OP1]) seemed to enhance cartilage formation.[104] At the time of this writing, BMP-2 and BMP-7 are the only members of the family that are approved by the FDA for human application. For veterinary use, the commercially available products are cost-prohibitive, and therefore most of the BMPs applied in veterinary medicine were either in experimental animals[105] or a select few clinical patients.[106] Because the BMP patent will expire within the next few years, costs may come down considerably and products may be developed for veterinary use.

Apart from BMP, other growth factors, such as PDGF, FGF a and b, IGF-1, and TGF-β were demonstrated to play a role in fracture healing.[107] Whereas PDGF and FGF are released mainly soon after the fracture and are responsible for cell recruitment and proliferation, IGF and TGF are responsible for callus formation (TGF), angiogenesis (IGF), and finally, fracture healing. PDGF is mainly released by platelets in the initial fracture hematoma; FGF is synthesized by cells of the soft callus; and IGF-1, TGF-β, and BMP-2 are liberated from the bone matrix during initial bone biodegradation of the fracture ends and later are produced by osteoblasts building up the new bone matrix. Additionally, BMP plays a major role in pushing osteoprogenitor cells toward differentiation into osteoblasts.

Because of their prominent role in physiologic fracture healing, PDGF, FGF, IGF, and TGF were also tested in research situations to enhance bone healing in fractures or bone defects. PDGF has gained some attention in clinics by the introduction of platelet-rich concentrates applied in trauma cases in the operating room.[108] Because this concentrate can be easily prepared by medical personnel and its autologous source does not evoke immunologic responses, this has become an attractive form of therapy. The concentrate contains high levels of PDFG, VEGF (vascular endothelial growth factor), and TGF, which are released by the platelets and are thought to increase migration of cells into the fracture hematoma; their proliferation enhances callus formation and angiogenesis. Nevertheless, the efficacy of platelet-rich concentrates is still debated and awaits clinical proof. IGF encapsulated in microspheres showed significant dose-dependent enhancement of bone-forming properties in a tibial defect model in sheep,[109] whereas TGF that was exogenously applied in tibia osteotomies in rabbits increased callus size but not osseous bridging of the fracture line.[110] Nevertheless, the combination of IGF and TGF enhanced cervical spine fusion when titanium cages coated with a polymer layer (PDLLA) were enriched with both.[111]

The local use of biomimetics other than growth factors is still rare, but one of the more promising molecules is based on parathyroid hormone (PTH). Although PTH is known to cause bone biodegradation to maintain systemic calcium homeostasis, in low and intermittent dosages it increases bone formation and is now clinically used to treat osteoporosis in menopausal women.[112] $PTH_{1\text{-}34}$ also has been studied as a local biomimetic to enhance fracture and bone defect healing[113] in combination with carriers, such as fibrin-based hydrogels alone or mixed with calcium phosphate granules ($PTH_{1\text{-}34}$ + hydrogel) vs. ($PTH_{1\text{-}34}$ + hydrogel + CaP granules). $PTH_{1\text{-}34}$ has been used successfully to bridge 1- to 2-cm tibial defects in experimental sheep (Figure 77-7), and a first clinical trial in humans confirmed its safety as well as its beneficial effects in healing human distal radius fractures.[114] Systemic application of low intermittent dosages of PTH during fracture healing also increased bone formation in laboratory rodents.[115] However,

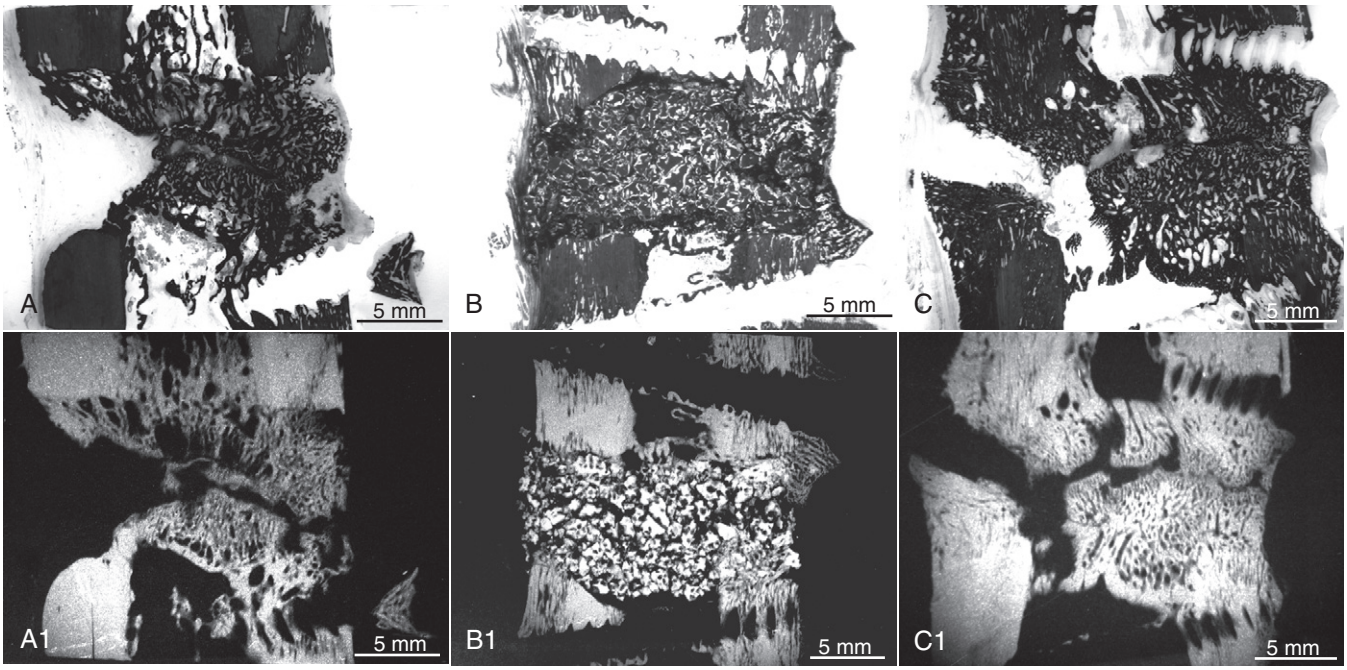


Figure 77-7. Hydrogel and composite bone replacements in a 1-cm tibia defect in sheep at 3 months after surgery. **A,** Fibrin hydrogel + biomimetic alone. **B,** Fibrin hydrogel + tricalcium phosphate and hydroxyapatite granules + biomimetic. **C,** Synthetic hydrogel based on polyethylene glycol (PEG) alone. *Top row:* Ground sections. *Bottom row:* microradiographs. **B** shows the best results for bone healing.

successful clinical use in horses is limited to a report of healing of a subchondral cystic lesion.[8]

Because the delivery of these growth factors as native proteins is costly and difficult (see earlier), alternative routes are to use gene delivery in the form of DNA plasmids in carrier systems[116,117] or to transfect cells *in vitro* and introduce the transfected cells into the desired area. Transfection of cells *in situ* can be achieved by either virus-mediated plasmid incorporation, often associated with adenoviruses,[118] or nonviral methods; the most promising of the latter include liposomes,[119] superparamagnetic iron oxide nanoparticles,[120] and CaP-based delivery systems.[121]

Tissue Engineering and Regenerative Medicine

Brigitte von Rechenberg

Tissue engineering emerged from materials science, and because of its increasing importance it has become a field of its own right. In a larger sense, the term *regenerative medicine* is used almost as a synonym. However, the difference is that tissue engineering is an effort to replace organ tissues with a combination of biomaterials, cells as well as biochemical and biomechanical factors; regenerative medicine concentrates more on methods using mesenchymal stem cells. Both involve classic interdisciplinary or translational approaches to improve functions of a diseased organ system in the body. In equine medicine and surgery, tissue engineering and regenerative medicine are still mostly exploratory[122] but are gradually finding their way into clinical applications involving, for example, tendons[123,124] and cartilage[125,126] and, less frequently, bone.[127]

Cells used for these applications are mostly either autologous primary cells, taken from the target organ itself (e.g., cartilage, bone), or stem cells. Although primary cells must be assessed for their ability to maintain phenotype and function through several passages during cell culture expansion, harvesting of stem cells from bone marrow or blood may prove difficult because of their limited availability in adults. Therefore, alternative sources, including adipose and umbilical tissues, are being characterized and may prove to be richer sources of stem cells. The use of embryonic or fetal tissue is an ethical issue in human medicine, but is less so in the veterinary field. The advantages of using fetal tissue are that they lack immunogenic properties[128] and it may be possible to establish cell banks for tissue engineering or regenerative medicine with veterinary applications. Because allographic tissues are used, this path is therefore highly attractive. Conversely, if autologous cells were the only source for cell expansion, the technical and financial difficulties encountered when trying to prepare tissue-engineered products or suitable cell constructs for clinical applications would severely limit their use in the veterinary field. Cell banks with fetal tissue could circumvent this problem to a large extent. Fetal cells were used successfully to engineer bone tissue constructs[129,130] *in vitro* and new bone formation *in vivo* using experimental sheep.[131]

Tissue engineering, especially in bone defect healing, normally involves 3-D scaffolds or matrices. Collagen, fibrin, and hyaluronic acid–based matrices (Hyaff), flexible polymer scaffolds enriched with CaP layers, and other composites were proved suitable for cell seeding. Modern technology (nanoconstructs) provides almost unlimited possibilities for creating porous structures as effective bone substitutes. However, independent of the scaffold type used, effective cell seeding is difficult to accomplish in the center of the grafts, where it is difficult to supply sufficient nutrients if only traditional cell culture conditions are used. Therefore bioreactors have been developed that maintain steady flow dynamics of the culture media and promote access of cell nutrients to the center of grafts.[132,133] Bioreactors also can be used to induce biomechanical loading of grafts, which stimulates cell differentiation. In combination with appropriate cell culture media, a permissive biochemical and physical environment is created for cells to

differentiate into bone or cartilage constructs that can be implanted in patients. Types of bioreactors may vary, such that compressive cylinders, spinner flasks, and rotating or stirring suspension bioreactors have been proposed to facilitate tissue engineering of bone substitutes.

With all of these exciting prospects for tissue engineering and regenerative medicine, it is important to realize that none of these technologies had yet to be routinely used in equine bone surgery at the time this chapter was written, but rather were still in their embryonic stages.

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CHAPTER
78 Synovial Joint Biology and Pathobiology

David D. Frisbie

The function of synovial joints depends on the integrity of normal anatomy and proper cellular function of each of its components. Bone, articular cartilage, synovial fluid, synovial membrane, fibrous joint capsule, and ligamentous structures make up these components (Figure 78-1). On either side of a synovial joint, subchondral bone is covered by articular cartilage, providing the nearly frictionless contact surfaces of the joint. Synovial membrane and ligamentous joint capsule surround the cartilage and attach to bone on either side of the joint, providing stability and a reservoir for the synovial fluid. The synovial joint is further stabilized by ligamentous and muscular structures that surround the joint; these structures are extracapsular in most instances. Three different *classifications of joints* exist: synarthroses (immovable), amphiarthroses (slightly movable), and diarthroses (movable). Diarthrodial joints account for the greatest number in the body and are the focus of this chapter. The articulations of diarthrodial joints facilitate movement of the rigid skeleton. A healthy joint provides a frictionless system with efficiency that is an order of magnitude superior to the best bearing surfaces known to modern engineering.[1] When pathologic states arise, it is usually because of dysfunction of one or more components. Because the health of the synovial joint depends on the sum of its parts working in unison, it is often referred to as an organ system, as all components must be functional for the health of the "organ." This chapter explores each component of the synovial joint both in health and disease in more detail.

ANATOMY OF SYNOVIAL JOINTS

Synovial Membrane and Synovial Fluid

The synovial membrane, or synovium, lines the joint cavity and is made up of two layers: intimal and subintimal. The *intimal layer* is largely responsible for the content of the synovial fluid, is typically one to four cell layers thick and does not have a basement membrane. The lack of basement membrane is relevant with respect to the role the synovium plays in determining the content of the synovial fluid in the joint and is covered in more detail later. The *subintimal layer* is made up of fibrous, areolar, and fatty tissues. A very good blood supply is present in the subintimal tissue, as well as innervation originating from both peripheral and muscular nerve branches (Figure 78-2).[2,3] In fact, using an arthroscope close up and focused directly on normal synovial membrane, one can see individual red blood

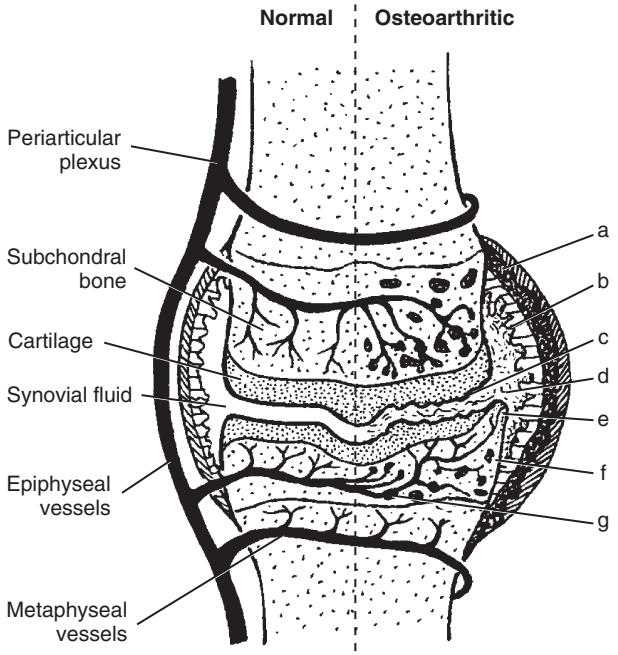

Figure 78-1. A normal synovial joint *(left),* including articular cartilage, synovial fluid, and synovial membranes, together with changes seen in a joint with osteoarthritis *(right).* In an osteoarthritic joint, the following abnormalities can be present: *a,* capsular fibrosis; *b,* synovitis; *c,* cartilage failure; *d,* depolymerized hyaluronic acid; *e,* osteophytes; *f,* subchondral cysts; *g,* vascular engorgement. (Modified from March L: Articular Cartilage in Health and Disease. p. 86. In Sambrook P [ed]: The Musculoskeletal System. Churchill Livingstone, New York, 2001.)

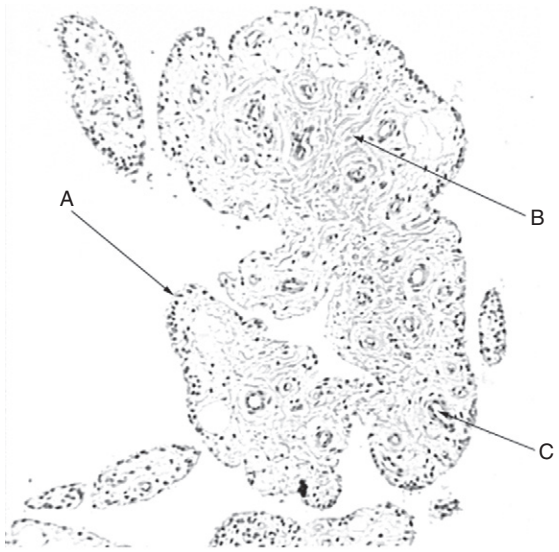

Figure 78-2. Photomicrograph of a 5-µm section of normal synovial membrane, hematoxylin and eosin (H&E). *A,* The intimal layer; *B,* the subintimal layer; *C,* a vessel in the subintima.

cells coursing through the capillary network of the villi. When viewed both grossly and arthroscopically, the synovium has areas that are flat intermixed with areas of loose collections of villi. These areas of villi are often described as "seaweed-like" when viewed arthroscopically in a fluid medium. The two appearances of the synovium are thought to be related to biomechanical characteristics of the joint area, since they remain relatively unchanged throughout the life of healthy joints.

The cellular population of the intimal layer is based on two basic functions: *phagocytosis* and *protein secretion*. The cell types have historically been classified as type A or B synoviocytes on the basis of ultrastructure and immunohistologic characteristics. Cells engaged in phagocytosis or pinocytosis are termed type A synoviocytes, whereas those responsible for protein secretion have been referred to as type B synoviocytes.[4] More recently, the description of type C cells has been reported, and these cells are thought to represent a cell type between type A and B. Because type A and B cells have demonstrated functions assigned to the other cell type, and because synoviocytes have been shown to be dynamic, it is likely that type C cells represent synoviocytes in a transition from type A to B, or vice versa.[5,6]

When the focus is on the function of the intimal layer of synoviocytes, the importance of its role in both health and disease of the joint becomes apparent. One function is phagocytosis—the mechanism whereby unwanted particles can be cleared from the joint. However, a central role of the synoviocytes is the secretion of proteins that contribute to both the anabolic and the catabolic metabolism of the joint as a whole. Like many other metabolic processes, when checks and balances are in place, these metabolic processes are part of normal turnover, but when unregulated or aberrant regulation occurs, these same processes can lead to the development of disease. The synoviocytes are responsible for the secretion of a diverse array of proteins that contribute to the synovial membrane proper, as well as to the composition of the synovial fluid, and they include hyaluronan, collagen, lubricin, pro-matrix metalloproteinases (pro-MMPs), interleukins, and eicosanoids (e.g., prostaglandin E_2).[7,8] The breadth of these proteins in the pathophysiology of the joint underscores the importance of this tissue.

Another important role of the synovial membrane is to *regulate the composition of synovial fluid.* Synovial fluid is often referred to as an ultrafiltrate of the plasma. This term implies a more passive process, but it is actually very dynamic and inter-related with many factors, including the molecules composing synovial fluid (e.g., hyaluronan size and concentration), degree of inflammation, and lymphatic drainage. Components of the plasma, such as glucose, oxygen, carbon dioxide, and proteins, typically less than 10 kDa in size, are allowed through the endothelium of the subintima and contribute to the composition of the synovial fluid, whereas large molecules are excluded. Routine cytologic examination techniques, as well as normal and disease-state levels of the constituents, are reported later. Most laboratories consider levels of less than 500 cells per deciliter of synovial fluid normal.

Molecules such as hyaluronan and lubricin are contributed to the synovial fluid directly by the synoviocytes. These molecules are thought to be important in the *steric exclusion* (i.e., steric hindrance: a process of separation of molecules by size) of larger molecules from the synovial cavity, thus regulating synovial fluid composition (Figure 78-3), and as *boundary lubricants* of the joint surfaces. The regulation of size and concentration of hyaluronan within the synovial fluid is unknown, but these two factors do affect the functional properties assigned to the molecule. A variety of methods have been used to determine both the concentration and the molecular weight (or size) of hyaluronan. The diversity of methods has provided a

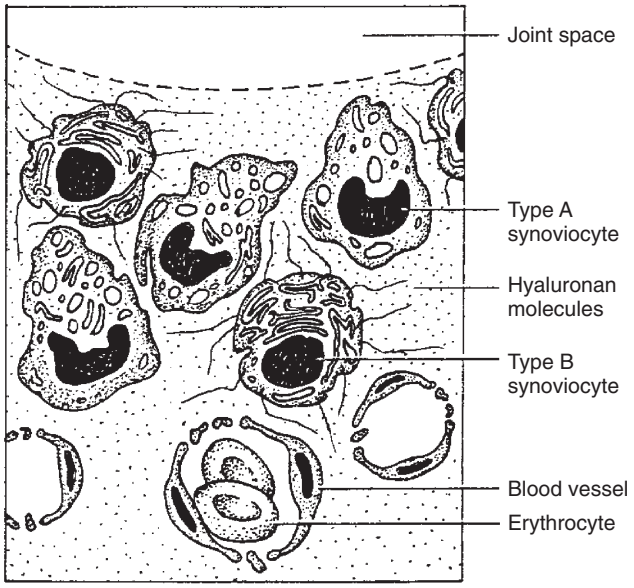

Figure 78-3. Hyaluronan secretion from type B synoviocytes, and its close association with the synovium, where it is thought to aid in excluding leukocytes and other solutes from the joint space through steric hindrance. (Adapted from Howard RD, McIlwraith CW: Hyaluronan and Its Use in the Treatment of Equine Joint Disease. p. 257. In McIlwraith CW, Trotter GW [eds]: Joint Disease in the Horse. Saunders, Philadelphia, 1996.)

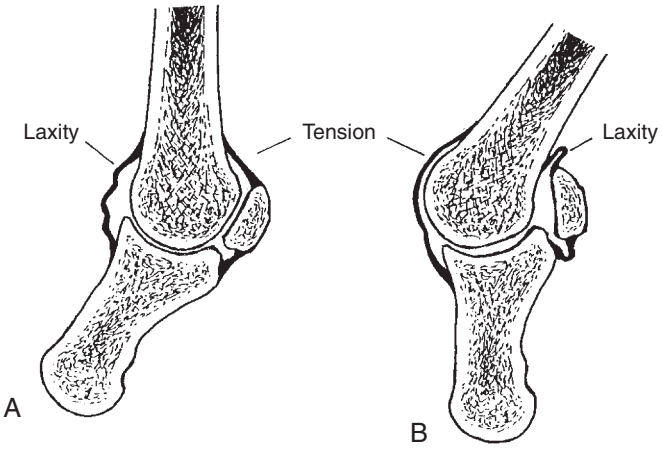

Figure 78-4. Synovial and joint capsule laxity or tension based on the anatomic position of the joint. The dorsal aspect of the joint capsule is lax in fetlock extension **(A)** and under tension in flexion **(B).** This figure also illustrates the change in joint congruity thought to occur during loading. In an unloaded position **(B),** it is thought that joints are not completely congruous, but as load is applied, a more congruous joint is formed. (Adapted from McIlwraith CW: Diseases of Joints, Tendons, Ligaments, and Related Structures. p. 459. In Stashak TS [ed]: Adams' Lameness in Horses. 5th Ed. Williams & Wilkins, Philadelphia, 2002.)

wide range of normal values, 0.33 to 1.5 mg/mL and several thousand kilodaltons, respectively.[7,9] The half-life of endogenous hyaluronan has been estimated by injecting radiolabeled hyaluronan into the joint and tracking both its degradation and its clearance. It appears that the majority of exogenous hyaluronan is cleared within 48 hours through the synovium and lymphatics, with degradation occurring in both the synovium and the liver.[7]

Periarticular Ligaments and Joint Capsule

Periarticular ligaments, the fibrous joint capsule, and surrounding muscles provide stability to synovial joints. The contribution to stability afforded by each of these structures differs according to its anatomic location. Joints higher in the limb obtain more stability from muscle than those lower in the limb, so the contour of the joint surface and stability from the joint capsule are more important in the lower limb, although muscular stabilization is a factor. The role of muscular stabilization cannot be overemphasized. For example, even with all periarticular ligaments and the joint capsule intact, desensitization of the suprascapular nerve results in subluxation of the shoulder joint.

The ligamentous and capsular configurations within a joint are different depending on the specific joint and the location within the joint, presumably based on range of motion and other biomechanical considerations. For example, in extension, the dorsal aspect of the metacarpophalangeal joint has capsular redundancy, but in flexion, the same region of the capsule is under tension (Figure 78-4).[10,11] It is easy to see how a decrease in compliance of the joint capsule because of increased fibrous tissue deposition could limit the range of motion within a particular joint.

The ligaments and fibrous joint capsule are composed mainly of type I collagen with some elastin fibers, accounting for 75% of the organic solids, with 23% being proteoglycan.[12] The organization is mainly in parallel fascicles with occasional fibrocytes and blood vessels. Most of the innervation of a joint is seen in association with perivascular structures.[10] The attachment of these structures to bone has been compared to Sharpey fibers, because the collagen fibers course through both fibrocartilage and calcified cartilage before their insertion in bone. This type of transition is credited with the biomechanical pullout properties (the amount of force needed to pull a ligament from the bone attachment) of the ligaments and joint capsule.[13] It is important to note that these tissues are metabolically active and undergo hypertrophy with activity and atrophy with immobilization. Immobilization has been shown to affect the microscopic, enzymatic, and biomechanical properties of ligaments, suggesting that immobilization should be used temporarily to maintain joint stability during a controlled return to use after injury, as is used in human medicine in the form of function splints (knee braces).[14]

Subchondral Bone

The subchondral bone plate, along with epiphyseal bone, provides contour and stability to the articular cartilage. The subchondral bone plate consists of *cortical bone with the haversian system running parallel to the joint surface*, unlike the diaphyseal cortical bone, where the haversian system runs perpendicular to the joint surface. The biochemical and histologic appearance of subchondral bone is similar to bone in other locations. In humans, the subchondral bone plate has been shown to be approximately 10 times more deformable than the cortical shaft.[10] Remodeling or stiffening of the subchondral bone plate

has been noted to occur with osteoarthritis in humans and is considered deleterious to the joint function.[15] This has sparked similar exploration in horses, and although remodeling and subchondral bone sclerosis is easily documented, especially in areas such as the third carpal bone, deleterious changes in proteoglycan content in this location have not been documented to date.[16] However, subchondral bone microcracks and other forms of bone remodeling have been demonstrated in this area in response to exercise, although their relationship to pathophysiology is not well defined.[17] The subchondral bone plate is integral to joint function as a whole; research in this area is quite active and may provide insights into its role in disease.

Articular Cartilage

Articular cartilage is the central structure that constitutes the joint surface and ultimately its function. In conjunction with the synovial fluid, articular cartilage is credited with providing the nearly frictionless movement of the synovial joint. Although other components of the joint, such as the subchondral bone, synovial membrane, joint capsule, and ligamentous structures, are integral for proper function and continued health of the joint, it is the state of the articular cartilage that is typically used to define joint health. Furthermore, although it is likely that disease may start in components of the joint other than the articular cartilage (e.g., subchondral bone or ligamentous structures), the degeneration and slow or poor healing response of the cartilage usually defines the level and progression of the joint disease.

On gross appearance, the articular cartilage appears smooth or glasslike (hence the term hyaline). In most areas, the articular cartilage has an opaque white color, with areas of thin articular cartilage appearing slightly pink because of the underlying subchondral bone color showing through. Although grossly the surface of articular cartilage appears flawless, a scanning electron microscope reveals it to be gently undulating with depressions that appear to correlate with interchondrocyte spaces immediately under the articular cartilage surface. These depressions have been estimated to be 20 to 40 μm in diameter and have a density of about 430/mm^2.[10] The thickness of articular cartilage varies by joint, location (related to the degree of weight bearing), and age, but it is in the 1- to 4-mm range.

The lack of vascular, lymphatic, and neural supply makes cartilage a unique tissue and dictates a *dependency on diffusion for nutrient supply and waste removal.* The nutritional supply is provided via the synovial fluid. The nutritional solutes diffuse from subintimal vessels through the intimal layer of the synovium to the synovial fluid, then through the articular cartilage matrix, until they finally reach the chondrocytes. The ultrastructure of the chondrocytes supports a diffusion gradient; for example, a decrease in the number of mitochondria in deeper chondrocytes suggests that they are less dependent on oxygen than more superficial chondrocytes.

The chondrocyte content of articular cartilage typically accounts for 1% to 12% of the volume of the cartilage, with the remaining component being termed extracellular matrix.[7] The *extracellular matrix* is somewhat complex but can be broken down into three main components: collagens, proteoglycans, and water. Water is the most abundant molecule of the extracellular matrix, making up about 70% of the wet weight in adults and closer to 80% in young and immature animals. On a dry weight basis, the extracellular matrix is composed of approximately 50% collagen, 35% proteoglycan, 10% glycoprotein (e.g., growth factors, cartilage oligomeric protein [COMP], proteinases), 3% minerals, 1% lipids, and 1% miscellaneous substances.[7]

Microscopic Appearance

On the basis of the microscopic appearance of both the chondrocytes and collagen orientation, articular cartilage has been historically divided into four contiguous zones (Figure 78-5):

1. The *superficial (tangential) zone* has the highest density of chondrocytes. These chondrocytes appear flattened and are oriented with the long axis of the cell parallel with the joint surface. The collagen here is more densely packed than in deeper layers, and the orientation of the fibers is also parallel

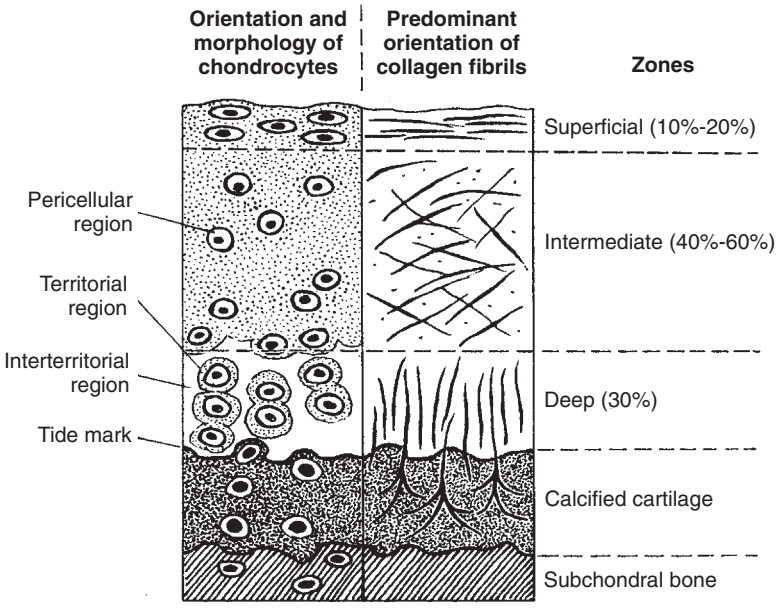

Figure 78-5. Normal articular cartilage with orientation and morphology of the chondrocytes, orientation of the collagen fibrils, and the relative contribution of the zone or layer. (Adapted from March L: Articular Cartilage in Health and Disease. p. 86. In Sambrook P [ed]: The Musculoskeletal System. Churchill Livingstone, New York, 2001.)

with the joint surface. There now is acceptance of a previously described but refuted acellular layer of collagen called the *lamina splendens.* This layer has fibril diameters that are distinctly different from those of the traditional cellular portion of the superficial zone and that can be consistently separated from the cellular portion of the superficial zone without damage to the collagen network. The lamina splendens is only loosely connected to the fibrous structure in the adjacent layer beneath. It is, however, firmly connected to the deeper cartilage matrix in the vicinity of the synovium, where it undergoes transition to synovial tissue.[18]

2. The *intermediate (transitional) zone* is characterized by larger, more ovoid to round chondrocytes.
3. The *deep (radiate) zone* has the largest chondrocytes, and the long axis of the cell is oriented perpendicular to the joint surface.
4. The *calcified zone* consists of mineralized cells and matrix. The *tidemark* refers to the junction of the noncalcified and calcified cartilage, which is a distinct transition seen on histologic sections. More recently, the tidemark has also been described arthroscopically.[19]

Cartilage Collagens

Collagen is considered the component of articular cartilage that provides the framework or lattice in which all other matrix molecules are constrained. It is also the component attributed to the counteraction of tensile stresses seen at the joint surface. As described earlier, the architecture of the collagen differs depending on its zone or depth within the cartilage. In 1925, Benninghoff described an arcade configuration of articular cartilage collagen, which is helpful in understanding the function of the cartilage. Although the overall accuracy of his model has been challenged, it is useful for demonstration purposes (Figure 78-6).[1] The superficial pattern of the collagen framework was described as "armor plating," referring to the tough, resilient, skinlike cartilage surface. The collagen content in this area is higher and the fibrils have a smaller diameter (about 31 nm) and an orientation parallel with the joint surface, which is different from collagen in deeper zones. Based on this configuration, minute openings exist in the superficial layer of cartilage, which have been calculated to be around 6 nm in diameter. This pore size would preclude large proteins such as hyaluronan from entering the cartilage from the synovial fluid in any significant quantities. However, small ions and molecules such as glucose could readily pass through an opening of this size.[20] As previously mentioned, the collagen fibrils of the intermediate zone are oriented in a more random pattern and are between 40 and 100 nm in diameter. The collagen fibrils of the deep zone have the largest diameter, are oriented perpendicular to the joint surface, and cross the calcified zone before attaching within the subchondral bone. This pattern of collagen orientation is supported by light and electron microscopy, with further confirmation by an interesting phenomenon called the *Hultkrantz line,* which appears when the articular surface is penetrated with a pin and stained with India ink.[21] The round hole made by the pin appears as an elongated slit, and the axis of the slit has been found to run perpendicular to the collagen fibril orientation and to the maximal surface tensile strains.[22] This method is currently the preferred test to determine the orientation of collagen in articular cartilage (Figure 78-7).

At least 16 different collagens have been described in mammalian species, and they are divided into two main categories on the basis of their primary structure and supramolecular assembly: *fibril-forming collagens* (including types I, II, III, V, and XI) and *non–fibril-forming collagens.*[23] Collagen turnover is much like that of other fibrous connective tissues, but it tends to be slower than that of other matrix components of the articular cartilage. It has been shown that significant collagen synthesis occurs in adult cartilage, although not much is understood about the process by which orientation is controlled in response to remodeling.

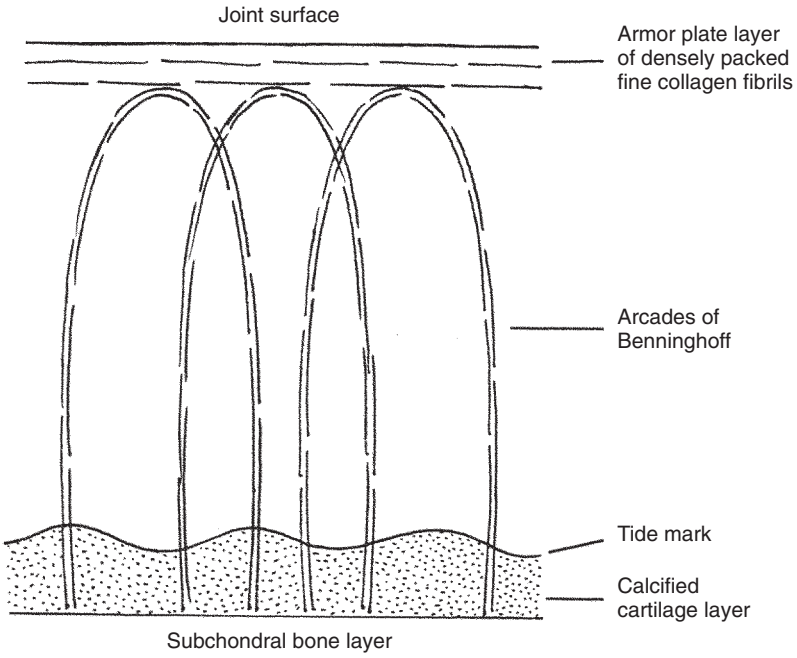

Figure 78-6. Simplistic illustration of the biomechanical function of the collagen fibrils in the different areas of articular cartilage. The tightly packed, smaller-diameter collagen fibrils are near the surface in the superficial layer (armor plate layer), notably in the lamina splendens. The larger-diameter, more loosely spaced fibrils of the deep layer (forming the arcades of Benninghoff) traverse the calcified layer and are anchored in the subchondral bone. (Adapted from Daniel DM, Pedowitz RA, O'Connor JJ, et al [eds]: Daniel's Knee Injuries: Ligament and Cartilage Structure, Function, Injury, and Repair. 2nd Ed. Lippincott Williams & Wilkins, Philadelphia, 2003.)

TYPE II COLLAGEN

The primary collagen of articular cartilage is type II and is produced by the chondrocytes. It constitutes 90% to 95% of the total collagen content.[24] The principal differences between type II and type I collagen (the latter found in the majority of other musculoskeletal tissues) are the number of hydroxylysine molecules and an increase in glycosylation. Type II collagen is a homotrimer composed of three identical collagen molecules that are constructed in such a manner that the start of each molecule is offset from the next by 25%, or by a "quarter stagger" (see Figure 80-3). Of the other types of collagen found in articular cartilage, types IX and XI are thought to be integral in the organization and mechanical stability of the type II collagen fibrillar network (Figure 78-8) and are intimately associated with type II collagen.

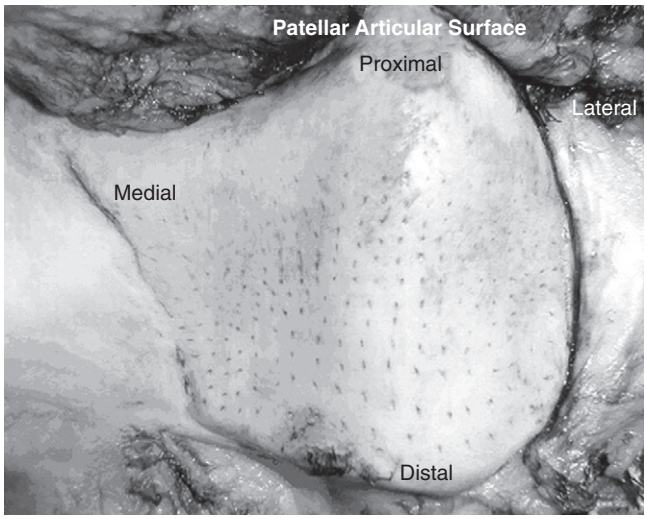

Figure 78-7. Gross photo demonstrating Hultkrantz lines in an equine patella following round perforations of the articular cartilage. The orientation of the slits after pin prick of the surface runs perpendicular to the orientation of collagen fibril. (Courtesy Dr. Chris E. Kawcak and Dr. Chad Lewis.)

MINOR COLLAGENS

The content of *type IX* collagen in articular cartilage decreases from being around 10% of the collagen protein in fetal tissue to being about 1% in the adult. This molecule is found on the outer surface of the type I triple helix and is thought to provide a covalent interface between the surface of the type II collagen fibril and other type II fibrils as well as the interfibrillar proteoglycan domain, thus providing mechanical stability of the fibrillar network.[1] Types VI, XII, and XIV collagen also are thought to have roles in the association between fibrillar collagen and other matrix components.[25,26] *Type XI collagen*, on the other hand, is found within the type II triple helix and acts as a core filament on which the type II molecules are deposited during formation.[27] Type XI collagen accounts for about 3% of mature collagen protein. Type VI collagen binds both hyaluronan and fibronectin and has been identified in the perilacunar area.[1]

Proteoglycans

Proteoglycans make up the other major component of the articular cartilage extracellular matrix, about 35% of the dry weight. As the name implies, these molecules are a combination of protein (*proteo* for the core protein to which glycosaminoglycans are attached) and glycosaminoglycan (*glycan* for the repeating disaccharide or polysaccharide). At first glance, the nomenclature of proteoglycans appears somewhat complex, but when broken down into its components it is relatively straightforward, and it is important to understand its evolution. The proteoglycan molecule aggrecan makes up about 85% of the proteoglycans that in turn make up the extracellular matrix, and it is mainly responsible for providing the resistance to compressive forces within the articular cartilage. The other proteoglycans are less studied but function in interactions with collagens and in various metabolic roles in cartilage.

AGGRECAN

A single (monomer) aggrecan molecule is made up of two main components: the core protein, which acts as the backbone of aggrecan, and the glycosaminoglycans (GAGs) that attach

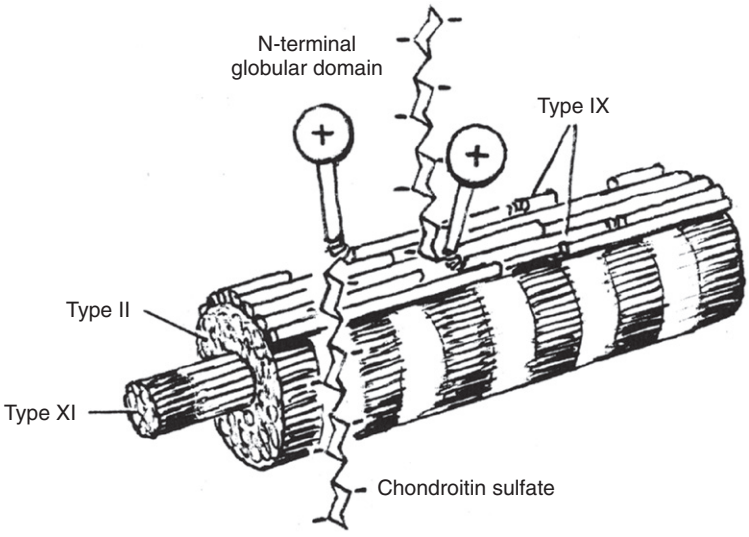

Figure 78-8. Arrangement of types II, XI, and IX collagen in articular cartilage. (Adapted from Eikenberry EF, Mendler M, Burgin R, et al: In Kuettner KE, Schleyerbach R, Peyron JG, et al [eds]: Articular Cartilage and Osteoarthritis. Raven Press, New York, 1992.)

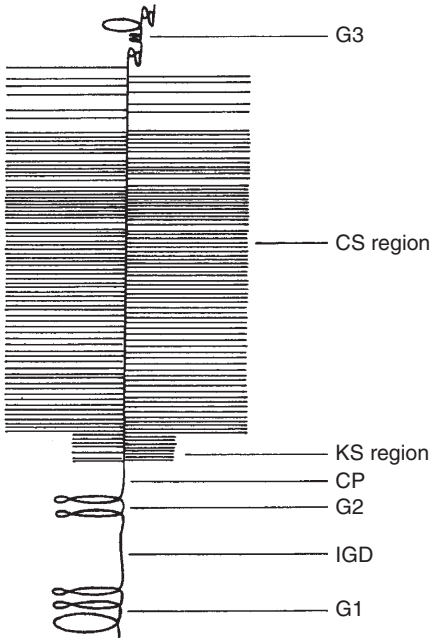

Figure 78-9. An aggrecan monomer consisting of a central core protein *(CP)* interrupted by three globular domains *(G1, G2,* and *G3).* Peripherally on the molecule are the keratan sulfate *(KS)* and chondroitin sulfate *(CS)* regions. Proteolytic cleavage of the molecule *in vivo* first occurs in the interglobular domain *(IGD).* (From Koopman WJ [ed]: Arthritis and Allied Conditions: A Textbook of Rheumatology. Vol 1, 13th Ed. Williams & Wilkins, Baltimore, 1997.)

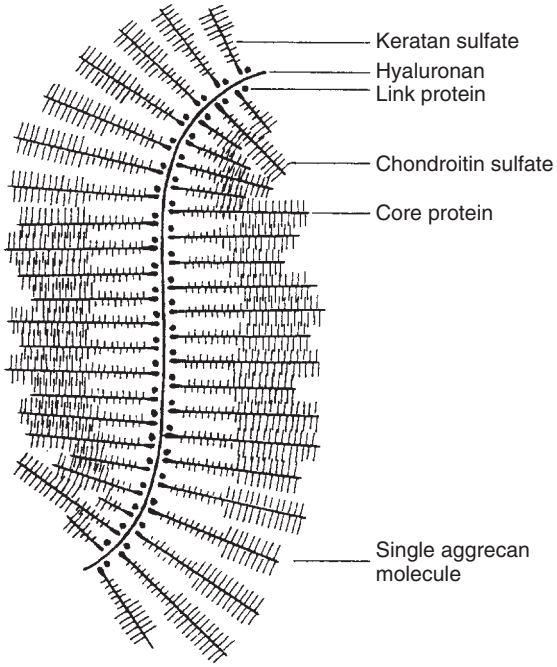

Figure 78-10. An aggregate containing many aggrecan molecules that are linked to hyaluronan via a noncovalent bond associated with the link protein. (From Rosenberg L: Structure of Cartilage Proteoglycan. In Simon WH [ed]: The Human Joint in Health and Disease. University of Pennsylvania Press, Philadelphia, 1978.)

radially to the core protein. The three main GAG molecules (Figure 78-9) that make up aggrecan are chondroitin-4-sulfate, chondroitin-6-sulfate, and keratan sulfate. Their ratio in cartilage differs with age. Of note is an increase in chondroitin-6-sulfate over chondroitin-4-sulfate as the cartilage becomes more mature. The GAGs covalently attach to the core protein, and about 100 chondroitin sulfate molecules preferentially attach at the carboxy end of the molecule. Likewise, approximately 100 keratan sulfate molecules are preferentially found closer to the N-terminal (amino-terminus) region of the aggrecan monomer.[11] These regions are sometimes described as chondroitin- or keratan-rich regions. Because of the high negative charge associated with the GAGs, they tend to repel each other (chemical expansive stresses) and attract water, creating positive pressure (Donnan osmotic pressure). The combination of these two forces has been termed *cartilage swelling pressure* and is roughly the equivalent of tire pressure.[28]

Current research has focused on delineating various functional and structural domains along the core protein. These regions or domains have been shown to help define the breakdown of various catabolic enzymes involved in normal and pathologic processes—for example, the cleavage site of *aggrecanase* is different from that of *stromelysin*, two important molecules associated with aggrecan turnover. Three main domains have been defined: G1, G2, and G3. The G1 region of the core protein interacts with hyaluronan, and this interaction is involved in arranging about 100 aggrecan monomers along a hyaluronan backbone. The G2 region is located on the N-terminal side of the keratan sulfate–rich region and is a key target of enzymes that break down aggrecan.[29] The G3 region is on the "end" or carboxy side of the aggrecan molecule and in fact is present in only some aggrecan molecules, suggesting a less-important role.[30]

AGGRECAN (PROTEOGLYCAN) AGGREGATES

Aggrecan monomers are further organized into large aggregates around a hyaluronan molecule. These aggregates can be in the range of 200 million daltons in size and comprise over 100 aggrecan monomers.[31] The noncovalent bond between the aggrecan monomers and hyaluronan is stabilized by a small group of link proteins in conjunction with the G1 domain of the core protein (Figure 78-10).[1]

SMALL PROTEOGLYCANS

About 5% of the articular cartilage proteoglycans are termed *small proteoglycans* or *nonaggregating proteoglycans.* Like aggrecan, they consist of a protein core and attached GAG chains. As the name implies, these molecules are smaller than aggrecan—in fact, the core protein is about 25% of that found in an aggrecan molecule. Examples are biglycan, decorin, and fibromodulin. Decorin and fibromodulin are found associated with collagen molecules in the superficial zones of the articular cartilage. *Decorin* has been shown to inhibit type I and II collagen formation, and *fibromodulin* inhibits fibrillogenesis. Biglycan and decorin have been shown to bind transforming growth factor-beta (TGF-β), thus making it unavailable to perform its biologic function. This may play an important role in the healing process of articular cartilage and needs to be better understood to improve intervention in cartilage healing.[1] The overall organization of the extracellular matrix is presented graphically in Figure 78-11.

Chondrocytes

The chondrocytes, as previously mentioned, represent a small proportion of the total volume of the articular cartilage. The morphology and metabolism of the chondrocytes vary

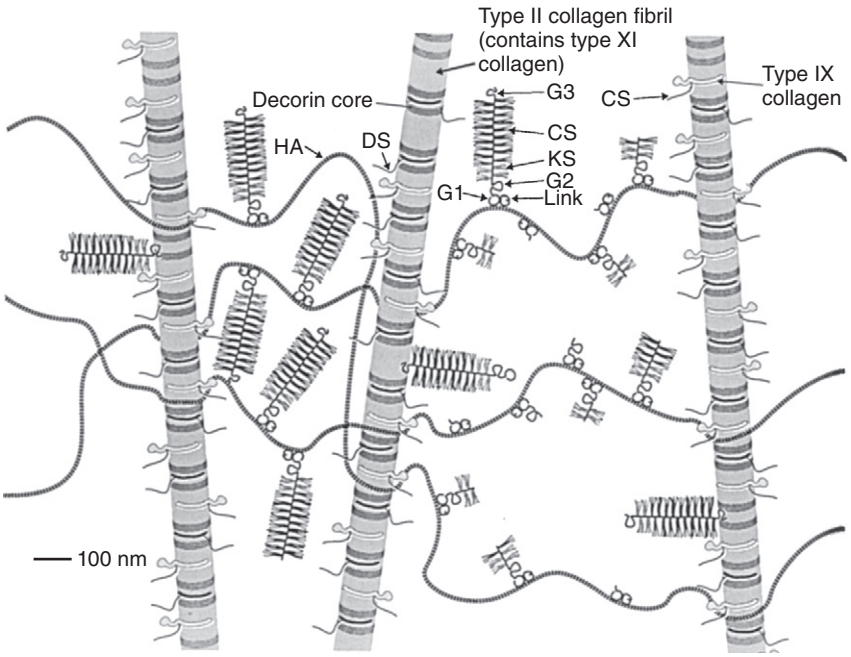

Figure 78-11. Organization of the major extracellular matrix components in articular cartilage. The principal collagen of cartilage is type II, and a network of these fibrils provides much of the tensile strength of the tissue. Aggrecan consists of a linear protein with three globular domains *(G1* to *G3),* to which are attached numerous glycosaminoglycan chains of chondroitin sulfate *(CS)* and keratan sulfate *(KS).* Supramolecular aggregates are formed by the noncovalent interaction of aggrecan with hyaluronan *(HA)* and stabilized by a link protein *(Link).* The negatively charged glycosaminoglycans *(CS* and *KS)* attract several times their weight in water, and this proteoglycan–water composite is responsible for the compressive stiffness of cartilage. Cartilage also has a number of minor proteoglycans and collagens (e.g., decorin and dermatan sulfate [DS]) whose functions are not fully characterized. Fragments of aggrecan, remaining bound to HA, are shown to illustrate the effects of proteolytic activity in cartilage. (From Koopman WJ [ed]: Arthritis and Allied Conditions: A Textbook of Rheumatology. Vol 1, 13th Ed. Williams & Wilkins, Baltimore, 1997.)

according to their depth within the cartilage, and the presence of lacunae represents a microenvironment around the chondrocytes that is more prevalent in the deeper layers. Chondrocytes have cytoplasmic processes that extend into the interterritorial region and appear to sense the biochemical and biomechanical environment, but, unlike the cytoplasmic processes of osteocytes, they do not make contact with cellular processes of other chondrocytes. These cytoplasmic processes may represent one mechanism through which the chondrocytes respond to *articular cartilage loading.* It appears that at a low level of mechanical stress, chondrocyte metabolism favors the catabolic processes, whereas at normal physiologic levels, anabolic processes are favored, and a balance of metabolism is possible. In contrast, at superphysiologic levels, the metabolism favors catabolism, potentially as a result of an overwhelmed anabolic pathway.[32] Alterations in extracellular matrix pressure caused by biochemical changes may also direct chondrocyte metabolism.

Turnover of the proteoglycan portion of the extracellular matrix occurs faster than in the collagen component. Specifically, proteoglycan turnover has been estimated at about 300 days in dogs and rabbits, versus an estimated 350 days for total turnover of the collagen in humans and 120 days in dogs.[11] These rates can be upregulated in disease states—in fact, upregulation of anabolism of both aggrecan and collagen synthesis appears to be one of the earliest changes detected in osteoarthritic processes.[33] More detail on cytokines and regulation of articular cartilage metabolism are covered later.

PHYSIOLOGY OF SYNOVIAL JOINTS

Intra-articular Volume and Pressure

The volume of the synovial space or joint cavity varies with its anatomic location, and, because of the elasticity of the joint capsule, it can change depending on various factors (e.g., disease, level of exercise). As an example, horses exercising at an increased level are often noted to have synovial effusion, notably in the fetlock and tarsocrural joints, without pathology being present. Some data on the "normal" synovial volume have been published, but, as previously noted, this can be somewhat subjective depending on the degree of joint capsule distention that is perceived as typical.[34] The mechanisms by which intra-articular volume and pressure are regulated are poorly understood. Most synovial joints are reported as having negative pressure in a neutral position, whereas at the extremes of flexion and extension the intra-articular pressure increases up to 30 psi in the human knee. These pressures are also sensitive to synovial effusion; increased pressures can be translated to the innervation of the joint capsule and subsequently to joint pain during range-of-motion activities.

Joint Mechanics

Joint mechanics are unique and very complex, largely because of the articular cartilage and its response to loading. The study of the loading properties of synovial joints is a very active field whose scope is beyond the reach of this chapter, but it is summarized here. Joint mechanics depend on three aspects: kinematics, kinetics, and joint lubrication. *Kinematics* involves the study of the motion of the articulating surfaces in relation to each other. *Kinetics* relates to the forces that are created during the motion of the joint and the loads that are created across the articular surface. *Joint lubrication* provides a nearly frictionless movement of the soft tissues and articular cartilage.

Kinematics

The kinematics of a particular joint are a function of the geometry of the joint surface—specifically, of the articular cartilage and the underlying subchondral bone, as well as the periarticular supporting structures such as ligaments and muscles. The types of motion that occur in joints fall into three categories: translational, rolling, and sliding motions. *Translational motion* refers to movement without rotation, where two surfaces move past each other. This type of motion somewhat describes the movement in the spine and shoulder. Both rolling and sliding

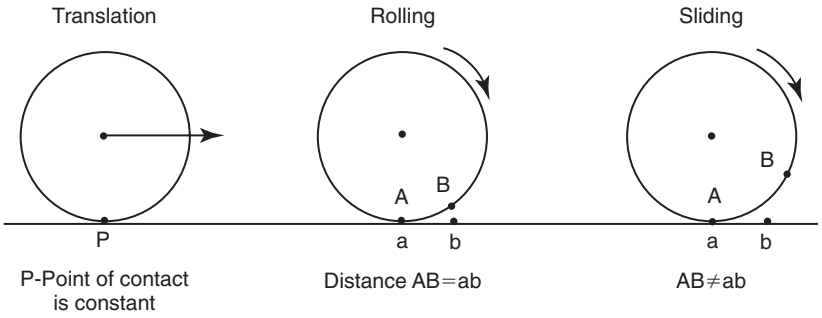

Figure 78-12. The three types of joint motion. (Adapted from Delahay JN: In Wiesel SW, Delahay JN [eds]: Principles of Orthopaedic Medicine and Surgery. Saunders, Philadelphia, 2001.)

imply some degree of rotational component, which adds another dimension and makes the mathematical models and understanding of these motions complex. Pure *rolling motion* implies that the instant center of rotation is always at the point of contact between the two surfaces and that the distance over which contact occurs is similar on both opposing surfaces. In *sliding motion*, the point of contact is not the instant center of rotation, and the areas of contact during the motion are not equal on both surfaces (Figure 78-12). Most synovial joints have some component of sliding and translational motion, and pure rolling motion is just theoretical.

Another factor that affects the kinematics of synovial joints is the observation that joint surfaces that are relatively congruent possess thinner articular cartilage than those with incongruent surfaces. Furthermore, in some joints, overall joint congruity appears less when they are unloaded than when they are under load. This is thought to allow better distribution of forces over a larger surface with increasing loads (see Figure 78-4).[10] It becomes quickly apparent that loss of any component of the synovial joint will alter the normal motion of a joint and its ultimate function.

Kinetics

The analysis of joint forces is even more complex, because it depends on the kinematics of the joint as well as on any change of normal position, which then alters the forces as they are applied to the joint. Kinetics takes into account the muscle forces pulling across a joint, the superimposed body weight, the force of the floor or surface pushing back against the joint in question, and the force felt by the cartilage in any given load-bearing situation (termed the J-force).[35] Joint modeling has been a focus in human research for some years and is currently being conducted in the equine carpus. This may provide valuable insight into the joint as a whole organ in the years to come.

Lubrication

Lubrication of the synovial joint can be broken down into two main components, that of the soft tissue and that of the articular cartilage. From a pure point of view, two types of lubrication function in the synovial joint: boundary and fluid-film lubrication. *Boundary lubrication* prevents adhesion and abrasions of two surfaces and is independent of the physical properties of the lubricant or contacting surfaces. Hyaluronan and lubricin (secreted by the synovial membrane) are believed to be the main boundary lubricants of the diarthrodial joint. Boundary lubrication is the main way of decreasing friction between soft tissue and bone, but it is believed to be relatively ineffective at providing a frictionless surface between articular cartilage surfaces at physiologic loads.[35]

Fluid-film mechanisms are believed to provide the low friction environment for the articular cartilage at physiologic loads, accomplished by creating a wedge of fluid between the two bearing surfaces. Several models have been proposed, including squeeze film, hydrodynamic, and elastohydrodynamic models (Figure 78-13). The *elastohydrodynamic model*, which is accepted as best representing lubrication of the articular cartilage, is based on the attraction of water by aggrecan molecules at the porous articular surface. In this model, when load or pressure is applied to the surface, water is squeezed from the articular surface and interposed between the bearing surfaces. With joint movement, a wedge of fluid is created, the leading edge of which is wider and exudes fluid, and at the thinner trailing edge, fluid is thought to be resorbed. Stated another way, as load is applied to the bearing surface of the articular cartilage, fluid is squeezed from the cartilage, separating the surfaces, and is resorbed as the load decreases on the back side of the motion.[35] The material properties responsible for the fluid flow in the articular cartilage have been studied in detail.

An air tent (like those used to cover tennis courts) has been used as an analogy to understand the biomechanical function of the articular cartilage as it relates to the components of the cartilage matrix and fluid flow.[1] The components required for the proper function of the air tent are a pump (intake portal and fan), an inflation medium (air), and some form of fabric enabling containment of the inflation medium, thus creating a pressurized system. These tents are engineered to have small openings in the fabric, which require the pump to move more air inward than is escaping to keep the tent inflated. In articular cartilage, the pump corresponds to the proteoglycans; their ability to attract solutes based on their strong negative charge is the driving force of the pump. The inflation medium is solutes from the synovial fluid made up mainly of water, and the containment material, or fabric, is the network of collagen. Instead of entering through a single intake port of a mechanical pump, solutes gain access to the articular cartilage through the fine network of pores formed in the superficial layer of collagen, and likewise these molecules exit via these pores. Both systems (air tent and articular cartilage) depend on all components functioning appropriately and in unison. For example, if a tear in the containment material is too large, the pump cannot continue to pressurize the tent and the system collapses; likewise, if the pump fails, the normal egress of inflation media ultimately ends in a collapse of the tent.

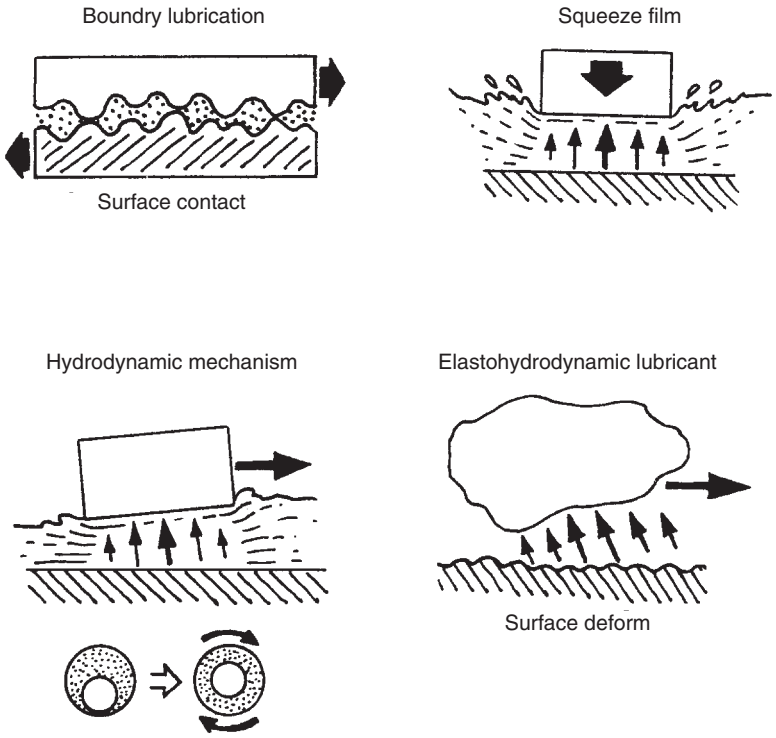

Figure 78-13. Models of lubrication: boundary, squeeze film, hydrodynamic, and elastohydrodynamic (a combination of squeeze film and hydrodynamic). (Adapted from Delahay JN: Basic Science. p. 1. In Wiesel SW, Delahay JN [eds]: Principles of Orthopaedic Medicine and Surgery. Saunders, Philadelphia, 2001.)

In a state of equilibrium, the articular cartilage maintains a balance of pressure created by the interaction of aggrecan and water that is counteracted by the constraints of the collagen network. When an external load is applied to the articular surface, water flows out of the surface pores until a new equilibrium is reached. This process is relatively complex and not linear in nature. For example, as water egresses out of the articular cartilage as a result of increased surface load, the proteoglycans are forced closer together and generate greater repulsive pressures as they come closer to each other, much like similar poles of magnets repel each other with increasing proximity. Such fluid movement in and out of the articular cartilage is of great interest, because it is integral to the lubrication of the joint surface, load-bearing functions of the articular surface, and nutrition of the chondrocytes.

The nutritional supply of the articular cartilage is thought to be provided through the exchange of solutes present in the synovial fluid during loading and unloading of the articular surface. It has been calculated that based on this type of supply, the articular cartilage was limited to 6 mm of thickness for chondrocyte viability to be maintained, a calculation that appears to be accurate in terms of synovial joints.[35]

The term *viscoelastic* is used to describe the biomechanical properties of articular cartilage, and as the name implies, it is a combination of viscous, or fluid, and elastic biomechanical properties. When a static load is applied in a laboratory setting, there is an initial rapid movement of water out of the cartilage in the fluid phase, followed by a slower loss of water and compression of the collagen known as the *creep phase*. A time–deformation curve is used to graphically represent the response of the cartilage to loading after the static application of a load (Figure 78-14). The loss of water and compression of the tissue, forcing the proteoglycans or aggrecans into closer proximity and repelling the forces of the proteoglycans from one another, also contributes to opposing the loading force in the creep phase. It has been estimated that 2- to 4-mm-thick cartilage can take 4 to 16 hours to reach creep equilibrium.[36]

PATHOPHYSIOLOGY OF OSTEOARTHROPATHY

Osteoarthropathy is a general term defined as any disease of the joints and bones. It is derived from the Greek words *osteo* ("bone") and *arthron* ("joint"). A more-specific term that relates to disease of the articular cartilage is osteoarthritis, defined as a disorder of movable joints characterized by degeneration and loss of articular cartilage. The suffix *itis* is somewhat misleading because it suggests a central inflammatory role, which is not present in many joint diseases of horses and humans. Human rheumatoid arthritis is a notable exception and does have a central inflammatory role. Some definitions of osteoarthritis even state that it is a noninflammatory disease process, which is also somewhat misleading because inflammatory factors do play a part in most arthropathies (defined as any joint disease). Because of these discrepancies, several terms are used to describe similar disease processes. Other common terms are degenerative joint disease (DJD), which circumvents these issues but is often not favored as a scientific term. *Osteoarthrosis* is also sometimes used, correctly not emphasizing the inflammatory component, but this is defined as a chronic condition, which is not always accurate. *Osteoarthritis* is, in my experience, the currently accepted term describing degeneration of the equine articular cartilage. Therefore this term is used in this chapter.

Many texts define specific mechanisms as being responsible for the pathogenesis of osteoarthritis. For example, one author describes three proposed mechanisms for osteoarthritis, the first being fundamentally defective cartilage with abnormal biomechanical properties. Thus the cartilage fails under normal

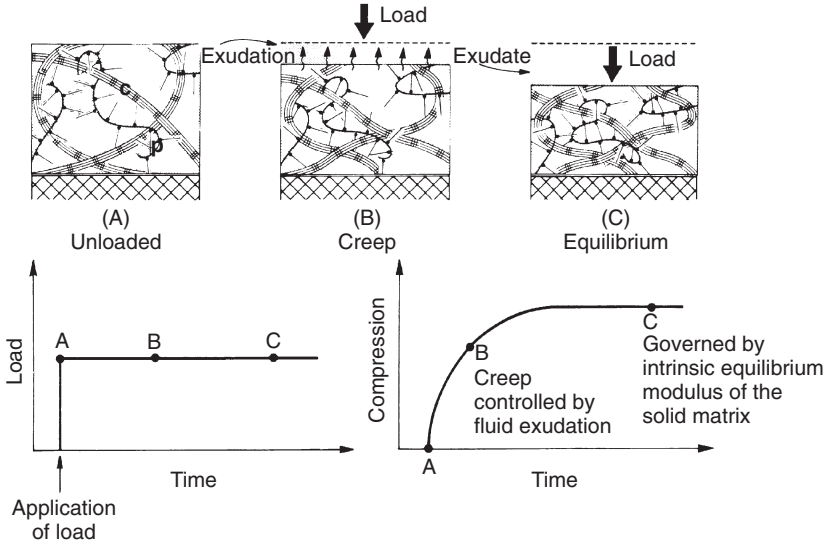

Figure 78-14. Schematic representation of time-dependent deformation of cartilage. The steep portion of the curve *(A* to *B)* represents initial loading. Subsequently, in the creep phase, there is further deformation of the matrix, which is a result of a slower flow of water through the matrix, until the equilibrium stage *(C)* is reached. (From Myers ER, Mow VC: Biomechanics of Cartilage and its Response to Biomechanical Stimuli. p. 313. In Hall BK [ed]: Cartilage. Vol 1. Academic Press, New York, 1983.)

loading conditions.[37] An example of *abnormal biomechanical properties* is the genetic defect described in humans that results in a type II collagen that is unable to withstand normal joint loading.[38] The second mechanism involves *abnormal change in the subchondral bone.* It is accepted that subchondral bone typically undergoes remodeling in response to exercise or to changes in load. It is thought that in some cases, the bone increases its density to a pathologic level, which in turn results in a stiffer or less compliant bone–cartilage unit that is prone to failure. The third proposed mechanism revolves around *normal cartilage that is exposed to abnormal forces*—for example, those that may be seen with abnormal joint congruity resulting from a collateral ligament strain. In this mechanism, abnormal forces overwhelm the normal metabolic repair mechanisms in the articular cartilage and ultimately lead to its failure.

Another author has also outlined discrete mechanisms whereby osteoarthritis may ensue, and many similarities exist.[8] More recently, he gave an example of a scenario that could lead to osteoarthritis and described how the different joint tissues may be involved, but he did not elaborate on previously defined mechanisms.[11] This trend for more general pathogenic descriptions is favored by the author. It is probable that most cases of equine joint disease have some involvement of most joint tissues: subchondral bone, articular cartilage, synovial fluid, synovium, ligamentous joint capsule, and external stabilizers of the joint, such as ligaments, tendons, and muscles. This is not to say that specific components do not play a leading role, and initial failure of a single component or tissue is not often the case, but rather each component has some capacity for repair, and clinical disease usually is characterized by involvement of multiple joint tissues in some degree of pathology.

Metabolism of Arthritic Cartilage

It is widely accepted that all joint tissues have normal metabolic turnover, and the turnover is a balance of anabolic and catabolic pathways. Osteoarthritis at its origin may be defined by the predominance of the catabolic pathway, which leads to the ultimate failure of the tissue. Two interrelated factors should be considered in the pathogenesis of osteoarthritis: abnormal mechanical loads and metabolic tissue failure. *Abnormal mechanical loads* may be in the form of mechanical instability that can overwhelm the normal repair process of the tissues it is acting on. It may range from microdamage because of slight imperfections in conformation, to a single traumatic event leading to immediate failure, often a catastrophic one. *Microdamage* is believed to accumulate over time, leading to failure of the tissue after a failed reparative effort, and it may be likened to microcracks in an airplane wing that ultimately lead to failure if not repaired in time. On the other end of the spectrum, immediate damage to the cartilage could occur with a single impact without chance for repair, such as an articular fracture caused by a traumatic event. *Metabolic tissue failure* revolves around normal tissue metabolism and the fact that all the tissues of the joint have some capability for normal turnover and thus repair. However, some tissues, such as bone, are very adept at repair, whereas others, such as articular cartilage, are less adept. Thus osteoarthritis could be defined as the point at which the anabolic repair processes are overwhelmed by the catabolic processes. As described earlier, the imbalance and failure can be initiated by mechanical forces or by an inherent defect caused by a genetic imperfection, such as a type II collagen defect. A continuum of disease severity and involvement of multiple joint tissues is often the case in osteoarthritis; the etiology is most likely multifactorial, and at some stage, it includes some degree of both abnormal mechanical loads and metabolic tissue failure. The current understanding for normal metabolism and, in the case of osteoarthritis, response to injury for each joint tissue is described next.

Mechanisms of Articular Cartilage Matrix Depletion

Synovial Membrane and Ligamentous Joint Capsule

The involvement of the synovial membrane and joint capsule in the pathogenesis of osteoarthritis is often not emphasized, with more focus being on the articular cartilage and bone. The synovium is a very dynamic tissue that has an integral role in influencing the joint environment, especially through the constituents of the synovial fluid, and thus it can have an especially

significant effect on the articular cartilage. The highly vascular nature of the synovium allows it to act as the conduit and regulator for inflammatory cells and peripheral mediators that influence metabolic processes, especially in the release of catabolic substances affecting the joint. Likewise, the synoviocytes themselves, much like the chondrocytes, are capable of producing a wide variety of anabolic and catabolic enzymes, including prostaglandins, cytokines, and MMPs.[8] *In vitro* experiments have shown the importance of mediators released specifically from the synovial membrane in regard to the degradation of articular cartilage. A limitation in determining the relative quantitative and temporal contributions of the synovial membrane to disease *in vivo* is our inability to identify the origin of the mediators *in situ* as being synovial or chondrocytic. To avoid redundancy, a more-detailed description of the mediators and their role in osteoarthritis is presented later, under "Chondrocytes."

Primary synovitis or *capsulitis* is thought to result from biomechanical damage, which may be on a continuum of repetitive trauma from discrete incidents. Severe trauma can lead to an effect on the joint capsule's role in joint stability and thus joint-wide abnormal biomechanical forces. The response of the joint capsule to injury is to form a fibrous repair tissue that is not biomechanically equivalent to the original tissue and can often result in a decreased range of motion for the particular joint. It is conceivable that this may have long-term ramifications of altered use of the joint and overall abnormal biomechanical forces to the joint tissues. Although the synovium is not thought to have any biomechanical stabilizing properties, it does respond to injury through cellular and enzymatic pathways. It is worth noting that synovitis and capsulitis can be secondary to other abnormalities in the joint. Synovitis may be secondary to other damage within the joint such as occurs with an osteochondral fragment in the carpus. In this example, physical damage to the tissues as well as liberation of debris and mediators from the damaged cartilage and bone will affect the synovial membrane and capsule. In concert with the lymphatic drainage of the joint, the synovial membrane is responsible for clearing debris from the synovial space. Neutrophils, macrophages, and mononuclear cells can also be routinely found in inflamed synovial membrane, contributing to both the phagocytic role of the membrane and the production of enzymatic mediators.

Histologic abnormalities seen in the synovial membrane can be characterized by edema, hyperplasia of the intimal cell lining, hypervascularity, cellular infiltration of inflammatory cells, and fibrosis of the subintima. The order in which these events appear loosely corresponds to the appearance of the abnormalities in a equine model of osteoarthritis and presumably in naturally occurring disease.[39] Changes documented histologically support alterations seen in other areas of the joint. For example, an increase in vascularity is typically associated with some degree of synovial effusion and an increased protein concentration in the synovial fluid. The increase in protein and fluid is thought to be associated with "gaps or leakiness" of the endothelial cell layer. Although ill defined, alterations in the lymphatic drainage of the synovial space is most likely a component of synovial effusion, with one proposed mechanism being an increased fluid pressure allowing less egress via the lymphatics. Intimal cell hyperplasia is also seen in cases of synovitis and is believed to be associated with a substantial metabolic response by the synoviocytes and most likely contributes to the central pathophysiologic events in osteoarthritis.

Chondrocytes

The joint chondrocytes are responsible for maintaining their surrounding environment through a complex interaction of anabolic and catabolic mediators as well as mechanical stimuli. In osteoarthritis, the net outcome favors the catabolic pathways, and degeneration of the articular cartilage ensues with *loss of the key matrix components, including aggrecan and type II collagen*. There has been a long-raging debate over the definition of the earliest sign of osteoarthritis as it relates to aggrecan or type II collagen loss (reminiscent of the chicken-or-egg argument). Both *in vitro* and *in vivo* studies have documented loss of aggrecan as an initial biochemical event.[33,40-42] Furthermore, it appears that upregulation of the synthetic pathways is one of the earliest metabolic manifestations, although the net loss of aggrecan and collagen prevail. The quality and quantity of both matrix components are affected, with an increase in water content and surface fibrillation also being early events in articular cartilage degeneration. This in turn affects the mechanical properties of the cartilage, making it less able to withstand normal loads, perpetuating the disease process, and inciting changes in other tissues such as bone and synovial membrane as the cartilage loses its functionality.

The proteolytic enzymes synthesized by the chondrocytes and synoviocytes, and their role in cartilage breakdown, have been extensively studied. They are described by their catalytic mechanisms and include MMPs, aspartic proteinases, cysteine proteinases, and serine proteinases, with the MMPs thought to be most involved in the pathogenesis of osteoarthritis.[8,43]

Matrix Metalloproteinases

The MMPs are grouped together by a requirement for zinc in their active binding site and are typically identified either by a descriptive name or by a numerical MMP assignment. To date, 25 different MMPs have been described.[44] The descriptive name is often based on the substrate the MMP was first observed to degrade, such as *collagenases*, *stromelysins*, and *gelatinases*. The MMPs are secreted in an inactive "pro" form and collectively possess the ability to degrade all of the major components in the articular cartilage; thus they play a major role in both health and disease of the articular cartilage. The synoviocytes and chondrocytes as well as other cell types are capable of MMP production. Increasing concentrations of MMPs have been correlated to areas of histologic abnormalities, signifying their active role in cartilage degradation.[45,46] The major MMPs that have been incriminated in osteoarthritis include collagenase 1 (MMP-1), collagenase 2 (MMP-8), collagenase 3 (MMP-13), stromelysin 1 (MMP-3), and two gelatinases (MMP-2 and MMP-9). A family of enzymes called "a disintegrin and metalloproteinase" (ADAM) is structurally and functionally related to the MMPs and is also a major factor in aggrecan cleavage. A summary of the nomenclature and function of the MMPs and related proteinases can be found in Table 78-1.

Interstitial or *tissue collagenase* (MMP-1) is able to break intact type II collagen molecules by cleaving all three collagen α-chains of the triple helix at a specific amino acid sequence (residues 775-776).[8] Collagenase 2 or human neutrophil collagenase (MMP-8) is released from polymorphonuclear leukocytes (PMNs) and was first shown to be active in human osteoarthritis and has been identified as having activity in equine cartilage.[47,48] Collagenase 3 (MMP-13) has been shown to be more

TABLE 78-1. Matrix Metalloproteinases (MMPs) Implicated in Cartilage Matrix Degradation

Enzyme	MMP Number	Function	TIMP Number
COLLAGENASES			
Interstitial collagenase (collagenase 1)*	MMP-1†	Collagens II and X (not IX and XI), denatured type II, aggrecan, link protein	TIMP-1 > TIMP-2
Neutrophil collagenase*	MMP-8	Collagen II, aggrecan, link protein	TIMP-1, TIMP-2
Collagenase 3*‡	MMP-13†	Collagens II, IV, IX, X; aggrecan; fibronectin	TIMP-3
STROMELYSINS			
Stromelysin 1*	MMP-3†	Aggrecan, fibronectin; denatured collagen II	TIMP-1 > TIMP-2
Stromelysin 2*	MMP-10	Collagens IV, IX, X, XI; procollagens; link protein; decorin; elastin; laminin and the function of stromelysin 1	TIMP-3
GELATINASES			
Gelatinase A (72 kD)*	MMP-2†	Denatured collagen II, collagens X and XI, elastin	TIMP-2 > TIMP-1
Gelatinase B (92 kD)*	MMP-9†	Aggrecan, fibroconectin, collagens IX and XI, procollagens, link protein, decorin, elastin	—

*Expressed by chondrocytes. All are expressed in synovium.
†MMPs characterized in the horse.
‡MMP-13 expression is relatively weak in equine system.
TIMP, Tissue inhibitor of metalloproteinase.

aggressive in type II collagen degradation, cleaving it 10 times faster than MMP-1. Also found in higher concentrations in diseased cartilage of horses and humans, MMP-13 synthesis is upregulated by interleukin-1 (IL-1) and tumor necrosis factor (TNF), key enzymes at the top of the articular cartilage catabolic pathway.[46,49] *Stromelysins* have been studied most notably for their ability to break down proteoglycans, but partially degraded collagen and other minor cartilage proteins can be substrates. Much of the credit for breaking down aggrecan in osteoarthritic cartilage has recently been given to members in the ADAM family of enzymes, specifically *aggrecanases*.[43] It is currently thought that aggrecanases play a pivotal role in proteoglycan depletion in diseased cartilage. The *gelatinases* also have a diverse range of substrates, including partially degraded collagens and elastins.[8]

Cytokines

Historically, the term *cytokine* denoted small regulatory proteins that were associated with catabolic pathways. More recently, the term has been broadened and is now considered to define catabolic, modulatory, and anabolic proteins that are produced by one cell while acting on another. Cytokine pathways are relatively complex, and more in-depth knowledge of these pathways, as well as their interactions with other molecules, is being published. These mediators play a pivotal role in the metabolism of the synovial membrane and articular cartilage in health and disease.

Most notable of the *catabolic cytokines* are IL-1 and TNF-α, which can be secreted from chondrocytes and synoviocytes. Both have been demonstrated to be upregulated beyond a normal level in osteoarthritis, promoting production of MMP, nitric oxide, and prostaglandin E_2 (PGE_2), as well as inhibiting aggrecan and type II collagen synthesis.[8] Although IL-1 and TNF-α have relatively similar actions, it is thought that IL-1 is the most important of the proinflammatory cytokines.[50] *In vitro*, the two molecules do appear to potentiate each other, and TNF-α has been shown to stimulate IL-1 activity.[51] In addition to having these effects, IL-1 has been shown to inhibit the production of natural antiarthritic molecules such as the MMP inhibitors (tissue inhibitor of matrix metalloproteinase, or TIMP) and interleukin-1 receptor antagonist (IL-1Ra), which utimately potentiates the catabolic cascade.

The *modulatory* or *regulatory cytokines*, such as IL-4, IL-10, and IL-13, have actions that balance or modulate the proinflammatory cytokines, namely IL-1 and TNF-α. They have been shown to inhibit the synthesis of IL-1 as well as to promote the synthesis of the natural inhibitors, specifically the TIMPs and IL-1Ra. IL-6 has a mixed mode of action that includes magnifying the effects of IL-1 while promoting synthesis of the TIMPs.[52]

Cytokines that promote the *anabolic* cascade of cartilage metabolism, such as insulin-like growth factor (IGF) and transforming growth factor (TGF), play a role in osteoarthritis. These molecules have been shown to promote chondrocyte production of matrix molecules such as proteoglycans and type II collagen. Thus anabolic cytokines can be helpful in reparative attempts in diseased joints. The use of anabolic cytokines in treatment protocols of osteoarthritis is an area of current research.[53,54]

Although not classified as cytokines, levels of *nitric oxide* (as well as other oxygen-derived free radicals) and *prostaglandins* are increased in joints with osteoarthritis. Free radicals play a role in the degradation of hyaluronan and collagen. Cells have been shown to produce nitric oxide in response to IL-1, and its production has been related to inhibition of chondrocyte anabolic activities. Association with activation of MMPs and the reduction in the natural inhibitors has also been observed with nitric oxide, although this molecule's specific role in osteoarthritis is still somewhat controversial.[55,56] Also associated with decrease in anabolic synthesis is the E series of prostaglandins. PGE_2 has been shown to be increased in the synovial fluid of horses with osteoarthritis and has been correlated with both synovitis and clinical lameness as well as being produced after stimulation with IL-1 and TNF-α *in vitro*.[8]

Natural Inhibitors of MMPs and Cytokines

The biological response to IL-1 and TNF-α occurs after the molecules bind to a specific receptor. Both molecules have at least one mechanism by which their activity is blocked by a natural inhibitor. One example of a natural inhibitor of IL-1 is *IL-1Ra*, which has affinity for the IL-1 receptor, but when it binds to the receptor, it does not elicit a biological response, and thus it acts as a natural inhibitor of IL-1. In the case of TNF-α, the membrane-bound receptors can be secreted or solubilized (in this form, there is no way to signal a biological response once they are bound to TNF-α). They maintain the affinity for the TNF-α molecule and, in thus binding the molecule, prevent it from binding a membrane bound receptor. Numerous *in vitro* and *in vivo* studies have been carried out using natural inhibitors of the cytokines with very promising results (see Chapter 79).[57,58]

Like the cytokines, natural inhibitors of the MMPs also exist, termed *TIMPs*. Four different TIMPs have been described, three of which (TIMP-1, TIMP-2, and TIMP-3) are thought to be active in inhibiting joint-related functions. Synthesized by numerous cells that include chondrocytes and synoviocytes, these inhibitors bind one-to-one with MMPs to form an inactive complex.[59,60] Believed to play an important role in the normal regulatory mechanism of the joint environment, these inhibitors have been shown to be present in abnormal levels in human osteoarthritic cartilage.[61,62] Therapeutic intervention using TIMPs has not appeared to advance as rapidly as cytokine inhibitor therapy, which may suggest a less-global role in osteoarthritis, although more research is needed.[63]

In summary, a vast number of mediators are involved in joint metabolism in both health and disease. This chapter has only outlined the major molecules and the current level of understanding. Given the discovery of new mediators and common pathways, substantial changes in our understanding of osteoarthritis should be expected in the next decade. Additional information on this subject can be found in Chapter 79.

Clinical Manifestations of Osteoarthritis

Sources of Pain

Joint-related problems account for the greatest single economic loss to the horse industry, much of which can be related to osteoarthritic pain. Although the articular cartilage is devoid of innervation, the surrounding tissues are rich in unmyelinated C fibers. The capsule, synovium, tendons, ligaments, periosteum, and bone have all been defined as sources of pain in osteoarthritis cases. Sensory nerves are known to respond both to *mechanical stimuli*, such as stretching, and to *chemical mediators.* Mediators such as kinins and the neuropeptides (e.g., substance P) have been shown to stimulate pain fibers directly. These mediators, along with others, such as PGE_2 and IL-1, sensitize fibers to be more reactive after mechanical stimulation. Upregulation of pain receptors and involvement of the central nervous system (i.e., the spinal nerves) has also been demonstrated in osteoarthritis.[64] Elevated levels of substance P have been observed in both equine and human patients with osteoarthritis, and they can stimulate monocytes to release other proinflammatory cytokines such as IL-1 and TNF-α.[65-67]

In both horses and humans, correlation between clinical signs and disease severity is poor. In humans, there is an increased chance of reporting pain with increasing radiographic changes consistent with osteoarthritis, but a significant number of people report pain despite normal radiographs, and many individuals with unequivocal knee osteoarthritis deny having pain. Similar parallels can be drawn from equine patients, although anatomic location in both horses and humans appears to play a part in the correlation between clinical pain and the objective parameters. One explanation for the lack of association between pain and structural damage is the lack of sensitivity of outcome parameters, such as radiographs. Many of the soft tissue structures responsible for the pain, such as joint capsule, ligaments, and menisci, are poorly imaged using radiographs. In fact, the importance of soft tissue in joint health is underscored by the fact that quadriceps weakness is a risk factor for human knee osteoarthritis. Multiple studies have shown that strengthening exercises are an effective method of reducing pain and improving function in people with osteoarthritis.[68-70]

It is also believed that the periosteum and bone can play a significant role in osteoarthritic pain. People report focal pain in the area of osteophyte growth, and it is believed that growth of the osteophyte may result in elevation and stretching of the richly innervated periosteum.[64] In some cases, the subchondral bone plate appears to be a source of pain, although not all horses with subchondral cystic lesions demonstrate clinical lameness.[71] Increases in intraosseous pressure have been demonstrated in osteoarthritis, and there is evidence to link this with pain in people, especially following reduction of symptoms after cortical fenestration.[64] Similar mechanisms have been proposed for horses, and decompression of cystic lesions has led to improved lameness scores.

Clinical Parameters

An increase in synovial fluid or synovial pressure is a common finding, especially in joints with excessive joint distention. Thought to be initiated by inflammatory events occurring in the joint, synovial effusion is in part a result of increased vascular permeability (ingress) and a decrease in lymphatic drainage (egress). This net increase in fluid results in an abnormal use of the joint for both mechanical and pain-related reasons. If not controlled, the inflammatory process may lead to changes in the synovium and the joint capsule, which are often observed as a decreased range of motion. Although an increase in synovial fluid can be solely responsible for decreases in range of motion, edema and a decreased pain threshold most likely also contribute. More chronic changes, such as fibrosis in the synovial membrane and joint capsule, can also be observed histologically.

Changes in the content of the synovial fluid are also observed in most cases of osteoarthritis, and *reduction in viscosity* is one of the oldest measures of a diseased joint. An evaluator can roughly determine viscosity by handling a tenacious string of synovial fluid between his or her fingers. The loss in viscosity is typically attributed to a decease in hyaluronan concentration as well as to depolymerization or shortening of the molecule.[72] The large variation in scientific methods and the time-consuming nature of measuring synovial fluid viscosity often make this measurement clinically impractical. The addition of acetic acid leading to precipitation of mucin or a "mucin clot" is a quick and easier but less-specific or sensitive way to measure synovial fluid quality. This, in combination with gross observation of the synovial fluid, which is usually pale yellow to clear and free of flocculent material, has been used in the field to assess quality. In a laboratory setting, the determination of synovial fluid total

protein is often useful in relation to the degree of synovitis and also has been correlated to articular cartilage damage arthroscopically.[73] Cytologic examination of the synovial fluid also is routinely performed in the laboratory. Although synovial fluid total protein and cytology is not diagnostically very specific or sensitive, it can be useful in some cases, especially those with extreme values (Table 78-2).

More-sophisticated methods of measuring cartilage-specific (aggrecan and type II collagen) synthetic and degradation molecules for use on synovial fluid and serum of horses with osteoarthritis involve biomarkers. These biomarkers show promise in early detection and staging of equine joint disease, although more clinical research is needed.[33,73]

As previously mentioned, the usefulness of radiographs is somewhat limited in osteoarthritis, but, because of their ease, they have historically been a method for evaluating joint disease. Although often not correlated to the severity of clinical signs, periarticular enthesiophytes, joint-space narrowing, subchondral bone sclerosis or lysis, and the presence of osteochondral fragments are typical features that can be present radiographically in an osteoarthritic joint (Table 78-3).

Other imaging modalities such as ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) have more recently gained acceptance in veterinary medicine. The ability of ultrasonography to image soft tissues, including articular cartilage, makes it an especially useful adjunctive diagnostic tool in osteoarthritis. The level of sophistication and general knowledge in equine ultrasonographic joint anatomy has greatly increased in the last decade, making joint ultrasonography a standard tool.[74] Likewise, with increased knowledge of normal anatomy and increased availability of equine-dedicated MRI units, this modality will surely gain greater acceptance. The use of MRI in the human orthopedic field is commonplace, largely because of the ability to image all of the joint tissues in a three-dimensional space.

Cartilage Repair

An observation made in 1743 that "cartilage once destroyed never heals" is still accurate today.[75] Modern nomenclature defines healing as the restoration of structural and functional integrity, whereas regeneration suggests the tissue will be identical to that of the original. Repair, on the other hand, has a more limited meaning and suggests that cells and tissue structures replace the damaged tissue, but that the tissue does not necessarily return to its original structure or function. The degree of damage to normal articular cartilage is typically described by the dimensions of the lesion and its depth into the tissue; both of these factors, as well as anatomic location and weight-bearing, play a significant role in the degree of healing and return to normal function.

It is important to note discrepancies in the cartilage repair literature regarding the definition of *full-thickness defects.* This term has been used by some to describe lesions including only the noncalcified cartilage (superficial, intermediate, and deep, but not the calcified cartilage layers) all the way to lesions that extend past the subchondral bone plate. Until recently the true depth was known only if histologic confirmation was performed. However, after studies using arthroscopic visualization of experimental cartilage defects were correlated directly to histologic sections, some degree of confidence about the depth of defects can be made. Today, most authors use "full-thickness articular cartilage defects" to describe a lesion through the calcified cartilage layer but not involving the subchondral bone plate (Figure 78-15).

Figure 78-15. A 15-mm-diameter full-thickness articular cartilage defect on the medial trochlea of the femur depicted through an arthrotomy.

It is believed that full-thickness defects greater than 3 to 5 mm^2 in surface area have a poor capacity for repair. In general, defects of this size range are difficult to identify grossly 1 year after they are created, whereas larger defects show good initial healing but degenerate within a year's time.[76,77] *Partial-thickness defects* are believed to have some minor capacity for healing, but typically they appear neither to be progressive nor to compromise joint function, and they are therefore not the focus of most cartilage repair procedures. Clinically, partial-thickness lesions are débrided of any surface fibrillation without débriding deeper. This is because currently used cartilage-repair processes do not provide a repair tissue that is clearly better than the tissue in a partial-thickness defect.

Historically, two different repair mechanisms are described for articular cartilage: intrinsic and extrinsic. Intrinsic, as the name implies, occurs from within the cartilage. Thus *intrinsic repair* relies on the limited capacity of the chondrocytes to divide and repair the damage. A type of intrinsic repair termed *matrix flow* describes healing lesions that have chondrocytes and surrounding matrix that appear to flow from the peripheral cartilage edges into the defect in an attempt to fill the lesion. Small defects appear to repair via this process. *Extrinsic repair* derives cells and other factors contributing to the repair process from sources other than the chondrocytes. One example is repair following surgical perforation of the subchondral bone plate, which is believed to allow stem or progenitor cells as well as growth factors access to the defect, thus enhancing the repair of large defects, which would otherwise exhibit poor healing.[19] The currently accepted method of subchondral perforation is called subchondral bone *microfracture.* Contrary to historic beliefs, mounting evidence suggests that progenitor cells may exist in the surface layer of articular cartilage, thereby challenging the distinction of intrinsic and extrinsic healing, although it is still accepted that large lesions most likely derive most of

TABLE 78-2. Synovial Fluid Cytology Corresponding to Various Clinical Conditions and Diagnostic or Therapeutic Manipulations

Parameter	Normal	Mild Synovitis (e.g., OCD)	Osteoarthritis	Septic Arthritis*	Arthrocentesis	Balanced Electrolyte Solution	Local Anesthetics†‡	Gentamicin†	DMSO† (10% Solution)
Total leukocytes (/μL)	50-500	20-250	≤1 × 10?	20-200 × 10^3	1-4 × 10^3	6-45 × 10^3 (typically 20 × 10^3)	2-10 × 10^3	8-40 × 10^3	6-20 × 10^3
Neutrophils (%)	<10	<10	<15	>90 (variable toxic changes)	50	80	60	50	>50
Mononuclear cells (%)	>90	>90	>85	<10	50	20	40	50	<50
Total proteins (g/dL)	0.8-2.5	0.8-3	0.8-3.5	4.0-8+	1.5-2.5	3-4	2.5-4	4.5-6	2.5-4

Listed ranges are approximate. Considerable variability exists in published reports.
*Significant elevations in leukocyte counts and total protein concentration occur within the first 12 hours in experimentally inoculated joints. Values shown represent those observed at 24 hours.
†Leukocyte counts and total protein concentrations correspond to the maximum values that typically occur within the first 24 hours.
‡Synovial response to lidocaine and mepivacaine are comparable.
DMSO, Dimethyl sulfoxide; *OCD,* osteochondrosis dissecans.

TABLE 78-3. Radiographic Features of Osteoarthritis

Radiographic Feature	Pathogenic Mechanism*
Periarticular osteophytosis	Endochondral ossification occurring at bony margins of unknown cause. Possible repair attempt modulated by altered cytokine milieu.
(Asymmetrical) joint space thinning	Cartilage degeneration and loss.† Usually at areas of weight bearing or high stress. May be absent when focal cartilage loss occurs.
Subchondral sclerosis	Deposition of new bone as a response to changes in force transmission and from healing of trabecular microfractures. Corresponds to areas of maximum stress. Significant sclerosis often corresponds to full-thickness cartilage loss.
Subchondral lysis	Less-common change of uncertain pathogenesis. Possibly pressure necrosis from synovial fluid gaining access to subchondral plate via fissures, or related to pressure necrosis from trauma to bone.
Osteochondral bodies	Disintegration of joint surfaces or fractured osteophytes. May represent inciting lesions (e.g., osteochondral fracture).
Advanced remodeling/ ankylosis	Articular response to advanced degeneration. Environment more consistent with fracture than synovial joint.

*Specific pathophysiologic mechanisms and reasons for disproportionate representation of changes among and between joints remain unclear.
†Seldom used as a marker of disease progression because of problems with technical aspects of radiographic positioning and focal-film distance.

their reparative capacity from extrinsic sources.[78] Another example of extrinsic repair is through the implantation of chondrocytes obtained from an external source, which are typically suspended in some form of scaffold and secured into the defect. Greater detail on specific cartilage repair procedures can be found in Chapter 80.

Repair of clinically significant lesions (i.e., greater than 5 mm in diameter), which requires extrinsic repair methods, appears to be hampered by a less-than-optimal aggrecan and type II collagen content and by a lack of re-formation of normal subchondral bone and calcified cartilage layers. As was previously described, all of these components are integral to the normal function of articular cartilage and hence the joint. Significant improvements in reestablishment of the type II collagen content and to some degree aggrecan have been made recently, but little has been done to improve, enhance, or even control the re-formation of the subchondral bone plate or calcified cartilage layer. Experimental defects have been shown to heal through an influx of granulation tissue, which is characterized by types I and III collagen, and by little proteoglycan or aggrecan in the first 6 weeks. The granulation tissue is then slowly replaced with increasing amounts of type II collagen and aggrecan by chondrocyte-like cells.[79] At best, a hyaline-like tissue is formed, but the biomechanical and biochemical properties of this tissue leave much to be desired. Although modern science has made great leaps in improving the *in vitro* character of tissue-engineered cartilage, the absence of normal physiologic mechanical stimuli appears to be a rate-limiting step *in vivo*, given the critical role it plays in tissue integration and long-term survival of these creations. Interestingly, it has been observed in humans that the quality of the repair tissue is not always directly correlated to the functional outcome of the patient. Thus less emphasis is now being placed on the regeneration of the articular cartilage and more on long-term improvement in function. In some older human patients, 5 years of dramatic pain relief, thus delaying joint replacement, is considered a success.[80]

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CHAPTER
79 Medical Treatment of Joint Disease

David D. Frisbie

There are two main goals for medical treatment of osteoarthritis (OA) in the horse: reducing pain (lameness) and minimizing progression of joint deterioration. When formulating a treatment plan, the optimization of these goals is influenced by an accurate and specific diagnosis, the stage of disease, severity, available treatment modalities, and rehabilitation time. Clinicians realize that treating joint disease is an art and does not follow any specific recipe.

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) for inhibiting the arachidonic acid cascade has been a mainstay for the treatment of joint disease for many decades. NSAIDs are typically used in cases of acute injury and are often accompanied by other treatment modalities. The renal and gastrointestinal side effects associated with NSAID administration limit their long-term use for treatment of joint disease. NSAIDs inhibit the production of prostaglandins and thromboxanes, especially the prostaglandin E (PGE) series, which is thought to be intimately involved in pain, altered cartilage metabolism, and ongoing inflammation in damaged joints (Figure 79-1).

Traditionally, inhibition of PGEs and the resulting symptomatic pain relief has been viewed as a beneficial goal in treating OA (Figure 79-2). Although relatively recent research has offered some suggestion that this inhibition may have long-term unfavorable effects on cartilage metabolism and does warrant further investigation, the current level of knowledge of these unfavorable effects most likely will not alter the use of NSAIDs in clinical practice.[1] Research has also suggested that NSAIDs may have a role in mediating joint pain at the level of the spinal cord as well as locally, although specific research in this area has not yet been conducted in the horse.[2] In the early 1990s, a major advancement in NSAIDs for treatment of OA occurred with the identification of isoenzymes for the cyclooxygenase (COX) pathway (COX-1 and COX-2), and this does have implications for current treatments in horses.

COX-1 has been associated with the housekeeping functions of the cyclooxygenase pathway. It is known to be constitutively produced and important in the balance of normal physiologic function of the gastrointestinal and renal systems, although it has a lesser role in the inflammatory cascade. COX-2 has mainly been associated with inflammatory events, especially those driven by macrophages and synovial cells, and has only minor roles in normal physiology, thus its "bad" or inducible role (Figure 79-3). These findings have led to a paradigm that the anti-inflammatory properties of NSAIDs are mediated through the inhibition of the COX-2 enzyme, whereas the untoward effects occur as a result of their action on COX-1.

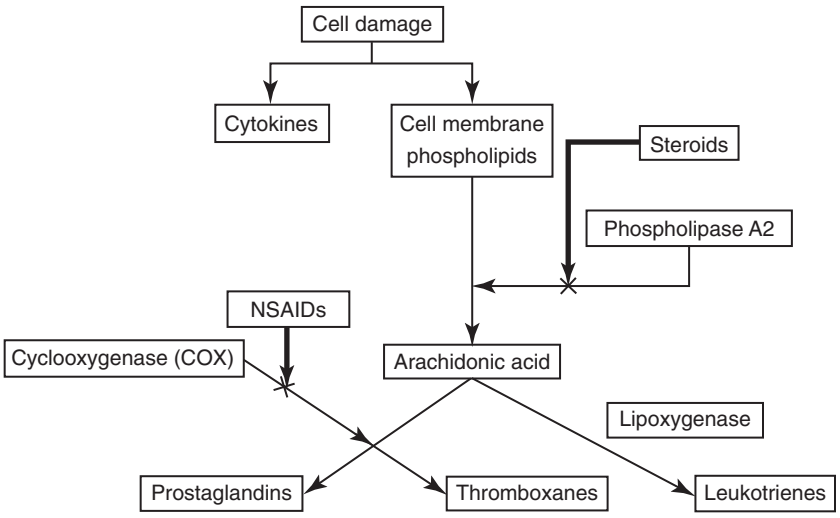

Figure 79-1. Schematic representation of inhibition of the inflammatory cascade by nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids.

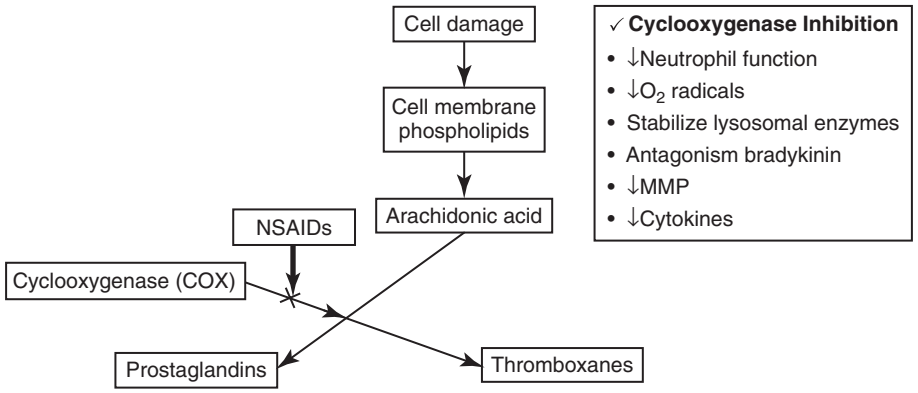

Figure 79-2. Schematic representation of effects associated with the administration of nonsteroidal anti-inflammatory drugs (NSAIDs). *MMP,* Matrix metalloproteinase.

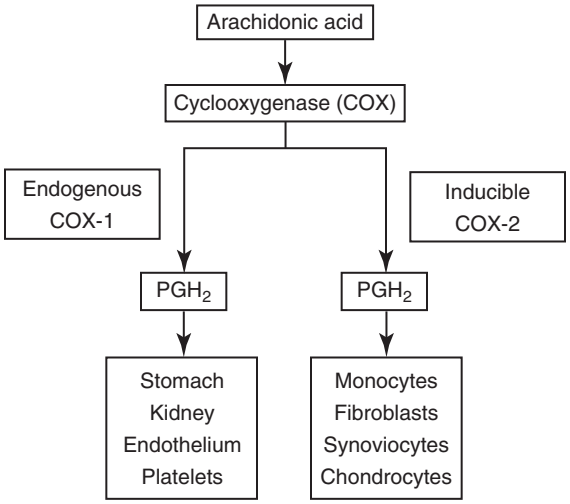

Figure 79-3. Schematic representation of the cyclooxygenase isoenzymes and target tissues. *PGH,* Prostaglandin H.

The protective role of COX-1 for the gastroduodenal mucosa has been shown both experimentally in laboratory animals and in human clinical trials. Furthermore, studies have shown that the greatest degree of damage to the gastroduodenal mucosa is generally caused by NSAIDs that preferentially inhibit COX-1.[3] In fact, the preferential COX-2 inhibitor rofecoxib (Vioxx) was shown in human patients to induce ulceration at a lower incidence than that produced in a randomized placebo group.[4]

As with most biological systems, complete inhibition of COX-2 may not be optimal; some value, albeit minimal, has been shown from expression of COX-2 as well as some demonstrable negative effects of COX-1 expression. More specifically, a low level of COX-1 expression is inducible during stress or inflammatory periods. Likewise, some constitutive expression of COX-2 has been demonstrated by the brain, kidney, and pancreatic islet cells, and it has a function in bone resorption. Furthermore, COX-2 suppression has been shown to delay gastric ulcer healing in rats. Thus, as Oscar Wilde said, "The pure and simple truth is rarely pure and never simple."

To summarize: COX-1 is mainly responsible for the protective prostaglandins, but COX-2 plays some accessory role that is more important than previously thought. However, these facts still might not outweigh the beneficial effects of selective COX-2 inhibition in joint disease.

Research in humans has indicated that topical NSAID application can be clinically beneficial and may reduce systemic side effects.[5] The effect of topical application of diclofenac liposomal suspension has been evaluated in both an equine clinical trial and a model of OA with promising results.[6,7] This product is indicated for horses with a finite number of affected joints, especially if prolonged treatment is not necessary.

Of products currently on the market, phenylbutazone remains one of the most popular NSAIDs for musculoskeletal pain in horses. Its cost, ease of administration, and relatively few side effects during short-term administration make it a good therapeutic choice. It should be noted that when phenylbutazone was compared to diclofenac cream during a 56-day treatment period in horses with OA, phenylbutazone was associated with significantly more pathology.[7] Thus the use of phenylbutazone to treat OA should most likely be limited to the acute period. Flunixin meglumine and ketoprofen both have uses in treatment of equine OA, especially with some reports of fewer side effects; however, the cost-to-benefit ratio often limits their use. Also the use of the first-generation COX-2 inhibitor carprofen (not U.S. Food and Drug Administration [FDA] approved for use in horses) had historically been indicated in horses experiencing side effects from NSAIDs or when extended treatment periods of these medications were expected, but now a selective COX-2 inhibitor (firocoxib) has been approved by the FDA and would be recommended.

Continued use of NSAIDs for treating acute inflammation associated with joint disease is likely for the foreseeable future. Selective COX-2 inhibitors such as firocoxib are being used more often for clinical applications. The NSAIDs currently prescribed in the horse are summarized in Table 79-1.

CORTICOSTEROIDS

More equine-specific research has been published on intra-articular (IA) administration of corticosteroids than any other equine joint medication. Since the introduction of IA corticosteroid use in the mid 1950s, controversy has existed over the risk-to-benefit ratio of this class of medication. Much of this controversy has been fueled from unsubstantiated statements in both the scientific and lay press. Studies showing potentially detrimental effects of corticosteroids on articular cartilage have been conducted using normal cartilage.[8] Current research suggests that inflamed or abnormal joints do not exhibit the

TABLE 79-1. Partial Listing of Currently Available NSAIDs*

Generic Name	Product Name	Formulations	Recommended Dosage (mg/kg)	Standard Availability	Cost
Phenylbutazone	Equi-Phar Phenylbutazone	1 g tablet	4.4 bid for 1 day, 2.2 bid for 4 days, 2.2 sid	Bottle 100 tabs	$9.98
	Equi-Phar Phenylbutazone injection 20%	Injectable 20%	2.2-4.4 bid (5 days)	100 mL	$8.89
	Phenylzone Paste	200 mg/g (20% paste), 12 g Rx	1-2.2 bid (4 days)	60 g	$8.95
	Phenylzone Paste	200 mg/g (20% paste), 6 g Rx	1-2.2 bid (4 days)	30 g	$4.78
Carprofen	Rimadyl	Injectable or tablets	0.7 daily (IV, PO)	Bottle of 180 tablets	$0.84/tablet
Flunixin meglumine	Flu-Nix D Rx (Flunixamine)	Injectable	1.1 daily (5 days) (IV, IM)	100 mL	$18.75
				250 mL	$38.50
	Banamine Paste	Paste	1.1 daily (5 days)	30-g tube	$21.17
Ketoprofen	Ketofen	Injectable	2.2 daily (5 days) (IV)	100 mL	$135.25
				50 mL	$80.76
Firocoxib	EQUIOXX	Paste	0.1 mg/kg daily (up to 14 days)	6.93 g syringe	$5.50

*Availability and costs are as of 2010.
bid, Twice daily; *IM*, intramuscular; *IV*, intravenous; *NSAID*, nonsteroidal anti-inflammatory drug; *PO*, by mouth.

TABLE 79-2. Corticosteroids Commonly Used for Intra-articular Administration

Corticosteroid	Product Name	Manufacturer	Concentration (mg/mL)	Dose (mg)	Potency Relative to Hydrocortisone
Betamethasone sulfate	Celestone Soluspan	Schering-Plough	6	3-18	30
Triamcinolone acetonide	Vetalog	Solvay	6	6-18	5
Flumethasone	Flucort	Syntex	0.5	1.25-2.5	120
Isoflupredone acetate	Predef 2X	Pharmacia and Upjohn	2	5-20	50
Methylprednisolone acetate	Depo-Medrol	Pharmacia and Upjohn	40	40-120	5

detrimental effects seen in normal joints when corticosteroid is administered, potentially explaining some discrepancies seen in experimental studies conducted on normal tissues compared to clinical impressions.[8]

It is well documented that corticosteroids inhibit the arachidonic acid cascade by blocking phospholipase A_2 and that they are selective COX-2 inhibitors.[9] The selective COX-2 inhibition and low incidence of side effects may be reasons why this class of medication is so useful in the horse. The use of corticosteroids for joint disease often brings up questions about three issues in particular: which preparation to use, what dose to prescribe, and what length of rest period to recommend.

Currently, three main corticosteroid preparations are available to the equine clinician: methylprednisolone acetate (Depo-Medrol), triamcinolone acetonide (Vetalog), and compounded betamethasone sodium phosphate and betamethasone acetate (Celestone Soluspan, formerly Betavet) (Table 79-2). Compounded betamethasone has been difficult to obtain commercially in the United States, but it remains available in other countries. As a result, it has been compounded by various pharmacies; however, significant problems have resulted from the use of some of these formulations.

Experimental studies have provided a relatively good comparison of these three products and found that they are not equivalent.[10-12] The three preparations were tested using a relatively similar osteochondral fragment model. Results of this work indicated that the most beneficial effects were seen with triamcinolone, and some protective effects were associated with this preparation. Methylprednisolone did have beneficial effects, but detrimental effects on the articular cartilage were also demonstrated. Compounded betamethasone had no measured effects, which suggests that this preparation has a less-potent effect on joint tissues compared to triamcinolone and methylprednisolone. Compounded betamethasone might not have caused significant improvement because the newly developed experimental model might not have elicited sufficient lameness to evaluate a difference. These *in vivo* studies coupled with *in vitro* work have fueled the recommendation to use triamcinolone, especially in high-motion joints. A recent survey of American Association of Equine Practitioners (AAEP) members[13] suggests that practitioners are in fact using triamcinolone in high-motion joints. Further, this study suggests that when used, methylprednisolone is applied predominantly in low-motion joints.

Recent research has also focused on low-dose corticosteroid administration for the treatment of equine joint disease. It appears to take about a tenfold change in concentration to alter *in vitro* effects of corticosteroids. As an example, joint dose between 1 and 10 mg of triamcinolone or between 20 and 200 mg of methylprednisolone would respond in a relatively similar manner. However, *in vitro* studies have indicated that lower concentrations of these corticosteroids might not inhibit the catabolic pathways driving joint disease.[14] The currently recommended doses appear to be within an acceptable range given in published information (see Table 79-2).

Corticosteroids combined with hyaluronan are very commonly administered. This combination is based on limited scientific evidence but good clinical responses. Combination treatment was supported by 60% of recently polled equine practitioners, reinforcing the favorable clinical response.[13]

The exercise or rest period following treatment with IA corticosteroids is also somewhat controversial. The controversy is based on the ability of some corticosteroids to transiently decrease the anabolic metabolism of chondrocytes and potentially blunt the normal metabolic responses to exercise. Although this is a concern, it is most likely overstated. A decrease in chondrocyte anabolic rate has not been proved to be detrimental. Furthermore, studies have not identified gross or histologic lesions subsequent to IA corticosteroid administration and exercise; however, changes in biochemical and biomechanical parameters have been identified.[15,16] Changes induced by methylprednisolone have been noted to last for more than 21 days, and this information has influenced exercise recommendations for all other corticosteroids. Anecdotal recommendations for rest periods after IA corticosteroid injection vary widely from immediate return to work to more than 30 days of rest. Even a relatively aggressive protocol, involving a high dose of corticosteroids repeated at 14 days along with concurrent strenuous exercise, has failed to demonstrate definitive detrimental effects.[10-12] Thus a rest period might not be absolutely necessary. Anecdotal clinical impression does suggest that confinement to a box stall or paddock for 7 to 10 days, with a slow return to full work in the subsequent week, can prolong the duration of action following injection, but it does not appear to alter the effect of the treatment.

Some consideration should be given to the reported incidence of corticosteroid-induced laminitis; however, no direct association has been proved. It has been suggested that the total body dose of triamcinolone should not exceed 18 mg, methylprednisolone should not exceed 200 mg, and compounded betamethasone should not exceed 30 mg. These numbers are based on some facts and some fiction, but one of the main goals is to eliminate corticosteroid-induced laminitis. Two publications have challenged this doctrine. The first study with long-term follow-up on 205 clinical cases of joint injections using either 40 mg or 80 mg of triamcinolone acetonide found that the incidence of laminitis was effectively 0.5%.[17] The second 2000 cases were treated with 20 to 45 mg of triamcinolone, resulting in 0.15% of the cases having reported laminitis, and all but one were ponies.[18] Because it is not possible to have a consistent model of laminitis following corticosteroid injection, we must rely on clinical reports and anecdotal suggestions for dosing regimens. Based on published literature and clinical experience, I routinely use 20 to 40 mg total body dose of triamcinolone in a healthy athletic horse. The frequency of treatment should be considered, and these higher total body doses should not be repeated frequently, especially in unfit or overweight horses.

HYALURONAN

Hyaluronan (HA) (disaccharide composed of D-glucuronic acid and *N*-acetyl-D-glucosamine) is a normal and necessary component of synovial fluid and proteoglycan aggregates (aggrecan) (see Figure 78-10). Based mainly on *in vitro* work, both the concentration and molecular weight (degree of polymerization) appear to be important in the normal function of HA. Although the exact mechanism through which endogenous or exogenous HA exhibits an effect is not known, additional effects of exogenous HA administration have been documented to include anti-inflammatory activity, such as inhibition of chemotaxis, inhibition of phagocytosis of granulocytes and macrophages, and reduction in the stimulation, proliferation, and migration of lymphocytes. Hyaluronan has also been shown to decrease the formation and release of prostaglandins from macrophages and to scavenge oxygen-derived free radicals in a dose-dependent manner.[19] Researchers also hypothesize that exogenous administration of HA increases the synthesis of high-molecular-weight hyaluronate by the synoviocytes, a theory that is yet to be definitively proved. In addition, some questions exist about the molecular weight requirement for effectiveness of exogenous HA.

Assessing the literature suggests that molecular mass less than 500 kDa has little effect, whereas HA with a larger molecular mass demonstrates a dose-dependent response. Other studies have failed to correlate molecular mass to outcome parameters. Summarizing the current literature, it appears that some basal molecular mass is needed for effectiveness.[20] I therefore use products with molecular mass greater than 500 kDa and selects products with yet higher molecular mass based more on economic consideration of the client (Table 79-3).

Cross-linked exogenous HA has been introduced in an attempt to improve viscosupplementation of joints. Briefly, viscosupplementation refers to the injection of HA or its derivative in an attempt to return the elasticity and viscosity of the synovial fluid to normal or higher levels. Cross-linked HA has been developed as an improvement to other synthetic HA. Because its rheologic properties are increased, it has a longer retention time in the synovial space and appears to be more resistant to free radicals. Hylan G-F 20 (Synvisc-One), a cross-linked HA with a molecular mass on average greater than 6 MDa, has demonstrated favorable results in large multicenter human clinical studies, showing effectiveness as a nonsteroidal medication.[21-23] An experimental study conducted in the horse failed to show beneficial effects of hylan G-F 20, but it does cite the difference in acute experimental synovitis compared to the more chronic OA (in the human trial) as one possible explanation for the study outcome.[24] The authors also mention that the severity of the model may have been too harsh to observe a significant treatment effect. Further work on case selection for viscosupplementation (cross-linked HAs) is needed.

The dosage of conventional HA (0, 1, 10, 20, and 40 mg per joint) has been tested using an equine OA model.[25] This study demonstrated that a dosage of 20 mg per joint was needed to demonstrate improvement in degree of lameness measured using a force plate. Dose–frequency studies are based on work in human clinical patients, suggesting that at least three doses

TABLE 79-3. Partial Listing of Available Hyaluron Products*

Product Name	Manufacturer	Concentration	Molecular Weight (daltons)	How Supplied	Recommended Dose (for Small- to Medium-Size Joints)	Standard Availability	Cost
Hylartin V	Pfizer	10 mg/mL	3.5×10^6	2-mL syringe 20 mg	20 mg	Each	$54.16
MAP-5 (used intra-articularly at this dose)	Bioniche	10.3 mg/mL (2 mL) 5 mg/mL (10 mL)	7.5×10^5	2-mL vial 10-mL vial	20 mg	10 mL 2 mL	$37.93 $17.78
Legend (Hyonate) Intravenous/ intra-articular use	Bayer Corporation	10 mg/mL	3×10^5	4-mL vial	40 mg (IV)	Box of 6	$388.08
Hyalovet (Hyalovet 20)	Boehringer	10 mg/mL	$4\text{-}7 \times 10^5$	2-mL syringe	20 mg	2 mL	$42.79
HyCoat (used intra-articularly)	Neogen	5 mg/mL	$>1.0 \times 10^6$	10-mL vial	30 mg	10 mL	$37.17
Hyvisc	Boehringer	11 mg/mL	2.1×10^6	2-mL syringe	20 mg	each	$53.04
HY-50	Bexco Pharma	17 mg/mL	—	3-mL syringe	51 mg	each	$35.00

*Availability and costs are as of 2010.

are required (administered 1 week apart) to see significant effects in clinical parameters of pain and effusion. A recent blinded, randomized, placebo study evaluated a high-molecular-weight product (Hyvisc), in which horses with experimental OA were dosed three times, 1 week apart. Though no symptom-modifying effects were seen, a decrease in gross articular cartilage fibrillation as well as improvements in synovial membrane parameters were noted.[26] Thus the currently recommended equine HA treatment protocol is 20 mg of HA once a week for 3 weeks. It appears that HA may be more effective when used in cases of mild synovitis or capsulitis and not as effective in severe synovitis or capsulitis and chronic OA.[26,27]

The use of intravenous (IV) HA in the treatment of joint disease is very common. An experimental study documented improvement in clinical lameness and synovial membrane histology, as well as better synovial fluid parameters, such as lower PGE_2 and total protein levels in a carpal chip model.[28] These improvements were seen 42 days following the last of three treatments (40 mg) that were given 1 week apart.

Research to elucidate the mechanism of action for IV HA has been conducted, although relatively few answers have been found. A recent survey of 20 equine practitioners did not reflect an overwhelming usefulness of IV HA; however, case selection or objective outcome parameters were not clearly defined.[13] From both scientific and anecdotal reports, it appears that the use of IV HA in cases of acute synovitis could yield a better chance for success than in cases of chronic OA, where significant cartilage loss is present.

The prophylactic use of IV HA has also been studied in both Quarter Horse and Thoroughbred racehorses. The Quarter Horse study enrolled 140 horses; subjects received either IV saline or HA every 2 weeks for the duration of the 9-month study.[29] HA-treated horses tended to race longer before requiring the first joint injection and to have a better speed index, higher average number of starts, and more money earned compared to placebo-treated horses. Anecdotal reports from trainers in various equine disciplines have been positive regarding the prophylactic use of IV HA.

POLYSULFATED POLYSACCHARIDES

Polysulfated polysaccharides include polysulfated glycosaminoglycan (PSGAG; Adequan), GAG peptide complex (Rumalon), and pentosan polysulfate (Cartrophen Vet; Pentequin). These drugs have been referred to as exhibiting chondroprotective effects or, by more recent definitions, are slow-acting, disease-modifying osteoarthritic drugs (SAMOADs). Such agents are meant to prevent, retard, or reverse the morphologic cartilaginous lesion of OA.

PSGAGs are manufactured from bovine lung and trachea extracts containing mainly chondroitin sulfate. Numerous equine studies have been carried out *in vitro* and *in vivo* using PSGAGs and have had contradictory results.[30,31] Some studies have shown PSGAGs to inhibit many of the key enzymes or cytokines involved in OA, including interleukin 1 (IL-1), matrix metalloproteinases (MMPs), and PGE_2, and to affect proteoglycan synthesis and degradation.[31] Because the testing systems, duration of experiments, and dosages varied from study to study, direct comparisons are difficult. The precise mechanisms of action of PSGAGs are uncertain, and the interaction of PSGAGs with cytokines involved in joint disease has not been well defined.[20]

Some of the earliest work involving IA PSGAGs in the horse investigated the effects of administering 250 mg biweekly for 3 weeks in clinical cases. This study and others using models of acute synovitis have shown improvements in clinical disease parameters following PSGAG treatment.[32-35] One study also looked at cartilage histology, observing improvement in chondrocyte morphology. The same authors, using a similar model, also tested PSGAG via an intramuscular (IM) route with less

impressive results.[20] In a group of ponies with experimentally induced osteochondral lesions, IA PSGAGs (250 mg once a week for 5 weeks) significantly improved clinical lameness, radiographic progression of OA, and joint capsule parameters, compared with untreated ponies. However, PSGAGs also reduced the endogenous repair of the cartilaginous lesions.[28] Therefore, care should be taken when using PSGAGs in cases where significant cartilage lesions exist on weight-bearing surfaces.

In an experimental study, IM PSGAGs administered every fourth day for 28 days, starting 14 days following OA induction, demonstrated some improvement in clinical lameness 56 days after initiation of treatment.[36] Although PSGAG-treated horses did show biochemical improvements compared to placebo-treated horses, overall more impressive improvements were observed with shock wave treatment of the affected joint (see "Extracorporeal Shock Wave Therapy," later). The study was repeated using an IA route of administration once a week for 3 weeks and did in fact show significant improvement in reducing synovial effusion and improvement of synovial membrane parameters when compared to placebo.[37] This further suggests a more potent response when used IA versus IM.

A survey to assess the perceived efficacy of PSGAGs by 1522 equine practitioners was published in 1996.[27] Practitioners treating Thoroughbred racehorses gave the highest efficacy scores, and practitioners treating pleasure horses gave the lowest efficacy scores. PSGAGs were considered more effective than hyaluronan for the treatment of subacute OA and less effective for idiopathic joint effusion and acute synovitis. The authors of the survey concluded that the efficacy of PSGAGs for incipient and chronic forms of OA is comparable to that of hyaluronan.

Important information has been published indicating that a lower bacterial inoculation is needed to establish septic arthritis when PSGAGs are administered IA.[38,39] This result is abolished with the addition of 125 mg amikacin to the PSGAG treatment. Therefore, administration of amikacin with PSGAGs administered IA is strongly recommended.

Like PSGAGs, pentosan polysulfate (PPS) is a heparinoid compound, but it is unique in that it is derived from beech wood hemicellulose instead of the animal or bacterial sources used for PSGAGs. These products have been administered mainly in horses in Australia, although efficacy reports have been anecdotal. To my knowledge, sodium pentosan polysulfate has been used only parenterally in horses. Although a form has been complexed with a calcium salt and tested in other species, the results indicate that pentosan appears to be bioavailable following oral administration. Clinical studies in dogs and people with OA have shown convincing evidence of clinical efficacy.[40] In the horse, an unpublished study using an experimental model of OA has shown favorable results.[41] In this model, parenteral PPS showed significant improvement in reducing articular cartilage fibrillation and a strong trend for overall improvement in the histologic appearance of cartilage. Furthermore, most parameters showed numerical improvements (lameness, joint flexion, synovial fluid total protein, synovial fluid collagen degradation products, and aggrecan synthesis), although statistical significance of greater than 95% was not obtained.

Clinically, PPS could be used to treat horses with mild or early-stage OA, particularly with multiple joint involvement, because it is a systemic therapy. At this time, a dosage of 3 mg/kg IM once weekly for 4 weeks is recommended for treatment of equine OA.

ORAL JOINT SUPPLEMENTS

A vast number of oral supplements are available for the horse. Many of these work on the premise of providing building blocks of molecules that are integral in the articular cartilage or joint fluid. Although the formulation, concentration, and source of the products differ widely, most contain chondroitin sulfate, glucosamine, or hyaluronan, or some combination thereof. Chondroitin sulfate has been assessed using radioisotope tracking and shown to be absorbed through the gastrointestinal tract, but it is thought to be in a smaller monomeric form compared to the ingested molecule. Glucosamine is even more bioavailable than chondroitin sulfate.[41-45]

A study was conducted to document absorption specifically in the horse, but how these levels relate to clinically therapeutic levels is yet to be determined.[42] Double-blinded controlled studies in humans have indicated that these substances are efficacious in the treatment of joint disease.[46-49] Unfortunately, only limited, poorly controlled equine clinical studies have been performed to date, but anecdotal reports and *in vitro* tests are favorable for the use of glucosamine and chondroitin sulfate.[50]

Oral hyaluronan products are relatively new on the market and have been tested less compared to chondroitin sulfate or glucosamine. However, a controlled experimental *in vivo* trial comparing IV Legend and Conquer, an oral hyaluronan, is under way. An interesting report in the human literature compared the label ingredient with the independent testing of the products and found little correlation to label claim and content or price and content. This potentially emphasizes the use of trusted brands that have at least undergone some testing.

Laboratory and live animal work as well as a human clinical trial have shown beneficial effects of oral avocado and soya unsaponifiables (ASU) on joint disease.[51] Likewise an equine-specific study showed significant improvement in the gross joint and cartilage health in response to oral ASU administration in horses with OA.[51] Platinum and Cosequin ASU are currently commercially available but provide 2 and 1 g/day respectively, compared to the 6 g/day administered in the experimental study showing efficacy.[51]

EXTRACORPOREAL SHOCK WAVE THERAPY

Recent experimental evidence and anecdotal clinical impressions of extracorporeal shock wave therapy (ESWT) for the treatment of OA have been reported.[52-54] Unpublished clinical studies in the dog have shown promising results, as have anecdotal reports of treating shoulder, pastern, and coffin joint OA in horses.[36] An equine-specific, controlled experimental OA study has been completed comparing ESWT to PSGAGs and sham treatments.[55] The study used an established short-term (70 days) OA model where an osteochondral fragment was created at time zero and treatments were initiated 14 days later.[12] IM PSGAG treatment was administered every 4 days for 28 days. ESWT was administered on days 14 and 28 using a VersaTron 12-mm probe, and a sham shock wave procedure was performed on the control horses on days 14 and 28. In the ESWT group, 2000 shock waves at the E4 energy level were administered on study day 14 and 1500 shock waves at the E6

level on study day 28. The energy was delivered mainly to the middle carpal joint capsule attachment, but some energy (approximately 20%) was delivered to the area of fragmentation. Significant improvement in clinical lameness, decreased synovial fluid total protein (as a marker of synovitis), and reduced GAG release into the bloodstream were observed with ESWT compared to both control and PSGAG-treated horses. These results appear to show promise for this type of therapy for use in localized joint disease in horses, although clinical studies are still needed.

BIOLOGICAL THERAPIES

Since the 1990s, research has led to a greater understanding of joint pathophysiology and identification of major mediators driving the disease process. Also, the identification of naturally occurring antagonists or synthetic analogs with the ability to block these degenerative mediators has been realized. Numerous novel medical treatments are being tested *in vivo* after successful *in vitro* trials.[56,57]

Treatment of generalized OA using novel methods of administering antiarthritic therapeutic proteins is one of the most rapidly expanding areas in treating equine joint disease. Given that the inhibition of IL-1 and tumor necrosis factors (TNFs) has been shown to be clinically beneficial, practical methods of administering these therapeutics *in vivo* are being explored. Specific examples that have shown clinical success in people with either rheumatoid arthritis or OA are the administration of IL-1 receptor antagonist (IL-1Ra, a natural antagonist of interleukin-1), soluble receptors to TNF (receptors that bind the parent molecule but have no further biological action), and antibodies directed at TNF.

In one study conducted in the horse, the administration of *autologous conditioned serum* (ACS), or interleukin-1 receptor antagonist protein (IRAP) (another abbreviation of IL-1Ra) as it is commercially known, resulted in demonstration of both beneficial symptom- and disease-modifying effects.[58] Though ACS has been shown to increase IL-1Ra levels, many other proteins are also upregulated during the 24-hour culture period. The specifics of this process are currently under investigation. Clinically, anecdotal reports of significant improvement in horses following treatment with ACS have been seen even in joints that are or have become unresponsive to corticosteroids. In some horses ACS is being used as a primary treatment of OA.

Platelet-rich plasma (PRP) has also recently been used to treat joint disease. However, little proof from basic science is currently available to support its use over a biological treatment such as ACS. It is most likely because of the decreased time (does not require a 24-hour culture period) and increased convenience (can be done in the stall with home commercial products) of PRP that this treatment is being used clinically. Further research on the beneficial effect of PRP in the equine joint is needed.

Stem cells are also now being used to treat equine joint disease. Early work using labeled *mesenchymal stem cells* (MSCs) has shown they do have an affinity for damaged joint tissues, and more recent *in vivo* studies have confirmed their ability to localize and participate in repair of damaged joint structures, including cruciate ligaments, menisci, and cartilage lesions.[59] Most of the *in vivo* studies using MSCs have focused on meniscal repair. In some cases, MSCs are delivered on a carrier or scaffold, whereas in others, they are directly injected into the joint.[60-62] These studies have shown positive outcomes when bone marrow–derived cells are used to treat meniscal damage. The degree of damage has ranged from experimental meniscal lacerations treated with bone marrow aspirates, separating and using only the nucleated cells,[63] to total medial meniscectomy treated with injection of bone marrow–derived and culture-expanded MSCs.[61] Early work indicated that MSCs deposited in a fibrin matrix would be useful in improving cartilage healing. Although a recent equine study demonstrated early benefit, no significant differences were noted when treatment using MSCs and fibrin was compared to fibrin alone at 8 months.[64] Based on this work, it appears likely that the matrix or cells will need to be modified to achieve long-term benefit of MSCs for cartilage repair.

Although a goat study demonstrated regeneration of the meniscus, it was aimed at evaluating the *in vivo* effects of IA stem cell injection on decreasing the progression of OA.[61] This study used medial meniscectomy and cranial cruciate transection to induce OA. The investigators concluded that the decrease in OA seen in the study appeared to be secondary to the regeneration of the medial meniscal tissues, which was dramatic in seven of nine cases. However, the design of the study did not lend itself to determining if the stem cells had a direct effect on the articular cartilage and progression of OA. Thus the authors completed an equine study that used an osteochondral fragment with bone and cartilage debris to induce OA,[65] unlike the study mentioned earlier, which relied on joint instability (medial meniscal model) to create secondary OA. The results of this study indicated significant improvement in synovial fluid PGE_2 levels in response to treatment with bone-derived cells. Also demonstrated was a negative response via an increase in synovial fluid TNF concentrations in response to adipose-derived cells. The beneficial response seen with bone-derived cells overall was interpreted as a nominal improvement in symptom or disease-modifying effects.[65] The results of this study combined with the meniscectomy study[61] suggest that the regeneration of the medial meniscus in the goat study may have been the reason for slower OA progression. These studies also suggest that MSCs by themselves do little to counteract the progression of acute OA mediated by enzymatic degradation and joint debris. It would appear that modification of the MSCs is needed if they are to be useful in treating OA. Treatment timing relative to the degree of pathology could also contribute to the insignificant results of the equine study. Specifically, because MSCs appear to have a tropism for damaged cells, including fibrillated articular cartilage, it may be that at day 14 (day of treatment) the degree of fibrillation was not great enough for an effect of MSC treatment to be realized. Evaluation in cases with more-advanced fibrillation would need to be conducted to answer this question. Because significant improvement in acute OA could not be demonstrated following IA treatment, I have a dampened enthusiasm for the use of MSCs in clinical cases of acute OA. I did conclude that the use of MSCs appears to be indicated with loss of soft tissue structures leading to instability, such as with meniscal damage, and have pursued this treatment modality specifically in a multicenter clinical trial.

The results of this prospective multicenter trial are promising.[66,67] Currently 39 cases have been treated with IA administration of autologous bone marrow–derived MSCs with a mean follow-up time after treatment of 21 months. Cases selected for this trial were meant to have failed routine treatments, be moderate to severely affected, and have surgical confirmation of the

diagnosis. Seventy-seven percent returned to some level of work. Thirty-six percent (14/39) returned to or exceeded their prior level of work, 36% (14/39) returned to work at a lesser level or required some level of additional medical treatment in the affected joint, and 28% (11/39) did not achieve the work status held before follow-up.[67] Stifle injuries accounted for 29 of the 39 of the cases. This work is an extension of the study previously presented in 2007 where there were 15 cases with 6-month follow-up and a 67% return to work.[67,68] These data suggest further exploration of MSCs for the treatment of joint-related soft tissue pathology. For additional information on biological therapies please review Chapter 8.

CHALLENGES OF MEDICAL MANAGEMENT IN EQUINE JOINT DISEASE

Some of the considerations in treating equine joint disease have been presented in this chapter. Challenges that equine clinicians face are numerous, but the rapid evolving knowledge of joint disease pathophysiology, the relative lack of specific equine research, economic considerations of the owner, and rules imposed by governing bodies of sanctioned equine events pose a unique set of challenges. More specifically, the pathophysiology of joint disease is extremely complex; the multifactorial nature of the disease presents additional experimental complications, and at best it is only partly understood in any species.[69,70] Even with the greatest understanding of the disease being in people, human physicians do not agree upon a course of treatment based on the joint affected, stage of disease progression, or age of the patient. Further removed is the equine practitioner, who is often making therapeutic decisions based on extrapolations from human studies and therapies, comparisons that are not always justifiable.

The lack of specific equine research often drives the need to extrapolate from other species on the class of substance to use, specific compound within a class (e.g., a specific corticosteroid), dose, and frequency of administration, and in some cases, a combination of medications are recommended. The equine clinician is often left to make a plan based on anecdotal information rather than substantial evidence showing safety or efficacy of a specific regimen. Advances in equine-specific studies are occurring, thus confirming or refuting many anecdotal or uncontrolled published studies, although as expected, much work is left to be completed.

Other factors do play a role in arriving at a therapeutic plan, but some of them have little to do with the defined treatment goals. Economic consideration and medication rules governing many sanctioned equine events are two such examples. The coordination of all of the presented factors and issues into a sound, effective therapeutic plan are what makes treatment of equine joint disease challenging. Through an understanding of the historic and current literature, anecdotal information, and up-to-date rules pertinent to equine events, advancements can be realized in the treatment of equine joint disease.

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CHAPTER 80

Surgical Treatment of Joint Disease

David D. Frisbie

Over the last decade, significant advancements have been made in the understanding of medical and surgical treatment of equine joint disease. Many of these advances originally were aimed at helping humans, but the principles have been adapted for use in the horse. Advances in understanding pathophysiology, novel surgical techniques, improved surgical equipment, and more-sophisticated imaging modalities have led to improved treatment of joint disease.

It is important to focus on the two main treatments for joint disease: the relief of pain to regain functional use of the diseased joint and the arrest of disease progression. In many cases, generalized osteoarthritis (OA) is treated using medical management and exercise protocols, whereas full-thickness articular cartilage damage usually involves some surgical intervention.

SURGICAL TREATMENT

Diagnostic and Surgical Arthroscopy

Equine arthroscopy was first described as a diagnostic tool to visualize lesions that were undetectable using radiography or other routine imaging procedures, but it was soon employed for therapeutic purposes, such as surgical removal of osteochondral fragments. Arthroscopy is now considered part of routine equine surgery and has, for the most part, replaced arthrotomy. It allows better visualization of the joint anatomy and inflicts less damage to the joint capsule and surrounding tissues, leading to quicker return to use and a more favorable outcome for the horse.[1] Since the 1980s, various publications have described arthroscopy in the horse; many of the early publications only described various techniques and approaches to joints, with more detailed outcomes and prognoses for specific pathologies following in subsequent publications. Most of the published work has been compiled in equine-specific texts.[1,2] The equipment used for arthroscopy and the surgical technique is discussed in Chapter 13.

Arthroscopy has a diagnostic and a therapeutic function in joint disease. Even with modern imaging modalities, it is still considered the gold standard for diagnosing equine joint problems. The cost and availability of arthroscopic equipment, along with arthroscopy's specificity and sensitivity when compared to more-complex imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI), and the expertise needed to perform definitive joint ultrasonography, will likely keep arthroscopy in the forefront of diagnostics for equine joint disease for many years. This is especially true in cases that show no demonstrable or questionable joint lesions using traditional imaging modalities, even though the horse has localized clinical pain based on diagnostic analgesia.

In recent years, additional joint pathology, such as meniscal and cruciate lesions, has been described. This may be because diagnostic arthroscopy, especially in the stifle joint, has become more accepted. Therefore, these types of lesions, which are not detectable using radiography, are being recognized more often. With an increased awareness of how often these types of lesions occur, further work is being focused on therapeutic treatments. This is especially true in the field of cartilage resurfacing and meniscal pathology, with many of the therapeutic avenues pursued in equine practice representing the current state of the art in human medicine.

Another advantage of the use of arthroscopy is the potential benefit of lavage. Arthroscopy is often performed using fluid to distend the joint for better visualization. The accompanying egress of joint fluid, which potentially contains cytokines and cartilage wear particles, may be therapeutically beneficial, even when lavage is performed as a sole treatment in cases of osteoarthritis. However, the benefit of lavage is controversial, and it does not work in cases with significant definable disease such as meniscal tears. Research also has shown that when lavage is performed with large-gauge needles (14 gauge), it is not as effective as using arthroscopic cannulas.[3-5] Distention of the joint can be accomplished using inert gas, which can be especially helpful if bleeding of intrasynovial tissues impairs visualization when a fluid medium is being used.

In general, arthroscopy is of more therapeutic benefit in acute than in chronic cases. For example, removing a freshly detached osteochondral fragment before OA can develop typically produces a better outcome than waiting until after OA has developed to remove an osteochondral fragment that has been present for some time. However, there are some indications that specific chronic pathologies, such as osteonecrosis and cartilage fibrillation of the third carpal bone, can be successfully managed using arthroscopy.

Another consideration regarding arthroscopic surgery is the documentation of the surgical procedure. Since the 1990s, reduction in cost and increase in availability of video and still-capture devices have allowed real-time documentation of arthroscopic surgery to be made and kept as part of the medical record. This real-time information is embraced by trainers and owners as a means of understanding the pathology and treatment procedures, as well as providing more specific information about the surgery.

Removal of Osteochondral Fragments

In most instances, osteochondral fragments are removed when they are diagnosed in conjunction with clinical lameness. Some fragments are considered relatively benign, and removal is not indicated without clinical lameness.[1,6,7] The routine use of arthroscopy has improved the outcome of osteochondral fragment removal as compared to removal using arthrotomy. Outcomes related to osteochondral fragment removal are presented in Table 80-1, and specific anatomic landmarks relating to arthroscopic approaches to the different joints can be found detailed in this text and in published references.[1]

In general, diagnostic visualization of the entire joint cavity is performed before fragment removal (Figure 80-1). When multiple fragments are present, typically the smaller fragments are removed first. This postpones the need to increase the size of the instrument portal and delays the potential for subsequent

TABLE 80-1. Prognosis* for Racing or Athletic Soundness Given by Various Authors for Intra-Articular Osteochondral (Chip) Fractures† in Various Locations and Joints

Fracture Location	Treatment‡	Prognosis	Reference
Extensor process of P3	M or S[a]	Guarded	8, 9, 10
	M or S[a]	Poor	11
	M or S[a]	Good	12
P2 (not involving DIP joint)	S[a]	Guarded to good§	13, 14
	S[a]	Guarded	11, 15, 16
P2 (involving DIP joint)	M or S[a]	Poor	11
Dorsal frontal P1	S[a] (displaced)	Good	17
	M (nondisplaced)	Good	17
Proximodorsal P1	S[a]	Excellent	18
	—	Good	19
	S[b]	Excellent	16, 20, 21
Plantar P1	S[a]	Excellent	22
	S[a]	Good	23
	S[b]	Excellent	12
	S[b]	Good	24
Palmar P1	M	Guarded	22
	S[a]	Excellent	25
	S[b]	Excellent	12, 26
	S[b]	Good	27
Apical sesamoid	M	Poor	28
	S[a]	Good	28, 29
	M or S[a]	S better than M	30
	S[b]	Good	31, 32
Abaxial sesamoid	S[b]	Good	33
Basilar sesamoid	M or S[a]	Guarded	34, 35
	S[b]	Guarded	36
Distal radius	M	Guarded	12
	S[a]	HR = 1¶	34
	S[a]	Guarded to good§	37
	Sb	Good	20
Proximal RC	S[a]	Good	37
	S[a]	HR = 2¶	20, 38
	S[b]	Good	20
Proximal IC	S[b]	Good	20
Distal RC	S[a]	Guarded	37, 35
	S[a]	HR = 1¶	38
	S[b]	Guarded	35, 20
Distal IC	S[b]	Good	20
C3	S[a]	Guarded to good§	36, 39
	S[b]	Guarded	36, 39
	S[a]	HR = 1¶	20, 38

*The prognoses have been graded as excellent (80% to 90% chance of athletic soundness), good (60% to 80%), guarded (40% to 60%), and poor (<40%) and usually refer to the ability to race after injury. These are approximate percentages extrapolated from the literature because these categories are not always used by each author and a definition of these categories is not usually given. "Favorable" has been graded with "good." In general, the prognosis given is for horses with no radiographic signs of osteoarthritis (i.e., the horse is given its best chance of recovery).

†These fractures do not cause major joint instability.

‡*a,* Arthrotomy; *b,* arthroscopy.

§Guarded to good used when the percentages spanned 2 ranges.

¶HR, Hazard ratio was used to compare the racing performance of the treated with that of a control group, taken from the race horse registry for the same period. A hazard ratio of approximately 1 means that the same number of treated horses dropped out of racing as the controls. A hazard ratio of 2 means that twice as many treated horses were lost as the controls.

C3, Third carpal bone; *DIP,* distal interphalangeal; *HR,* hazard ratio; *IC,* intermediate carpal bone; *M,* medical; *P,* phalanx; *RC,* radial carpal bone; *S,* surgical.

Adapted from Caron JP: In Auer JA, Stick J (eds): Equine Surgery. 2nd Ed. Saunders, Philadelphia, 1999.

reduced visualization until the end of the surgery. Very large fragments can be broken into smaller pieces using a rongeur or osteotome to prevent the need for excessive portal size. Hand curettes are typically used to débride the parent bone down to the appropriate level as well as to smooth any edges that might traumatize opposing joint surfaces postoperatively. Before finishing arthroscopic surgery, typically the joints are lavaged with fluids while a final inspection of the joint is completed to ensure all debris has been removed. Debris often accumulates in certain locations within a specific joint, and knowledge of these locations often aids in finding debris that has inadvertently escaped removal by lavage alone.

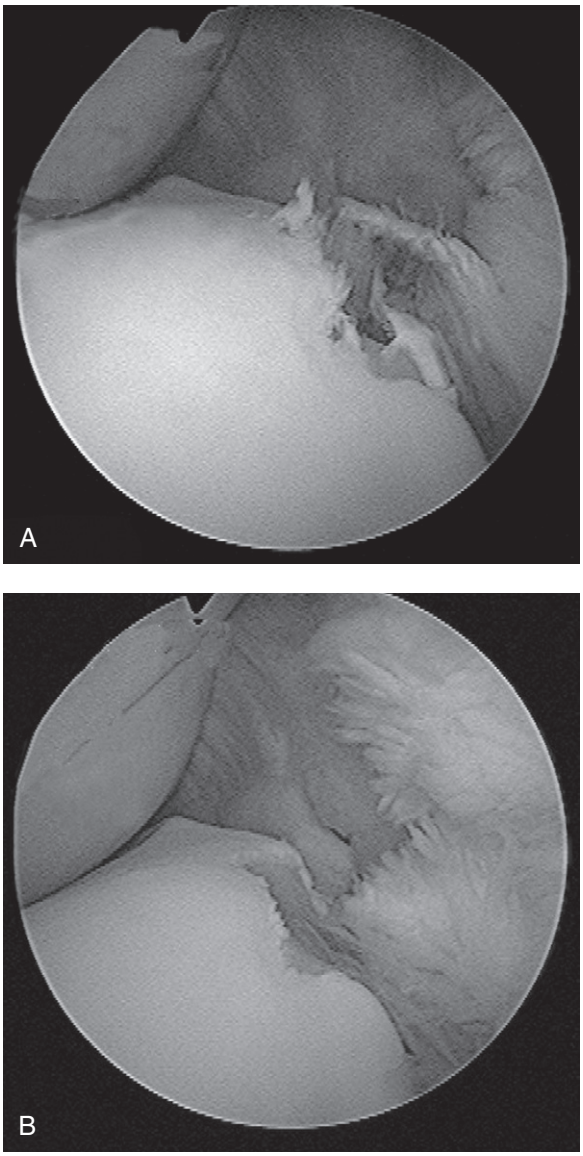

Figure 80-1. Arthroscopic image of a chip fragment off the distal radial carpal bone before **(A)** and after removal **(B).**

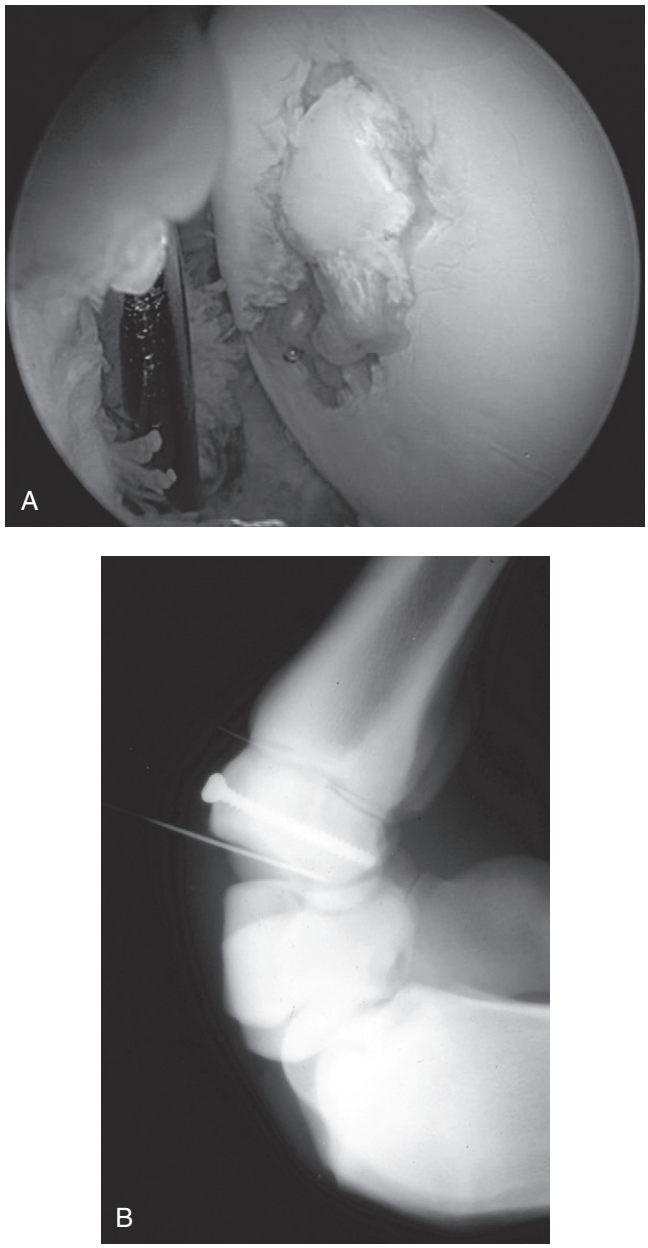

Figure 80-2. A, Arthroscopic image of a slab fracture of the third carpal bone before reduction. **B,** Lateromedial radiographic view of the slab fracture seen in **A** after reduction and fixation using a 4.5-mm cortex screw in lag fashion.

Reconstruction of Intra-Articular Fractures

Once damaged, bone can be repaired so that it is indistinguishable from the original tissue. However, articular cartilage does not have the same ability to heal. Therefore correct anatomic alignment and reconstruction of the fracture gap, especially the articular component, is integral to the repair process and determines the long-term resolution of intra-articular fractures.

Arthroscopic visualization during the reconstruction of the articular component of the fracture is recommended to ensure the best possible outcome in a high-motion joint. In many cases, loose debris is removed and flushed from around and in the fracture gap, allowing more anatomically correct reduction and better stabilization of the fracture. In some cases, such as with slab fractures of the third carpal bone, assessment of the articular component dictates the surgical procedure, namely repair or removal of the fracture fragment (Figure 80-2).

Common intra-articular fractures encountered in equine athletes are condylar fractures of the third metacarpus (MCIII) and third metatarsus (MTIII), third carpal bone slab fractures, and sagittal fractures involving the proximal phalanx. Most cases of intra-articular fractures are repaired using internal fixation with screws inserted using lag technique (see Chapter 76). These fracture repairs are covered in other chapters of the text, but some treatments and outcomes are listed in Table 80-2.

Synovectomy

Equine synovium is occasionally removed locally (partial synovectomy) to help arthroscopic visualization, but synovectomy can also be performed as a therapeutic treatment, most often in cases of septic arthritis. With sepsis, the synovial membrane can be laden with fibrin. Fibrin is thought to harbor bacteria; thus removal of the involved synovium is thought to aid in the treatment and potentially prevent recurrence of the septic process.

TABLE 80-2. Prognosis* for Racing or Athletic Soundness Given by Various Authors for Intra-Articular Fractures

Fracture Location and Type	Treatment†	Prognosis	Reference
Distal phalanx (all except fractures of extensor process fractures)	M	Good (horses <3 yr old)	8
Distal phalanx (intra-articular, except extensor process fractures)	S	Good (horses >3 yr old)	8
P3 (type II)	M	Guarded	11, 40
Middle phalanx (comminuted)	M or S	Guarded	11
	S	Poor to guarded‡	15
	M	Poor	15
Proximal phalanx (simple, nondisplaced)	M or S	Good	11, 17, 41, 42, 43, 44
Proximal phalanx (noncomminuted, displaced)	S	Good	17, 45
Proximal phalanx (comminuted)	S	Poor	11, 43
Proximal sesamoid (midbody)	M or S	Poor	34, 43
MCIII/MTIII (noncomminuted)	S	Good	34, 46, 47
MCIII/MTIII (with comminution)	S	Guarded	34
	S	Good	48
MTIII (medial condyle)	S	Good	49
MCIII/MTIII (displaced)	M or S	Poor	41
MCIII/MTIII (complete, nondisplaced)	S	Good	41
MCIII/MTIII (incomplete, nondisplaced)	S	Excellent	41
	M	Excellent	41
C3 slab (frontal)	S (TB)	Guarded to good‡	50
	S	Guarded to good‡	12, 51
		Guarded	52
	M	Poor	53
	M or S (SB)	Good	50
C3 slab (sagittal)	M	Guarded	12, 45

*The prognoses have been graded as excellent (80% to 90% chance of athletic soundness), good (60% to 80%), guarded (40% to 60%), and poor (<40%) and usually refer to the ability to race after injury. These are approximate percentages extrapolated from the literature because these categories are not always used by each author and a definition of these categories is not usually given. Favorable has been graded with good. In general, the prognosis given is for horses with no radiographic signs of osteoarthritis (i.e., the horse is given its best chance of recovery).

†Articular fractures of long bones (except for the metacarpus and metatarsus) have not been included because of insufficient numbers from which to derive prognoses or other complicating factors, or both.

‡All fractures repaired surgically with stab incisions or arthrotomies, or both, except C3 slabs, in which repair was guided by arthroscopic visualization. Two grades are given when percentages spanned two categories.

C3, Third carpal bone; *M,* medical; *MC,* metacarpus; *P,* phalanx; *S,* surgical; *SB,* Standardbred; *TB,* Thoroughbred.

Adapted from Caron JP: Principles of Treatment of Joint Disease. p. 678. In Auer JA, Stick J (eds): Equine Surgery. 2nd Ed. Saunders, Philadelphia, 1999.

Some septic equine joints appear to produce more fibrin than others (tarsocrural, distal interphalangeal, and elbow joints are among the most prolific), which requires visualization of the joint space and synovectomy rather than ingress–egress flushing using large-gauge needles. In some cases of chronic osteoarthritis, marked hypertrophy of the synovium can be encountered, and current practice is to perform a subtotal or complete synovectomy of the affected membrane to reduce cytokine production as well as physical impingement. The carpal joints and the metacarpophalangeal and metatarsophalangeal joints appear to benefit clinically from this type of treatment.[8]

The role that the synovial membrane plays in equine joint disease is not as well characterized as it is in humans. In human osteoarthritis, the synovial membrane is thought to play a secondary role to the cartilage lesion in the pathogenesis. Conversely, in rheumatoid arthritis, the synovial membrane is thought to be the primary instigator and to propagate the ongoing degeneration of the cartilage. Thus an effective method of controlling rheumatoid arthritis in people is through synovectomy, although it has a limited duration of clinical effectiveness.[9-12] A rheumatoid condition has not been identified in the horse.

Different methodologies have been used to accomplish synovectomy, including use of surgery, chemicals, and radioisotopes. Typically, surgical methods in horses use motorized synovial resection. Experimentally, synovectomy has been performed in normal horses and has shown no ill effects, but the regeneration of the synovial membrane was slower than expected when compared to other species.[8,13,14]

Joint Resurfacing

Partial-thickness lesions and full-thickness articular cartilage lesions greater than 5 mm in diameter do not heal spontaneously in the horse. In spite of the recent interest in assessing treatments for chondral defects, efforts have been under way for more than 250 years to heal articular cartilage with only moderate progress.[15] The reason is related to the highly specialized nature of articular cartilage and the need for an intact structure to perform its biochemical and physiologic functions.

Damage to articular cartilage is common in horses and typically is described in two forms: chronic degenerative lesions and acute damage. Chronic lesions are often considered OA, and whereas these lesions would benefit from joint resurfacing, this

type of lesion is the most challenging in which to achieve long-term repair because of generalized joint pathology that is not addressed with most current joint-resurfacing techniques. Conversely, acute lesions typically comprise a discrete or focal area of cartilage loss without other chronic manifestations of joint degeneration. Such lesions are currently best treated with equine joint resurfacing techniques.

The outcome of cartilage repair is typically assessed on biochemical content (including type II collagen and aggrecan), histologic appearance (resemblance to hyaline cartilage), biomechanical properties, and functional outcome of the joint and patient. As a general rule, it appears that cartilage in younger patients has a better capacity for repair compared to that of older patients.

When intra-articular osteochondral fragmentation occurs in the horse, surgical treatment involves removal of the fragment and débridement of the damaged cartilage and subchondral bone. In general, because of a generalized poor regenerative response, articular cartilage is usually minimally débrided, doing no more than is necessary to remove damaged cartilage. Partial-thickness cartilage lesions are typically not converted into full-thickness lesions, but rather the fibrillated cartilage is removed, leaving the intact cartilage below the fibrillated cartilage in place. In the case of a full-thickness lesion, the edges of the damaged articular cartilage are débrided until cartilage that is firmly attached to the subchondral bone plate is reached. The edges of the cartilage are débrided so that they have a sharp vertical border. The bone is always débrided down to a level of the subchondral bone plate so as to fully remove the calcified cartilage, which has been shown to impede the repair process. When necrotic bone is encountered, it is débrided aggressively to ensure that healthy bleeding bone is being left in the lesion.

Chapter 79 covered general concepts regarding treatment based on the lesion depth, size, and basic repair or treatment options. Two basic approaches are historically considered in joint resurfacing: stimulated endogenous repair and transplantation or grafting of tissues. Although these techniques are covered as separate entities, many cutting-edge approaches are combining the techniques as well as augmenting either or both techniques with growth factors.

Stimulated Endogenous Cartilage Repair

Because bone marrow has a good supply of both stem cells and growth factors thought to be integral to cartilage health and repair, direct communication of articular lesions to these elements beneath the subchondral bone plate has been a cornerstone of stimulated cartilage repair. Growth factors believed to be important in cartilage repair include insulin-like growth factor-1 (IGF-1), transforming growth factor-β (TGF-β), and bone morphogenetic proteins (BMPs) 2 and 7. Access to these marrow elements has been facilitated by various surgical techniques, including *abrasion arthroplasty*, which involves débridement to the level of the subchondral bone plate; *spongialization*, which is débridement past the subchondral plate into cancellous bone; focal drilling to the depth of cancellous bone in discrete locations throughout the cartilage lesion (*osteostixis*); and subchondral bone *microfracture*, which also penetrates to the level of the subchondral bone in discrete locations. Current literature and clinical practice do not favor spongialization, in part because it is thought to destabilize the subchondral bone plate.[16,17]

Based on the human and equine literature, the current recommendations for stimulated endogenous repair of an articular lesion are débridement of lesions to the level of the subchondral bone plate (abrasion arthroplasty) alone or in conjunction with subchondral bone microfracture.[17] If the lesion crosses the subchondral bone plate into the cancellous bone, the addition of subchondral bone microfracture is probably not necessary. In the presence of sclerotic bone, the lesion is débrided to a depth that produces petechial bleeding (in the absence of fluid pressure) but does not enter the cancellous bone. In this case, subchondral bone microfracture is also used.

Currently, subchondral bone plate drilling is not widely used based on subsequent formation of subchondral bone cysts and poor histologic appearance of repair tissue, especially when compared to other, more recently developed techniques such as subchondral bone microfracture.[18,19] The greater depth of penetration, smooth penetration through the bone, and heat generated with the drilling process probably contribute to the less-than-optimal tissue repair. Bone cyst formation associated with the drilling technique is probably a result of synovial fluid movement into the bone through the drill holes (see Chapter 89).

Subchondral bone microfracture allows access to the cells and growth factors beneath the subchondral plate, without disrupting the subchondral plate's biomechanical stability (Figure 80-3). In addition, the penetration of the stainless steel bone awl causes cracks in the bone as well as spicules of bone that protrude from the penetration site, both of which are believed to aid in the attachment of the repair tissue. Experimental studies have demonstrated that large articular cartilage defects (1 to 2 cm in diameter) débrided to the level of the subchondral bone have significantly greater volume of healing tissue following subchondral bone microfracture compared to defects that were débrided to the level of the subchondral bone plate but did not undergo microfracture. Biochemical analysis of the repair tissues also has shown a greater type II collagen content in repair tissue of microfractured defects in two experimental

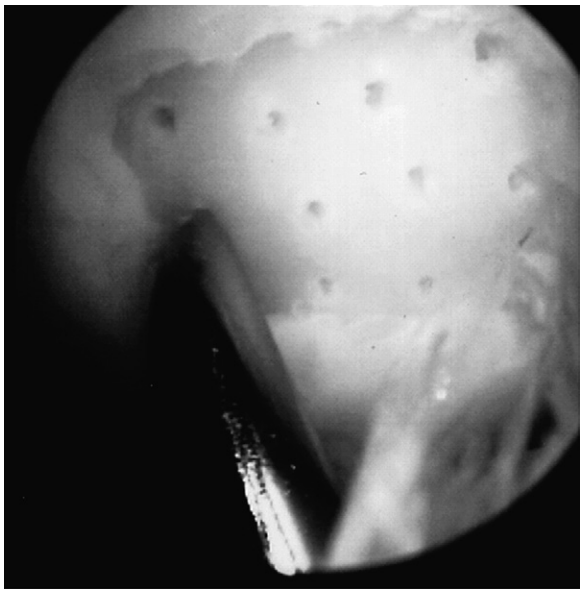

Figure 80-3. Arthroscopic image of microfracture spacing on the medial femoral condyle.

studies, although histologic appearance of the repair tissues was similar.[20,21]

Further improvement in the repair tissue obtained following subchondral bone microfracture has been achieved by supplementing IGF-1 and interleukin-1 (IL-1) receptor antagonist using gene transfer.[22] Experimental work assessing subchondral bone microfracture has also confirmed the poor attachment of repair tissue in areas where the calcified cartilage layer had been incompletely removed. Confirmation of the level of débridement can be achieved using a microarthroscope or focusing close to the defect margins with a standard arthroscope. A granular appearance of the defect should be evident, differentiating the subchondral bone plate from the glasslike appearance of the calcified cartilage layer. Following débridement of the lesion, microfracture holes are spaced 2 to 3 mm apart, avoiding communication between sites and penetrating approximately 2 mm into the bone (see Figure 80-3).

To date, no long-term follow-up on clinical results after resurfacing therapy have been published relating specifically to horses, although anecdotal reports have been promising. However, human data compared long-term follow-up of the most commonly used cartilage resurfacing techniques: autologous cartilage implantation (ACI) as described by Brittberg,[23] and subchondral bone microfracture first described by Steadman.[24,25] The short-term and long-term results of this study show minor significant improvement with subchondral bone microfracture over ACI but no significant differences in histologic appearance of repair tissue or patient outcome between the two techniques. Both of these techniques are considered better than débridement alone for most human and equine patients. Because subchondral bone microfracture does not require a second surgery and is less technically challenging compared to ACI, it is favored by most equine surgeons.

Articular Cartilage Grafting

Many different tissues have been transplanted or grafted into cartilage defects; they include periosteal and perichondrial autografts, osteochondral, chondral, or isolated chondrocyte autografts or allografts, and stem cell transplants from bone marrow or fat. Periosteal and perichondrial grafts have been performed in laboratory animals with some success, but results in the horse have been very disappointing and are no longer a focus of ongoing research.[26,27]

Osteochondral grafting procedures have been well developed for use in people, but they have had limited success in the horse. Early work in the horse demonstrated short-term success but resulted in long-term failures.[28-30] Many of the failures with osteochondral grafting have been attributed to lack of congruity of the recipient and donor tissues as well as difficulties with surgical technique. Some concern also revolves around morbidity in the donor graft site. Typically, in people, a non–weight-bearing region is used for donor harvest, reducing morbidity, but lack of suitable non–weight-bearing donor tissue has been a limitation in horses. More recently, with the advent of specialized surgical tools designed for human osteochondral grafting, studies are under way using this technique in the horse. Surgical technique and donor site selection are the main hurdles yet to be overcome before this technique reaches mainstream practice.[31-34]

Chondrocyte transplantation has been a very active area of equine research since the 1990s. Techniques using both allografts and autografts have been reported, but most work, especially in humans, has focused on autografts. The technique described by Brittberg and marketed by Genzyme is the most well studied grafting technique.[23] This technique uses autologous chondrocytes harvested from a non–weight-bearing region, usually the trochlea of the distal femur, followed by a 4-week *in vitro* expansion of chondrocytes. The expanded cell population is then implanted during a second surgical procedure and held in the defect beneath autologous periosteum, which is sutured to the cartilage bordering the defect to create a watertight seal. Although this technique has been performed in horses with outcomes similar to those seen in people, the cost, laboratory facilities, need for multiple surgeries, and technical challenges of the procedure have limited its usefulness in clinical cases.

Techniques using frozen chondrocytes harvested from neonatal foals, which are implanted in a fibrin glue to help retain the cells in the chondral defect, have had some success in a limited number of chondral defects. The technique is being used more commonly in cystic defects to date.[35] Other materials used to fill cystic lesions are discussed in Chapter 89.

Techniques using autologous chondrocytes harvested from the non–weight-bearing region of the lateral trochlea of the distal femur, implanted into 15-mm-diameter defects, have been successful in experimental equine trials. One of the tested procedures uses fibrin glue holding minced cartilage to a bioresorbable scaffold, which is subsequently stapled to the subchondral bone of the defect in a one-step surgical procedure.[36,37] In comparable equine experimental trials this technique has been superior to the ACI technique. This technique is now undergoing human clinical trials, and because of the ease, cost, and promising results in equine experimental trials, it is likely to be used in equine clinical cases in the near future.

Considerable research is being directed toward the use of *mesenchymal stem cells* for implantation in cartilage defects. This cell population has been shown to improve healing in experimental animals, but gaining access to a sufficient number of stem cells without *in vitro* expansion is a hurdle yet to be resolved in horses.[35]

Arthrodesis

Assisted fusion of a joint is sometimes indicated when destruction to the joint is beyond any other treatment. Although most commonly achieved through surgical methods typically involving internal fixation, arthrodesis can be carried out using chemical or laser-based methods as well.[2,38-40]

In high-motion joints, such as the antebrachiocarpal, midcarpal, metacarpophalangeal, and distal interphalangeal joints, surgical fusion using internal fixation is required (see Chapter 81). The expectation of this procedure is to alleviate the pain associated with movement of the joint and to salvage the animal for nonathletic function. Conversely, arthrodesis of low-motion joints often carries a reasonable prognosis for athletic soundness and can be accomplished using surgical, chemical, or laser-based techniques. This is especially true for the proximal interphalangeal, tarsometatarsal, carpometacarpal, and distal intertarsal joints. Some risks have been identified with chemical fusion of joints because unexpected anatomic communications with other synovial and nonsynovial structures can be encountered. Thus contrast studies outlining the structure and extent of the communication should be performed before chemical fusion, and caution should be used not to create further injury by overdistending the joint capsule.

In the distal tarsal joints, some initial studies have been performed comparing surgical drilling to ablation of the joint space using a laser in both clinical and experimental cases.[41] Early results indicate that some decrease in time to resolution of clinical lameness may be seen for the laser-based procedure, but actual bony union of the joint space appears to occur more quickly with drilling. Further research comparing these techniques is needed, but the morbidity associated with the early reports using the laser favors the continued use of surgical arthrodesis in the distal tarsal joints, using the modified drilling technique. Further discussion of the principles of arthrodesis can be found in more detail in Chapter 81.

Joint Replacement

When reparative surgical procedures have failed, and the patient is unresponsive to medical management, many end-stage cases of osteoarthritis in people are treated using joint replacements, especially in the knee and hip. Joint replacement procedures are typically postponed until the patient is old enough that the implant will not fail during the expected remaining lifetime. Implant wear is improving, but it is still in the 10- to 20-year range, depending on the location and type of implant. Even in humans, where a large number of joint replacements are carried out annually, and to a lesser extent in dogs, relatively high morbidity is associated with such procedures, especially related to implant loosening. With respect to equine joint replacement, cost of implant design and manufacturing, difficulties with prolonged non–weight-bearing, and surgical morbidity continue to keep equine joint replacement out of mainstream clinical practice.

Aftercare

The aftercare for surgical joint treatment is often as important as the central treatment itself. Treatment goals and pathophysiology of healing tissue should be kept in mind when the aftercare protocol is being designed. Each aftercare protocol varies depending on the procedure, but a common focus is to minimize the duration to full return to function without compromising the athletic soundness of the patient. Following surgery, the goals are to provide support to the weakened areas, often through bandaging, casts, and controlled exercise, which can range from strict stall rest to specific graded exercise programs.

It is also well accepted that decreasing postoperative inflammation is beneficial in the healing process, as is controlling postoperative pain. Given that maximal inflammation in a surgical wound typically occurs 3 to 5 days postoperatively, nonsteroidal anti-inflammatory medication is administered systemically for at least this time period. The postoperative use of corticosteroids, although providing potent anti-inflammatory activities, is often contraindicated in the first 4 weeks because of their role in decreased cell metabolism, which affects healing time, as well as the decreased ability of the immune system to combat infection.

Other medications believed to have chondro-enhancing or chondroprotective qualities, such as hyaluronan or polysulfated glycosaminoglycans (Adequan), have also been studied in an attempt to demonstrate enhanced postoperative healing. However, they have not shown significant benefit in most studies, with the exception of decreasing adhesions in tendon sheaths.[42-44]

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CHAPTER
81 Arthrodesis Techniques

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Arthrodesis is a salvage procedure that is used for relief of pain associated with severe degenerative joint disease, stabilization of a limb after loss of supporting soft tissue structures, and/or treatment of complicated fractures involving a joint.

By definition, ankylosis involves fusion of bones of a joint damaged by disease or injury, or it may be performed intentionally through a surgical procedure.[1] A genetic predisposition for natural or spontaneous ankylosis in horses has been suggested.[2,3] Procedures that aim to accelerate bony fusion in a particular joint by removing the articular cartilage or destroying chondrocytes without providing additional fixation to the joint may be categorized as facilitated ankylosis techniques. The most commonly used technique is mechanical removal of cartilage, which can be achieved by drilling across the joint space or removing the cartilage with a curet or a motorized burr. Other techniques to induce ankylosis include the intra-articular injection of chemical substances (e.g., monoiodoacetate, ethyl alcohol), the direct application of laser[4] or electrosurgical energy to the cartilage or, as recently investigated *in vitro*, a hydrothermal intra-articular lavage procedure.[5] Arthrodesis, which is another type of iatrogenically induced ankylosis, typically involves cartilage removal and surgical stabilization of the joint to promote fusion of the joint surfaces.

In selected high-motion joints, surgical arthrodesis is carried out to salvage valuable breeding stock or pet horses. However, certain low-motion joints can be fused without unduly compromising an athlete's performance.[6]

GENERAL GUIDELINES

Arthrodesis is most successful when two cancellous bone surfaces are aligned, compressed, and stabilized to faciltate fracture healing.[7] For any arthrodesis, as much cartilage as possible should be removed, allowing maximum bone-on-bone contact, because articular cartilage inhibits vascular invasion and ossification.[7-11] Immobility of a joint with persistent articular cartilage results in malnutrition of the cartilage and its eventual degeneration. However, this is a very slow process, and islands of articular cartilage can persist between bone bridges across a joint for several years. Therefore, in low-motion joints, such as

in the small tarsal joints[11] or the carpometacarpal joint,[12] partial removal of cartilage by drilling along the joint surface produces a functional ankylosis with relief of pain symptoms, although the convalesence time and the radiographic fusion of the joint is highly variable. This technique involves drilling several 2.5- to 3-mm-diameter holes across the dense subchondral bone plate of each of the opposing bone ends. This type of osteostixis promotes ingrowth of blood vessels across the bone plate and accelerates fusion.

A cancellous bone graft or an alternative osteoinductive material (see Chapter 77) may be used to improve bone fusion. However, if a cancellous bone graft is used, it should be loosely packed around the arthrodesis site and not squeezed between opposing bone ends, because this will mechanically damage viable cells.

Joint immobility and compression are best accomplished by placing a combination of multiple transarticular screws in lag fashion and applying bone plates. Generally, it is believed that increased stability of the fixation may improve postoperative comfort and minimize patient morbidity. Newer locking implants seem to be considerably more stable, which may facilitate the use of less-invasive arthrodesis techniques and therefore further improve the success rate of these procedures.[13,14]

DISTAL INTERPHALANGEAL JOINT

Indications

Indications for arthrodesis of the distal interphalangeal joint (DIP) are rare. This surgical intervention represents a salvage procedure and does not permit the patient to assume any form of athletic competition. The indications include severe osteoarthritis, injuries to the collateral ligaments that result in joint instability, luxation of the DIP joint after rupture of the deep digital flexor tendon at the level of the distal sesamoid bone, chronic intra-articular fractures, and septic arthritis.

Surgical Techniques

Arthrodesis of the DIP joint in adult horses has been reported using three transarticular 5.5-mm cortex screws[15,16] or stainless steel baskets with transarticular 4.5-mm cortex screws.[17] Facilitated ankylosis and immobilization in a half-limb cast for 50 days (initially for 4 weeks in a transfixation cast) resulted in complete ankylosis in a 3-month-old foal with septic arthritis.[18]

The two recommended techniques are described later.

Dorsal Hoof Wall Approach

We prefer a lag technique through a dorsal hoof wall approach.[15] For this arthrodesis, the hoof capsule is thoroughly cleaned the day before the procedure, including removing the superficial layer of horn with a rasp. The hoof is subsequently scrubbed using antiseptic products and covered with a sterile bandage.

The surgery is performed the following day with the horse in lateral recumbency with the affected limb positioned uppermost. The articular cartilage is removed arthroscopically using a 5.5-mm spherical burr through both a dorsal and a palmar/plantar approach. It is a difficult and time-consuming procedure to remove the cartilage from the palmar/plantar aspects of the joint, but 80% of cartilage could be removed using both arthroscopic approaches.[15] A 10-mm drill bit is used to create three parallel holes in the dorsal aspect of the hoof wall under fluoroscopic guidance, one axially, one dorsomedially, and one dorsolaterally. With the 5.5-mm drill bit, a glide hole is prepared across the distal phalanx in a dorsodistal to palmaro/plantaroproximal direction through each of these hoof wall openings. The beginning of the glide hole is at the junction of the proximal to the middle third of the distance from the extensor process to the tip of the distal phalanx. The glide hole is aimed toward the center of the distal articular surface of the middle phalanx. Accuracy of the depth and location of the glide holes can be observed arthroscopically through the dorsal pouch of the DIP joint.

Subsequently the 4.0-mm drill sleeve is inserted into the center glide hole and used to align the distal and middle phalanx in a normal weight-bearing position, then the thread hole is drilled across the middle phalanx. Threads are hand tapped and the three 5.5-mm cortex screws inserted and firmly tightened (Figure 81-1). Autogenous cancellous bone grafts may be placed through the dorsal and palmar/plantar arthroscopic portals into the joint space. The hoof wall defects are filled with polymethyl methacrylate impregnated with 1 g gentamycin sulfate, and the surface edges are sealed with cyanoacrylate. Perioperative intravenous regional perfusion with antimicrobials (with the horse still on the surgery table) is recommended to reduce the risk of postoperative infection. The horse is recovered in a half-limb cast, which should be maintained for approximately 2 months. Radiographic evidence of complete fusion of the joint was observed 8 months postoperatively in one horse.[15] At that time the horse was sound at walk.

Palmar/Plantar Approach

An alternative, but more invasive technique has been described using two surgical approaches.[16] Through a dorsal approach to the DIP joint, as much articular cartilage as possible is removed. An 8-cm (3-inch) semicircumferential horizontal skin incision is made 1 cm proximal and parallel to the coronary band, centered over the midline of the foot. The incision is extended through the common digital extensor tendon into the dorsal pouch of the DIP joint. The incision is further extended through the joint capsule, transecting the collateral ligaments, allowing subluxation of the joint. Approximately 70% of the articular cartilage can be removed with a curet through this approach, but the distal sesamoid bone and the palmar aspect of the middle phalanx cannot be accessed for cartilage removal.

Then the implants are inserted through a palmar approach to the proximal and palmar/plantar aspect of the middle phalanx, entering the tendon sheeth of the deep digtal flexor tendon. The initial 8-cm skin incision parallel to the long axis of the limb in the median plane centered over the proximal interphalangeal (PIP) joint is extended into the digital tendon sheath, where the deep digital flexor tendon is transected and reflected. Three stab incisions are made through the distal sesamoidean ligament and the insertion of the superficial digital flexor tendon, respectively, to provide access to the proximal palmar/plantar rim of the middle phalanx.

While maintaining the DIP joint in reduction, the first thread hole is prepared under fluoroscopic control across the middle phalanx and DIP joint in a distodorsal direction into the distal phalanx. The thread hole across the middle phalanx is enlarged into a glide hole, with the joint opened to prevent inadvertent

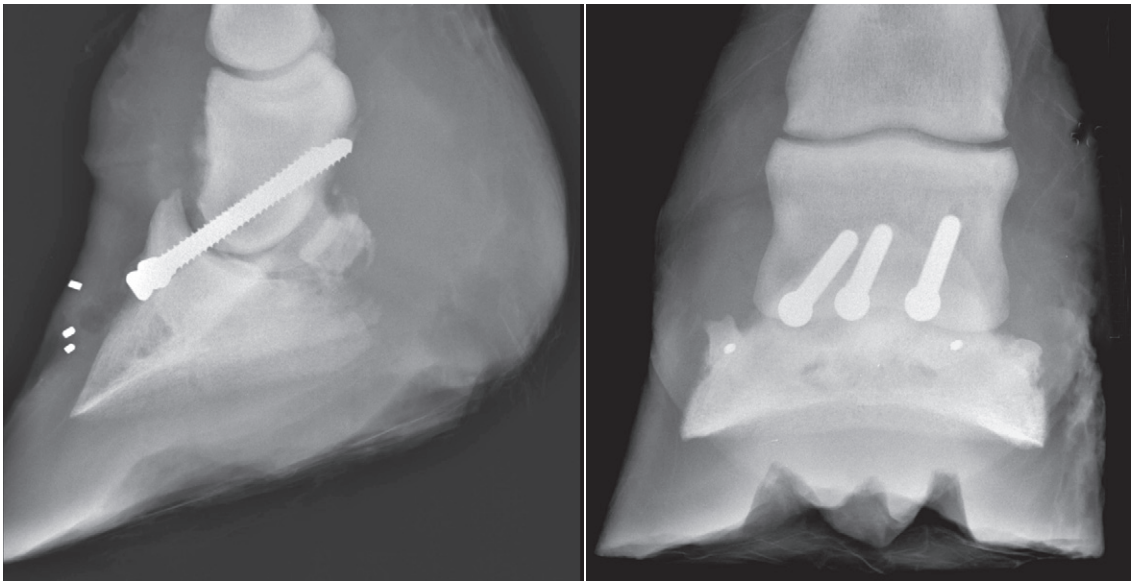


Figure 81-1. Arthrodesis of the distal interphalangeal (DIP) joint using three parallel 5.5-mm cortex screws placed in lag fashion through a dorsal hoof wall approach. Note the metallic markers in the hoof wall that served as landmarks for screw placement.

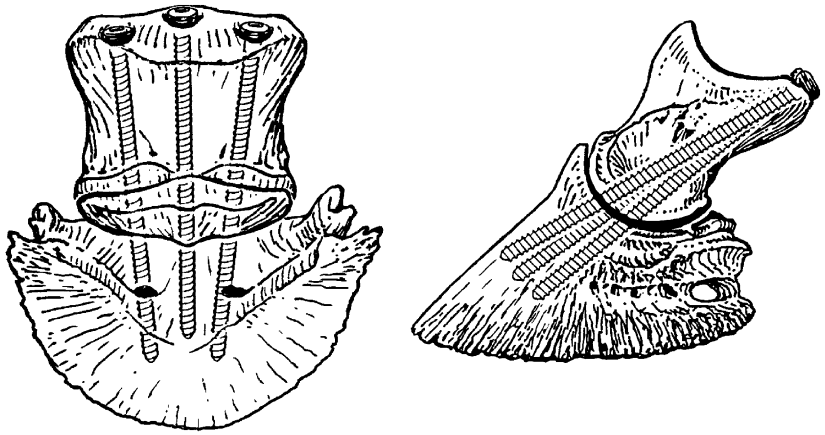


Figure 81-2. Graphic illustration of an arthrodesis of the DIP joint using three parallel 5.5-mm cortex screws placed between the proximal and palmar aspect of the middle phalanx and the distal phalanx.

enlargement of part of the thread hole in the distal phalanx. Two more glide holes are drilled parallel to the first one across the middle phalanx. The center hole is finished using lag technique, and a 5.5-mm cortex screw of correct length is inserted and solidly tightened. The other two 5.5-mm cortex screws are implanted using lag technique (Figure 81-2). A cancellous bone graft or an alternative osteoinductive material may be placed in the dorsal and the palmar/plantar aspect of the joint before closing the joint.

The dorsal incision is closed in three layers: extensor tendon and joint capsule, subcutaneous tissue, and skin. Although an attempt should be made to suture the ends of the deep digital flexor tendon with interrupted sutures of 0 polydioxanone, the tendon ends often retract, making accurate anatomic reconstruction impossible. The palmar incision is closed in two layers: tendon sheath and subcutaneous tissues in one layer, followed by the skin layer. The limb is placed into a half-limb (transfixation) cast for 3 months, ideally with only one cast change. Routine anti-inflammatory therapy and antibiotic prophylaxis are used.

In one horse that suffered from nonresponsive progressive arthritis of the DIP joint, this technique resulted in solid fusion. The horse was able to ambulate with a mechanical lameness but was pain free for more than a year at the time of reevaluation.[16]

The advantages of the dorsal hoof wall approach over the two-incision technique are the minimal soft tissue damage and the greater purchase of the screws, because the thread hole in the middle phalanx is much longer compared to the short thread hole in the distal phalanx, when screws are placed in proximal-palmar/plantar to distal-dorsal direction. The disadvantage of the dorsal hoof wall technique is associated with the hoof wall holes that have to be prepared and may lead to infection. With a more widespread use of this technique, this potential danger can be better assessed.

PROXIMAL INTERPHALANGEAL JOINT

Indications

Indications for arthrodesis of the PIP joint include osteoarthritis (high ring-bone), comminuted fractures of the proximal or middle phalanx, and luxation or subluxation of the PIP joint.[6,19-21]

Because of the low-motion[22] and high-load nature of this joint and the lack of interdigitation, attempts to manage the majority of common conditions of the PIP joint by methods

other than arthrodesis usually fail to provide long-term success. Unlike arthrodesis of high-motion joints, the goal of PIP joint arthrodesis is to return the horse to athletic performance.

Fractures entering the PIP joint through either the proximal or the middle phalanx often result in osteoarthritis or subluxation. Therefore arthrodesis is often performed as part of the definitive treatment of the fracture. In comminuted fractures of the proximal phalanx, the middle phalanx serves as the distal fragment, achieving additional stability for the fixation. Conversely, the proximal phalanx can serve as the proximal fragment in comminuted middle phalanx fractures.

Lag Technique

Over the years, a variety of techniques have been advocated for PIP joint arthrodesis.[16-19] The first technique routinely applied since the 1980s used two or three transarticular cortex screws placed in lag fashion in a parallel or diverging pattern.[16-18]

A *cadaveric* study compared interfragmentary cortex screws applied in lag fashion in forelimbs and hindlimbs.[23] Three parallel 4.5-mm cortex screws were compared to two parallel 5.5-mm cortex screws in three-point bending to failure. The bending moments were greater in the forelimbs than in the hindlimbs. The difference between the two techniques was not statistically significant. However, more 4.5-mm cortex screws failed than 5.5-mm counterparts. Further cadaveric studies revealed that two parallel 5.5-mm cortex screws provided an equally strong PIP joint arthrodesis compared with three 5.5-mm cortex screws inserted in a similar manner, when tested in three-point bending to failure[24] and in cyclic loading.[25] In these studies, no discernible mechanical difference between forelimb and hindlimb constructs was observed. Insertion of two parallel headless, tapered, varible-pitched titanium compression screws were biomechanically comparable with two parallel 5.5-mm stainless steel cortex screws inserted in lag fashion across the PIP joint.[26] In contrast to previous studies, forelimb constructs were significantly stiffer than hindlimb constructs in this testing series using 8 forelimbs and 12 hindlimbs.

The lag technique may be the fixation technique of choice for PIP joint arthrodesis in young foals.[27] Recent clinical case series suggest that the lag screw technique remains a viable option for arthrodesis[28-31] of adult horses with severe osteoarthritis and significant periarticular new bone formation. This is especially true when the procedure is performed through a minimally invasive approach, such as inserting the screws through stab incisions[29a,30] and in combination with laser-facilitated, minimally invasive cartilage destruction.[31]

However, insertion of three transarticular screws in lag fashion using the conventional open approach increases the need for cast support when compared with plating techniques for PIP joint arthrodesis.[31] The almost linear alignment of the screws in the frontal plane provide minimal stability, especially at the dorsal aspect of the joint. This results in patient discomfort because of excessive new bone formation impinging on the extensor tendon or the coffin joint, or both.[32] This presumably reflects decreased stability.[33]

Combination of a Plate and Lag Technique

An arthrodesis technique using an axial plate with two abaxial transarticular cortex screws inserted in lag fashion was tested *in vitro* and compared to a method using three transarticular screws. There was a substantial increase in stability across the dorsal aspect of the PIP joint in addition to a significantly longer fatigue life compared to the three-transarticular screw method.[33,34]

The combination of transarticular screws and dorsal plate has a biomechanical advantage over a single plate: it provides compression across the entire joint because the tensile forces at the palmar aspect of the joint induced by the plate are neutralized by the two oblique transarticular screws inserted in lag fashion. It has a similar advantage over the three-screw technique because the dorsal joint instability seen with that technique is obviated by the plate.

Positive clinical results achieved on horses suffering from osteoarthritis, subluxations, and some stable middle phalanx fractures support the *in vitro* results.[31] Postoperatively, the patients were more comfortable on the limb, and the cast was removed earlier compared to patients treated with the three parallel 5.5-mm cortex screws.[31]

Other attempts to improve stability induded the use of two plates,[35] a T-plate,[36] a Y-plate,[37] and most recently a spoon plate,[38] but currently the best clinical results have been reported by using a dorsal three-hole narrow dynamic compression plate (DCP)[39] or locking compression plate (LCP)[14] combined with two transarticular abaxial 5.5-mm cortex screws inserted in lag fashion. Therefore we recommend this technique for arthrodesis of the PIP joint with an open approach. The potential increase in stability achieved in a clinical case has to be weighed against the cost of the implants and the surgical trauma. If these aspects are considered, the recently developed PIP arthrodesis plate is the implant of coice.

Surgical Techniques

APPROACH

The joint is approached through an inverted-T skin incision that begins just distal to the level of the metacarpophalangeal (MCP) or the metatarsophalangeal (MTP) joint and ends at a horizontal skin incision made 2 cm proximal to the coronary band. The horizontal incision extends 4 cm (1½ inches) on either side of the midline. The subcutaneous tissues are separated sharply down to the common digital extensor tendon. The two triangular skin flaps are dissected free from the common digital extensor tendon with the help of scissors and sutured to the intact skin on either side of the incision.

The common digital extensor tendon is transected with an inverted V-shaped tenotomy at the level of the insertion of the extensor branches of the suspensory apparatus (Figure 81-3).[40] The proximal tendon stump is reflected proximad using scissors, taking care to spare the attachments of the extensor branches of the suspensory ligament. The distal tendon stump is sharply incised along its lateral and medial sides and reflected distad, exposing the dorsal pastern region. If necessary, bony proliferations on the dorsal aspect of the pastern are removed with a chisel and a mallet.

The dorsal attachments of the PIP joint capsule are sharply incised parallel to the joint surface, the medial and the lateral collateral ligaments are transected, and the joint is disarticulated. As much cartilage as possible is removed from both articular surfaces with a curet. By removing the articular cartilage layers, the radii of the two opposing bones are altered (Figure 81-4, *A*); the radius of the proximal phalanx is reduced, whereas that of the middle phalanx is increased. Theoretically, this results in reduced contact between opposing bones (see Figure

81-4, *B*). By rasping the subchondral bone of the distal articular surface of the proximal phalanx, the contact area can be increased (see Figure 81-4, *C*). Osteostixis of both subchondral bone plates is performed at 0.5-cm intervals using a 2.5-mm diameter drill bit.

ARTHRODESIS

The preferred technique for arthrodesis of the proximal interphalangeal joint involves the application of a 3-hole narrow plate with two additional transarticular cortex screws. Although the technique was originaly described with a DCP (Figures 81-5 and 81-6),[33] the introduction of the LCP concept led to the development of a PIP arthodesis LCP.[14] For more information on the LCP technology, please review Chapter 76. This plate has a wider space between the stacked combi-hole (most distal hole), usually placed over the proximal aspect of the middle phalanx, and the remaining two regular combi-holes. The principles and steps for implant application are the same for either plate and are described later with particular details for the LCP.

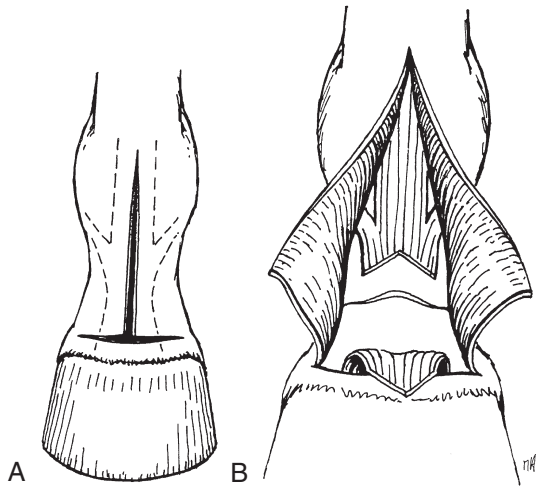


Figure 81-3. A, The surgical approach for arthrodesis of the proximal interphalangeal joint involves an inverted-T skin incision with the horizontal portion located 1.5 cm proximal to the coronary band. A sagittal vertical incision is extended to the level of the metacarpophalangeal joint. **B,** An inverted-V tenotomy of the common digital extensor tendon, just distal to the insertion of the extensor branches of the suspensory ligament, allows access to the joint. The distal part of the tendon (shaded vertically) is reflected distally, whereas the proximal part (also shaded vertically) is still in its original position.

The drill sleeve for the 5.0-mm locking head screw is fastened to the stacked combi-hole and the previously contoured 3-hole LCP is placed axially over the joint, with the stacked combi-hole positioned at the proximal and dorsal aspect of the middle phalanx. By positioning the plate as far proximad as possible, interference between the extensor process of the distal phalanx and the distal end of the plate is prevented. Additionally, the risk of inadvertent injury to the distal sesamoid bone at the palmar or plantar aspect of the middle phalanx is reduced. With the joint opened, allowing a good view along the axial ridge of the proximal articulation of the midddle phalanx, the 4.3-mm thread hole is drilled across the bone and followed by insertion of the 5.0-mm locking screw using the power-tapping technique. The screw is not completely tightened.

With the phalanges maintained in neutral position, the location of the two transarticular screws are marked on either side of the plate on the distal aspect of the proximal phalanx, making sure to leave enough space for countersinking the bed for the screw heads.

With the joint opened, drilling of the 5.5-mm glide hole for the first transarticular screw to be inserted on one side of the plate is started perpendicular to the bone surface of the proximal phalanx, followed by a gradual redirection of the drill bit to the desired oblique orientation. Because the middle phalanx is wider than the proximal phalanx, the drill holes are prepared in slightly diverging directions (dorsoaxial to palmaro/plantaroabaxial). Drilling is continued until the drill bit enters the joint to ensure an ideal lag effect without drilling into the palmar and proximal aspect of the middle phalanx. The hole should enter the joint midway between the dorsal and palmar/plantar cortices. The same procedure is repeated on the other side of the plate for the second lag screw.

The countersink is used to prepare depressions in the bone for the heads of the transarticular screws. Care is taken to work predominantly on the proximal rim. The joint is closed to its normal anatomic position and the plate is aligned axially between the glide holes. The load drill guide is placed in the dynamic compression unit (DCU) portion of either the middle or proximal combi-hole of the plate, and then a perpendicular 4-mm thread hole is prepared across the proximal phalanx. The selected 5.5-mm cortex screw is inserted following final preparation of the thread hole, but it is not fully tightened.

The thread hole for one of the transarticular screws is drilled, the hole is tapped, and the screw is inserted and tightened. The second transarticular screw is implanted in the same manner

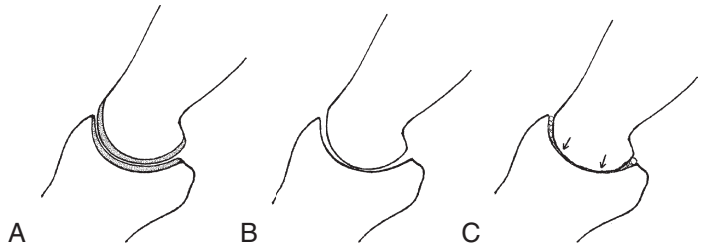


Figure 81-4. A, The articular cartilage covering the two opposing bones normally allows congruency of the surfaces during joint loading and maximum contact surface. **B,** Removal of the articular cartilage from the bone ends results in an incongruity between the two surfaces. Insertion of three lag screws after this type of preparation results in an inadequate fixation because of cyclic screw failure. **C,** Rasping of the distal subchondral surface of the proximal phalanx *(between the two arrows)* substantially enlarges the contact surface. Cancellous bone may be placed between the two bone ends.

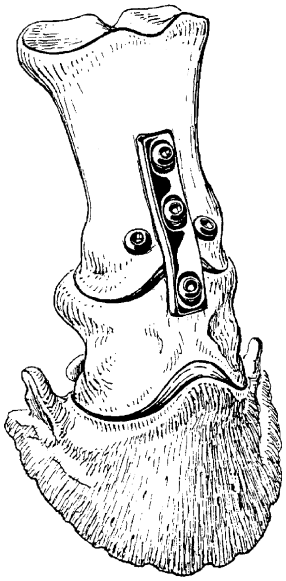


Figure 81-5. Graphic illustration of an arthrodesis of the proximal interphalangeal joint, using a 3-hole narrow dynamic compression plate (DCP) and one 5.5-mm cortex screw applied in lag fashion across the joint on either side of the plate. The plate is applied axially over the dorsal aspect of the joint with the most distal screw inserted into the proximal aspect of the middle phalanx.

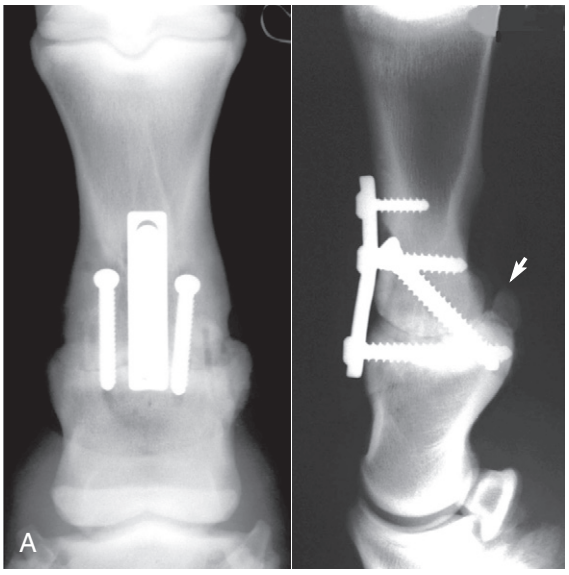
on the other side of the plate. The two plate screws are now tightened as well, bringing about transarticular compression. Finally, a second 5-mm locking head screw is inserted orthogonally across the bone through the threaded part of the remaining empty combi-hole and tightened (Figure 81-7).

CLOSURE AND AFTERCARE

Closure of the V-plasty of the tendon is accomplished with size 1 polydioxanone suture material using a simple-continuous pattern, and the skin is apposed using a combination of size 0 polypropylene (Prolene) and skin staples. A standard distal limb cast is applied for the immediate postoperative period to protect the fixation during recovery and, most importantly, to support healing of the soft tissues. The cast is removed at about 2 weeks, and every other skin suture is taken out at that time, with the patient standing. The limb is protected with a three-layer Robert Jones half-limb bandage, and the remaining sutures are removed at the next bandage change. In the absence of complications, the horse can be discharged from the clinic about 3 weeks postoperatively with instructions to maintain the bandage for another 3 weeks. The owner is advised to confine the horse for 3 months in the box stall. During the first 6 weeks, grazing in hand is allowed. A progressive program of hand-walking exercise is instituted during the second 6 weeks of confinement. A minimum of 3 additional months of paddock exercise is recommended before retintroducing the horse to riding activities.

Proximal Interphalangeal Joint Arthrodesis for Fracture Repair

When treating fractures of the middle or proximal phalanx with a PIP joint arthrodesis technique, it may be advisable to apply two plates, one dorsomedially and one dorsolaterally, once the



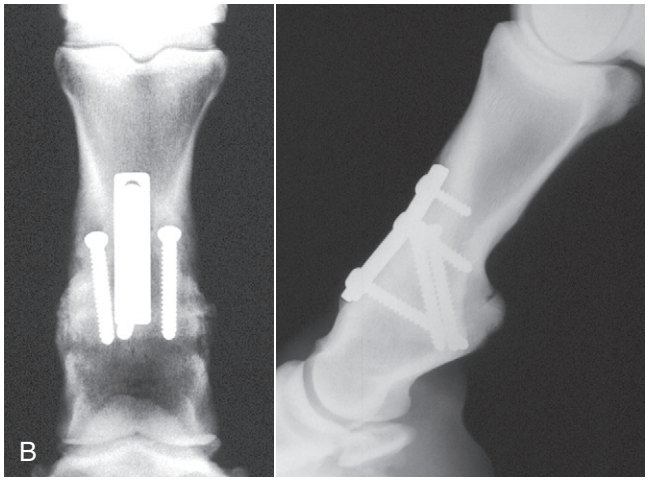


Figure 81-6. A, Immediate postoperative dorsopalmar and lateromedial radiographs of a PIP joint arthrodesis in a 2-year-old Thoroughbred filly with a proximal palmar fracture of the middle phalanx. The arthrodesis was accomplished with a 3-hole narrow DCP and two 5.5-mm cortex screws. The plate used two loaded bicortical 5.5-mm screws and a single unicortical 4.5-mm screw in the proximal hole. The two transarticular 5.5-mm cortex screws were inserted in lag fashion to provide plantar compression. The *arrow* points to the palmar fragment of the middle phalanx. Note the slightly radiolucent osteostixis holes in the distal aspect of the proximal phalanx. **B,** The 11-month follow-up dorsopalmar and lateromedial radiographs show complete fusion of the joint, with negligible periarticular new bone formation. (Courtesy J. Watkins, Texas A&M University.)

bone has been reconstructed with strategically placed screws using lag technique.[41,42] Care is taken to prevent interference of the interfragmentary screw heads with the plates. Therefore, 3.5-mm cortex screws are often used because they permit the screw heads to be buried within the cortex.

A biomechanical *in vitro* study compared two seven-hole, 3.5-mm broad DCPs with two 5-hole 4.5-mm narrow DCPs. The DCPs were placed dorsomedially and dorsolaterally, respectively. No significant difference was detected either in composite stiffness or maximal bending moment between the two fixation

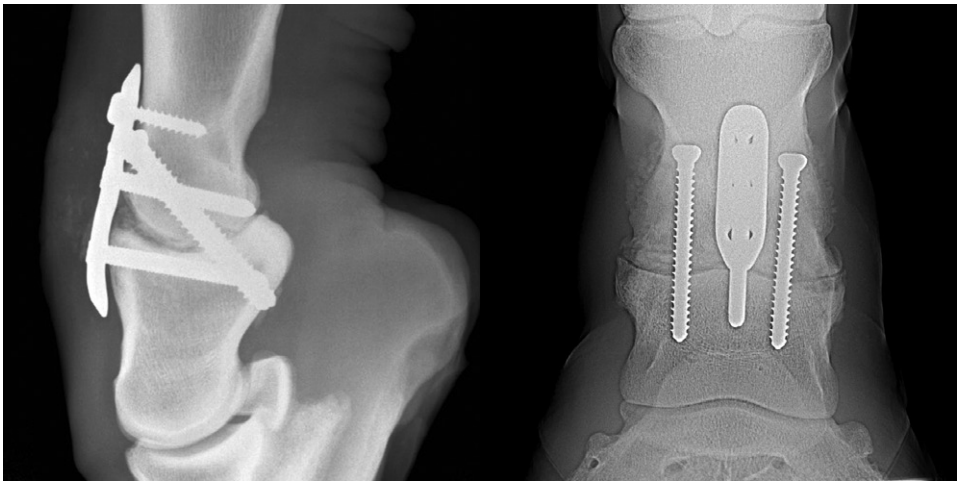


Figure 81-7. Postoperative lateromedial and dorsopalmar radiographic views of a 3-hole PIP arthrodesis LCP application combined with two transarticular 5.5-mm cortex screws. This represents the present state-of-the-art surgical technique.

techniques.[35] Therefore either plate size could be used for arthrodesis of the PIP after fractures to the pastern.

The closure of soft tissues is routine. The type of external coaptation is dictated by the complexity of the fracture and the quality of repair. For complex fractures, a transfixation cast might be necessary. The time period will vary depending on stability of the repair and progress of healing.

Minimally Invasive Appliction of LCPs

Horses with fairly advanced osteoarthritis with significant cartilage loss are candidates for minimally invasive plate application using an LCP technique.[13] This technique is more difficult and requires good intraoperative imaging facilities for cartilage removal and implant placement. Although in the PIP joint it is difficult to remove a large amount of cartilage with drill bits, a combination of lateromedial and dorsopalmar/plantar passes with a 5.5-mm drill bit seems to result in adequate cartilage destruction.

The two 5.5-mm transarticular screws are first placed in lag fashion through stab incisions abaxially using intraoperative imaging in lateromedial and dorsopalmar/plantar direction to ensure that there is enough room centrally for the plate. After preparing a subtendinous tunnel along the dorsal midline through a small axial incision down to the bone, the previously described PIP arthrodesis LCP is placed using a plate-passing tool. The most distal locking screw is inserted in the proximal aspect of the middle phalanx through a stab incision across the stacked combi-hole and firmly tightened. Subsequently, a 5.5-mm cortex screw is placed through the middle or the proximal combi-hole in a load position to achieve maximal axial compression over the dorsal aspect of the PIP joint. Finally a 5.0-mm locking head screw is placed in the remaining empty plate hole. The small incisions are closed with skin sutures (Figure 81-8), and a half-limb cast is applied. It has been reported that osseous fusion of the joint with this type of arthrodesis progresses more slowly than that seen with the traditional open technique, and lameness remains for several weeks, requiring cast coaptation for more than 6 weeks.[43]

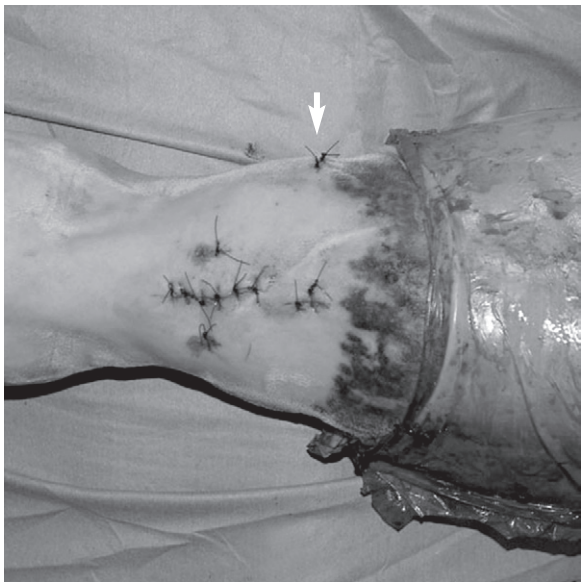


Figure 81-8. Immediate postoperative appearence of the PIP region following minimally invasive application of a 3-hole LCP. The plate was applied through the small axial incision and the transarticular screws through the stab incision on either side of the central incision. The *arrow* points to the incision on the lateral side that was used to remove cartilage with a drill.

Arthrodesis for Septic PIP Joints

PIP joint sepis secondary to wounds that penetrate the PIP joint are fairly common. In the case of joint instability, the use of implants for surgical arthrodesis is not recommended in the acute stage because of increased risk of a generalized infection of the implants.[6,44] These cases are best immobilized with a cast or transfixation cast in combination with aggressive débridement of the joint, cancellous bone graft, and systemic and local antimicrobial treatment.[6] Once the infection has resolved, surgical arthrodesis might be considered to treat the persistent lameness caused by the osteoarthritis of the joint.[39]

Prognosis

Early postoperative complications related to surgical arthrodesis include cast sores, incisional infection, implant infection, and construct failure.[31,39] Reported long-term complications are persistent postoperative lameness, cast-related morbidity, and excessive new bone formation.[32] Some horses develeop pathology of the distal interphalangeal joint after successful fusion of the PIP joint; therefore good case selection is important, which means that early signs of concurrent arthritis of the DIP joint must be recognized before surgery.[6]

The period from surgery to return to intended use r anges from 6 to 12 months and is related to indication for arthrodesis, arthrodesis technique, and the intended use of the horse.[30,31,39]

Long-term follow-up results after arthrodesis of the PIP joint using transarticular screws placed using lag technique revealed a range of success rates from 50% to 85% in the forelimbs and from 80% to 89% in the hindlimb.[28,31,32,45,46] Combining all four studies, arthrodesis was considered successful in 76 of 97 (78%) cases (28 of 41 [68%] forelimbs, and 48 of 56 [86%] hindlimbs). In a study on PIP joint arthrodesis using a combination of plates and transarticular screws, long-term outcome was evaluated in 53 horses.[39] Eighty-seven percent returned to intended use with a success rate of 81% for forelimb arthrodesis and 95% for hindlimb arthrodesis. Horses in this study consisted of American Quarter Horses (43%), Arabians (13%), Peruvian Pasos (11%), and similar types of working horses (33%). Their intended use was predominantly for pleasure riding (23%), showing (21%), and breeding (19%). This illustrates that direct comparsion of outcomes is not always feasible, because criteria for success, type of horse, and use may be different. However, there is a lack of information on the long-term outcome for PIP arthrodesis in larger Warmblood horses or Thoroughbreds used for show jumping, dressage, racing, or similar athletic activities. We assume that the success rates in those horses might not be as favorable as the ones presented in the studies discussed earlier.

METACARPOPHALANGEAL OR METATARSOPHALANGEAL JOINT

Indications

MCP or MTP joint arthrodesis is performed predominantly in the management of breakdown injuries of the suspensory apparatus. This injury occurs almost exclusively in the forelimbs of Thoroughbred racehorses, although it can be acquired by any horse running at high speed.[9,47] Fatigue of the digital accessory ligaments and flexor muscles supporting the MCP/MTP joint and digit leads to higher stresses in all components of the suspensory apparatus and permits development of the injury. Comminuted proximal phalanx fractures, advanced osteoarthritis, and severe flexural deformities of that region represent other indications for the arthrodesis.[6] In flexural deformities, an osteotomy might be needed to achieve an acceptable joint angle before arthrodesis.

The major physical finding after breakdown injuries of the suspensory apparatus is dorsiflexion or hyperextension (dropping) of the MCP/MTP joint as the horse attempts to bear weight. Many horses become anxious or even frantic as they attempt to control the injured limb. The disruption can occur as a result of fracture of both proximal sesamoid bones in the injured limb or, less often, rupture of the distal sesamoidean ligaments or the two suspensory ligament branches.

Breakdown of the suspensory apparatus should always be considered a career-ending injury. Because of the massive trauma that occurs with this injury, a myriad of complications are expected. It is therefore important to explain the situation to the owner so that an informed decision can be made concerning treatment. Horses can be saved as pasture-sound or breeding animals, but treatment is often prolonged and expensive, regardless of the therapeutic approach selected.

Surgical Techniques

The most frequently used technique for MCP/MTP arthrodesis involves the use of a 10- to 14-hole, 4.5-mm or 5.5-mm broad plate with an approximately 10-degree angle applied to the dorsal aspect of the limb and the creation of a tension band on the palmar/plantar aspect of the joint (Figure 81-9).[47] Dorsal plating without a tension band on the palmar/plantar aspect of the MCP/MTP joint usually results in failure, because the plate cycles in bending on the joint's dorsal surface and eventually breaks. The different techniques used to create the tension band depend on the type of injury and are discussed later.

Reports of experimental use of a dynamic hip screw,[48] a dynamic condylar screw,[48] or a prototype intramedullary pin

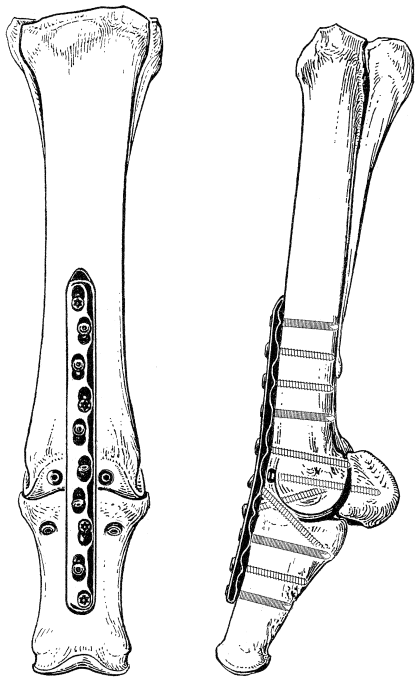


Figure 81-9. Graphic illustration of an arthrodesis of the MCP joint using a dorsally applied 10-hole 5.5-mm LCP. Note the stacked combihole at the distal end of the plate allowing insertion of either a cortex screw or a locking head screw. (This hole is filled with the latter in this illustration.) Two locking head screws are applied in MCIII and in the proximal phalanx. The remaining plate screws are of the cortex screw type. Two cortex screws are implanted transarticularly through the plate, and an additional cortex screw is inserted from the dorsal aspect of the MCIII into each proximal sesamoid bone. An additional cortex screw on either side of the plate (not shown) may be implanted transarticularly from the proximodorsal aspect of the proximal phalanx into the palmar or plantar aspect of the MCIII to improve stability.

plate[49] have been published, but reports on clinical use are currently not available. LCP is currently the treatment of choice for arthrodesis of the MCP/MTP joint.[14,50,51] The recently developed 5.5-mm LCP is more rigid than the standard 4.5-mm plate and represents a good option for an arthrodesis of the MCP/MTP joint. The price difference between locking compression technology and conventional internal fixation implants is found in the locking head screws. The plate itself is only slightly more expensive than a limited contact DCP (LC-DCP) or DCP. However, it is not necessary to use only locking head screws with the LCP; two such screws on either side of the MCP/MTP joint are adequate to provide the stability desired. The rest of the screws can be of the 4.5- or 5.5-mm cortex type. However, if exclusively locking head screws are used, except for the transarticular screws applied in lag fashion, the cost is about three times that of a conventional construct.[51]

Surgical Approach

A straight skin incision is made over the lateral digital extensor tendon, extending from the proximal aspect of the third metacarpal or third metatarsal (MCIII/MTIII) bone to the proximal interphalangeal joint. The incision is continued through the lateral digital extensor tendon, splitting it longitudinally, and extended, splitting the lateral extensor branch of the suspensory ligament.

The joint capsule of the MCP or MTP joint is transected parallel to the articular surface. Transection of the lateral collateral ligament and the lateral metacarposesamoidean or metatarsosesamoidean ligament allows disarticulation of the joint and removal of all articular cartilage from the proximal phalanx, the distal MCIII/MTIII, and the proximal sesamoid bones.

The cartilage is removed with a curet or an oscillating saw, and multiple osteostixis holes are drilled across the subchondral bone plates of all of the bones. The proximal dorsal end of the proximal phalanx and the intermediate ridge of the distal MCIII or MTIII are contoured with the help of an osteotome to improve the contact area and reduce the need to excessively contour the plate.

Plate Application

The 10- to 14-hole, 5.5-mm LCP (see Figure 76-17) is slightly bent between the fourth and fifth holes from the rounded end of the plate to an angle of about 10 degrees. One experienced equine surgeon uses a broad 10-hole LCP for this procedure with good success.[52] The plate is aligned to the dorsal surface of the proximal phalanx and MCIII/MTIII so that the bend is located directly over the dorsal aspect of the articulation, allowing four holes to be positioned over the proximal phalanx. One cortex screw is inserted through the DCU portion of the combi-hole next to the stacked combi-hole and solidly tightened. This presses the plate onto the bone surface. The two adjacent plate holes are filled with locking head screws. These three screws attach the plate firmly onto the proximal phalanx. The hole adjacent to the joint should be left without a screw.

Application of the Tension Band

For both procedures described later, the joint is maintained in a neutral or slightly flexed position (5 degrees).

LAG TECHNIQUE THROUGH THE PROXIMAL SESAMOID BONES

When a suspensory apparatus is intact or when the suspensory apparatus disruption is associated with suspensory ligament disruption proximal to the sesamoid bones, one 5.5-mm cortex screw is placed in lag fashion from the dorsal aspect of the distal MCIII or MTIII into each proximal sesamoid bone.

CERCLAGE WIRE

With comminuted fractures of proximal sesamoid bones, screw insertion is not effective. Also, in cases where the distal sesamoidean ligaments are ruptured, inserting the screws into the proximal sesamoid bone using lag technique does not produce a tension band across the palmar/plantar joint surface. In such cases, a double strand of 1.25-mm-diameter cerclage wire is placed in figure-of-eight fashion across the palmar/plantar aspect of the joint. A 3.2-mm hole is drilled in a frontal plane parallel to the joint surface about 4 to 6 cm (1½ to 2½ inches) proximal and distal to the joint surface across the MCIII/MTIII and the proximal phalanx, respectively. Care is taken not to interfere with the screws placed in the proximal phalanx and the ones inserted into MCIII/MTIII.

The wire is guided through these holes and directed toward the palmar or plantar joint surface using the large wire passer to place the wires between the bone and the suspensory apparatus in a figure-of-eight pattern. It is important to not induce acute bends in the wire, because these resist effective tightening of the wire. The wires ends are tightened with the joint maintained in a neutral or slightly flexed position. One set of wire ends is tightened on the lateral side of the MCIII/MTIII and the other set on the medial side of the bone. Before final tightening, all the slack has to be taken out of the wire to ensure the development of an effective tension band function. Alternatively, one 1-mm diameter stainless steel cable with crimping sleeve has been used successfully for tension band application.[51]

Completion of Plate Application

Once the tension band is created using either screws or cerclage wire, the angulated tension device is applied to the most proximal hole of the plate and attached to MCIII/MTIII with a unicortical cortex screw. With the help of the socket wrench, the device pulls the plate proximad, and in doing so the palmar/plantar tension band is further tightened. If the tension band is accomplished with screws in the proximal sesamoid bones, tension is transferred via the distal sesamoidean ligaments to the suspensory ligament. The second plate hole from the top and the sixth plate hole from the bottom are filled with 5.5-mm cortex screws, allowing the plate to be firmly pressed onto the bone surface. At least two locking head screws are inserted into MCIII/MTIII. The tension device is removed, and the remainder of the screws can be either of the cortex or the locking head type. Across the two juxta-articular empty plate holes, cortex screws are implanted transarticularly using lag technique. Additionally, two or three transarticular screws are inserted next to the plate to increase stability across the joint to be fused.

A longer plate may be used when an arthrodesis of the MCP/MTP joint and the PIP joint is desired. Indications include fixation of severely comminuted proximal phalanx fracture, or breakdown injuries with suspected damage to soft

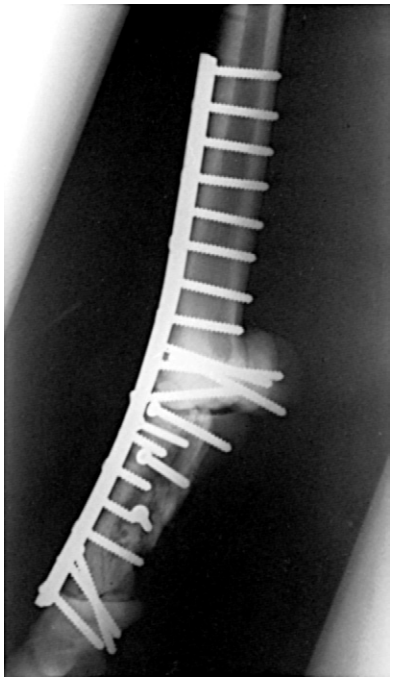


Figure 81-10. Radiograph of a comminuted proximal phalanx fracture repaired with an 18-hole DCP. The plate was applied to the dorsal aspect and spanned the PIP and MCP joints. This repair is used as a salvage procedure.

tissue support structures of the PIP joint to prevent its luxation (Figure 81-10).

Minimally Invasive Technique

With the introduction of the LCP to equine orthopedics, minimally invasive techniques[50] or less-invasive techniques[13] have been described for MCP/MTP joint arthrodesis. The prebent plates are introduced through a small incision after a tunnel is prepared for the future plate bed with the help of a sharp chisel-like instrument. The screws are inserted across the plate hole through stab incisions. These incisions are best prepared through an identical plate applied over the inserted one. The authors reported shorter surgery time, lower infection rates, and higher survival rates when compared to the open-approach technique.[50] However, none of the differences were statistically significant.

Prognosis

The major risks of the procedures are deep infection[53] and subsequent instability of the construct, controlateral limb laminitis, and PIP joint luxation. Survival rates are 25% (open approach, DCP),[50] 33% (minimally invasive technique, DCP),[50] 65% (open approach, DCP),[54] 67% (open approach, LCP),[51] and 75% (open and minimally invasive approach, LCP).[51] The prognosis is better for horses that were treated with MCP/MTP arthrodesis as a primary treatment than in situations where nonoperative treatment was tried initially and the MCP/MTP arthrodesis was elected as a last resort.[54] The prognosis was also better for horses treated for osteoarthritis than for horses treated for rupture of the suspensory apparatus, because the former have less trauma to the limb before surgery.[54]

CARPUS

Indications

In the carpal region, two types of arthrodeses are performed: the pancarpal arthrodesis, where all the joints in the carpal region are fused, and the subtotal or partial arthrodesis, involving only one or two of the joints.[6,55,56] Carpal arthrodeses are indicated for treatment of comminuted carpal fractures as well as luxations and subluxations of this region. In most animals, these operations should be viewed as salvage procedures, with the exception of animals with carpometacarpal luxations and carpometacarpal osteoarthritis. Partial carpal arthrodeses are recommended for severe osteoarthritis of the carpometacarpal joint, in cases in which the distal or proximal row of carpal bones has comminuted fractures and accurate anatomic reconstruction is impossible,[57,58] or in luxations of the carpometacarpal and the antebrachiocarpal joints.

Surgical Techniques

Facilitated Ankylosis for Osteoarthritis of the Carpometacarpal Joint

Carpometacarpal osteoarthritis (CMC-OA) is characterized by progressive lameness that responds poorly to medical treatment. With conservative management, prognosis for use is poor and prognosis for life is unfavorable.[59,60] In a recent report, 12 horses with CMC-OA in 15 joints were treated with a drilling technique that produced a bony ankylosis in 6 of 9 joints that were available for follow-up radiographs 10 months (range, 6 to 14 months) after surgery.[61] Long-term follow-up by owner interview (mean, 28.6 months after surgery) revealed that 10 of 12 horses (83%) were considered sound, and 8 of 12 horses (67%) returned to their original activity. In an *in vitro* study, three drilling techniques to remove cartilage from the CMC joint were evaluated using 4.5-mm or 5.5-mm drill bits in different drilling patterns. The mean percentage of damage caused by the three drilling techniques to the articular surface and calcified cartilage ranged from 16% to 33% and 14% to 23%, respectively.[62]

Partial Carpal Arthrodesis

Depending on the injury, partial carpal arthrodesis involves either fixing the proximal row of carpal bones to the distal row and MCIII or fixing the distal row of carpal bones to the MCIII (Figure 81-11). Additionally, partial carpal arthrodesis can be used for antebrachiocarpal luxations, where the proximal row of carpal bones is fastened to the distal radius (epiphysis and/or metaphysis). For these arthrodesis techniques, a variety of plates may be used. Currently the preferred plate is the locking compression type. Selection depends on size of the animal, availability of the plates, and preference of the surgeon. Generally 6- to 8-hole plates are used.[6,58] The horse is positioned in dorsal or lateral recumbency. General surgical and specific anatomic considerations guide the surgeon in selecting the proper approach.

Typically, the carpus is approached through two vertical skin and subcutaneous incisions, one on either side of the extensor

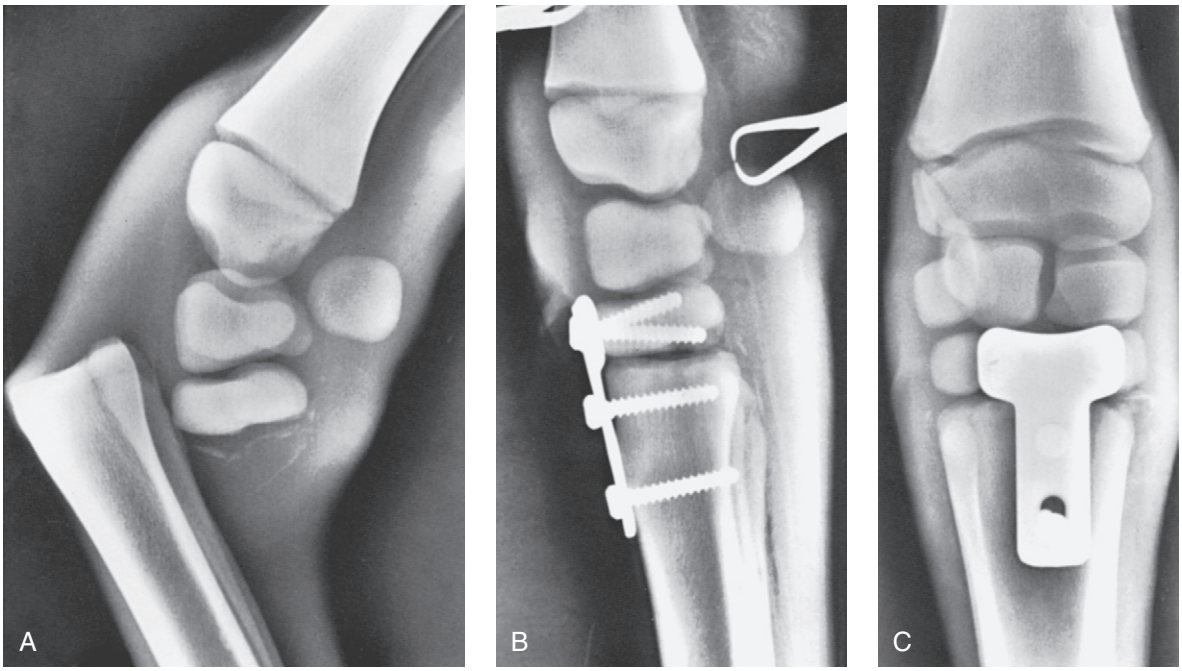


Figure 81-11. A, Lateromedial radiograph shows a carpometacarpal luxation in a 2-week-old foal. Lateromedial **(B)** and dorsopalmar **(C)** radiographs of the repair depict a 2-hole T-plate applied to the dorsal aspect of the distal row of carpal bones and the proximal aspect of MCIII. (From Auer JA, Taylor JR, Watkins JP, et al: Partial carpal arthrodesis in the horse. Vet Comp Orthop Traumatol 3:51, 1990.)

carpi radialis tendon. The incisions are extended through the joint capsule of the affected joints.Whenever possible, articular cartilage should be removed to facilitate fusion of the joint surfaces. Osteostixis as well as application of a cancellous bone graft or an alternative osteoinductive material are indicated to enhance fusion. In comminuted carpal bone fractures, strategically placed interfragmentary screws inserted in lag fashion across large bony fragments contribute considerably to the stability of the construct. In most cases, it is prudent to maintain alignment by applying one or two bone plates across the joint(s) involved. The same holds true for selected subluxations with signs of avulsion fractures and displacement of specific bones (Figure 81-12).

In a mare with severely comminuted fractures, all the fragments of the distal row of carpal bones were removed, with subsequent fixation of the proximal row of carpal bones to the MCIII. After 3 months in a tube cast, the mare developed fusion at the arthrodesis site and served for several years as a successful brood mare.[56]

One of the problems in carpal arthrodesis is the closure of the skin over the two plates that are routinely used. The placement of tension sutures in the skin over the distal carpus and the proximal metacarpus 24 hours before the surgical procedure has been effective in creating more available skin to cover the dorsal carpus. This should partially relieve the tension on the skin incision during closure over the plates.[6] If needed, relief skin incision(s) distant to the surgical approach in the carpal region facilitates skin closure.

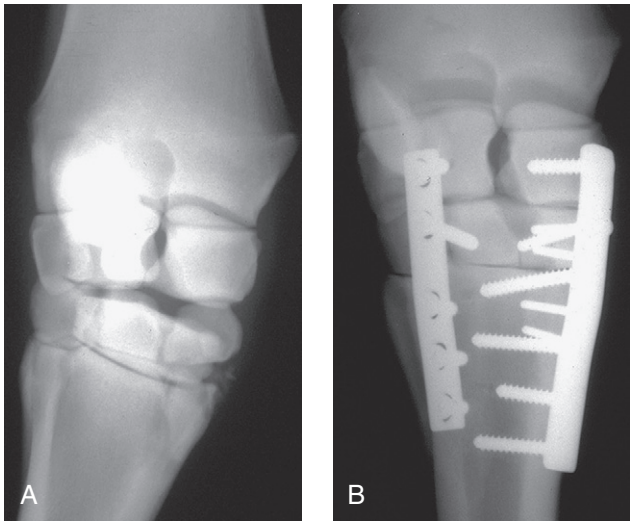


Figure 81-12. A, Dorsopalmar radiographic view of subluxation of the middle carpal joint in a 6-year-old brood mare. Note the avulsion fracture at the medial aspect of the carpometacarpal joint. The second carpal bone is also partially luxated. **B,** Postoperative dorsopalmar radiographic view showing a broad 6-hole 4.5-mm DCP applied to the dorsomedial aspect and a narrow 5-hole 4.5-mm DCP applied to the dorsal aspect of the carpal region to achieve a partial carpal arthrodesis.

Pancarpal Arthrodesis

Pancarpal arthrodesis using two long, broad DCPs or LCPs[63] is advocated for comminuted fractures involving both rows of carpal bones (Figure 81-13) and for severe degenerative carpal joint disease in older horses that develop a carpus valgus or varus deformity.[56] In severely comminuted fractures, the purpose of the plates is to buttress the unstable limb and maintain a straight axis. The 5.5-mm LCP and DHS or DCS plates are excellent options for those cases, because they are stronger than the DCP. Plate luting is recommended in pancarpal arthrodeses (see

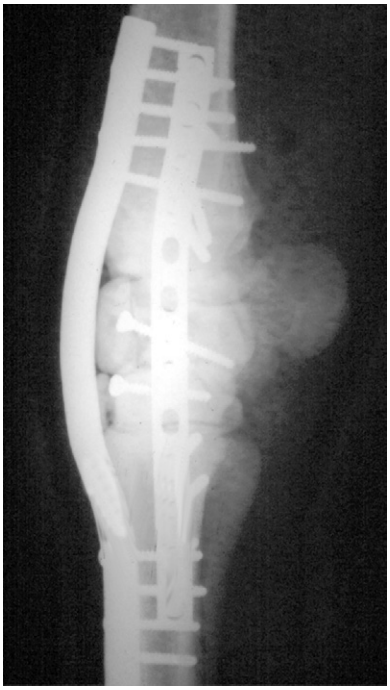


Figure 81-13. Lateromedial radiograph depicting pancarpal arthrodesis used to repair a comminuted carpal fracture. A 12-hole broad DCP bone-lengthening plate was applied to the dorsocranial aspect of the limb, and a narrow 12-hole DCP was applied laterally. Cortex screws were used to provide interfragmentary compression across slab fractures.

Chapter 76).[64] Simple cast application is not effective in supporting collapsed comminuted intercarpal fractures.[56]

The approach to the carpus can be achieved by a single, 40-cm (16-inch) vertical skin incision on the dorsal aspect of the limb and centered over the carpus. Because a long incision is used, both plates are placed through this incision along the dorsomedial and dorsolateral aspect of the limb, on either side of the extensor carpi radialis tendon, and centered over the carpus.[63] To facilitate skin closure, several towel clamps can be used to fatigue the skin edges. In extreme cases, a relief skin incision is made at a location distant to the main skin incision, avoiding an area where pressure necrosis may develop under the splint or cast (see later).

Postoperative Management

Splints, casts, or transfixation casts may be necessary, depending on the injury that led to the partial or pancarpal arthrodeses.

Complications are common with carpal arthrodeses, including supporting limb lameness, incisional infections, and implant infection. Approximately 67% of horses survive and achieve comfortable weight bearing.[65] Timing of surgery is important: a prolonged period of non–weight bearing because of conservative management of severe osteoarthritis or comminuted carpal fractures reduces the prognosis for successful outcome of the arthrodesis.[6]

SCAPULOHUMERAL JOINT

Indications

Advanced osteoarthritis and persistent luxations and subluxations of the scapulohumeral (SH) joint (Figure 81-14, *A*) in Miniature or small horses may be treated by arthrodesis with

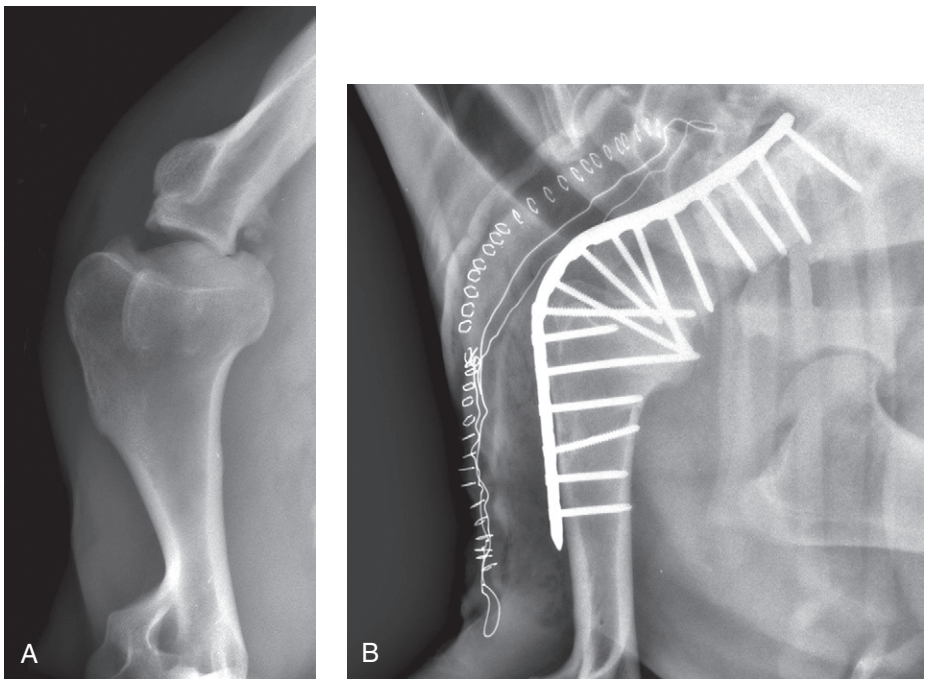


Figure 81-14. A, Craniocaudal radiographic view of a scapulohumeral subluxation in a Miniature Horse stallion. **B,** Lateromedial radiographic view of the scapulohumeral arthrodesis performed in the Miniature Horse stallion. A 16-hole 3.5-mm LCP was applied to the cranial aspect of the joint region. Over the joint, transarticular screws were implanted through the plate, providing additional stability to the fixation.

the goal of rendering the animal free from painful lameness.[66-68] This technique is not well suited for horses, because the quality of bone in this region is inadequate to withstand the tremendous mechanical forces that would be exerted on the implants. There is a report of a successful SH arthrodsis in a Paint Horse treated with two LCPs.[69]

Surgical Technique

A 10- or 11-hole, 4.5-mm narrow DCP or LCP and 4.5-mm cortex screws are applied to the cranial surface of the scapula and cranial surface of the humerus after osteotomy of the intermediate tubercle. An alternative technique involves the use of a 16-hole broad LCP (or reinforced 3.5-mm DCP) applied to the cranial aspect of the proximal humerus and the scapula following transection of the biceps tendon at the level of the shoulder joint.[70] The broad 3.5-mm plates have the same cross-sectional area as the 4.5-mm narrow plates, but more screws can be inserted through these plates, which provides a biomechanically superior fixation. Additionally, because the plate holes are smaller, there is more metal surrounding the DCP holes. In most patients, one or two plate holes located over the joint should be filled with transarticular screws inserted in lag fashion.

A report of four Miniature Horses suffering from SH osteoarthritis, some with concurrent shoulder luxation or subluxation, described SH arthrodesis as a successful technique, despite some complications.[68] The animals developed a mechanical lameness but were able to ambulate well after surgery. A recent report on a Miniature Horse suffering from persistent shoulder subluxation associated with severe osteoarthorsis resulted in the horse's becoming sound and able to gallop around the pasture without any gait anomaly following application of a 3.5-mm broad LCP (see Figure 81-14, *B*).[70]

TARSUS

Indications

The primary indication for arthrodesis in the tarsus is osteoarthritis of the tarsometatarsal (TMT) and the distal intertarsal (DIT) joint that is unresponsive to medical therapy.[8,71-73] Occasionally, luxations of the tarsus, especially involving the TMT and DIT joints, necessitate an arthrodesis.[74] Talocalcaneal arthrodesis has been described as a promising treatment for talocalcaneal osteoarthritis.[75,76]

Facilitated Ankylosis Techniques

Chemically Induced Ankylosis

Chemically induced ankylosis of the TMT and DIT joint involves the injection of a chemical agent that with time destroys the articular cartilage and facilitates ankylosis of the joints. Before such an agent can be injected into a joint, it has to be ascertained that no communications with other joints exist, such as between the DIT and the proximal intertarsal (PIT) joint. This can be established by a contrast arthrography. Two agents have been used to induce ankylosis in the tarsal region as treatment for bone spavin: monoiodoacetate (MIA) and ethyl alcohol.

Intra-articular injection of 100 to 250 mg of MIA, a chemical compound that causes cartilage death and stimulates fusion, was introduced as a treatment for osteoarthritis of the TMT and DIT joints.[77,78] Attributes of this treatment that limit its acceptance as a method for treatment of bone spavin include the severity of discomfort of the horse immediately after treatment, postinjection soft tissue swelling, progression of osteoarthritis in the PIT joints, severe soft tissue necrosis that can occur with extra-articular injection, variable length of convalescence, and inconsistent outcome.[4,77-79]

Two reports recommend the use of intra-articular administration of ethyl alcohol for treatment of bone spavin in horses.[80,81] The mechanism of action of ethyl alcohol is through nonselective protein denaturation and cell protoplasm precipitation and dehydration.[80] Ethyl alcohol also functions as a neurolytic agent that results in a sensory innervation blockade at the intra-articular level. The neurolytic properties and nonselective destruction of proteins contribute to its success at disrupting the cartilaginous matrix, causing necrosis of the chondrocytes and facilitating arthrodesis.[79] Ethyl alcohol is inexpensive and readily available to most practitioners. This newer method of faciltated ankylosis of the TMT joint has been invesigated in eight healthy horses.[80] In each horse, one TMT joint was injected with 4 mL of 70% ethyl alcohol, and the opposite joint was treated with 4 mL of 95% ethyl alcohol. A mild swelling at the injection site persisted for 24 hours in all horses, but no obvious signs of discomfort were observed. Mild lameness was detected in 2 of 16 limbs during the first week of the study. Postmortem examination 12 months after treatment revealed that there was gross and histopathologic evidence of local bony fusion in all but 1 of the 16 TMT joints. However, complete obliteration of the TMT joint was not detected in any of the joints investigated. A second study reported radiographic evidence of ankylosis 4 to 6 months following injection of 3 mL of 70% ethyl alcohol in 16 horses.[81]

Laser-Facilitated Ankylosis

Laser-facilitated ankylosis using a neodymium:yttrium-aluminum-garnet (Nd:YAG)[81] or diode[4,82] laser to destroy articular cartilage by superheating and vaporizing synovial fluid, which should result in chondrocyte death, has also been performed. Clinical studies are currently not available, but experimental studies in sound horses demonstrated that laser application promotes partial ankylosis of the DIT joint within 5 to 12 months.[4,82] However, diode laser–facilitated ankylosis resulted in significantly less fusion of the distal tarsal joints compared to MIA injection or transarticular drilling (see next).[4]

Transarticular Drilling Technique

Transarticular drilling of the distal tarsal joints to facilitate ankylosis is a commonly performed surgical treatment.[83] The animal is positioned in lateral recumbency, providing access to the dorsomedial aspect of the tarsus involved. If the procedure is performed in both hindlimbs at the same time, the horse is positioned in dorsal recumbency. After routine preparation of the surgical site for aseptic surgery, a 3-cm (1¼-inch) vertical skin incision is made on the dorsomedial aspect and centered over the TMT and DIT joints. The sites for drill bit entry are midway between a line extending from the groove between the proximal MTII and MTIII and the most dorsal aspect of the distal tarsus (at the level of the TMT and DIT joints) (Figure 81-15). This is adjacent to the plantar margin of the saphenous vein, which has to be displaced dorsally to avoid iatrogenic

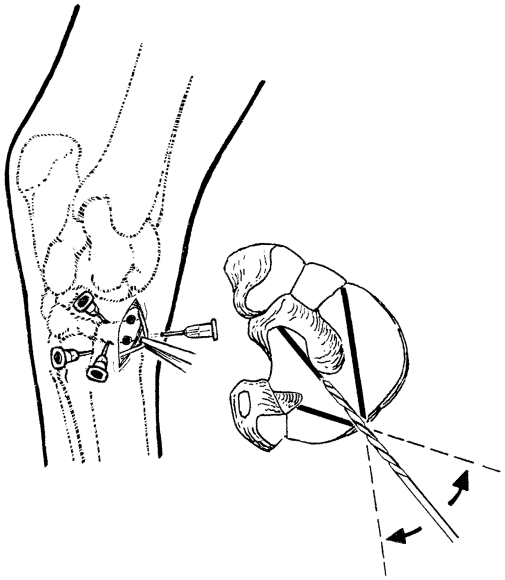


Figure 81-15. Graphic illustration of the drilling technique for tarsal arthrodesis. The sites for drill bit entry are midway between a line extending from the groove between the proximal MTII and MTIII and the most dorsal aspect of the distal tarsus (at the level of the TMT joint and DIT joint). Three diverging drill holes are made along the articular surfaces of the distal tarsal joints using intraoperative imaging.

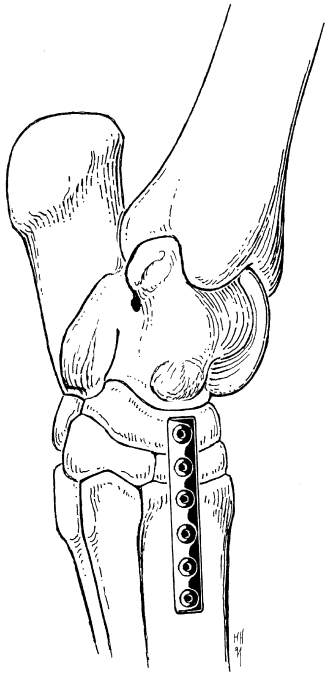


Figure 81-16. Graphic illustration of a tarsal arthrodesis with a six-hole narrow DCP applied medially. One screw penetrates both the central and third tarsal bones. The remaining screws are placed into the proximal MTIII.

trauma. Placement of a Penrose drain around the vein facilitates its manipulation during the surgical procedure. The TMT and DIT joints are identified with hypodermic needles using intraoperative radiographs or fluoroscopy.

A 4.5-mm drill bit is passed into the joint space in three directions from a single entry point on the dorsomedial aspect of the tarsus, creating a fanlike pattern of holes.[11] The tracts are best drilled in pairs (TMT and DIT): first, a 20-mm long tract directed toward the most lateral palpable extremity of MTIV; second, a 20-mm tract angled 30 degrees to the first in a plantar direction; and third, a 35-mm tract angled 30 degrees to the first in a dorsal direction. For intraoperative imaging, it is important to orient the x-ray beam perpendicular to the drill tract and to angle as necessary in a distal to proximal direction to bring the joint margins into convergence. The number of images can be reduced if two drill bits are used and tracts in the TMT and DIT joints are imaged simultaneously. The procedure described resulted in consistent destruction of articular surface of the TMT and DIT joint.[84] The average destruction of joint surface was 18.0% (14.5% to 23.8%) for the proximal MTIII and 21.7% (15.1% to 30.4%) for the proximal third tarsal bone, respectively.[84]

Drilling too deeply may lead to penetration of the tarsal canal, resulting in unnecessary periosteal reaction or profuse hemorrhage from disruption of the perforating branch of the cranial tibial artery. Excessive side-to-side drilling causes instability, severe pain, and prolonged recumbency and is associated with excessive periosteal new bone formation.[85] Most of the horses in which excessive drilling has been performed never regain complete soundness.[85]

The estimates of outcome following transarticular drilling suggest that this treatment may be successful in 47% to 85%, depending on the technique and study design.[71,86-88]

Fixation Techniques

Plate Application for Arthrodesis

One technique consists of applying a narrow DCP or LCP over the medial aspect of the tarsal region (Figure 81-16). Fibrous covering of the dorsomedial aspects of the joints together with any exuberant exostoses are partially removed with an osteotome and mallet. This facilitates identification of the joint spaces involved, provides a greater plate–bone contact area, and reduces the amount of plate contouring needed. The PIT joint space is identified through placement of two hypodermic needles or two 2-mm drill bits using intraoperative imaging technique.

As an optional procedure, two drill holes, 4.0-mm in diameter are prepared along each of the joints as previously described.[85] These holes may be filled with a bone graft plug harvested from the proximal tibia or the tuber coxae. Placement of such a plug, hydroxyapatite granules, or biodegradable bone cement will enhance osseous union of the two articulations by means of spot welds.

The selected 5-hole narrow 4.5-mm DCP is applied to the arthrodesis site.[89] The thread hole for the most proximal plate screw is drilled with the 4.0-mm drill bit protected by the corresponding drill guide within the body of the central tarsal bone, parallel to the joint surfaces. In smaller horses, 4.5-mm screws may be used instead of 5.5-mm screws. In that case, a 3.2-mm thread hole is prepared. (It is important to use 5.5-mm cortex screws whenever possible, because they resist cyclic loading better than 4.5-mm screws. Screws 4.5 mm in diameter often fail in an adult horse.)

After tapping the thread hole, a 30- to 40-mm-long cortex screw is inserted through the most proximal plate hole and tightened. Care is taken to select screws shorter than the

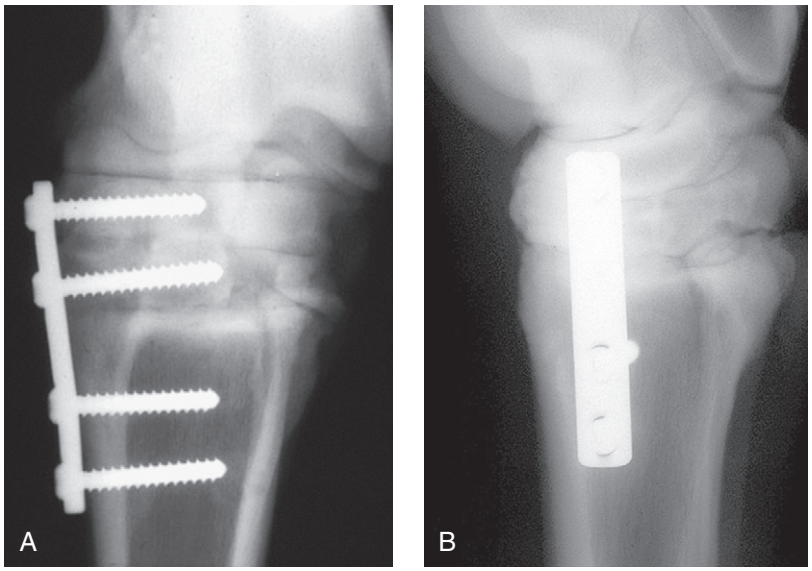


Figure 81-17. Dorsoplantar **(A)** and lateromedial **(B)** 4-week postoperative radiographic views of a tarsal arthrodesis using a four-hole narrow DCP in a 9-year-old Andalusian gelding. One 5.5-mm cortex screw is inserted into both the central and the third tarsal bones. Two identical screws are placed into the proximal MTIII. Note that the drill holes into the two distal joints are already filling in with new bone and becoming less obvious on these radiographs.

measured blind-ending hole in the bone to prevent impacting upon the dead-end hole, which prevents the screw from compressing the plate onto the bone. The tension device is applied subsequently at the distal end of the plate, secured to MTIII, and tightened. One or two plate screws are subsequently inserted into MTIII. After the tension device is removed, the remaining plate holes are filled with screws. If the DIT and TMT joints are to be fused, one screw is inserted into the central tarsal bone and one into the third tarsal bone (Figure 81-17).

After flushing the surgical site, the subcutaneous tissues and the skin are closed in routine fashion. The surgical site is covered by a bandage.

Postoperatively, the limb is kept under a bandage for 2 to 3 weeks. The skin sutures or staples are removed 10 days after the surgery. Lameness can persist for several months. Rehabilitation must be conducted gradually over several months.

The disadvantage of this techniques is that the only implant bridging the different joints is the plate at one loaction. It can be argued that this fixation technique does not sufficiently counteract rotational forces acting parallel to the joint surfaces. Therefore the insertion of oblique transarticular screws through the plate and adjacent to the plate should result in a greater area of fixation throughout the tarsal region. Up to now, no *in vitro* studies have been conducted to prove this theory.

The use of LCPs is presently encouraged for this procedure. A prototype of an LCP (Figure 81-18) that includes seven fixed-angle locking screws that cross the joint spaces of the DIT and the TMT joint is currently under investigation. The plate is applied to the dorsomedial aspect of the central and third tarsal bone as well as to the proximal aspect of MTIII. The second hole from the bottom is an elongated combi-hole and it allows dynamic axial compression. The remaining three plate holes over MTIII are filled with locking head screws. The placement of the screw holes within the head of the plate and their orientation in proximodistal as well as in lateromedial direction was tested in several cadaveric bones to ensure correct arrangement. No clinical case has yet been treated with this plate.

An alternative technique involves implanting perforated stainless steel cylinders filled with autogenous cancellous bone in the distal tarsal joints.[53] This technique was applied in four horses. Graft cell survival was poor 2 weeks after surgery in one horse. In two horses at 10 months, there was partial arthrodesis of the joints, with incorporation of the implants into the osseous union. The implants were filled with vascularized bone. These two horses were sound 9 months after surgery, but one horse fractured its third tarsal bone and still had a positive response to a hock flexion test 12 months after surgery.

A more involved technique that is occasionally used in tarsal luxations involves the application of a 12- to 14-hole broad DCP or 5.5-mm LCP to the plantarolateral aspect of the calcaneus and proximal metatarsal region (Figure 81-19). This technique allows extension of the plate farther proximad, resulting in a stronger arthrodesis. In selected cases, MTIV is removed before plate application. However, care must be taken to avoid damage to the greater metatarsal artery coursing between MTIII and MTIV. The surgical approach is more extensive than that for the technique described earlier, and the surgery itself is more demanding. Care must be taken to avoid penetrating the tarsocrural joint with a screw. Selected screws can be placed in lag fashion across the more distally located joints, facilitating interfragmentary fixation.

Talocalcaneal Arthrodesis

For talocalcaneal arthrodesis, the horse is placed under general anesthesia in lateral recumbency, with the affected limb positioned uppermost.[75] Application of a tourniquet proximal to the tarsus is optional. A slightly curved incision is made over the distal half of the calcaneus, and the tissues are sharply divided down to the bone.

Needle markers are used to determine the correct angulation of the future screws under fluoroscopic guidance. Computer-assisted guidance greatly facilitates preparation of the drill holes (see Chapter 13). The drill bit is aimed toward the plantaromedial aspect of the medial trochlear ridge of the talus, avoiding penetration of the tarsocrural joint at the intertrochlear groove.

Two or three 5.5-mm cortex screws are inserted in lag fashion across the lateral facet, using routine technique (Figure 81-20).

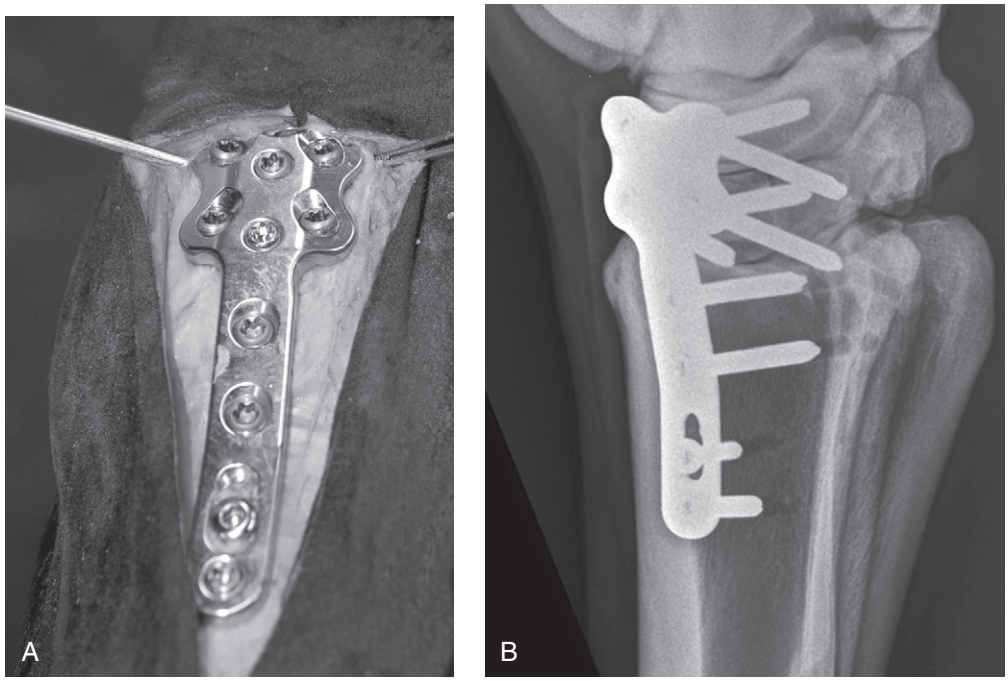


Figure 81-18. A, The new tarsal arthrodesis plate applied to the tarsal region. Seven locking screws are applied into the small tarsal bones at different angles. The second hole from the bottom is oblong and allows dynamic axial compression. The remaining three plate holes over the MTIII are filled with locking head screws. **B,** Oblique radiographic view of the tarsal region depicting the orientation of the screws.

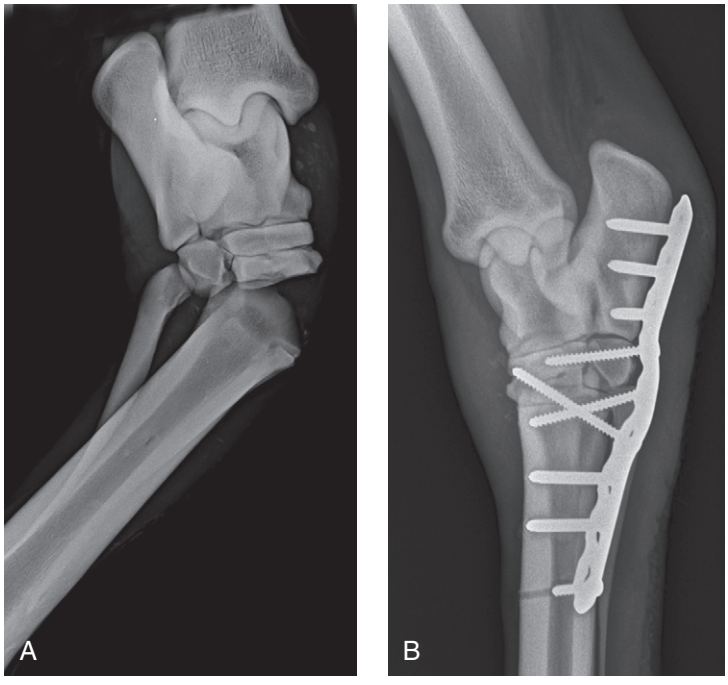


Figure 81-19. A, Craniocaudal radiographic view of a tarsometatarsal luxation in a pony. **B,** Oblique postoperative radiographic view of the tarsal region after application of 9-hole narrow 4.5/5.0-mm LCP to the palmarolateral aspect of the calcaneus and proximal MTIII region.

There is an adequate amount of solid bone present to achieve stable transarticular compression. By slightly altering the direction of the screws, an increased compressive effect can be achieved. To prevent weakening of the calcaneal bone, washers may be applied, which negate the need for countersinking.[75] Alternatively, the screws may be inserted through a plate contoured to the calcaneal surface. Once the screws are in place and solidly tightened, the incision is closed using routine technique.[75]

A slightly different technique was presented in another report, where two clinical cases were also treated with 5.5-mm cortex screws applied in lag fashion.[76] In a pony, three parallel screws were inserted through a dorsomedial arthrotomy to the talus. The screws were placed across the medial facet into the

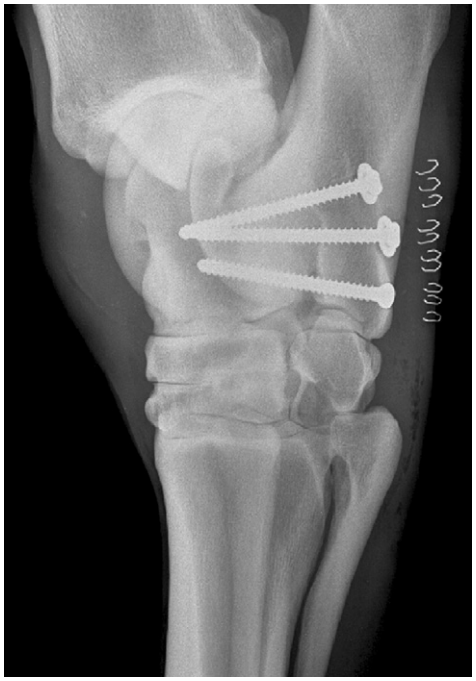


Figure 81-20. Oblique postoperative radiographic view of a talocalcaneal arthrodesis performed through three converging 5.5-mm cortex screws inserted in lag fashion across the lateral facet of the talocalcaneal joint. Washers were used in the two proximal screws to increase the contact area of the implants and reduce stress concentration at the bone–screw head junction. (Courtesy R. Smith, Royal Veterinary College, London.)

calcaneus; in a horse, identical screws were augmented with two additional screws similarly inserted as described in the first technique.[76] Both techniques resulted in successful fusion.[6,89] Talocalcaneal arthrodesis could develop into an effective treatment for local talocalcaneal osteoarthritis. Early results are encouraging.

COMPLICATIONS

Complications of arthrodesis include postoperative infection,[90] implant failure, laminitis in the opposite limb, long-term lameness, and development of angular limb deformities. Accidental insertion of bone screws into a joint space can provide a continuous source of pain and should be avoided. This can be done by meticulous surgical technique and careful intraoperative radiographic control.

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CHAPTER
82 Management of Bursitis

Gary M. Baxter

STRUCTURE AND FUNCTION OF BURSAE

A bursa is a closed sac lined with a cellular, synovial-like membrane, interposed between two moving parts or at points of unusual pressure such as between bony prominences and tendons.[1] This synovial sac can be completely enclosed beneath the skin or tendon, or it can partially surround the tendon in the form of a synovial sheath (Figure 82-1). Bursae differ from tendon sheaths because they do not completely surround the tendinous structure and are located on one side of the tendon only, between it and a bony prominence.

Bursae in horses are often classified as to when they form (congenital versus acquired) and their anatomic location (subcutaneous versus subtendinous versus subligamentous). *Congenital* bursae are those that are part of the normal anatomic structure and develop before birth. They are in a constant position, are often associated with deep structures, such as fascia, muscles, and tendons; and are viewed as "true" synovial bursae. Examples are the navicular bursa, bicipital bursa, and calcaneal bursa. *Acquired* bursae are those that occur after birth, usually from trauma. They develop over bony prominences, are usually located subcutaneously, and are viewed as "false" bursae. Examples are carpal hygroma, elbow hygroma, and capped hock. A subcutaneous bursa is nearly always acquired and has also been termed "reactive" or "functional" since they usually develop from mechanical effects after birth.[1] Movement of the skin after some form of trauma contributes to tearing of the subcutaneous connective tissue, gap formation under the skin, and fluid accumulation. The fluid exudate accumulates in the subcutaneous space and becomes encapsulated by fibrous tissue, forming a false bursa.[1] Ultimately, a synovial-like membrane develops and produces jointlike fluid, although it differs in viscosity and mucin clot formation.[1] Subtendinous bursae are congenital or true bursae associated with origins and insertions of muscles and tendons and are critical in normal movement of the limbs. They are considered synovial structures similar to tendon sheaths and joints and have the most significant clinical implications in the horse. Subligamentous bursae are the least problematic type of bursa and have few clinical implications in horses. The most notable examples are the atlantal and supraspinous bursae associated with the ligamentum nuchae.[1]

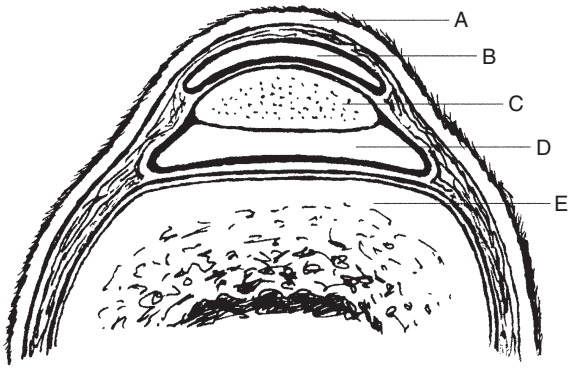

Figure 82-1. Diagram illustrating the anatomic location of a subcutaneous bursa and a subtendinous bursa as they occur at the tuber calcanei. *A,* Skin; *B,* subcutaneous bursa; *C,* tendon (SDFT); *D,* subtendinous or calcaneal bursa; *E,* bone (calcaneus).

Chronic bursitis is characterized by the accumulation of bursal fluid and thickening of the bursal wall by fibrous tissue; fibrous bands and septa may develop in the bursal cavity, and the subcutaneous tissues around the bursa may continue to thicken. Bursal enlargement usually develops as a painless swelling that does not typically interfere with function unless it becomes greatly enlarged. In most cases, the acquired bursa is only a cosmetic blemish unless it becomes infected. The bursae may become infected by a puncture or iatrogenic incident.[2] An infected bursa is usually painful, which contributes to lameness, and it may break open to drain, resulting in a fistula (see Chapter 28).

ETIOLOGY OF BURSA-RELATED CONDITIONS

Bursitis is defined as an inflammatory reaction within a bursa and may range in severity from a cosmetic blemish associated with a capped hock to a non–weight-bearing lameness secondary to infectious bursitis. Trauma is the most common cause, and in general, horses with noninfectious bursitis are much less symptomatic than those with infectious bursitis. Trauma may occur in the form of repetitive use injury as seen with navicular disease or syndrome, repetitive external injury such as occurs with elbow hygroma, single event external blows often seen with carpal hygroma, and external wounds causing infection commonly seen in the navicular and calcaneal bursae.[1,3-6] Infection within a bursa, however, may also occur from hematogenous spread of bacteria similar to other synovial cavities.[7-10]

Repetitive Use Trauma

This type of problem is most commonly identified in the navicular bursa associated with navicular disease or syndrome, the cunean bursa associated with distal tarsal osteoarthritis, and the trochanteric bursa secondary to other lameness conditions in the hindlimb. Bursitis in these horses is usually a secondary manifestation of the primary problem and not the only cause of the lameness. For instance, navicular bursitis in horses with navicular disease may result from pathology of the navicular bone, the deep digital flexor tendon, or the podotrochlear apparatus. It is rarely the only pathologic process (see Chapter 90).

Repetitive External Trauma

This form of trauma causes problems at bony prominences such as the point of the ulna (elbow hygroma; shoe boil) and tuber calcanei (capped hock) and contributes to the development of a subcutaneous or false bursa in these locations. For instance, the shoe of the affected limb constantly hitting the point of the elbow when the horse is lying down contributes to elbow

hygromas. The repetitive nature of the trauma also worsens the problem and makes treatment difficult unless the predisposing cause is eliminated.

Single Event External Trauma

This tends to occur most commonly over the cranial aspect of the carpus (horse falling to its knees) contributing a carpal hygroma, the cranial aspect of the shoulder (kick injury or running into a solid object) causing bicipital bursitis, and the tuber calcanei (hit a stall door or wall) contributing to a capped hock.

Wounds or Penetrating Injuries

Any type of wound that enters a bursal sac may contribute to infection. This can occur with both congenital and acquired bursae, and in any location. Infectious bursitis secondary to wounds most commonly involves the navicular bursa, calcaneal bursa, and bicipital bursa. Iatrogenic infection of subcutaneous bursae may occur following medical (drainage and injection of corticosteroids) or surgical (Penrose drain placement) treatments. Hematogenous spread of infection has been reported most commonly in the bicipital bursa and the supraspinous bursa. I have seen several foals suffering from infectious bicipital bursitis consistent with the "joint ill" syndrome. *Brucella abortus* infection of the supraspinous bursa can be associated with fistulous withers.[11]

DIAGNOSIS OF BURSA-RELATED CONDITIONS

The diagnosis of bursitis depends on the specific bursa that is affected and the initiating cause. For instance, the diagnosis of subcutaneous bursitis can often be made based on the history and physical findings of a soft, fluctuant swelling in specific anatomic locations where they tend to occur. Acute and chronic bursitis of true bursae, such as the navicular, calcaneal, bicipital, and trochanteric bursae, usually cause lameness in horses. Local swelling, heat, and pain may be evident in horses with bicipital and calcaneal bursitis but are often not present with navicular and trochanteric bursitis. In addition, bursitis at any of these locations is often secondary to other musculoskeletal problems that usually require additional imaging modalities, including radiology, ultrasonography, and magnetic resonance imaging (MRI). (See the discussion of specific bursal problems next.)

In general, most bursal problems can be diagnosed with a combination of radiology (including contrast radiology) and ultrasonography. The exception to this is the navicular bursa, where MRI is often needed to document the extent of the damage within the bursa (Figure 82-2). With subcutaneous bursae, ultrasonography can document fluid accumulation beneath the skin and rule out involvement of deeper structures. Ultrasonography is also helpful to determine primary tendon or ligament pathology, especially with bicipital, infraspinatus, and calcaneal bursitis, and to identify involvement of the bursa from wounds and penetrating injuries. Injection of sterile contrast media combined with radiographs can also be used to determine the extent of a subcutaneous bursa but is especially beneficial to document bursal involvement from penetrating injuries. This is especially true for injuries involving the navicular and calcaneal bursae (Figure 82-3).

Horses with infectious bursitis from wounds are typically much more lame than those with noninfectious bursitis, and they often present with clinical signs consistent with any synovial infection.[10] Their lameness grade is often 4 to 5 out of 5, and they often have significant heat, pain, swelling, and drainage associated with the infected bursa. Although any bursa can become infected, those involved most frequently include the navicular, calcaneal, and bicipital bursae. The diagnosis,

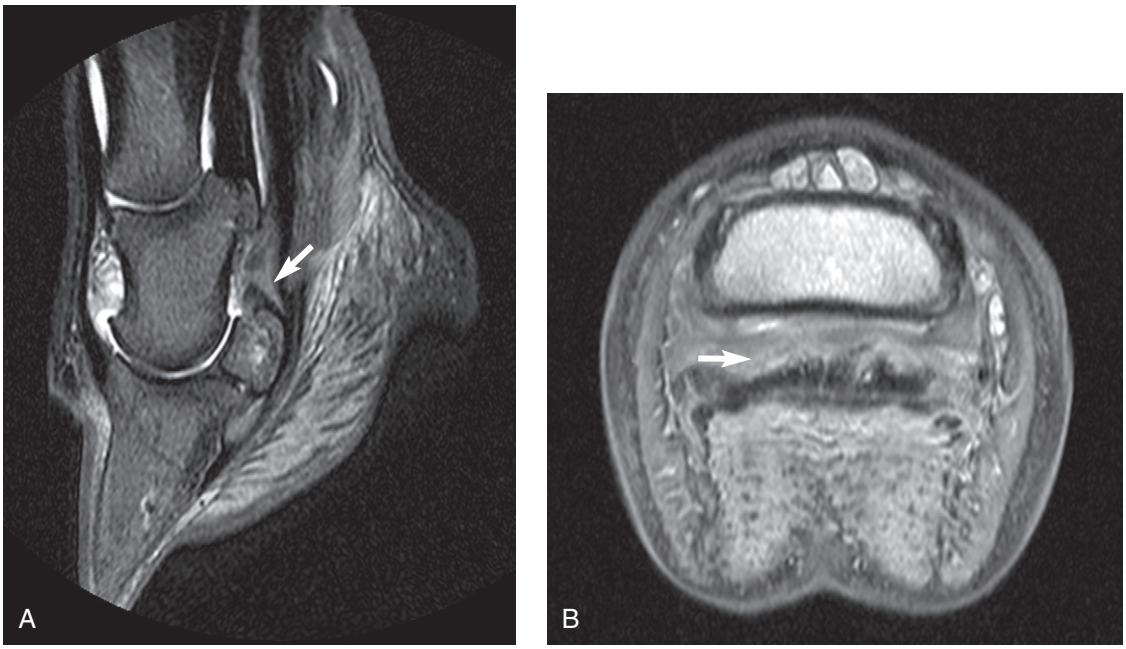

Figure 82-2. Lateral **(A)** and dorsopalmar **(B)** MRIs of an 8-year-old Quarter Horse gelding showing obliteration of the navicular bursa with scar tissue *(arrows)* secondary to chronic navicular disease and deep digital flexor tendinitis.

treatment, and prognosis of infectious bursitis are similar to those of any other synovial cavity in the horse.

GENERAL TREATMENT OF BURSA-RELATED CONDITIONS

Treatment of bursitis varies considerably depending on the location, duration, and presence of infection or other primary musculoskeletal problems. In general, conservative treatments are usually attempted first unless synovial infection is present. Acute *subcutaneous bursae* of the elbow, tarsus, and carpus usually respond to topical and systemic anti-inflammatories, pressure bandaging, and removal of the inciting cause. More chronic hygromas may be treated with drainage, injection of corticosteroids or iodine-containing compound and bandaging. Surgical drainage using Penrose drains may also be used, although I do not advocate it, and surgical extirpation of the entire subcutaneous bursal sac is recommended if other treatments fail.[1,2] Treatment of *subligamentous bursae* primarily involves the supraspinous bursa (fistulous withers), which is usually infected and usually requires surgical extirpation of the bursa and fistulas together with systemic treatment with antimicrobials.[12,13]

Treatment of nonseptic and septic bursitis affecting true or synovial bursae is similar to that for synovitis of joints and tendon sheaths. *Acute nonseptic bursitis* can be treated with systemic, topical, and/or intrasynovial anti-inflammatories, such as corticosteroids and hyaluronan, combined with rest and pressure bandaging. *Infected bursae* should be treated with wound débridement, synovial lavage and drainage, and systemic, intrabursal, and regional antimicrobials.[10] Approaches for bursoscopy of the navicular, calcaneal, and bicipital bursae have been described and are preferred over open techniques in most cases.[4,14-16] In addition, bursoscopy is recommended as both a diagnostic and treatment tool in many cases of chronic bursitis where a definitive cause cannot be determined.

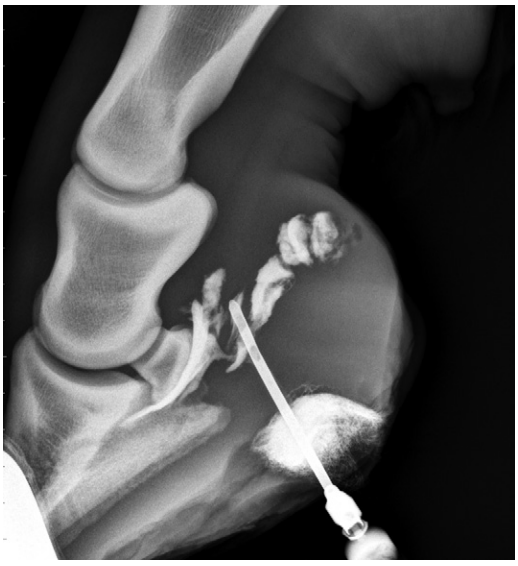

Figure 82-3. Lateral contrast radiograph of the foot of a horse with drainage from the lateral sulcus of the frog of 6 weeks' duration. Placement of a teat cannula into the defect and injection of contrast media confirmed communication with the navicular bursa.

PROGNOSIS OF BURSA-RELATED CONDITIONS

In general, bursitis in small subcutaneous and subligamentous bursae usually responds well to treatment and has an excellent prognosis for complete resolution. More chronically problematic subcutaneous bursae or those that become infected can present a greater clinical challenge, but most can be successfully resolved without causing lameness. Recurrence of infection tends to arise most commonly in horses with supraspinous bursitis, and leads to redevelopment of draining tracts in the withers area.[12,13] The prognosis for horses with infectious bursitis is nearly always more serious than those with noninfectious bursitis, regardless of the location. Infection within bony structures nearly always worsens the prognosis, especially within the navicular, calcaneal, and bicipital bursae.

MANAGEMENT OF CLINICALLY SIGNIFICANT BURSAE

Subcutaneous Bursae

Olecranon Tuberosity (Olecranon Bursitis)

Shoe boil or capped elbows are two common names for olecranon bursitis.[17] It has been regarded as a problem in draft breeds, but a recent report detailed 10 cases in Quarter Horses.[2] It is usually seen as a movable swelling over the point of the olecranon tuberosity that is usually trauma-induced from the shoe of the affected limb hitting the point of the elbow during motion or more commonly when the horse is lying down. The swelling may contain fluid, primarily fibrous tissue, or both, in the chronic stages (Figure 82-4). Lameness is usually not present unless the bursa is greatly enlarged or infected. The diagnosis can usually be made on physical findings alone. However, if the bursa appears infected, radiographs should be

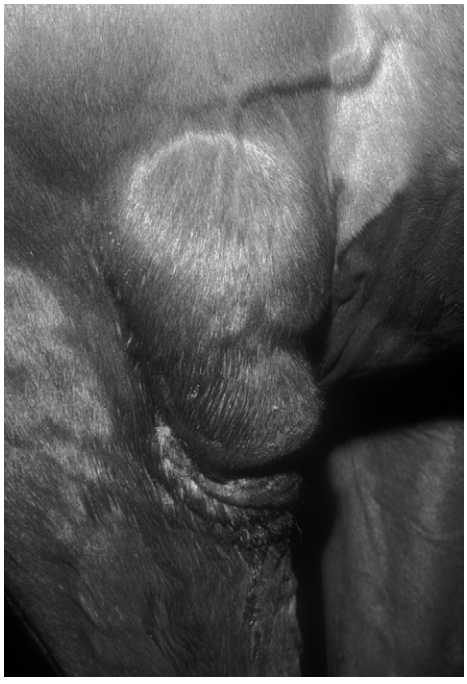

Figure 82-4. Large "shoe boil" on the olecranon process that was soft, fluctuant, and nonpainful.

taken to rule out trauma or infection involving the olecranon process.

In the acute stage, the condition may resolve by preventing further trauma to the region, with the use of a shoe boil roll or boot. The fluid can be drained aseptically and corticosteroids injected, and this can be repeated multiple times.[2] However, in one report, bursitis in 7 of 10 horses treated with intrabursal steroids did not resolve.[2] Anecdotally, injecting 7% iodine or iodine-based radiographic contrast material into the lesion or packing the incised bursa with iodine-soaked gauze have also been recommended with variable success.

Surgical intervention either by drain placement or *en bloc* resection appears to have the greatest success.[2,18] *En bloc* resection is the treatment of choice for large and mature acquired olecranon bursae. Preferentially the procedure is performed standing. Local anesthesia is administered and a curved skin incision over the lateral aspect is made. A plane of dissection is made to remove the mass in its entirety. Excess skin is removed as needed, and the deep and subcutaneous layers are closed with absorbable sutures. The skin is then closed in a tension-reducing manner, such as widely placed vertical mattress sutures. A stent bandage is placed to protect the suture line and provide some compression. The horse can be maintained in cross ties or an overhead wire until satisfactory healing is attained, usually for 2 to 3 weeks.[2] *En bloc* resection is regarded as a superior way to manage the lesion with good cosmesis.[2]

Tuber Calcanei (Capped Hock)

The subcutaneous bursa on the tuber calcanei is located between the skin and the superficial digital flexor tendon and is analogous to the bursa at the point of the olecranon. However, the subcutaneous bursa may communicate with the subtendinous bursa at the point of the hock in about one-third of horses.[19] Direct trauma to the point of the hock from a kick or the horse hitting a hard object such as a stall wall (this may be a bad habit and occur repeatedly) or fence is the most common cause of a capped hock.[20] These injuries may or may not be associated with a wound.

A nonseptic capped hock is usually characterized by a soft, fluctuant swelling located directly at the point of the hock. Subcutaneous edema in acute cases and thickening and fibrosis of surrounding tissues in chronic cases may make the diagnosis more difficult. Lameness may or may not be present, depending on the time since injury, but it is usually minimal after a few days. In general, signs of lameness are much less for nonseptic injuries of the subcutaneous bursa than the calcaneal bursa. Penetrating injuries to the tuber calcanei may involve the subcutaneous bursa, the calcaneal bursa, or both, and close evaluation of these injuries is important to determine the ideal treatment protocol.

Treatment of horses with a capped hock can be variable depending on the severity. Small swellings may merely be a cosmetic concern and not require treatment. However, measures to eliminate further trauma to the tuber calcanei are suggested to prevent the capped hock from worsening.[1] Larger swellings are treated in a manner similar to olecranon bursitis and carpal hygromas. Topical application of anti-inflammatories such as ice, dimethyl sulfoxide (DMSO), or diclofenac liposomal cream (Surpass) combined with bandaging may be beneficial in the acute stage. Other options include aseptic drainage and injection of corticosteroids or iodinated contrast agents.[1] Counterpressure with bandaging is recommended for at least 2 weeks but can be difficult in this high motion area. A compressive sleeve type of bandage can be beneficial in these cases. Surgical drainage using Penrose drains or complete removal of the bursa is rarely recommended because of the problems with wound healing in this location. The prognosis for acute swellings is usually very good with appropriate treatment. Superficial wounds to the subcutaneous bursa usually resolve with routine wound care and have an excellent prognosis.[6] Chronic swellings at the tuber calcanei can be problematic but are more of a cosmetic than functional problem in most horses. However, puncture wounds to the tuber calcanei can contribute to infection within the subcutaneous and calcaneal bursae and subsequent osteitis or osteomyelitis of the calcaneus. Treatment of severe infection in this location can be problematic and may greatly limit future performance.

Carpal Hygroma

A hygroma is an acquired bursa on the dorsum of the carpus caused by trauma from falling, getting up and down, or hitting a fence or by chronically pawing and hitting the dorsum of the carpus, such as on a feeder or water trough.[18,21] A nonpainful fluctuant, uniform soft tissue swelling develops on the dorsal aspect of the carpus that is not associated with joints or tendon sheaths (Figure 82-5). Range of motion of the carpus may be mechanically reduced, but lameness is unusual. However, if the hygroma is infected, then lameness can be moderate to severe. Injection of a radiopaque contrast agent into the hygroma usually confirms its extra-articular position, although ultrasonography can be used. If a hygroma is suspected of being infected, a fluid sample should be submitted for cytologic evaluation and culture, similar to the procedures performed for septic arthritis.

Some hygromas resolve on their own, but in most cases drainage and injection of anti-inflammatory agents are used,

Figure 82-5. A hygroma on the cranial aspect of the carpus that developed from trauma to the area.

and in many horses repeated injections are necessary.[18,21] I have seen spontaneous resolution of carpal hygromas after injection of iodine-containing contrast agent. Injection of atropine (7 mg total dose) may also help to resolve the swelling. Owners should be warned that bandaging and possibly splinting is an essential component of treatment and that long-term chronic thickening may occur. Draining the mass with a Penrose drain and bandaging have also been used successfully for recurrent hygromas. In recalcitrant cases, surgical excision is best accomplished if the fluid sac is left intact (complete extirpation) and is dissected free *in toto* from the other tissues. Soft tissue and skin closure are routine, and a splint or sleeve cast should be used to prevent carpal flexion for at least 10 days or until the sutures are removed. The limb is subsequently kept under a bandage for 2 more weeks.

Subtendinous Bursae

Navicular Bursa

The navicular bursa (bursa podotrochlearis) is a true synovial bursa interposed between the deep digital flexor tendon (DDFT) and the fibrocartilaginous distal scutum covering the flexor surface of the navicular bone. It has a large outpouching that extends proximally almost to the distal aspect of the digital flexor tendon sheath (see Figure 82-3). The volume of the navicular bursa is small (2 to 3 mL), and although no direct communication exists between it and the distal interphalangeal joint, both medication and local anesthesia can diffuse into the bursa from the joint.

The cause of *nonseptic navicular bursitis* is repetitive-use trauma in horses with navicular disease or syndrome. Pathology of the flexor surface of the navicular bone and the DDFT at the level of the navicular bone is common with navicular disease or syndrome, and both can contribute to acute or chronic navicular bursitis (see Chapter 90).[22,23] Adhesions between the navicular bone and the DDFT, thickening of the bursal lining, and collapse of the bursal space are not uncommon in horses with chronic navicular disease or syndrome (see Figure 82-2). A definitive diagnosis of navicular bursitis and pathology secondary to navicular problems is best determined with MRI,[22] but is often assumed to be present in most horses with the disease. Treatment of nonseptic navicular bursitis is usually aimed at the primary navicular syndrome but often includes injections of anti-inflammatories into the navicular bursa.[22,24] Corrective trimming and shoeing to alleviate the pressure of the DDFT on the navicular bone (and therefore the bursa) is often performed concurrently.

The causes of *septic navicular bursitis* include penetrating injuries of the foot (see Chapter 90) or iatrogenic infection following intrabursal injections. The classic example is puncture of the central third of the frog with a nail that enters the navicular bursa. Bacteria and possibly other debris are deposited within the bursa, which contributes to synovial infection and severe lameness. If possible the location, direction, and depth of a nail or other metallic object are best determined with radiographs before removal. Additional diagnostics that can be performed to confirm involvement of the navicular bursa include distention of the bursa with saline to detect leakage from the wound, plain radiographs (presence of gas), radiographs with a metallic probe inserted in the previously thoroughly débrided wound, or contrast radiography (fistulogram; see Figure 82-3).[10] Concurrent fractures and osteolysis as a result of infection should also be assessed with radiography. Treatment should be aggressive and is similar to any infection within a synovial cavity. Less-invasive approaches to treat septic navicular bursitis, such as endoscopy or bursoscopy, is recommended and is usually effective.[3,4] Débridement of a large defect within the DDFT ("street nail" procedure) to provide drainage should be avoided whenever possible.[10] There are, however, still clinics that perform that surgery with good success (see Chapter 90)

Bicipital Bursa

The bicipital bursa is located between the bilobed tendon of origin of biceps brachii muscle and the M-shaped tubercles at the cranioproximal aspect of the humerus (Figure 82-6). Although uncommon, communication can exist between the shoulder joint and the bicipital bursa. Trauma to the cranial surface of the shoulder region is believed to be the most common cause of a primary bursitis.[9,25-27] Other suggested causes include a stretch or tear of the bursa or biceps tendon during the cranial phase of the stride with the limb in full extension, or by a fall or slip that results in flexion of the shoulder with extension of the elbow. Dislocation of the biceps brachii tendon possibly associated with congenital hypoplasia of the minor tubercle has also been reported to cause bicipital bursitis.[28,29]

Reported causes of infection and inflammation of the bicipital bursa include an open or penetrating wound or hematogenous spread to the bursa.[8,9,26] Other reported and associated causes include *Brucella abortus,* in very old reports[7]; influenza or other viral respiratory disease outbreaks, in one case following a long trailer ride[9]; septic tendinitis and arthritis of the scapulohumeral joint; and tendinitis and humeral osteitis.[30,31]

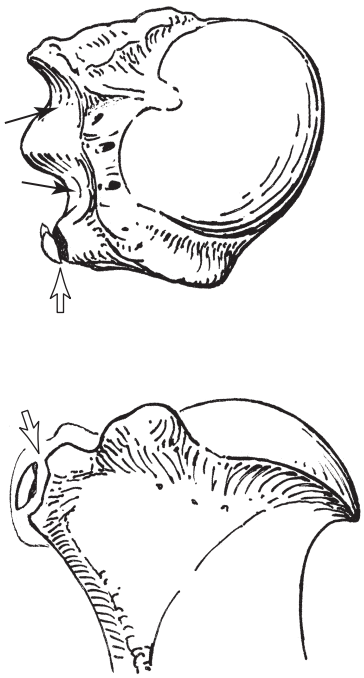

Figure 82-6. Diagram illustrating the location of the bicipital bursa between the M-shaped humeral tubercles *(black thin arrows).* Fractures of the lateral tubercle may contribute to bicipital bursitis *(fat white arrows).*

The signs of lameness usually have an acute onset, and swelling over the cranial aspect of the shoulder region may be evident with or without a wound being present. Pressure applied over the biceps tendon and bursal region and manipulation of the elbow into hyperextension may stress the biceps brachii tendon, which helps localize the lesion.[32] The lameness grade is often 3 to 4 out of 5 (greater if septic) and is often characterized by a shortened cranial phase of the stride, decreased height of the foot flight arc, reduced carpal flexion, and a fixed shoulder appearance during movement. If the localizing signs are not obvious, bursitis can be confirmed by centesis and local anesthesia of the bursa. A proximal approach to the bursa is considered the most successful for bursal centesis (Figure 82-7).[33]

Radiographs of the shoulder are recommended to identify any osseous changes in the tubercles, bicipital groove, or bursa; to rule out the possibility of fractures of the supraglenoid tuberosity or proximal humerus; and to rule out ossification of the biceps tendon.[9,25,26] Radiographic changes associated with bursitis include mottled changes in the tubercle, demineralization of the greater tubercle, osseous densities in the periarticular region, osseous cysts in the cranioproximal humerus, osteitis in the bicipital groove, ossification of the tendon, and calcification of the bursa (see Figure 82-6).[25,27] Radiographs may appear normal if the disease involves the bursa only. An ultrasonographic examination of the biceps tendon, bursa, and bicipital groove can be very informative, even when radiographs appear normal.[34-36] Ultrasonographic changes associated with bursitis include edema or hemorrhage in the biceps tendon or bursa, disruption of the tendon architecture with peritendinous thickening, an irregular surface of the bicipital groove, and hyperechoic material in the bicipital bursa.[9,37]

Noninfectious bursitis without radiographic abnormalities of the humerus may respond favorably to rest, parenteral administration of nonsteroidal anti-inflammatory drugs (NSAIDs), and in some cases intrasynovial injection of the bursa with corticoids and hyaluronan.[26,38] Application of topical NSAIDs may also help alleviate the inflammatory response. Manipulating the limb passively through a range of motion may also be helpful.[26,27,38] In cases where the tendon has been injured, rest periods up to 3 months followed by paddock rest for another 3 months may be required to allow healing of the tendon.

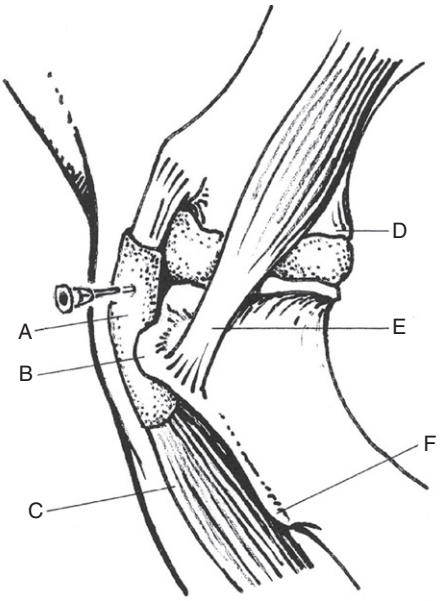

Figure 82-7. Centesis of the bicipital bursa can be difficult, but the proximal approach adjacent to the lateral tubercle is recommended. *A,* Bicipital bursa; *B,* lateral tuberosity of the humerus; *C,* biceps brachii muscle; *D,* glenoid; *E,* tendon of the infraspinatus muscle; *F,* deltoid tuberosity.

Bursitis from either a displaced fracture, osseous changes associated with the proximocranial aspect of the humerus, or sepsis generally requires surgery to resolve the problem. Incisional as well as arthroscopic approaches to the intertubercular bursa have been used and described.[15,16,25,26] If a fracture is present the fragment should be removed and the bed débrided and smoothed (see Figure 82-6). With septic bicipital bursitis, bursoscopy is recommended to permit débridement, lavage, and drainage followed by instillation of a broad-spectrum antimicrobial drug into the synovial cavity.[15]

Though acute cases of nonseptic bursitis in the absence of a fracture usually respond favorably to conservative treatment, this approach appears less satisfactory for more chronic cases of nonseptic or septic bursitis.[9,25] In one report, all three horses that were suffering from chronic nonseptic bicipital bursitis and were treated surgically became pasture sound.[27] Surgical intervention with débridement, lavage, and appropriate antimicrobial therapy usually results in a favorable prognosis in horses with septic bicipital bursitis.[8,9,30,31]

Infraspinatus Bursa

The infraspinatus bursa is located between the tendon of the infraspinatus muscle and the caudal eminence of the greater tubercle of the proximal humerus. The bursa is not visible in most horses except when inflamed or septic, and it is a rare cause of lameness.[39] Severe adduction of the forelimb and direct trauma to the region are considered the most likely causes.[39,40] The involved forelimb in affected horses may be held abducted, presumably in an attempt to reduce the pressure on the infraspinatus bursa. During exercise, a moderate lameness is usually present with an obvious decreased cranial stride.[39,40] Adduction of the limb reportedly elicits a painful response and results in increased signs of lameness at exercise.[40] If the bursa is septic, the lameness exhibited is severe.[39] Ultrasonographic evaluation and comparison with the opposite limb may be required along with ultrasound-guided centesis and local analgesia for a definitive diagnosis.[39]

In the acute stage, synoviocentesis and administration of corticosteroids into the bursa is recommended. With sufficient stall rest (6 weeks or more) and parenteral administration of NSAIDs, a good end result can be expected. With chronic cases, varying degrees of lameness may remain.[40] Septic involvement of this bursa will require endoscopic débridement and flushing. The initial distention of the bursa may require ultrasonographically guided needle placement followed by fluid instillation. The bursa is small and movement of instruments is restricted by the overlying muscle and infraspinatus tendon.[39] In one report, three horses returned to soundness after surgical treatment of septic infraspinatus bursitis.[39]

Calcaneal Bursa

Swelling at the point of the hock (calcaneus) is usually attributable to damage to the subcutaneous calcaneal bursa (capped hock) or to problems within the subtendinous calcaneal bursa

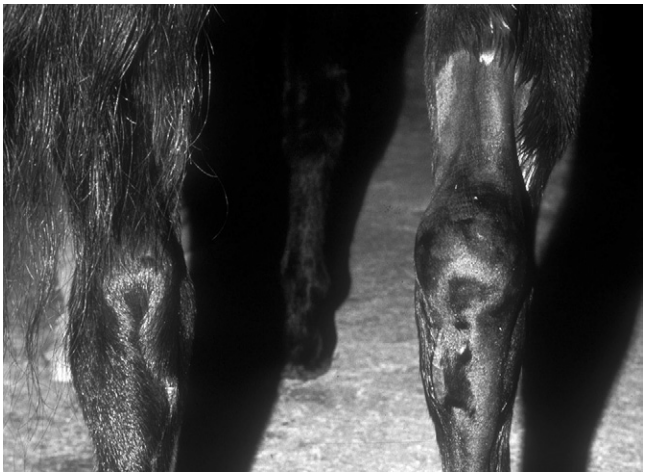

Figure 82-8. Horse with significant swelling and effusion within the right calcaneal bursa from lateral luxation of the superficial digital flexor tendon.

located beneath the superficial digital flexor tendon (SDFT). The subtendinous calcaneal bursa may be further divided into the gastrocnemius calcaneal bursa and the intertendinous calcaneal bursa, but in most horses these two bursae communicate and should be considered as one synovial structure.[19] The subcutaneous bursa at the tarsus is analogous to the bursa at the point of the olecranon but communicates with the subtendinous bursa in about 30% of horses.[19] The subtendinous calcaneal bursa is a true synovial cavity analogous to the bicipital bursa and navicular bursa, and problems within this anatomic structure are much more problematic than those within the subcutaneous bursa. The subtendinous calcaneal bursa extends approximately 7 cm (3 inches) distad and 9.6 cm (4 inches) proximad, relative to the tuber calcanei (point of the hock).[19] An acquired subcutaneous calcaneal bursa is usually located over a much smaller area. Therefore it is very important to determine the location of the swelling over the point of the hock to best address treatment and prognosis. This is especially true for traumatic wounds in this location.

Subtendinous Calcaneal Bursitis

True calcaneal bursitis (subtendinous) may be secondary to trauma such as that seen with luxation of the SDFT from the tuber calcanei and damage to the attachment of the gastrocnemius tendon to the tuber calcanei (Figure 82-8)[41,42] Nonseptic osteolytic lesions within the calcaneus at the insertion sites of the gastrocnemius tendon and the plantar ligament have also been reported to cause calcaneal bursitis.[14,42] Horses with nonseptic subtendinous calcaneal bursitis can be a diagnostic challenge because fluid distention of the bursa is often not readily apparent. However, the area above and below the retinaculum that keeps the SDFT on top of the tuber calcanei is usually enlarged compared to the opposite calcaneus, and fluid distention is usually palpable either above or below the retinaculum on the medial or lateral aspects of the tuber calcanei. In affected horses, direct palpation of the bursa often elicits a pain response. They are lame at the trot, and they hyperreact to tarsal flexion. Intrasynovial anesthesia directly into the subtendinous calcaneal bursa is usually the best approach to confirm the location of the lameness (Figure 82-9).

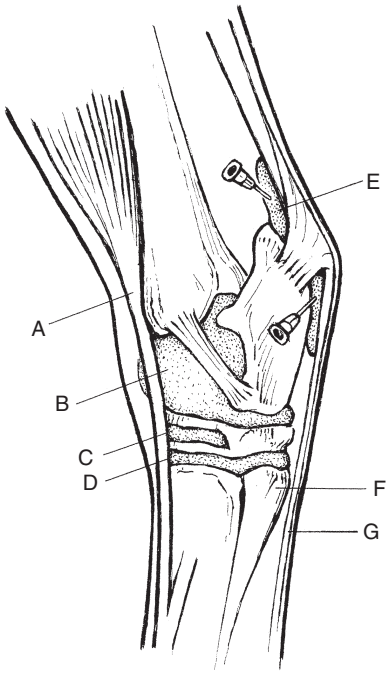

Figure 82-9. Diagram illustrating the injection sites for centesis and blocking the subtendinous calcaneal bursa above or below the retinaculum. *A,* Long digital extensor tendon; *B,* tarsocrural joint; *C,* distal intertarsal joint; *D,* tarsometatarsal joint; *E,* calcaneal bursa; *F,* head of fourth metatarsal bone; *G,* superficial digital flexor tendon.

A definitive diagnosis of the cause of nonseptic subtendinous calcaneal bursitis usually requires a combination of radiography and ultrasonography. However, endoscopy of the bursa is recommended as a diagnostic tool if other imaging results are negative.[14] Scintigraphy and MRI may also be beneficial but are not necessary in most cases. The cause of nonseptic subtendinous calcaneal bursitis may not be determined in all cases. Treatment of nonseptic subtendinous calcaneal bursitis depends on the initiating cause, but it is similar to that for other types of synovial inflammation. Acute bursitis without a defined cause can be treated with intrasynovial triamcinolone and hyaluronan combined with a short period of rest. Known causes of subtendinous calcaneal bursitis, such as osteolytic lesions within the calcaneus, should be débrided endoscopically.[14,42] In some chronic cases, endoscopy should be used as both a diagnostic and treatment tool for lesions within the gastrocnemius tendon, SDFT, and other soft tissue injuries within the bursa. Based on a very limited number of cases, horses with osteolytic lesions of the calcaneus and chronic wounds involving the bursa have a guarded prognosis for athletic activity.

Penetrating wounds that enter the subtendinous calcaneal bursa appear to be the most common cause for swelling and lameness referable to this structure.[6] These injuries often involve small puncture wounds that do not seem to be very important initially but subsequently result in infection within the bursa (Figure 82-10). Secondary osteitis or osteomyelitis of the tuber calcanei is not uncommon. The lameness may be mild initially but becomes severe with the onset of synovial infection. The point of the hock is usually swollen and painful to palpation, and purulent exudate often exits the wound. The diagnosis of infection is determined similarly to other sites of synovial

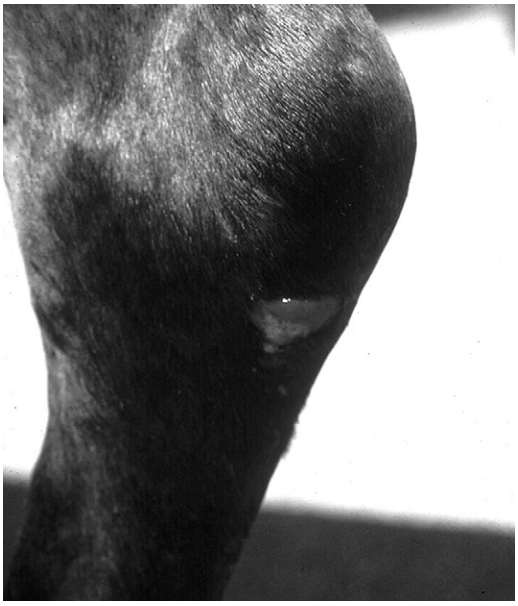

Figure 82-10. Small wound just distal to the tuber calcanei that entered the distal aspect of the calcaneal bursa. The horse presented with severe lameness and swelling and pain associated with the calcaneal bursa.

infection and includes a combination of plain and contrast radiography, ultrasonography, aspiration of synovial fluid, and culture.

Horses with septic calcaneal bursitis should be treated with a combination of synovial lavage (endoscopy), local and parenteral antibiotics, including IV regional perfusion, and NSAIDs.[10,14] Secondary osteomyelitis of the calcaneus should be débrided as part of the treatment protocol. In one study, 75% of horses with wounds involving the calcaneal bursa survived, but only 44% of horses with secondary osteomyelitis of the tuber calcanei survived.[6] Horses with septic calcaneal bursitis tend to have a fair to guarded prognosis for athletic use because of the high motion required in this area during limb flexion.

Subcutaneous calcaneal bursitis was discussed earlier.

Trochanteric Bursa (Trochanteric Lameness, Whirlbone Lameness)

The trochanteric bursa is located beneath the strong, flat tendon of the middle gluteus muscle as it passes over the convexity of greater trochanter of the femur.[43] The inflammatory response may involve the tendon of the middle gluteus muscle as well as the cartilage over the trochanter. In many cases, there is a concurrent source of chronic lameness in the same limb, or chronic forelimb lameness may cause the horse to place more strain on the hindlimbs.[43,44] A bruise resulting from the horse falling on the affected side, or strain of the tendon during racing or training, may also contribute to the bursitis.[44]

Lameness is usually mild, but pain may be evident when pressure is applied over the greater trochanter.[43] At rest, the limb may remain flexed, and the inside wall of the foot may be worn more than the outside wall.[43] The horse tends to travel "dog fashion," with the hindquarters moving toward the sound side, because the stride of the affected limb is shorter than that of the sound side. The gluteal muscles can atrophy with chronicity. The condition is difficult to differentiate from inflammation of the coxofemoral joint and may be confused with spavin lameness. Injection of a local anesthetic into the bursa may be helpful in confirming the location of the lameness. Addressing other concurrent sources of lameness is an important aspect of treatment. However, injection of the bursa with corticosteroids appears to be the most effective method to treat the bursitis. The prognosis is variable and often influenced by the second source of lameness.

Cunean Bursa

The cunean bursa is located on the medial surface of the distal tarsus between the medial collateral ligament of the tarsus and the medial branch of the tibialis cranialis (cunean) tendon. The bursa is relatively small and is not routinely anesthetized or treated alone, because it often communicates with the distal intertarsal (DIT) joint.[45] Cunean bursitis infrequently occurs as a separate clinical entity but is often considered part of the distal tarsal osteoarthritis complex. However, some clinicians treat the cunean bursa concurrently when medicating the DIT joint in horses with distal tarsal osteoarthritis.

Bursa of the Long Digital Extensor Tendon

The bursa of the long digital extensor is beneath the common origin of the long digital extensor and peroneus tertius muscles over the lateral surface of the tibia.[1] It communicates with the lateral femorotibial joint and can be seen during arthroscopy of the joint. Problems within this bursa are rare in horses.

Bursa of Common Digital Extensor Tendon

The bursa of the common digital extensor tendon is beneath the common digital extensor tendon at the level of the fetlock and pastern joints. Intersynovial fistulas between this bursa and the fetlock and pastern joints have been reported to occur[1] but are extremely rare.

Bursa of Extensor Carpi Radialis Tendon

The bursa of the extensor carpi radialis tendon is beneath the extensor carpi radialis tendon and the third carpal bone. It has been recommended to avoid this bursa when performing a carpal arthrotomy.[1]

Subligamentous Bursae

Supraspinous Bursa

The supraspinous bursa is located beneath the nuchal ligament over the third and fourth thoracic vertebrae at the withers. Fistula development secondary to infection is the most common clinical entity associated with this bursa and is referred to as *fistulous withers* (see Figure 28-8). The cause of the infection is not always readily apparent as a wound or penetrating injury and therefore often not part of the history. Infection with *Brucella abortus* was found in 9 of 24 horses with fistulous withers in one study, and seropositive horses were more likely to be pastured with cattle and have vertebral osteomyelitis than seronegative horses.[11] In another study, beta-hemolytic *Streptococcus* spp. were the most common bacterial isolates found.[12]

Most horses with supraspinous bursitis present with either swelling alone or a draining fistula over the withers. Direct palpation of the area usually elicits a pain response, but the horse is usually not lame or systemically ill. Septic bursitis alone or septic bursitis with osteomyelitis of the dorsal thoracic vertebral processes are possible; therefore, radiographs of the withers are recommended. Treatment with systemic antimicrobials alone is usually unsuccessful, and surgical débridement of the bursa and draining tracts is recommended.[11-13] The surgery can be performed in the standing or recumbent horse, multiple surgeries may be required, and the most common complication is recurrence of the fistulas.[12,13] Preoperative injection of a diluted vital dye, such as Evans or methylene blue, facilitates intraoperative recognition of the associated cavities within the infected tissue. The goal of the surgical intervention is to remove all the discolored membranes and to facilitate drainage.

Atlantal or Nuchal Bursa

The atlantal bursa is located between the nuchal ligament and the rectus capitis dorsalis muscle at the level of the first cervical vertebra (poll).[1] The layman's term for bursitis in the atlantal bursa is "poll evil." It has also been called the "nuchal bursa" and is thought to be secondary to pathology of the nuchal ligament at the level of the first or second cervical vertebrae.[46] Nuchal bursitis is considered to be a rare condition, but veterinarians in the field of equine sports medicine diagnose bursitis of the nuchal ligament quite frequently.[46] It is difficult to confirm the diagnosis on clinical signs and ultrasonographic examination alone. A common treatment for nonseptic nuchal or atlantal bursitis is ultrasound-guided injection of corticosteroids into the bursa. Calcification in the bursa region may be observed on radiographs of the poll and may be associated with recurrent injections of the bursa.[46] Surgical débridement may follow the same path as described earlier for supraspinous bursitis.

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CHAPTER
83

Diagnosis and Management of Tendon and Ligament Disorders

Charlotte S. Avella and Roger K. W. Smith

TENDON BIOLOGY

Structure of Tendons and Ligaments: From Molecule to Organ

Tendons and ligaments are complex organs built up in a hierarchical arrangement of increasingly smaller subunits (Figure 83-1). Tendon is highly hydrated, having a water content of around 70%, and the remaining 30% (the dry weight) is composed predominantly of type I collagen. Collagen is synthesized as procollagen molecules consisting of an α-helical chain and amino-terminal and carboxy-terminal extensions that are cleaved by procollagenases immediately before three α-chains are combined to confer a single collagen fibril. This process appears to take place postnatally outside the cell, but recent evidence has suggested that the original structural template for tendon formation is laid down by cells in the embryo that secrete formed fibrils into the extracellular space.[1] Thereafter, growth of the tendon occurs via a "seed-and-feed" mechanism, whereby collagen fibrils form extracellularly and accrete onto the sides of the template fibrils.

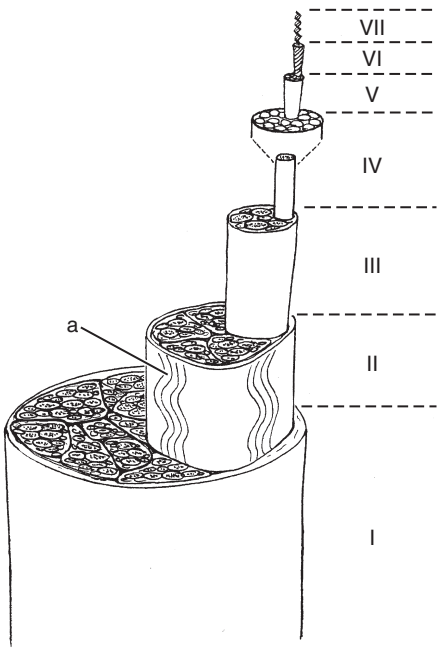

Figure 83-1. The structural hierarchy of tendon. The tendon is composed of increasingly smaller subunits, from fascicles visible to the naked eye (see Figure 83-3), to fibers seen under light microscopy, to individual collagen fibrils seen by electron microscopy. *I,* Tendon unit surrounded by paratenon in extrasynovial locations and epitenon in synovial locations; *II,* third-degree fascicle (1 to 3 mm diameter); *III,* second-degree fascicle (400 to 1000 μm diameter); *IV,* first degree fascicle (15 to 400 μm diameter; *V,* collagen fiber (1 to 20 μm diameter); *VI,* collagen fibril (20 to 150 nm diameter); *VII,* collagen triple helix (1 nm diameter); *a,* crimp.

The structural and material properties of tendon depend on a large number of factors, including the spatial organization of the collagen fibrils and their cross-linking. The determination of collagen fibril size, organization, and cross-linking is provided by an array of noncollagenous proteins, many of which are yet to be characterized fully. The next most abundant protein in young tendon is cartilage oligomeric matrix protein (COMP),[2] which is believed to be involved in the organization of the collagen fibril framework during formation and growth. In support of this proposed action, experimental data have shown that COMP accelerated collagen fibril formation *in vitro*.[3] From these data, it was proposed that COMP molecules bring together five separate collagen molecules in the "quarter-stagger" that is characteristic of collagen fibrils. Once these collagen molecules have formed a fibril, COMP no longer functions in binding and is displaced from the collagen fibril. In keeping with this role, levels of COMP in tensional regions of equine digital flexor tendons increase dramatically during growth and peak at around 2 years of age.[2] The importance and relevance of this protein during growth is suggested by a correlation between ultimate tensile strength of the tendon and the level of COMP at skeletal maturity.[4]

The extracellular composition varies along the length of the tendon, reflecting different biomechanical environments. Hence, the small proteoglycans (decorin, fibromodulin, lumican, and mimecan) predominate in the tensional regions, whereas the large proteoglycans (aggrecan and versican) are mainly, but not exclusively, located in the regions of the tendon that are subjected to compression as the tendon changes direction over a bony prominence, reflecting the fibrocartilagenous nature of the tendon in these regions (e.g., the deep digital

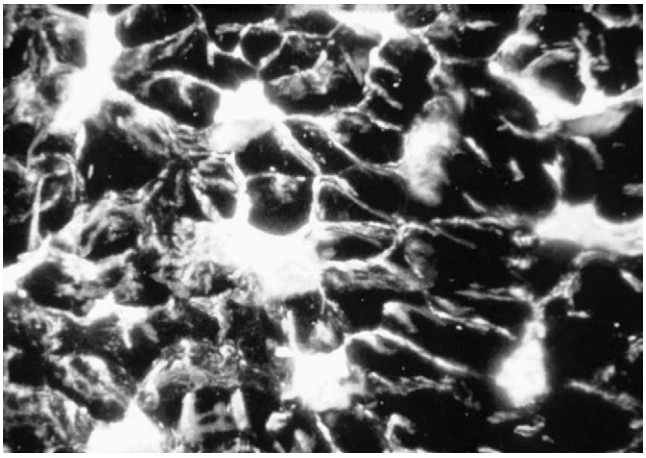

Figure 83-2. Transverse section of tendon stained to show the cytoplasmic membrane of the tenocytes. Note the large cell bodies and the extensive network of cytoplasmic extensions, which connect adjacent cells via gap junctions to produce a syncytium capable of orchestrating coordinated responses to loading. (Courtesy of Dr. Jim Ralphs, Cardiff University, UK.)

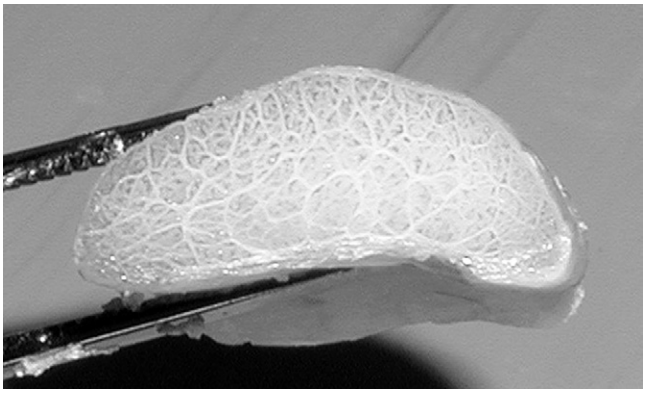

Figure 83-3. Cut surface of a frozen piece of superficial digital flexor tendon revealing the extensive interfascicular tissue, the endotenon, which contains the internal vascular and nervous supply to the tendon and is responsible for interfascicular gliding movements (where most of the tendon's stretch occurs). (From Smith RKW, Goodship AE: Tendon and Ligament Physiology. p. 130. In Hinchcliff KW, Kaneps AJ, Geor RJ (eds): Equine Sports Medicine and Surgery. Saunders, Philadelphia, 2004.)

flexor tendon [DDFT] at the level of the metacarpophalangeal joint).[5,6]

Bundles of collagen fibrils are grouped together into fibers that are separated by cytoplasmic extensions of the cellular components of tendon, e.g., tenocytes[7] (Figure 83-2). Little is known about these cells, although several different types are identified on light microscopy on the basis of their nuclear morphology.[8,9] These different nuclear shapes may represent stages of activation or differently differentiated cells. Type I cells, which have thin, spindle-shaped nuclei characteristic of adult tendon, are most likely to be relatively inactive forms of the type II cells, whose nuclei are more oval and are abundant in young, growing tendon. The fibrocartilagenous regions of adult digital flexor tendon, subjected to external compression, contain type III cells, which have round nuclei characteristic of chondrocytes. Other cell types are found within the endotenon (see later) and are associated with aging changes (e.g., chondroid metaplasia).

On a large scale, collagen fibers are grouped together in increasingly larger subunits divided by looser connective tissue known as the *endotenon.* It is these subunits, known as *fascicles,* that are visible to the naked eye on a cut section of tendon (Figure 83-3). In relaxed tendon, the fascicles have a waveform known as *crimp,* which is best seen under polarized light and which is partly responsible for tendon elasticity. However, the majority of tendon elongation comes from the sliding of the fascicles over one another rather than stretching of the fascicles themselves, and so the endotenon is believed to be critically important for this role.[10] The endotenon carries blood vessels and nerves and contains a different type of cell, one that is more rounded; these cells are believed to be the source of mesenchymal stem cells within the tendon. The endotenon contains higher levels of certain growth factors, such as transforming growth factor-β (TGF-β), than the content of the fascicles.[11]

The endotenon is continuous with the layer of connective tissue surrounding the outside of the tendon, known as the *epitenon.* Outside this layer, around tendons that are not contained within a tendon sheath, a thicker layer known as the *paratenon* is found. The paratenon is able to stretch considerably and is therefore rarely ruptured when a tendon suffers a strain injury. This layer is believed to play particularly important roles, both in reducing frictional forces between the tendon and the surrounding soft tissues and as a supplier of new blood vessels and cellular elements for repair. It is hypothesized that the lack of this layer intrathecally and within the synovial fluid environment results in slower healing of these regions.

Where tendon changes direction over a joint, the tendon is usually enclosed within a tendon sheath (Figure 83-4). This structure provides a synovial environment for the smooth gliding of tendon over a bony prominence. Associated with this sheath, there is usually a smooth fibrocartilaginous pad covering the bony prominences, best exemplified by the scuta of the distal limb—the proximal scutum or intersesamoidean ligament at the level of the metacarpophalangeal or metatarsophalangeal (MCP/MTP) joint, the middle scutum attached to the middle phalanx, and the distal scutum attached to the distal sesamoid (navicular) bone. The tendon sheath, like a joint, consists of an outer fibrous wall and an inner synovial membrane, which also surrounds the tendons within the tendon sheath. Dividing the sheath into complete or incomplete compartments are a number of mesotenons that are composed of two layers of synovium and frequently, but not always, carry the blood supply to the tendon.

Blood Supply

The blood supply to the tendon comes from its muscular origin and osseous insertion and variably from accessory ligaments, the paratenon, and mesotenon attachments. However, work on equine digital flexor tendons showed that stripping the paratenon failed to cause any ischemic damage to the tendon,[12,13] indicating the importance of the intratendinous supply for this tendon. Based on the microvascular anatomy determined by microradiographs,[14] this intratendinous blood supply is most abundant around the periphery of the tendon, which has led to the hypothesis that the central region of the tendon is relatively

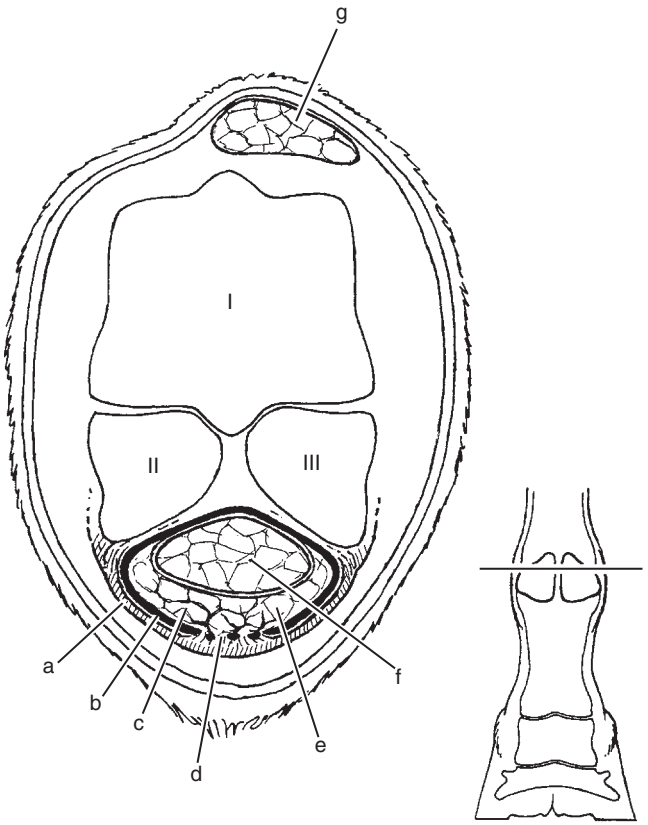

Figure 83-4. The anatomy of a tendon within a tendon sheath. Note the absence of a paratenon and the presence of mesotenon attachments, which are incomplete along the length of a sheath and through which blood supply gains access to the tendon. *a,* Palmar annular ligament; *b,* digital flexor tendon sheath wall; *c,* superficial digital flexor tendon (SDFT) endotenon; *d,* mesotenon attachment; *e,* SDFT fascicle; *f,* deep digital flexor tendon; *g,* common digital extensor tendon; *I,* MCIII; *II* and *III,* proximal sesamoid bones.

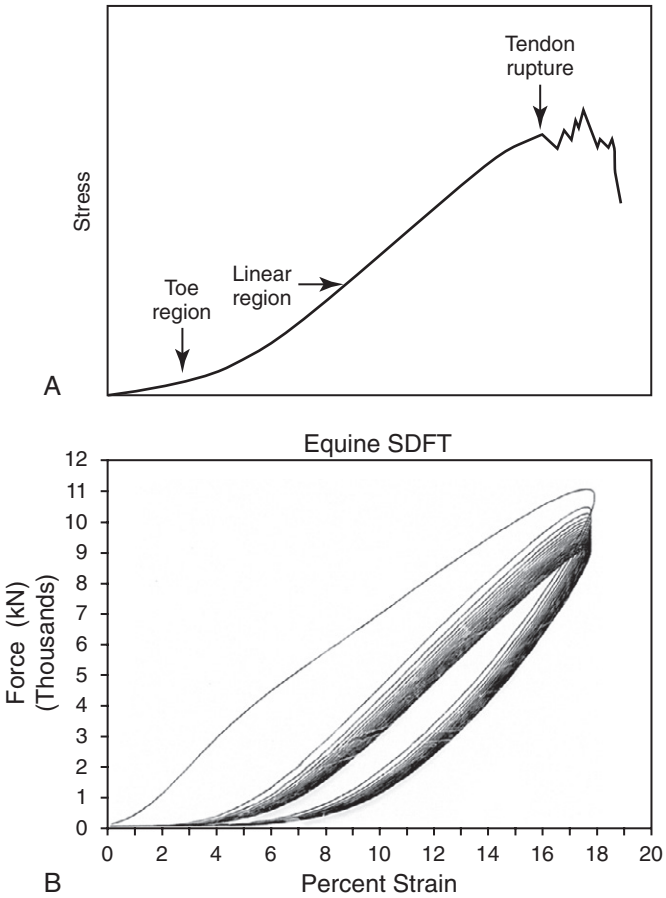

Figure 83-5. A, The stress–strain curve for tendon showing its viscoelastic properties. The toe region is associated with elimination of "crimp," and the linear region is where the tendon is operating in an elastic fashion. At the end of the linear region, the yield point is reached, where irreversible damage starts to occur before the tendon ruptures completely. **B,** Hysteresis and conditioning. The loading and unloading curves are not the same, resulting in an energy loss, which is represented by the area between the two curves (hysteresis loop). If the tendon continues to be loaded repeatedly, the curve moves to the right until it reaches a steady state, when the tendon is more elastic. *SDFT,* Superficial digital flexor tendon. (From Smith RKW, Goodship AE: Tendon and Ligament Physiology. p. 130. In Hinchcliff KW, Kaneps AJ, Geor RJ (eds): Equine Sports Medicine and Surgery. Saunders, Philadelphia, 2004.)

hypoxic, and that this is one of the reasons for the common manifestation of tendon disease as a central core lesion. However, there is little supporting evidence for this hypothesis, and, in fact, the superficial digital flexor tendon has a blood supply, as determined by xenon clearance, similar to that of resting skeletal muscle and is capable of increasing flow during exercise and after injury.[15]

Functional Characteristics of Tendons and Ligaments

Tendons and ligaments have a variety of functions: they transmit forces to move the equine skeleton, they provide support of the distal limb in the case of the digital flexor tendons, or, as ligaments, they maintain joint alignment and integrity. Their structure is optimized to perform these mechanical roles.

Tendons and ligaments have viscoelastic properties, which means that their mechanical properties vary as they are stretched. A typical force–elongation curve for tendon is shown in Figure 83-5. Initially, the tendon stretches under relatively little load, which is associated with the elimination of crimp in the fascicles. Thereafter, the curve is almost linear. It is from this area of the curve that the *structural property* of stiffness is determined (load divided by deformation). As the load increases further, there is a change in the gradient such that the tendon becomes less stiff. This is known as the *yield point,* and it is thought to arise from covalent crosslink rupture and irreversible slippage of collagen fibrils. If the tendon is stretched past this point, irreversible damage occurs. In other words, if loading is continued past this point, the tendon ruptures, which for the equine superficial digital flexor tendons (SDFT) is approximately 12 kilonewtons (kN) or 1.2 tons (ultimate tensile strength).[16,17]

If the cross-sectional area and length of the tendon is known, the stress (force per unit area) can be plotted against strain (change in length over original length), from which the *material properties* of ultimate tensile stress (N/mm^2) and Young's modulus of elasticity (E [MPa]) can be calculated. The approximate *ultimate tensile stress* of 100 MPa for the equine SDFT is similar to previously documented figures in other species (20 to 144 MPa).[18,19]

In vitro, equine flexor tendons can extend 10% to 12% of their length before they rupture (*ultimate tensile strain*), and values of up to 20% have been reported.[8] However, the ultimate

tensile strain reflects only the final strain before rupture and includes the yield portion of the stress–strain curve, which represents irreversible damage to the tendon tissue. The normal strains in the digital flexor tendons, recorded *in vivo*, are in the region of 3% to 8% at the walk, 7% to 10% at the trot, and 12% to 16% at the gallop.[20] Such strains, far greater than usually expected in tendons from most other species, may reflect the importance of the digital flexor tendon as an elastic energy store, where maximal deformation stores the most energy but also makes them operate close to their functional limit.

In addition to these basic functional characteristics, tendon has other mechanical properties of potentially important biological relevance. There is a difference between the stress–strain relationship when the tendon is loaded and the relationship when it is unloaded, which is known as hysteresis (see Figure 83-5). The area between these two curves represents the energy lost during the loading cycle. This is usually about 5% in equine tendons. Much of this energy is lost as heat and is responsible for the rise in temperature in the tendon core when the horse exercises. These temperatures can rise to as high as 44° C, which is potentially damaging to both tendon matrix and tenocytes. However, tenocytes recovered from the center of equine SDFT remain viable when subjected to rises in temperature of this magnitude, whereas those recovered from the periphery of the tendon do not.[21] This property is also present in fetal tenocytes, which suggests that the tendon has an inherent genetic adaptation to this physical process.

The loading rate has only a minimal effect on tendon biomechanics: a rapid loading rate results in a stiffer tendon, whereas repeated loading results in a less-stiff tendon, a process known as *conditioning* (see Figure 83-5). This effect appears to occur *in vivo* in humans,[22] although it is not clear whether this is also the case in horses. Because they are rarely recumbent, they usually constantly load their flexor tendons. The change is recoverable, but significant resting time is necessary.[8]

The Relationship of Structure to Function

It would appear that all tendons are formed *in utero* in a similar fashion and with a similar composition, analogous to the "blank joint" concept for articular cartilage.[6,23,24] However, as soon as the animal is born and stands, tendons and ligaments receive very different mechanical loading. It is believed that different mechanical environments drive growth and differentiation of tendons, so that at skeletal maturity, different tendons have different compositional and structural properties that relate to their function. Thus mature tendons are not all composed of the same material and hence do not all have the same functional characteristics. Furthermore, different loading patterns induce anisotropy within certain tendons. Thus, as mentioned earlier, where the digital flexor tendons change direction across the palmar/plantar aspect of the MCP/MTP joint and are subjected to external compression, they develop a cartilage-like matrix.[25]

The horse's distal limb is designed like a spring to store energy from weight-bearing for the subsequent stride.[26] The horse essentially bounces up and down on its forelimbs, whereas its hindlimbs provide propulsive force. This system is optimized by having a hyperextended MCP joint and by the energy being stored in the palmar soft tissue structures, principally the SDFT and suspensory ligaments (Figure 83-6). The superficial digital flexor muscle contains a high proportion of fibrous tissue, and data have indicated that its maximal contraction shortens the muscular unit by only 2 mm.[26] Hence, the muscle acts not to flex the MCP joint but rather to fix the origin of the SDFT and dampen high-frequency damaging oscillations. Although the muscle within the suspensory ligament is considered vestigial, it may also have a similar role in dampening the damaging oscillation during high-speed exercise.

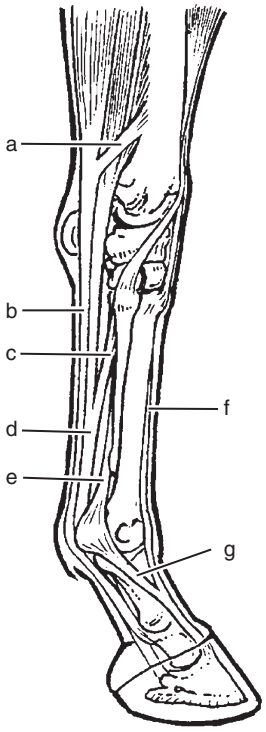

Figure 83-6. The anatomy of the equine distal limb showing the important weight-bearing tendons and ligaments on the palmar aspect of the limb. It is these structures that most frequently suffer strain-induced injury. *a,* Acessory ligament of the SDFT; *b,* SDFT; *c,* accessory ligament of the DDFT; *d,* DDFT; *e,* suspensory ligament; *f,* common digital extensor tendon (CDET); *g,* extensor branch of the suspensory ligament.

Consequently, these two structures have very similar matrix compositions and configurations: both contain high COMP levels and a combination of small and large collagen fibrils. In contrast, the common digital extensor tendon (CDET), a low-load tendon, and the deep digital flexor tendon (DDFT), which also has a different role, have lower levels of COMP and predominantly large collagen fibrils. These compositional differences are reflected in their mechanical properties. Tendons can be divided into weight-bearing tendons that function like springs (and are less stiff, or more elastic) and those that position the skeleton (which requires greater stiffness). Thus the CDET is almost twice as stiff as the SDFT.

Etiopathogenesis of Tendon Injury

Tendons and ligaments can be injured in one of two ways: from overstrain or percutaneous trauma. Overstrain injuries are believed to occur by one of two mechanisms. They can result from a sudden overloading of the structure, which overwhelms its resistive strength. This type of injury is probably the

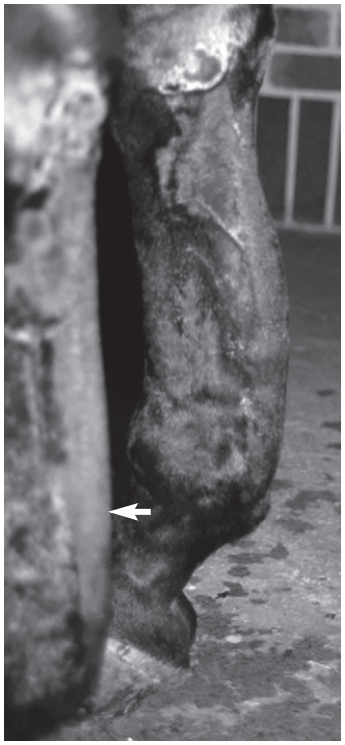

Figure 83-7. Superficial digital flexor tendinopathy. Note the bowing, or swelling, of the palmar border of the right limb. Careful attention must also be given to the contralateral limb, which may exhibit a smaller swelling that can be easily missed *(arrow)*.

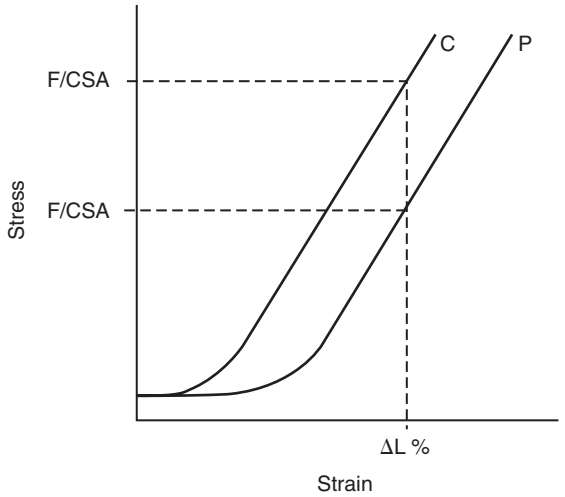

Figure 83-8. One possible explanation for the occurrence of a core lesion in the superficial digital flexor tendon. With aging and exercise, the central *(C)* crimp pattern, which is responsible for the toe region on a stress–strain curve (see Figure 83-5), is reduced more than that in the periphery *(P)*. Consequently, as the tendon is loaded, the central fibers are loaded first and take the most load for a given strain. Hence, it would be expected that they would rupture first, giving rise to a core lesion. ΔL%, Percent change in length; *F/CSA*, force per cross-sectional area. (From Wilmink J, et al: Functional significance of the morphology and micromechanics of collagen fibres in relation to partial rupture of the superficial digital flexor tendon in racehorses. Res Vet Sci 53:355, 1992.)

mechanism for most ligament and some DDFT injuries in the horse. However, for the most common strain-induced injuries in the horse, involving the palmar soft tissue structures of the metacarpal region, the clinical injury is believed to be preceded by a phase of molecular degeneration ("molecular inflammation"[27]), which induces neither a clinically evident inflammatory reaction nor any reparative response, but instead progressively weakens the tendon.[28] The evidence for this degeneration is based on four observations:

- "Asymptomatic" lesions are present both grossly and microscopically in postmortem studies of normal horses.[29] However, analysis of these lesions suggests that they are the result of healing of low-grade clinical injuries rather than true degeneration.[30]
- Many clinical strain-induced tendinopathies are bilateral, but one limb is more severely affected than the other (Figure 83-7). Careful ultrasonographic examination often reveals changes in the contralateral limb. Furthermore, in seemingly unilateral cases, blood flow studies have demonstrated increases in the "normal" contralateral tendon, which would suggest that it is not totally unaffected.
- Epidemiologic studies in both horses and humans have shown strong associations between injury rates and age.[31-33]
- A number of experimental exercise studies and postmortem analyses of normal tendons have indicated that increasing age and amounts of exercise induce tendon matrix degeneration rather than adaptation in the adult horse.[28]

Response of Tendons to Aging and Exercise

When evaluating the gross mechanical properties of the SDFT in a large group of normal horses, there is a reduction in tendon strength with age, but this is statistically insignificant because of the high variance. Therefore, in a population of normal horses, there is more than a twofold variation in ultimate tensile strength of the SDFT. This high variance arises from inaccuracies in measuring ultimate tensile strength *in vitro* and the true variation in tendon strength, which is influenced by genetic components,[34] but probably most importantly by environmental factors of aging and exercise.

The collagen crimp angle and length showed a regional reduction in the central core with exercise and age, both acting synergistically.[35,36] This results in the central fibers being straightened first under loading, and hence they are the most likely to rupture first, giving rise to a possible mechanism for the generation of the "core lesion" frequently seen clinically (Figure 83-8).

Within the tendon matrix, regional differences in collagen fibril diameter were seen in mature horses that trained for 18 months on a high-speed treadmill compared to age- and sex-matched controls that were only walked.[37] The higher proportion of small fibrils in the central region of long-term exercised horses does not appear to be associated with increased collagen content, and the collagen-related fluorescence (a measure of collagen age) is unaltered, suggesting that this change results from disassembly of the larger-diameter fibrils.

Collagen content varies little with age and exercise. The collagen in the equine SDFT has recently been shown (using aspartate racemization) to have a half-life of 198 years, whereas the noncollagenous matrix has a much shorter half-life of 2.2 years.[38] Thus, in longer-term exercised and older horses, the noncollagenous component of tendon shows changes in the

center of the tendon where clinical injury is seen and where there is an accelerated loss of glycosaminoglycans (GAGs) and COMP. This is in contrast to the central discolored regions seen as an occasional coincidental finding at postmortem examination, which contain higher hydration and increased GAG levels more consistent of reparative (scar) tissue formation.[27]

In contrast, exercise in immature horses appears to stimulate tendon formation and adaptation, and experimental long-term exercise studies have failed to induce any degenerative change.[39]

From the results of these studies, it is hypothesized that the midmetacarpal region of the equine SDFT can adapt to exercise during skeletal development, but it has little or no ability to do so after skeletal maturity[23,33] when the synergistic effect of aging and exercise causes an inevitable accumulation of microdamage (degeneration). In support of this hypothesis, analysis of matrix gene expression in bovine digital flexor tendons has shown prominent matrix gene expression at birth and during growth, but a dramatic reduction in the tensile (but not compressed) regions after skeletal maturity.[40]

Mechanisms for Tendon Degeneration

Cumulative tendon microdamage arises from the induction of tendon matrix damage and the failure of the resident cell population to repair this damage. The mechanism for the failure of tenocytes to repair or adapt to exercise-induced microdamage in the adult is unclear, but it possibly involves a combination of factors including reduced cell numbers and gap junctions that enable a coordinated anabolic response,[41] an absence of appropriate growth factor stimulus, and a degree of cellular senescence. Investigations into the synthetic response by equine tenocytes to mechanical load (biaxial stretch) and growth factors (e.g., TGF-β) *in vitro* have demonstrated that tenocytes recovered from aged flexor tendons do demonstrate a significantly reduced response.[42] Interestingly, this age effect is not apparent in tenocytes recovered from digital extensor tendons, which appear to continue to remodel and adapt during maturity.

The mechanism of degeneration of the tendon is currently unknown, but there are several possibilities. The close association between age and exercise suggests that the number of loading cycles is important, and it is logical to presume that the highest loading rates are likely to be the most damaging, so the amount of time spent at the fastest gaits (canter and gallop) is likely to contribute the most to degeneration. The actual mechanisms can be either physical or metabolic. The physical energy imparted to the tendon under weight-bearing load can directly damage the matrix by disrupting fibers, cross-links, or actual matrix proteins.[43,44] An indirect physical effect of weight bearing occurs via the energy lost through hysteresis.[45] The previously described increase in temperature (see earlier, "Functional Characteristics of Tendons and Ligaments") within the core of the SDFT up to 44° C during galloping could be damaging to matrix proteins or cell metabolism (Figure 83-9). Cyclical loading of tendon explants *in vitro* induces the upregulation of both synthesis and activity of proteolytic enzymes, potentially inducing a reduction in strength.[46] Furthermore, cleaved matrix proteins, generated either from direct physical forces or from enzymatic cleavage, also can provoke further matrix degradation, giving rise to an exercise-induced cycle of matrix degeneration.[47] Further work is necessary to elucidate the mechanisms of soft tissue degeneration so that preventive strategies can be developed.

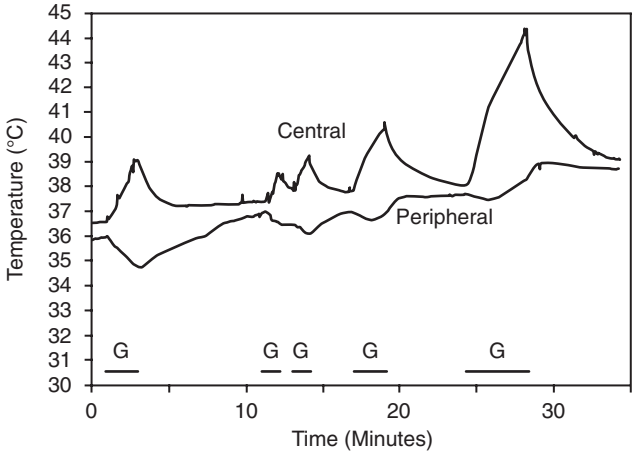

Figure 83-9. The hyperthermia theory of tendinopathy. When the horse is galloped *(G),* the temperature in the superficial digital flexor tendon rises as high as 44° C, which could damage the tendon matrix and be responsible for tendon degeneration. (Courtesy Dr. Alan Wilson.)

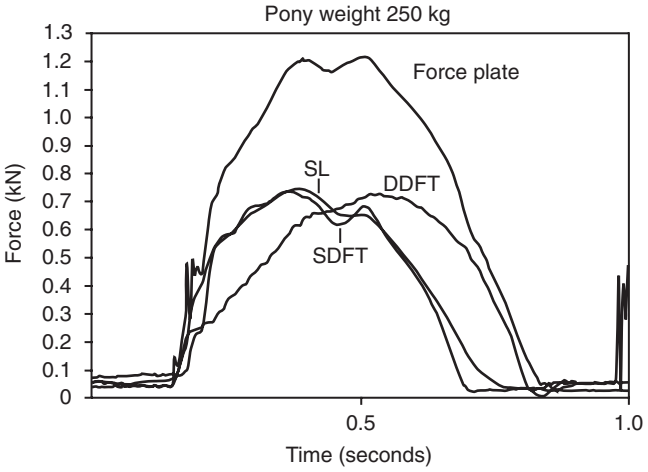

Figure 83-10. The force of individual tendons and ligaments of the equine distal limb during stance. Note the rapid rise in loading in the superficial digital flexor tendon (SDFT) and suspensory ligament (SL) (including what is thought to be damaging high-frequency "transients" at foot placement), whereas loading rises more slowly in the deep digital flexor tendon (DDFT). This may help explain why injuries are much more common in the SDFT and SL than in the DDFT. (Courtesy Dr. Alan Wilson.)

Mechanisms of Clinical Injury

STRAIN-INDUCED INJURY

Degeneration is usually the first phase of a tendinopathy. Any change in the structural properties of the tendon does not have to be large, because the tendon is already operating close to its tolerance limit. Clinical injury occurs when the highest stresses encountered by the tendon overwhelm its structural integrity, resulting in irreversible damage. At heel strike, the loads rise most quickly in the soft tissue structures that support primarily the metacarpophalangeal joint: the SDFT and the suspensory ligament (SL)[8] (Figure 83-10). The load in the DDFT rises later during stance, which may help to explain why the SDFT and the SL are the most prone to injury.

Once the peak load on the tendon overcomes its structural strength, there is physical disruption to the tendon matrix. The clinical injury varies in degree from fibrillar slippage, with

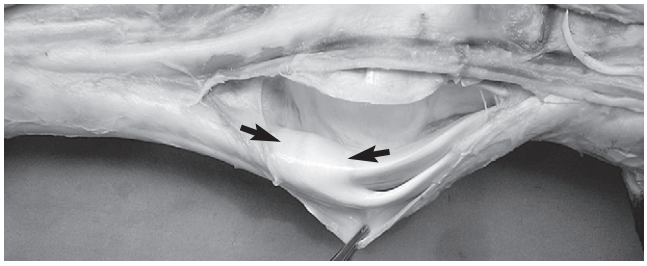

Figure 83-11. The manica flexoria *(between arrows).*

breakage of cross-linking elements, to fibrillar rupture and, in some cases, complete separation of tendon tissue. Once this occurs, the damage induces a repair process similar to that found in other soft tissues, such as skin, characterized by inflammation followed by fibroplasia (scar tissue formation).

This process allows the incorporation of risk factors that have been identified for inducing tendinopathy. These risk factors act to increase the peak loads on the SDFT, thereby increasing the risk of structural disruption. One of the most important factors is the speed of the horse. The faster the horse is going, the greater the risk of developing tendinopathy. Thus a hard track surface is associated with tendinopathy, because it increases the speed of the horse and also increases the peak impact loading.[31] Slower surfaces (including soft track surfaces) tend to be protective. Other factors, such as the weight the horse is carrying, fatigue, and the shoeing, can all influence peak tendon loads in this way. Heel elevation has been shown to decrease the load on the DDFT and cause a variable increase in the degree of extension in the MCP joint,[48,49] with toe elevation having the opposite effect. It may be thought, therefore, that low heels would be protective of tendinopathy. However, epidemiologic studies have shown an association between low heel and long toe conformation and musculoskeletal injury, of which the most common were tendon and ligament injuries.[50]

TENDON TEARS

The mechanism for generating tendon or ligament tears within synovial cavities is much less clear. The most common site is the DDFT (mainly in the forelimb) and the manica flexoria of the SDFT (predominantly in the hindlimb) in the proximal digital flexor tendon sheath (Figure 83-11). For the former, higher pressures within the compressed portion of the DDFT as it passes over the palmar aspect of the MCP joint may cause "bursting" of the lateral (or, less commonly, of the medial) borders of the tendon. Manica tears may develop when the structure gets caught as it passes into the fetlock canal. Because many of these tears have been seen after a period of tenosynovitis, they may be exacerbated by synovial hypertrophy or plantar/palmar annular ligament constriction within the sheath (making this passage even more restricted) or, less commonly, by the presence of adhesions.

PERCUTANEOUS TENDON INJURY

Percutaneous tendon injuries arise most frequently from trauma to the distal limb because of the minimal soft tissue cover. These injuries are usually associated with considerable contusion. Traumatic injuries involving overreaching, wires, jumping injuries, and kicking injuries are the most frequent causes. Tendon laceration usually requires the tendon to be taut, so tendons are most frequently partially or fully severed when under weight-bearing load. The exception to this is the lacerations caused when limbs are caught (e.g., in wire), and repeated attempts to free the limb can saw through any associated tendon or ligament.

The most serious consequences follow trauma to the palmar aspect of the metacarpus or pastern. Up to 50% of the tendon can be lacerated and still provide full tendon function at the walk. In contrast, extensor tendon lacerations on the dorsal aspect of the distal limb, although common (especially in hindlimbs caught in wire), rarely have long-term functional consequences.

Pathophysiology of Tendon Repair

All tendon injuries result in disruption of the tendon matrix, with initial intratendinous hemorrhage, usually followed rapidly by a pronounced inflammatory reaction (Figure 83-12). This inflammatory reaction results in an increase in blood flow; the development of edema; the infiltration of neutrophils, macrophages, and monocytes; and the release of proteolytic enzymes. Although this is the earliest stage of repair, designed to remove damaged tendon tissue, the response is thought to be excessive, further damaging the tendon. The inflammatory phase is usually short, and it overlaps the reparative phase, which begins within a few days and lasts several months. This phase is associated with a pronounced angiogenic response and fibroblastic cellular infiltration (extrinsic repair). It is believed that intrinsic repair mechanisms (i.e., from the tendon itself) are limited. The result is the synthesis of scar tissue with a composition that is different from that of tendon, with a higher ratio of collagen III to collagen I (about 50% compared with 10% for normal tendon), a higher hydration, and higher levels of GAGs[27] (see Figure 83-12).

The reparative phase of tendon healing merges with the remodeling phase, during which there is a gradual but incomplete transformation to a higher proportion of type I collagen as the scar tissue matures (see Figure 83-12).[51] The new collagen fibrils become thicker and cross-links increase in number and mature. Although mature scar tissue tends to be less stiff than tendon, because large amounts are formed, the scarred tendon is often stiffer than the original. As a result, the healed tendon becomes strong but it is functionally inferior to normal tendon, which predisposes it to reinjury, often at sites adjacent to the original injury, and potentially reduces performance.

This process is less efficient when injuries occur to an intrathecal tendon, which is thought to be the consequence of the absence of a paratenon and reduced extrinsic repair. In addition, any surface defect within a tendon sheath will be bathed in synovial fluid, which will slow or even halt repair.

DIAGNOSIS

Clinical Evaluation

Diagnosis of strain-induced tendon injury is usually based on history (frequently a preceding period of exercise) and the development of the signs of inflammation (pain, heat, swelling, and lameness) over the affected structure. Lameness, which is often severe in the early stages, is not always evident when a patient is presented to a clinician, and it tends to be related to the degree of inflammation rather than to the degree of damage.

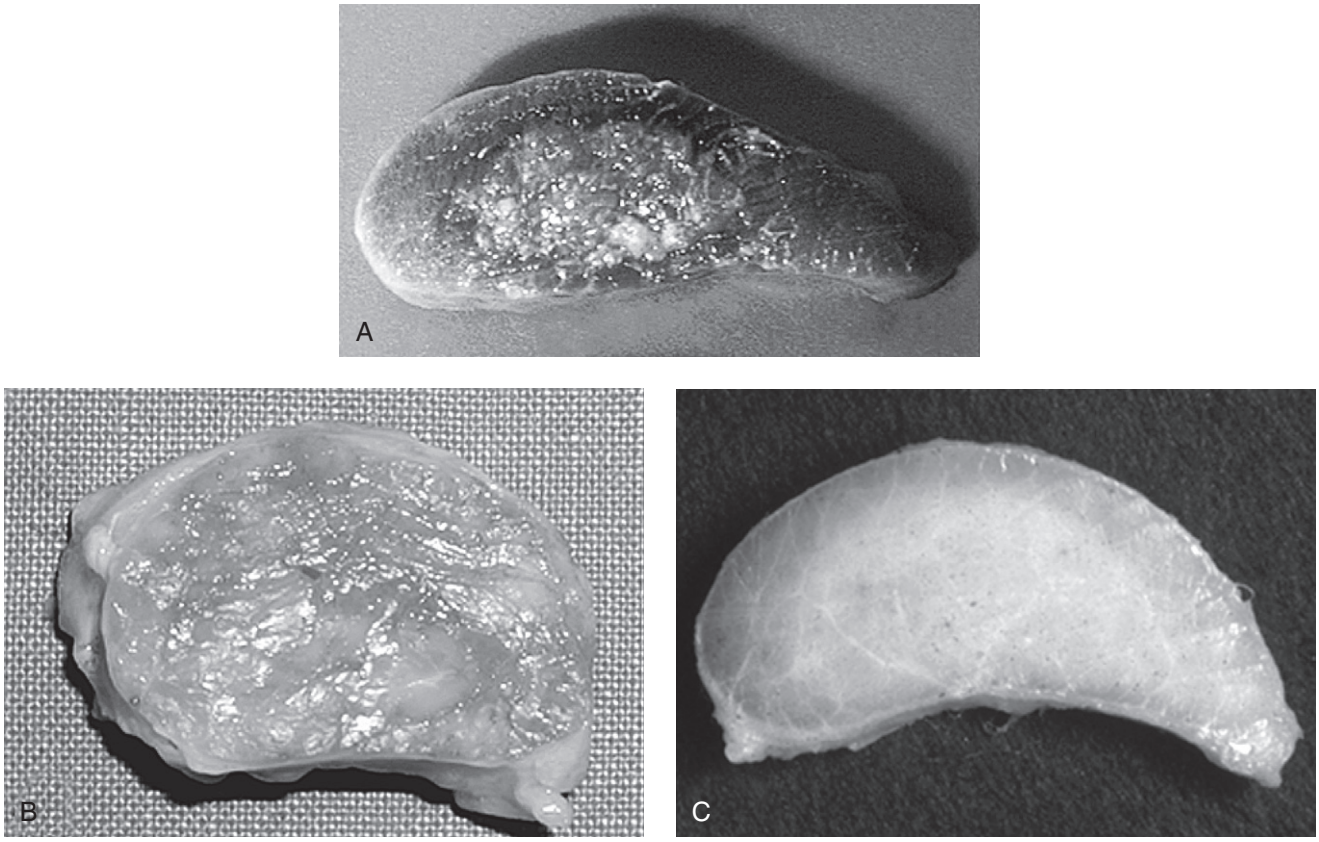

Figure 83-12. The different stages of superficial digital flexor tendinopathy. **A,** Acute. **B,** Subacute. **C,** Chronic. Note the replacement of tendon tissue with white fibrous tissue in **C.**

Similarly, once the inflammatory phase has passed (within 1 or 2 weeks), lameness usually resolves rapidly.

The posture and function of the limb may be altered depending on the structure damaged and on the severity of the injury. In the case of severe superficial digital flexor tendinopathy, resting MCP joint angle may be normal because of the action of the other supporters of this joint (SL and DDFT). However, when loading on the limb increases (e.g., when the contralateral limb is raised off the ground), the affected limb shows greater than normal overextension of the MCP joint (Figure 83-13). Severe damage to the SL has a greater effect on MCP joint extension. Complete disruption of the peroneus tertius produces a characteristic mechanical lameness because of disruption of the reciprocal apparatus. This allows tarsal extension when the stifle is flexed. Similarly, complete rupture of the extensor carpi radialis tendon results in hyperflexion of the carpus during limb protraction.

For subtle cases, careful palpation of the tendons and ligaments in the limb should be made both when the limb is bearing weight and when it is raised. Careful attention should be given to pain response, subtle enlargement, and consistency of the structure (soft after recent injury; firm after healing). The horse must be relaxed so that muscle activity does not tense the tendons and make them appear artificially firm. Careful visual assessment of bowing of the palmar contour of the metacarpal region can help to identify subtle superficial digital flexor tendinopathy. This assessment also should include the contralateral limb, because many strain-induced injuries are bilateral but with one limb more severely affected than the other.

Percutaneous tendon injuries are usually associated with moderate to severe lameness and may or may not have a concurrent wound. If a wound is present, it should be initially cleaned and explored digitally with sterile gloves to ascertain which structures are damaged. Small wounds may hinder full evaluation, because the site of the tendon laceration, sustained under full weight-bearing load, is unlikely to be visible in the wound when the horse is severely lame. In such cases, concurrent ultrasonographic examination is very useful. Penetration injuries or partial severance of a tendon does not alter the function of the tendon, so, other than lameness, there are few alterations of limb conformation. Complete transection, however, is associated with significant alterations in limb conformation under loading (see later). If the laceration is complete, the proximal part of a lacerated tendon often recoils as a result of the action of the associated muscle. It is necessary to assess whether any synovial structures have been penetrated, because this is a common complication of trauma to the distal limbs.

Ultrasonography

Clinical examination may not detect the most subtle injuries of strain-induced tendinopathies, and it provides a poor objective assessment of severity. Because prognosis is most dependent on the severity of the initial injury,[52] it is prudent to evaluate the damaged tendon ultrasonographically. This can be performed any time after the injury, but the optimal baseline scan for severity is best performed approximately 1 week after the onset of

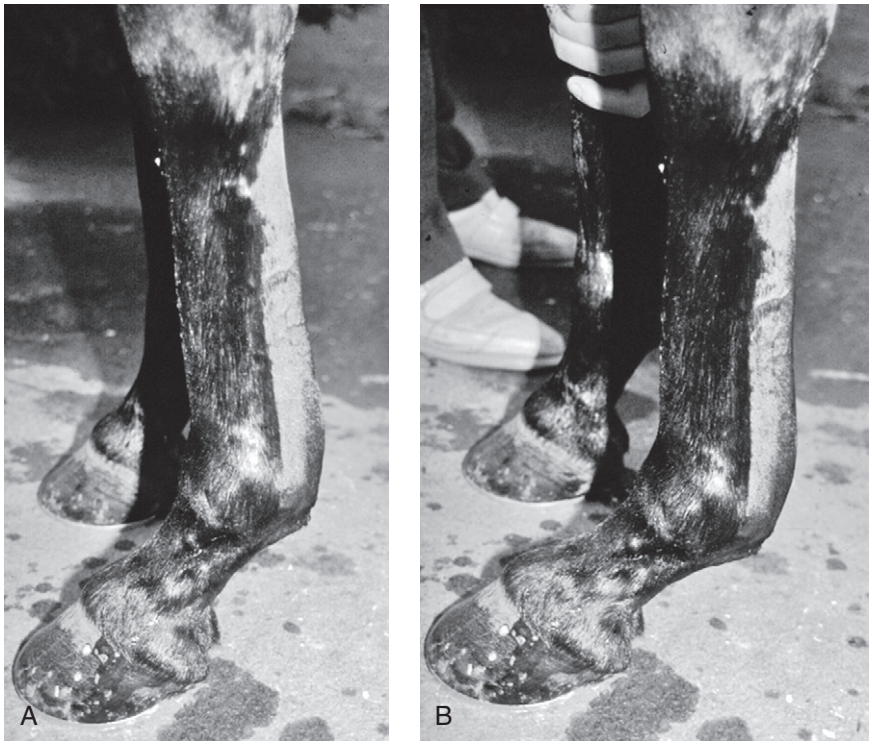

Figure 83-13. Demonstration of significant damage to the superficial digital flexor tendon (and/or suspensory ligament). **A,** Weight is evenly distributed over both limbs. **B,** Increasing the load on the affected limb by lifting the contralateral limb causes an abnormal increase in metacarpophalangeal joint extension.

the injury, because many lesions expand during the initial few days. Modern ultrasound machines with a 7.5- to 14-MHz linear transducer produce excellent-quality images of the flexor tendons and SL. Although the metacarpal region can be evaluated ultrasonographically without clipping the hair, it is best to prepare the limb by clipping and washing (with a surgical scrub and alcohol) to obtain the best-quality images. The horse should stand square, and both transverse and longitudinal images should be obtained methodically throughout the length of the region containing the injured tendon. For the metacarpal region, the area is divided into seven zones, each with characteristic anatomy. Alternatively, the distance between the transducer and the accessory carpal bone can be recorded. The palmar soft tissue structures of the metacarpus can be evaluated satisfactorily from the palmar aspect of the limb, except for the SL branches, which should be evaluated from the medial and lateral aspects of the limb, respectively. Both limbs should *always* be examined, because approximately one third of cases of strain-induced tendon injury have bilateral components but with one limb more severely affected.[53] Detailed information on ultrasonography of tendons and ligaments can be found in Chapter 68.

Acute tendon pathology is manifest ultrasonographically by enlargement, hypoechogenicity (focal or generalized), reduced striated pattern in the longitudinal images, and changes in shape, margin, or position (Figure 83-14). Chronic tendinopathy is associated with variable enlargement and echogenicity (often heterogeneous), and an irregular striated pattern indicates fibrosis (see Figure 83-14).

Ultrasonography is particularly useful for the assessment of tendon lacerations, which are associated with small wounds that limit digital exploration. However, ultrasonographic assessment may be impeded because of air artifacts and skin contusions.

Molecular Markers

Molecular markers have the potential to contribute useful parameters to the evaluation of equine tendon disease. They may be used to provide a diagnosis when other tests are negative (rarely needed for first-time injuries of the SDFT, but they may be useful for detecting reinjury). Additionally, molecular markers offer the following benefits:

- They may be useful for screening as part of a preventive strategy; early detection may enable introduction of measures (e.g., altered training) to minimize the risk of a career-ending injury.
- They may allow a more objective assessment of the severity of the pathology and therefore help to determine prognosis.
- They may provide information about the stage of the disease process, optimizing rehabilitation protocols.
- They may assist in the choice of treatment and provide an objective assessment of its efficacy.

To date, the diagnosis and assessment of stage, severity, and prognosis of equine tendinopathy have been correlated to semiquantitative ultrasonographic changes,[52] but this procedure is time consuming and has a relatively low sensitivity. In contrast, molecular markers have the potential to reflect specific processes, anabolic or catabolic, within the tissue of interest. Initial investigations assessed markers of both collagen I synthesis (carboxy-terminal propeptide of type I collagen [PICP]) and degradation (cross-linked carboxy-terminal telopeptide of type I collagen [ICTP]) after tendon injury.[54] Tendinopathy resulted in a significant rise in PICP concentrations, whereas ICTP concentrations were not different from those in the control group. These results indicated that serum concentrations of PICP are able to reflect changes in type I collagen formation in healing

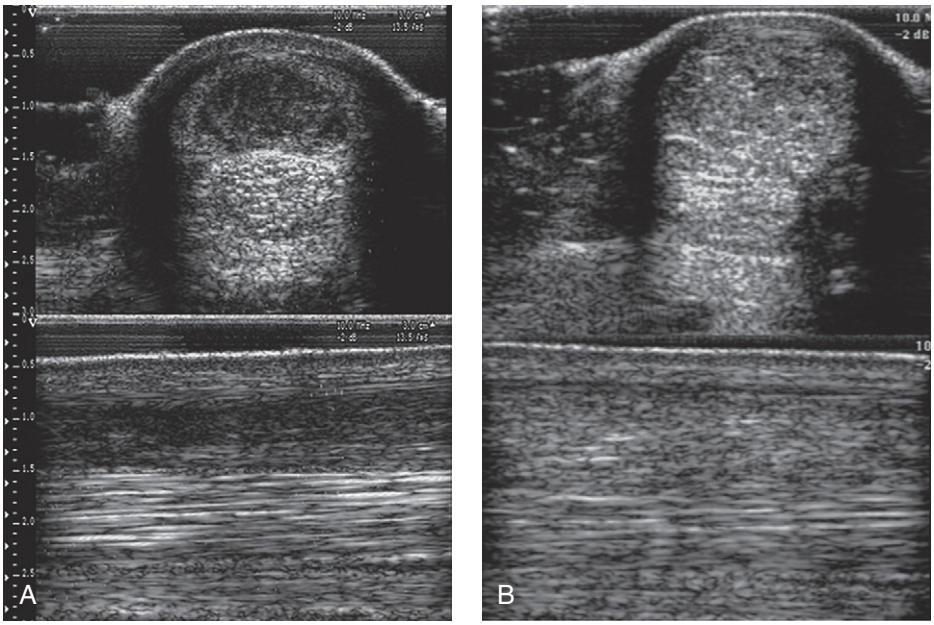

Figure 83-14. Ultrasonographic appearance of superficial digital flexor tendinopathy. **A,** Acute injury is manifested by enlargement, a reduction in echogenicity (especially centrally), and a disrupted striated pattern on longitudinal views. **B,** Chronic injury. The tendon is variably echogenic (usually heterogeneous), is enlarged, and has a poorer striated pattern than normal tendon, which is thought to represent the quality of the healed tendon.

connective tissue in one area of the body. The results also suggest that PICP is not an entirely bone-specific marker, if repair in tissues other than bone can contribute to concentrations in serum.

Another candidate, COMP, which as mentioned earlier is abundant in young tendon, also has been assessed.[55] Synovial fluid levels were more than tenfold higher than serum levels and have been shown to be significantly elevated in the synovial fluid from the digital flexor tendon sheath in cases of intrathecal tendon and ligament tears.[56] In older horses, increased levels of COMP within the DFTS synovial fluid appeared to be strongly suggestive of the presence of a tear even when there is no evidence of this ultrasonographically. In contrast, serum COMP levels were not significantly elevated with tendon disease, which is related to high natural levels of COMP in blood. Longitudinal studies with this marker have shown variable results, but rather than reflecting specific tendon pathology, serum COMP levels might be more useful for detecting joint disease or for determining whether training levels are too excessive and are damaging multiple soft tissues.

Clinical Characteristics of Specific Strain-Induced Injuries

For the SDFT, the clinical injury may be focal or generalized, one of the more common manifestations being a central region of injury (the so-called core lesion seen ultrasonographically). Usually, the most severe level is just below the midmetacarpal region, but it can extend throughout most of the length of the metacarpal extrasynovial tendon and give rise to swelling on the palmar aspect of the metacarpal region (the "bowed" limb). Regions of the SDFT enclosed within a tendon sheath (carpal sheath proximally, digital sheath distally) are much less often affected, although this can be more common when the metacarpal region was previously injured.

Desmopathy of the accessory ligament of the DDFT may occur as an isolated injury or in conjunction with superficial digital flexor tendinopathy. Although ponies rarely suffer strain-induced tendinopathies, they do have a relatively high incidence of desmopathy of the accessory ligament of the DDFT. Lameness is often mild, or even absent, in this injury. Swelling is restricted to the proximal half of the palmar metacarpal region, immediately dorsal to the SDFT and surrounding the DDFT.

The SL can fail at any site along its length. Injury to its proximal region at its origin at the palmar/plantar aspect of the third metacarpal/metatarsal bone (MCIII/MTIII) is often restricted to this area, whereas injuries to the branches usually occur separate from proximal suspensory desmopathy and may extend into the body of the ligament. Some branch injuries may also "tear" into the MCP/MTP joint and result in persistent joint effusion and lameness (Figure 83-15). The synovial fluid environment dramatically slows down healing processes.

In contrast to the SDFT and the SL, the DDFT is most frequently injured within the digital flexor tendon sheath (DFTS) or distal sesamoidean bursa. MRI has facilitated diagnosis of DDFT lesions within the foot.[57] Two manifestations within the DFTS are seen clinically. The first arises within the substance of the tendon (although it may extend to the borders of the tendon) and is similar to other clinical manifestations of tendinopathy.[58] The second arises at the medial or lateral borders of the tendon, usually in the region of the MCP joint.[59] Both lesions are invariably associated with a distended digital sheath, but the latter can be difficult to identify ultrasonographically if the tear does not extend into the body of the tendon (Figure 83-16). In a study of 264 horses with lameness localized to the foot, 82.6% had DDFT lesions identified on MRI examination.[60] These lesions within the DDFT were most commonly located at the level of the collateral sesamoidean ligament and the distal sesamoid bone.

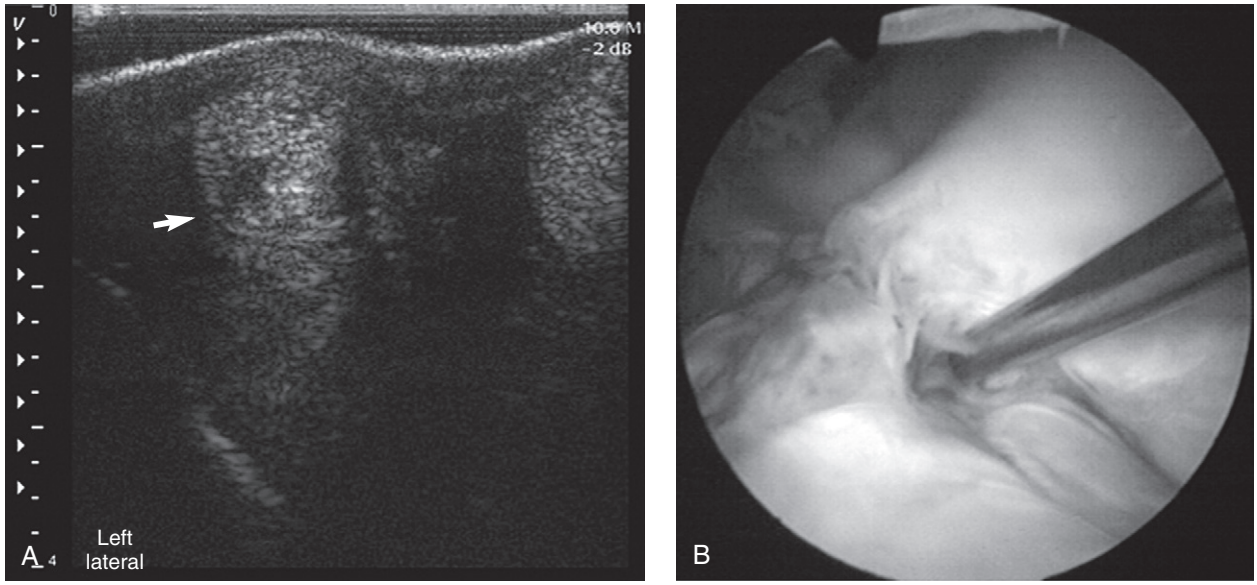

Figure 83-15. A, A hypoechoic defect *(arrow)* in the lateral suspensory ligament branch, which communicates with the metacarpophalangeal joint, causing joint distention, persistent lameness, and slow healing. **B,** This communication was confirmed by arthroscopy.

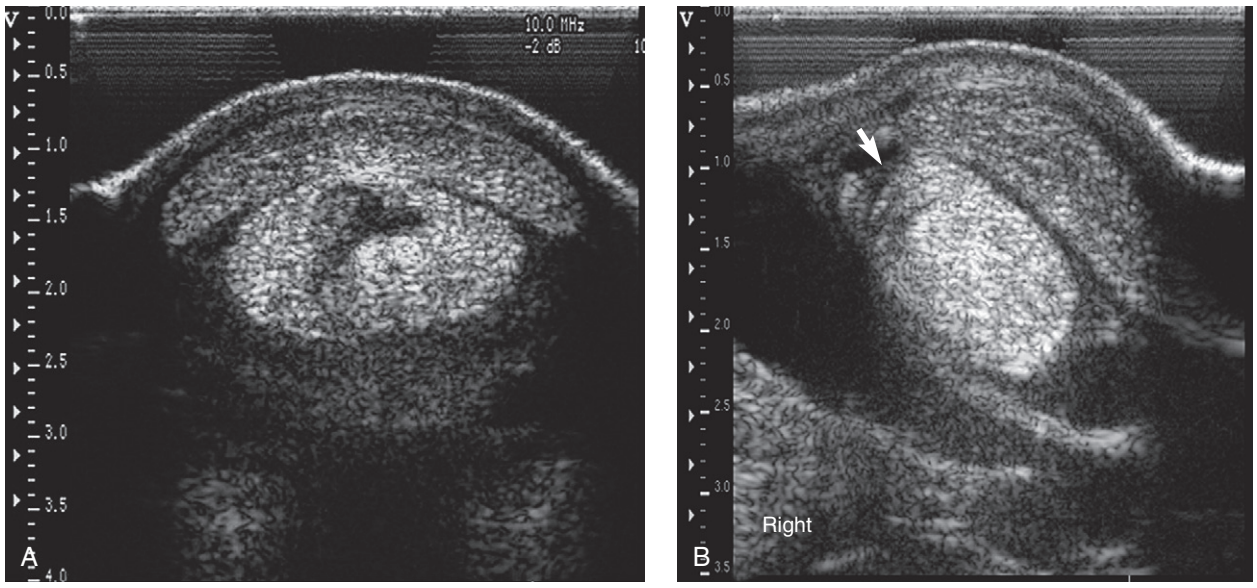

Figure 83-16. Deep digital flexor tendon injury. **A,** Mid-substance strain injury, **B,** Surface tear *(arrow).* The surface tear can be easily missed using ultrasonography and requires tenoscopy for confirmation and débridement.

Other tendons can suffer from strain-induced injury, although much less commonly than that affecting the palmar soft tissue structures of the metacarpal region. Ligament injuries tend to occur when the joint they span is loaded inappropriately, resulting in a degree of subluxation or even complete luxation. Collateral ligament injuries are most commonly diagnosed in the distal interphalangeal, MCP/MTP, and tarsocrural joints. The consequences of these injuries vary from an isolated desmopathy to more severe injury resulting in significant instability of the associated joint. Alternatively, because of their subsynovial location, there can be partial ligament rupture into the adjacent synovial cavity. Both of these consequences may result in secondary osteoarthritis.

Clinical Characteristics of Specific Tendon Lacerations

Complete transection of specific palmar metacarpal tendons and ligaments produces characteristic alterations in limb conformation, which may indicate the tendon or ligament that has been lacerated (Figure 83-17). With SDFT laceration, the MCP/MTP joint is hyperextended (dropped) relative to the normal limb when the limb is loaded, but in the standing horse no difference compared to the joint in the contralateral limb can be detected. If the SDFT and DDFT are lacerated concurrently, in addition to the signs described for SDFT laceration, the toe is raised from the ground. If the SL is completely transected in

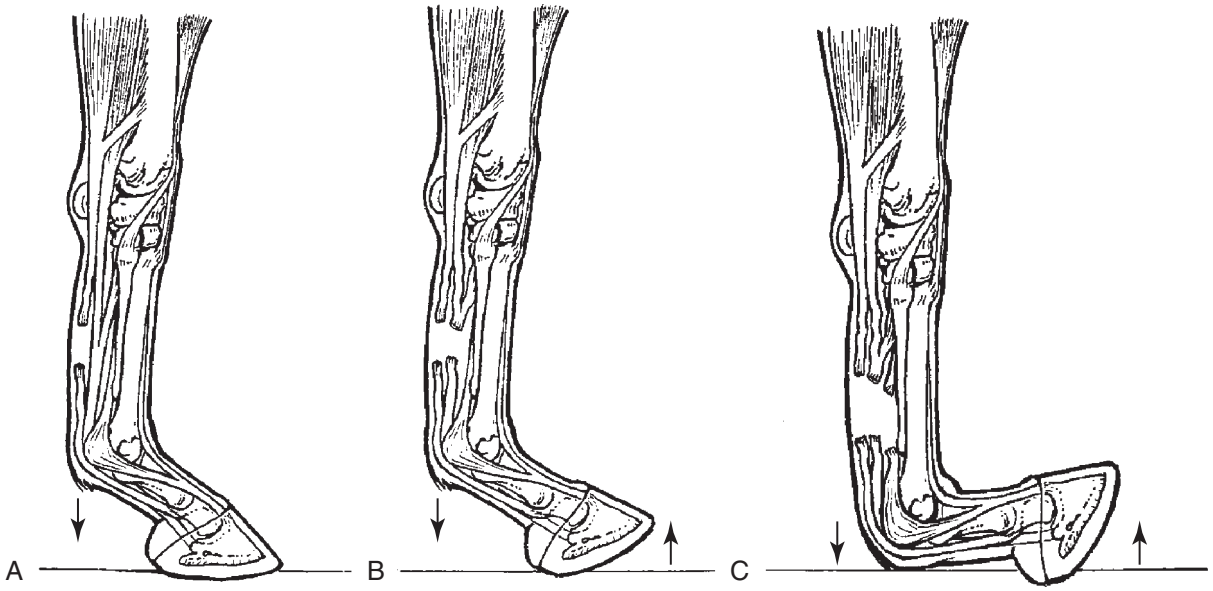

Figure 83-17. A, There is a degree of hyperextension of the metacarpophalangeal joint when the superficial digital flexor tendon has been transected proximal to the joint. **B,** Relative hyperextension of the joint and elevation of the toe off the ground when both the superficial and the deep digital flexor tendons are transected. **C,** Complete loss of metacarpophalangeal joint support when both digital flexor tendons and the suspensory ligament have been disrupted.

addition to the SDFT and the DDFT, there is complete loss of support of the MCP/MTP joint, which is hyperextended to the extent that the MCP/MTP joint contacts the ground during the stance phase. Complete loss of MTP/MCP joint support often causes the horse to be severely distressed.

Extensor tendon lacerations frequently involve the metacarpal and metatarsal regions, although lacerations proximal to the dorsal aspect of the carpus involving the extensor carpi radialis or common digital extensor tendons are not uncommon. Lacerations proximal to the tarsus may involve the cranialis tibialis tendon, the long digital extensor tendon, or the peroneus tertius tendon. Lacerations of the extensor tendons generally do not result in significant gait abnormalities or lameness, although they may cause the horse to stumble onto the dorsal aspect of the MTP/MCP joint region at a walk and to be unable to protract the limb completely.

TREATMENT OF TENDON AND LIGAMENT INJURIES

Strain-Induced Tendinitis (Tendinopathy)

In 1964, Asheim described the common treatment of tendon and ligament injuries as "phlebotomy, local cooling, plaster bandaging and rest."[61] Over the last four decades, many treatments have been proposed for the management of tendon injury, although few, if any, have convincing supporting evidence of efficacy, and some can even be considered deleterious or detrimental to healing. However, the basic principles described by Asheim of cooling, support, and rest remain integral parts of the management protocol. Acute tendon and ligament injuries are medical emergencies requiring prompt and appropriate treatment to reduce inflammation rapidly, because persistent inflammation may be responsible for further damage to the tendon.

Nonsurgical Therapies

PHYSICAL THERAPIES

Cold therapy

In the acute inflammatory phase of tendon injury, cold therapy is an important aspect of treatment as it is both anti-inflammatory and analgesic, largely through its ability to increase vasoconstriction, decrease enzymatic activity, reduce the formation of inflammatory mediators, and slow down nerve conduction.[62] The optimal frequency and duration of cold treatment for musculoskeletal disorders in horses has not been determined. The authors recommend 20 minutes of cold hosing several times daily during the acute phase of tendon and ligament injuries.

Research data have indicated that cold hydrotherapy is superior to the use of ice packs because of the increased contact and evaporation.[63] It is also less likely to cause adverse effects such as superficial tissue damage and cold-induced nerve palsy. Prolonged exposure to cold temperatures can cause a reflex vasodilatation, which can accentuate tissue swelling and edema. For this reason, it is recommended not to apply cold therapy for longer than 30 minutes. One highly effective way of providing cold hydrotherapy is the use of equine spas, which are currently gaining popularity. They provide both cold and compression using hypertonic saline at 5° to 9° C.[64] In addition to equine spas, underwater treadmills, used as part of controlled exercise programs in tendon and ligament injury rehabilitation, allow conditioning of the musculoskeletal system with minimal concussive forces.

Compression and coaptation

In the acute phase of injury, pressure applied to the affected limb reduces inflammation and edema by increasing interstitial hydrostatic pressure. A modified Robert Jones bandage is suitable in most cases of acute tendon and ligament injury.

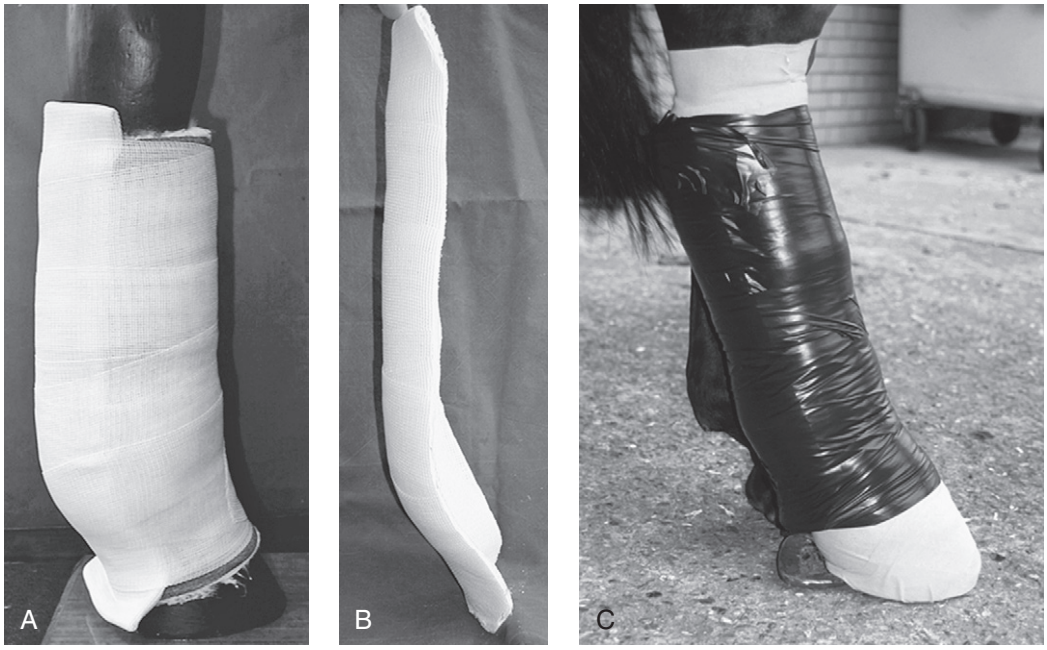

Figure 83-18. The use of a palmar splint to protect overloading of the superficial digital flexor tendon or suspensory ligament. **A,** The splint is fashioned out of casting tape on the back of a bandaged limb with normal conformation (either the contralateral limb or a "dummy," as shown here). **B,** It is then cut from the limb, the edges are smoothed off, and it is taped to the palmar aspect of the bandaged affected limb. **C,** This limb has also been fitted with a heel extension shoe to protect the deep digital flexor tendon, because this was damaged concurrently.

With severe injuries when there is hyperextension of the MCP joint, a palmar/plantar splint or cast may be applied to the bandaged limb to support the MCP joint (Figure 83-18). A palmar splint can be fashioned from two rolls of 7.5-cm (5-inch) casting tape. One roll is layered on top of itself over a length that matches the length of the limb from the carpus to the bulbs of the foot. This is placed on the palmar aspect of a 2.5-cm-thick support bandage on the contralateral limb. The contralateral limb can be used as a template for making the splint, as it has the optimal degree of extension of the MCP joint. A second roll of cast tape is wrapped around the palmar splint and bandage and left to set. The palmar splint is then cut away from the bandage using a cast saw and applied to the palmar aspect of the bandaged affected limb. A similar effect can be achieved more easily with a specially designed support boot.[65] A full distal limb cast may be used as an alternative in the most severe cases, such as suspensory ligament rupture, when all MCP joint support has been lost.

External coaptation is important in cases of joint instability resulting from collateral ligament injury. In cases where collateral ligament desmopathy of the distal interphalangeal joint that has not significantly destabilized the joint, corrective farriery has been advocated to reduce strain on the injured ligament. Fitting a shoe with increased width on the side of the affected ligament has been proposed to reduce strain on the damaged ligament by impeding downward vertical movement of the wider side of the shoe when the horse is exercised on a soft surface.

Controlled exercise

Controlled exercise is an intrinsic part of the rehabilitation of tendon and ligament injuries, helping to resolve residual inflammation, maintain gliding function, and promote optimal collagen remodeling.[66] Most SDFT injuries require at least 8 to 9 months of rehabilitation before resumption of full athletic function, although some require up to 18 months. It is rarely necessary to prolong rehabilitation longer than this, because healing appears to be complete by this stage.

A suitable exercise rehabilitation program should be created on the basis of the severity of the ultrasonographic appearance of the lesion. The aim of the program is to provide a controlled and ascending exercise regimen that optimizes scar tissue function without causing further injury. Because variability between animals makes this difficult to predict, the program should be adapted on the basis of serial ultrasonographic monitoring and clinical signs such as lameness, heat, and swelling.

Ultrasonographic monitoring of tendon or ligament injuries should include measurements of tendon cross-sectional area (CSA). An increase in CSA of greater than 10% between examinations suggests a degree of reinjury, and in such cases the level of exercise should be reduced. Because there is a sudden increase in strain levels in the SDFT and SL with an upward transition in gait from walk to trot, trot to canter, and canter to gallop, ultrasonographic examinations before and after these transitions can help to determine if the injured tendon or ligament can withstand the increased strain levels.

The importance of a controlled exercise program was illustrated by Gillis, who demonstrated that of 28 Thoroughbred racehorses with SDFT tendinopathy placed in a controlled exercise program, 71% returned to racing, compared with only 25% of 8 horses managed with uncontrolled turnout.[66] An example of rehabilitation programs for racehorses and sports horses is shown in Table 83-1.

Extracorporeal shock wave therapy

Extracorporeal shock wave therapy (ECSWT) has been established for a number of years for the treatment of human orthopedic conditions, especially insertional desmopathies. In this

TABLE 83-1. Typical Exercise Program* after Tendon Injury

Exercise Level	Weeks	Duration and Nature of Exercise
0	0-2	Stall rest
1	3	10 minutes walking
1	4	15 minutes walking
1	5	20 minutes walking
1	6	25 minutes walking
1	7	30 minutes walking
1	8	35 minutes walking
1	9	40 minutes walking
1	10-12	45 minutes walking
WEEK 12: REPEAT ULTRASOUND EXAMINATION		
2	13-16	40 minutes walking and 5 minutes trotting daily
2	17-20	35 minutes walking and 10 minutes trotting daily
2	21-24	30 minutes walking and 15 minutes trotting daily
WEEK 24: REPEAT ULTRASOUND EXAMINATION		
2	25-28	25 minutes walking and 20 minutes trotting daily
2	29-32	20 minutes walking and 25 minutes trotting daily
WEEK 32: REPEAT ULTRASOUND EXAMINATION		
3	33-40	45 minutes exercise daily with slow canter, gradually increasing in amount
3	41-48	45 minutes exercise daily with fast work three times a week
WEEK 48: REPEAT ULTRASOUND EXAMINATION		
4	48+	Return to full competition/race training

*This program is shortened or lengthened depending on the severity of the lesion and the progress of the patient.

therapy, shock or pressure waves are transmitted into the tissues where the handpiece is applied. Both focused and nonfocused units have been used, although the data that compare these machines are too limited to indicate a significant difference. The mechanism of action of ECSWT on tissues is unclear, but it is most likely related to induction of analgesia through an effect on sensory nerves.[67] The most frequently reported use of ECSWT in horses has been for the treatment of proximal SL desmopathy, and a significant improvement in prognosis over conservative treatment for chronic hindlimb proximal SL desmopathy has been reported. Investigation into ECSWT as a treatment for chronic SL desmopathy resulted in 41% of hindlimb patients returning to full work within 6 months of diagnosis,[68] compared with the previously reported 13% for conservatively managed cases.[69] There is evidence that ECSWT can be deleterious to normal tendons and ligaments, resulting in matrix disorganization; however, it was acknowledged that this initial disorganization could be a stimulus for repair in chronically injured tissue.[70]

Therapeutic ultrasound, laser, and magnetic fields

The effect of ultrasound, laser, and magnetic fields on tissues is not completely understood. It is thought that the main effect of ultrasound is the conversion of sound energy into thermal energy. Although there is a paucity of scientifically convincing research into the effects of ultrasound for the treatment of equine musculoskeletal disorders, a study by Morcos and Aswad showed that in experimentally split equine tendons, therapeutic ultrasound resulted in increased vascularization and fibroblastic proliferation compared with controls.[71]

Low-level laser (light amplification by stimulated emission of radiation) therapy has been shown to stimulate cellular metabolism and to enhance fibroblast proliferation and collagen synthesis *in vitro*.[72] However, there are no clinical trials demonstrating a significant difference between laser-treated and control cases of tendinopathy or desmopathy.

Magnetic therapy has not been demonstrated in any clinical trials to enhance tendon or ligament healing, despite its widespread use by horse owners and anecdotal reports of its efficacy.

Counter-irritation

Counter-irritation, in the form of chemical or thermal cauterization, has long been used in equine practice for the treatment of tendon and ligament injuries. Topical iodine and mercury-based compounds have been used for chemical cauterization or "blistering" of tendon injuries. Thermal cauterization (or "firing") is performed under general anesthesia or standing sedation with local analgesia, using heated bars or pins, which are applied to the skin over the injured tendon or ligament. In some cases, the tendon is penetrated with the heated pins. Studies have shown that there is no histologic difference between the collagen arrangement in the scar in cases of tendinopathy treated with firing and that of the controls.[73] It has been postulated that any benefits from firing result from the enforced rest, the local release of inflammatory cytokines, or a protective "bandage" of fibrous tissue or skin that supports the tendon. However, it has been demonstrated that firing causes thinner and weaker skin in the region that was cauterized.[73] The limited controlled studies that have been performed on firing have concluded that it is not an effective treatment for tendon and ligament injuries.

PHARMACOLOGIC MANAGEMENT

Systemic medication

Both systemic corticosteroids and nonsteroidal anti-inflammatory drugs can be considered for the management of tendon or ligament inflammation during the acute stages. Phenylbutazone is commonly used at a dose of 2.2 mg/kg twice daily, but the clinical effects of this drug appear to be more analgesic than anti-inflammatory.[74] Systemic corticosteroids can be administered within the first 24 to 48 hours after injury, but they should be avoided after this time because they also inhibit fibroplasia and hence repair of the tendon. The most commonly used systemic steroid is dexamethasone at 0.1 mg/kg as a single dose. The induction of laminitis with systemic steroids represents a small, but nevertheless real, risk. Topical or intravenous dimethyl sulfoxide (DMSO) may reduce the inflammation, but a study has shown that 40% to 90% topical medical grade DMSO may weaken normal tendon tissue.[75]

Intralesional medication

Various intralesional treatments have been advocated for the management of tendinopathy. These treatments include polysulfated glycosaminoglycans (PSGAGs), hyaluronan (HA), and beta-aminopropionitrile fumarate, with recent interest being focused on novel growth factor and cell-based therapies. Intralesional tendon and ligament treatment can be performed under standing sedation and local analgesia or under general anesthesia. Weight-bearing potentially assists injection, as the tendons are then taut. Although the technique is frequently performed blindly, by injecting where the least resistance in the tendon is detected, accurate placement of the needle in the center of the lesion is best achieved using ultrasonographic guidance. The skin overlying the tendon or ligament to be injected should be clipped and aseptically prepared, and if ultrasonographic guidance is used, a sterile sleeve should be placed over the probe. A 2.5-cm, 22-gauge hypodermic needle can be used for most intratendinous treatments, but this depends on the viscosity of the agent. Intralesional treatment should not be administered until 3 days after the injury, because there is the potential to increase hemorrhage. The volume injected into the tendon or ligament depends on the extent of the lesion. Large volumes can be damaging to the healing tendon.[76]

PSGAGs have been shown to inhibit collagenases and metalloproteinases and to inhibit macrophage activation,[5] but they were shown to have no effect on fibroblasts.[77] Hence, this drug can be viewed as a soft tissue anti-inflammatory agent. PSGAGs have been widely used for the treatment of tendinopathy and desmopathy, both intralesionally and intramuscularly. In one study of 73 horses, treatment with PSGAG either intramuscularly or intralesionally resulted in 76% of horses returning to work versus 46% of the control animals, although these results were not statistically significant.[78] Another study demonstrated improved echogenicity of collagenase-induced superficial digital flexor tendinopathy treated with intralesional PSGAGs, with faster resolution of core lesions.[79] However, another study demonstrated no significant difference in reinjury rates between horses treated with PSGAGs and those treated with controlled exercise alone.[80]

HA consists of repeating units of D-glucuronic acid and repeating units of *N*-acetyl-D-glucosamine and is a component of the tendon matrix. HA has been administered peritendinously, intralesionally, intrathecally, and systemically to treat tendinopathy. A study showed no significant difference between the reinjury rates of horses with SDFT tendinopathy treated with intralesional HA and those treated conservatively.[80] In a study of collagenase-induced digital flexor tendinopathy, HA was found to minimize tendon enlargement compared with controls, although histopathologic examination of the tendons failed to demonstrate a significant difference in the degree of inflammation.[81] Peritendinous HA has been shown to have no effect on ultrasonographic or histologic appearance, biomechanical properties, or molecular composition of tendons in collagenase-induced tendinopathy compared with controls, although it did appear to reduce lameness.[82]

HA has been shown to decrease the extent of adhesions when administered intrathecally to treat collagenase-induced deep digital flexor tendinopathy within the digital sheath.[83] Horses treated with intrathecal HA showed decreased inflammatory cell infiltrate and less intratendinous hemorrhage than controls.[83]

Methylprednisolone injected into and around normal equine tendons has been shown to cause dystrophic tissue mineralization and tissue necrosis, most likely a consequence of the carrier.[84] Hence, the local use of this steroid should be avoided. Nondepot preparations may be used peritendinously with caution.

New advances: Tissue engineering approaches

The use of *growth factors* is a relatively recent approach. Insulin-like growth factor-1 (IGF-1) has been investigated to assess its effect on tendon healing both *in vitro* and in collagenase-induced models of tendinopathy.[85] IGF-1 stimulates extracellular tendon matrix synthesis, and it is also a potent mitogen.[85] In collagenase-induced models of tendinopathy, initial swelling was decreased after intralesional injections of IGF-1 compared with controls, although no differences were found at later time points and there was no difference between the quantities of type I and type III collagen synthesized.[85] There are no published long-term follow-up data regarding reinjury rates of tendinopathy treated with IGF-1.

Recombinant equine growth hormone administered intramuscularly has demonstrated a negative effect on some of the biomechanical properties (decreased yield point and ultimate tensile strength) of the SDFT during the early phases of healing in collagenase-induced tendinopathy, although elasticity was maintained.[86] However, these properties were assessed at 6 weeks after treatment, which may have been too soon to detect any beneficial effects.

TGF-β has been considered another appropriate growth factor treatment, but clinical experience has been limited. Horses treated with TGF-β_1 (a proscaring isoform of TGF-β) showed significant enlargement of the tendon and, although reinjury rates were similar to those in conservatively managed horses, these reinjuries were all on contralateral, untreated limbs.[87]

Platelet-rich plasma (PRP) is defined as plasma with at least twice the platelet concentration of normal plasma[88] and can be created using centrifugation or gravity filtration of autologous blood. PRP is a rich source of certain growth factors (in particular, platelet-derived growth factor [PDGF], TGF-β, and vascular endothelial growth factor [VEGF]), which can stimulate cell proliferation and matrix synthesis. In an experimental model, modulation of composition, organization, and biomechanical properties of PRP-treated tendon was demonstrated in a surgically induced model of tendinopathy.[89] These preparations have become popular for the intralesional treatment of mainly suspensory ligament injuries, but long-term clinical data on a sufficient number of cases have yet to be published, although reports on small case series suggest efficacy.[90]

ACell Vet is a novel intralesional treatment for tendinopathy using acellular tissue components derived from porcine urinary bladder submucosa. This preparation has been suggested to deliver appropriate growth factors to the injured tissue and to attract mesenchymal stem cells. There is only anecdotal evidence that this treatment improves tendon and ligament healing, and controlled clinical trials are required to further assess efficacy.

Treatment with intralesional *bone marrow* has been investigated for SL desmopathy.[91] The aim was to stimulate ligament regeneration rather than scar tissue formation by the delivery of stem cells to the damaged tendon or ligament. Bone marrow is a rich source of growth factors[92] but contains very few

mesenchymal stem cells (1 in 10^4 nucleated cells). Hence, this treatment more closely resembles a growth factor treatment.

The use of intralesional bone marrow as a source of *mesenchymal stem cells* (MSCs) led to the development of culturing MSCs from bone marrow before implantation. The use of autologous MSCs for intralesional treatment of SDFT and SL lesions offers the prospect of tissue regeneration rather than repair. MSCs have the potential to differentiate into tenocytes and to regenerate tendon matrix, thereby creating a repair that is functionally superior to fibrous scar tissue. Initial studies have included the isolation of MSCs from either bone marrow or fat, followed by either direct implantation or expansion *in vitro* before implantation.[93] A recent study evaluated the efficacy of cultured bone marrow–derived stem cells for the treatment of superficial digital flexor tendinopathy in racehorses in the United Kingdom. The reinjury rate was 26%[94] in 113 National Hunt racehorses with 3 years of follow-up after treatment, which was significantly better than data previously reported for a similar population of National Hunt horses treated conventionally and analyzed in the same way.[80,95]

Surgical Therapies

TENDON SPLITTING

Tendon splitting was first performed in the 1940s and the technique was published in 1967.[96] It was initially advocated as a treatment for chronic tendinopathy to improve blood flow to damaged tendon tissue that is relatively avascular. The technique fell out of favor when subsequent research demonstrated extensive granulation tissue formation, and increased trauma to the tendon tissue and persistent lameness after treatment.[97] Tendon splitting is therefore no longer recommended for the treatment of chronic tendinopathy, but it is now thought to be more relevant to the management of acute cases when there is an anechoic core lesion evident on ultrasonographic examination, indicating the presence of a seroma or hematoma. It has been hypothesized that the presence of a core lesion within a tendon produces a compartment syndrome, resulting in decreased perfusion and ischemia of the region.[98] The aim of tendon splitting in acute cases is therefore to decompress the core lesion by evacuating the serum or hemorrhage and to facilitate vascular ingrowth.[98] Removal of the fluid in the core lesion may also reduce proximodistal propagation of the lesion. In a collagenase-induced model of tendinopathy in six horses, tendon splitting using the knife technique resulted in a faster resolution of the core lesion, quicker revascularization of the lesion, and increased collagen deposition than in controls.[99]

Tendon splitting may be performed under standing sedation or under general anesthesia. It can be done blindly or using ultrasonographic guidance, which minimizes damage to normal tendon tissue by enabling the needle or knife to be inserted at a point where the core lesion is closest to the periphery of the tendon. A No. 11 scalpel blade or double-edged blade is inserted into the tendon and "fanned" proximad and distad (Figure 83-19). Alternatively, the procedure can be achieved with multiple insertions of a 23-gauge needle, possibly with less damage to the remaining, relatively intact tendon tissue. Needle splitting may also be combined with various intralesional treatments.

After tendon splitting has been performed, a modified Robert Jones bandage should be applied and the horse rested in a stall for 10 to 14 days, after which a controlled exercise program should be initiated.

DESMOTOMY OF THE ACCESSORY LIGAMENT OF THE SUPERFICIAL DIGITAL FLEXOR TENDON

Superior check ligament desmotomy was first described as a treatment for SDFT tendinopathy by Bramlage in 1986.[100] The aim of the surgery is to produce a functionally longer musculotendinous unit to reduce strain on the SDFT. However, it has been shown in equine cadaver models that desmotomy of the accessory ligament of the SDFT (DALSDFT) actually increases the strain on the SDFT during loading because of increased extension of the MCP joint.[101] The biomechanical alterations of

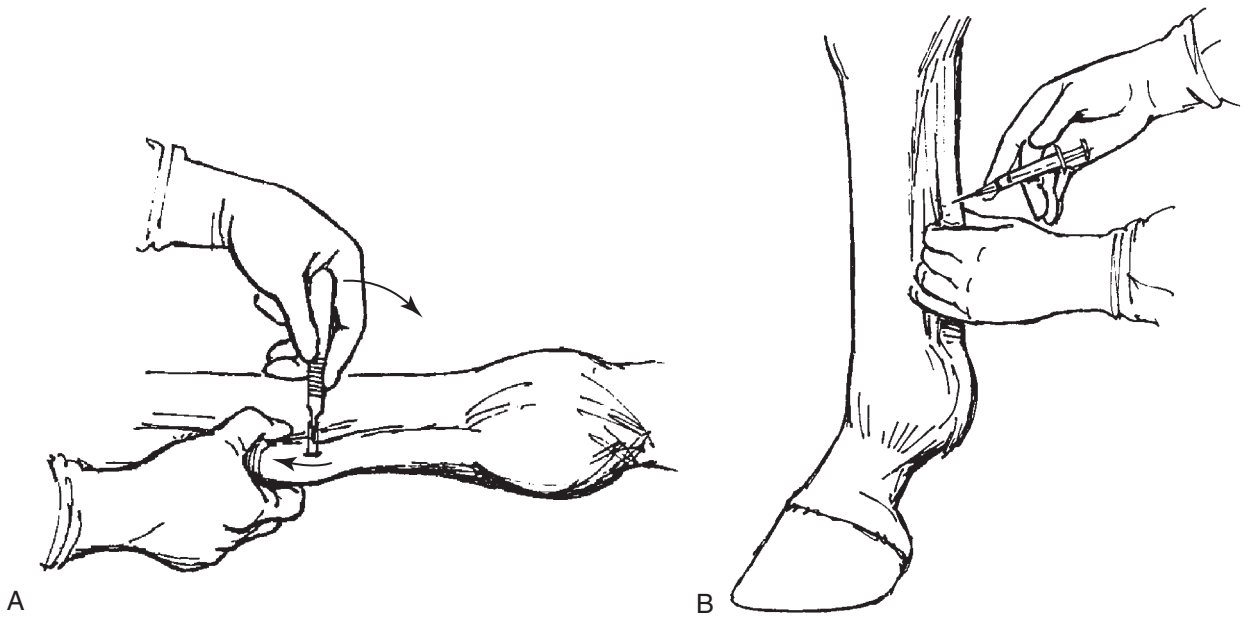

Figure 83-19. Tendon splitting can be performed with a blade **(A)** or with needles **(B).** With needles, the splitting procedure can be combined with an intratendinous treatment, if appropriate.

DALSDFT therefore are complex, and studies using cadaver limbs may not represent the biomechanical events in a fatigued galloping racehorse. However, increased risk of injury of the suspensory ligament after DALSDFT has also been demonstrated *in vivo*.[102] Initial results from Bramlage demonstrated that 79% of horses suffering from superficial digital flexor tendinopathy treated with DALSDFT competed in two races without recurrent tendinopathy.[100]

DALSDFT can be performed with the horse in lateral or dorsal recumbency. A 10-cm skin incision is made between the cephalic vein and the caudal radius on the medial aspect of the limb. The incision should extend from the level of the medial malleolus of the radius to midchestnut proximally. A transverse branch of the cephalic vein may require ligation to improve access. The cephalic vein is retracted caudad to expose the flexor carpi radialis (FCR) sheath, which is incised to reveal the FCR tendon. The FCR tendon is retracted caudad to expose the accessory ligament of the SDFT, which is located adherent to and deep to the lateral aspect of the FCR sheath. Both the transverse and the deeper oblique fibers of the accessory ligament of the SDFT are transected sharply, with or without the assistance of forceps to elevate the latter. Ideally the artery and vein of the proximal SDFT are identified on the proximal border of the accessory ligament and preserved.[103]

The FCR sheath and the fascia should be closed in two layers using a simple-interrupted or simple-continuous pattern and with 2-0 or 0 (3 or 3.5 M) synthetic absorbable suture material for each layer (e.g., polyglactin 910). The skin is then closed with a simple-interrupted or vertical mattress suture pattern using 2-0 or 0 (3 or 3.5 M) nonabsorbable material (e.g., monofilament nylon).

A well-padded bandage capable of providing compression is applied over a sterile dressing for 10 to 14 days to reduce the risk of seroma formation at the surgical site, which is a common complication of this procedure. The initial rehabilitation period involves 2 weeks of stall rest, followed by a controlled exercise program as previously described.

More recently, this procedure has been carried out tenoscopically through the carpal sheath.[104] With the affected limb uppermost and partially flexed, an arthroscopic portal is created into the carpal sheath 2 cm proximal to the distal radial physis on the lateral side of the limb. An instrument portal is made immediately proximal to the distal radial physis. With the limb in 90 degrees of flexion, the accessory ligament is visualized on the medial aspect of the sheath and cut using a No. 10 scalpel blade on a long handle or a meniscectomy knife. The very proximal portion of the ligament cannot be visualized directly but is transected by careful dissection using punch biopsy forceps, taking care to avoid the perforating blood vessel at the proximal limit of the accessory ligament. Experience is needed to perform this aspect of the surgery, and the operator may not sever all of the attachments of the accessory ligament of the SDFT, resulting in a poor outcome.

TENOSCOPY

The increasingly widespread use of tenoscopy has demonstrated a high frequency of intrathecal tendon tears that are associated with tenosynovitis and lameness. Hence, horses presenting with tenosynovitis with pain on palpation of the proximal digital sheath and lameness should always be considered candidates for tenoscopic evaluation. Based on a recent survey of cases, tears in the DDFT are more commonly found in forelimb digital sheaths, whereas tears to the manica flexoria are more commonly found in hindlimbs (Figure 83-20). Ultrasonographic evaluation usually reveals nonspecific changes of synovial hypertrophy, and it is frequently not possible to identify tears on ultrasonographic examination with confidence. However, when a tendon tear extends into the center of the tendon, it can be identified as an anechoic lesion (as it is filled with synovial fluid). Manica tears are particularly difficult to identify ultrasonographically, but occasionally disruption of the attachment of the manica to the SDFT can be demonstrated. In addition, tears can also be found and débrided tenoscopically in the SDFT and in the distal sesamoidean ligaments within the digital sheath.

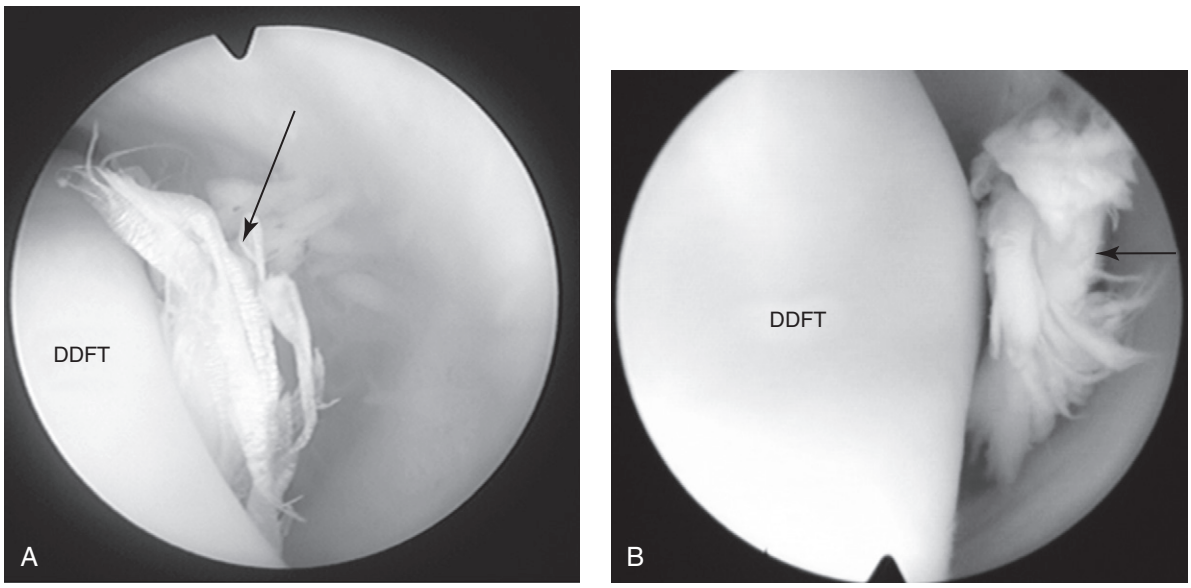

Figure 83-20. Commonly detected abnormalities that cause tenosynovitis of the digital sheath. **A,** Surface tear *(arrow)* of the deep digital flexor tendon *(DDFT),* most commonly found in forelimb tenosynovitis. **B,** Rupture *(arrow)* of the attachments of the manica flexoria, most frequently seen in hindlimb tenosynovitis.

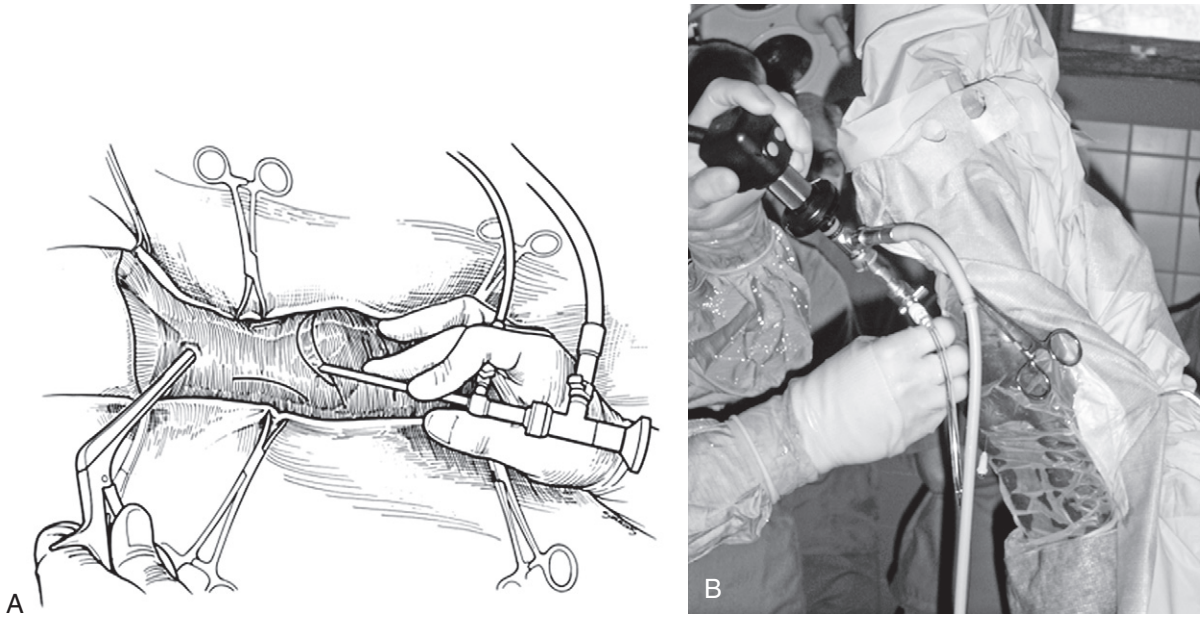

Figure 83-21. Tenoscopy of the digital sheath, which is now a routine procedure. **A,** The original description was in lateral recumbency, and the arthroscope was introduced into the digital sheath immediately distal to the palmar annular ligament, 1 to 2 cm palmar to the neurovascular bundle. **B,** However, dorsal recumbency allows more flexibility to evaluate both sides of the digital sheath and an easier bilateral approach for surgical removal of the manica flexoria. (From Nixon AJ: Endoscopy of the digital flexor tendon sheath in horses. Vet Surg 19:267, 1990.)

Tears of other tendons and ligaments communicating with a synovial cavity have been seen to be associated with synovial distention and lameness (e.g., in the suspensory ligament branches into the MCP/MTP joint) (see Figure 86-15).

Tenoscopy of the digital sheath requires general anesthesia and can be performed in lateral or dorsal recumbency. However, dorsal recumbency allows easier access to both sides of the digital sheath and is recommended. Arthroscopic portals are created immediately distal to the proximal sesamoid and 1 to 2 cm palmar/plantar to the neurovascular bundle[105] (Figure 83-21). This allows evaluation of the proximal and distal parts of the digital sheath, although distal visualization is sometimes easier with the arthroscope inserted through a portal in the proximal digital sheath (e.g., as for a proximal instrument portal). Instrument portals are created where appropriate to allow débridement of any tendon tears with a mechanical resector. This may require the instrument portals to be extended through the synovial reflection that attaches to the proximal border of the manica flexoria to allow access to DDFT tears located within or proximal to the manica. Although manica tears can also be débrided as is done for DDFT tears, this has been associated with poorer outcome than complete removal. Because no adverse effects have been observed with complete removal, this is recommended for all but the most minor manica tears. Removal requires transection of both medial and lateral attachments to the SDFT, as well as transection of the synovial attachment to the proximal border of the manica. This is facilitated by an assistant who maintains tension on the manica with rongeurs through the contra-axial proximal instrument portal. Transection can be achieved with arthroscopic scissors, bistoury, or a hook knife.

Chronic tenosynovitis can be associated with synovial masses attached to the synovial plicae in the proximal digital sheath. These, together with isolated adhesions, can also be resected during tenoscopic evaluation, and the palmar or plantar annular ligament can be transected if it is believed to be involved in the pathology. Postoperatively, the horse should be strictly rested for at least 2 weeks, after which hand-walking can be started and gradually increased over a period of at least 6 weeks. Thereafter, the duration of rehabilitation depends on the severity of the injury. The prognosis for simple DDFT tears (about 40%) is worse than for simple manica tears (about 80%).[106]

BURSOCOPY

Intrabursal lesions of the DDFT identified on postmortem examination most commonly consisted of tears and fibrillation of the dorsal surface of the tendon, followed by torn DDFT fibers adhered to the distal sesamoid bone, and DDFT core lesions.[107] A 2005 study[108] demonstrated that the prognosis for return to full athletic function without recurrence of lameness was 28%. DDFT lesions associated with distal sesamoid bone pathology had a poor prognosis. Treatment consisted of corrective farriery, controlled exercise, and in some cases medication of the distal sesamoidian bursa with hyaluronan, with or without corticosteroids. The technique for distal sesamoidian bursoscopy has been described using a transthecal approach[109] and has been associated with an improved prognosis.[110]

ANNULAR LIGAMENT DESMOTOMY

Annular ligament desmotomy has been advocated for the management of DDFT and SDFT lesions in the region of the MCP and MTP joints. The procedure is indicated if the annular ligament is impeding the normal gliding function of the flexor tendons. Annular ligament desmotomy is performed under general anesthesia and is ideally performed tenoscopically using a hook knife, with or without a slotted cannula (Figure 83-22), rather than via closed or open techniques, because it is less traumatic, ensures accurate transection of only the palmar or plantar annular ligament, and allows evaluation of the tendons to identify any primary causes. For a more detailed

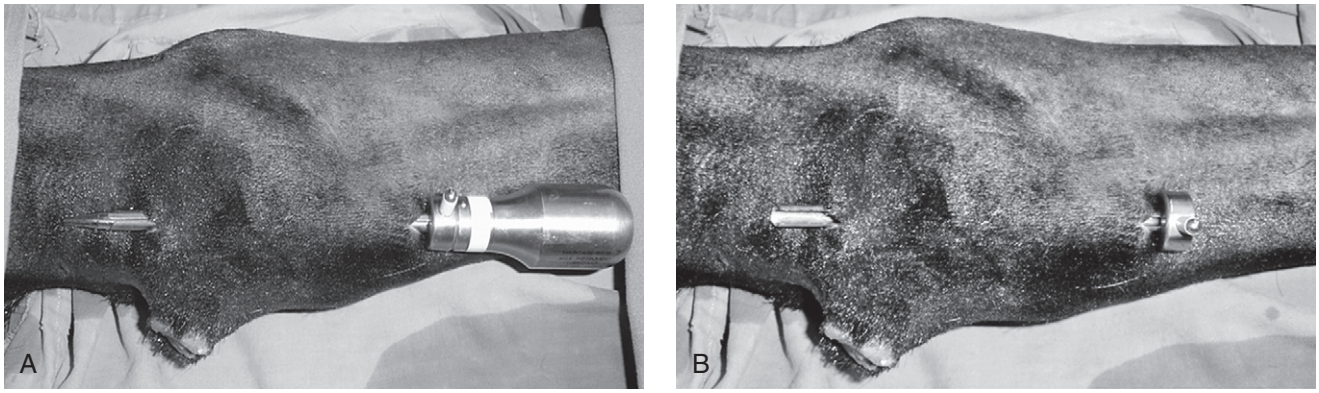

Figure 83-22. The use of the slotted cannula for transection of the palmar annular ligament. **A,** The cannula is guided through the sheath from proximal to distal under arthroscopic control to ensure that it does not lie inside the manica flexoria. **B,** The central obturator is removed and a hook knife is introduced (knife not shown) to cut the palmar annular ligament via the slot in the cannula. This can be introduced from either a proximal or a distal direction, with the arthroscope (without its sleeve) in the opposite end to view the procedure. This technique allows closed and accurate transection of the ligament without damaging other structures.

discussion of annular ligament desmotomy and its indication, please review Chapter 91.

FASCIOTOMY AND NEURECTOMY OF THE DEEP BRANCH OF THE LATERAL PLANTAR NERVE FOR THE TREATMENT OF PROXIMAL SUSPENSORY LIGAMENT DESMOPATHY

Surgery has been advocated in cases of proximal suspensory ligament desmopathy (PSLD) of the hindlimb that are unresponsive to conservative management. It has been reported that horses have returned to high-level competition after tibial neurectomy to treat PSLD.[111] However, a more specific neurectomy of the deep branch of the plantar nerve has been described.[112] This is performed under general anesthesia with the horse in dorsal recumbency. A 4- to 6-cm incision is made adjacent to the lateral border of the SDFT, originating proximally from the level of the chestnut (Figure 83-23). The plantar metatarsal fascia is incised and the incision is extended deep to the SDFT by blunt dissection, facilitated by retraction of the SDFT. The lateral plantar nerve is located in connective tissue, the deep branch is identified and transected using a scalpel, and a 3-cm section is removed. The connective tissue fascia covering the suspensory ligament is cut (fasciotomy) adjacent to the lateral splint bone to decompress the origin of the suspensory ligament, because hindlimb proximal suspensory desmopathy is believed to be associated with a compressive compartment syndrome involving the plantar metatarsal nerves.

Postoperatively, only a short period of strict rest is needed (about 2 weeks) to allow the surgical incisions to heal. Thereafter, the horse can gradually begin a controlled ascending exercise program. Nonpublished reports have suggested that this technique is associated with a high level of success in returning affected animals to full work with minimal risk of exacerbating the desmopathy.

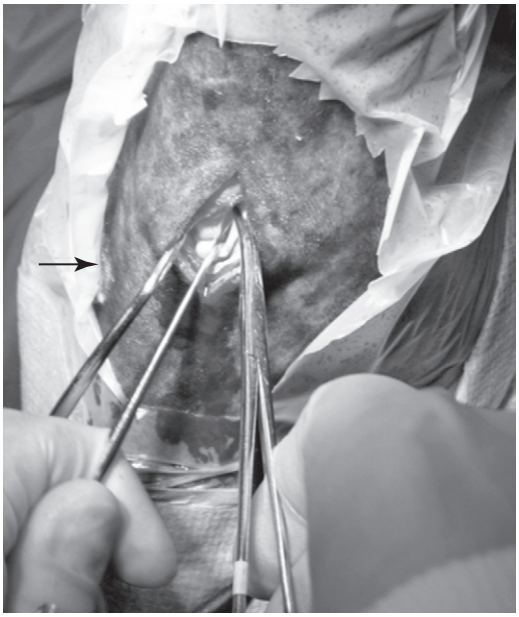

Figure 83-23. Cadaver specimen showing surgical approach for neurectomy of the deep branch of the lateral plantar nerve. Note the level of the incision compared to the location of the chestnut *(arrow)*. The nerve is held in a spay hook, and after bathing in local aesthetic, gentle traction is used to retract the nerve to allow removal of up to 5 cm.

DESMOTOMY OR DESMECTOMY OF THE ACCESSORY LIGAMENT OF THE DEEP DIGITAL FLEXOR TENDON

Conservative management of accessory ligament of the DDFT (ALDDFT) desmopathy is usually successful. However, in some cases, desmopathy recurs or causes adhesions between the ALDDFT and the SDFT or a flexural deformity. In such cases desmotomy, or preferably desmectomy, of the accessory ligament of the DDFT may be considered.[113]

ALDDFT desmotomy is performed under general anesthesia in lateral recumbency. The ALDDFT can be approached medially or laterally. A medial approach results in improved cosmesis but an increased risk of vascular damage to the medial palmar artery, and the lateral technique has the opposite effect. After aseptic preparation of the surgical site, a skin incision is made in the proximal half of the metacarpus adjacent to the palmar border of the DDFT (see Figure 87-20. The incision is continued deeper by blunt dissection to expose the ALDDFT. The ALDDFT is elevated using forceps and is transected with a scalpel. Greater exposure can allow complete removal of the accessory ligament if necessary. The wound is closed in two layers—first the fascia with a simple-continuous pattern using an absorbable suture

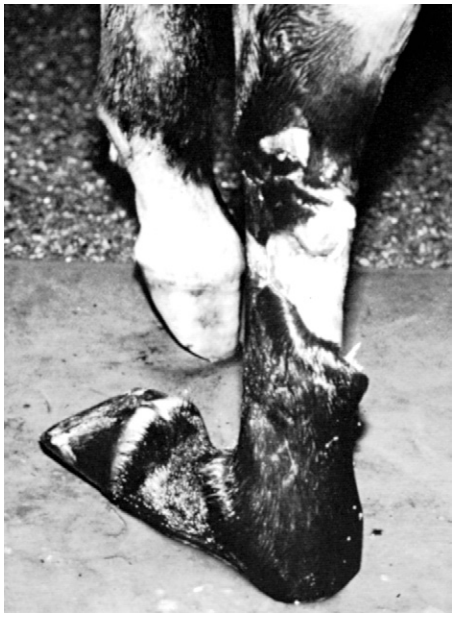

Figure 83-24. Loss of digital extensor function secondary to extensor tendon laceration, resulting in weight-bearing on the dorsum of the hoof and phalangeal region.

material, and then the skin. A modified Robert Jones bandage should be applied to the distal limb postoperatively for 3 to 4 days.

Tendon Lacerations

Lacerations of the digital extensor tendons can result in the partial or complete loss of protraction of the distal limb or the animal may place the limb in an abnormal position (Figure 83-24). However, many cases show minimal signs. In contrast, horses suffering from flexor tendon lacerations are usually in severe pain and need immediate attention.

Emergency Treatment of Tendon Lacerations

The limb should be stabilized before the horse is moved to an appropriate place for treatment. It is important that the limb be supported to ensure the comfort of the horse by restoring some biomechanical function to the limb to prevent further tissue damage. It is particularly important to avoid further trauma to the neurovascular structures of the distal limb. For flexor tendon lacerations, a palmar or dorsal splint or a commercial splint (e.g., a Kimsey splint [see Figure 73-6]) should be applied. Any difficulty in limb protraction with an extensor tendon laceration can be managed with a simple splint bandage. For more information on the first aid of traumatized horses, review Chapter 73.

Surgical Repair

FLEXOR TENDONS

Surgical repair of flexor tendon lacerations involves débridement, suturing of the tendon, and closure of the wound and is usually performed under general anesthesia with the horse in lateral or dorsal recumbency. The aims of tenorrhaphy are to restore tendon gliding function, minimize gap formation between the tendon ends, minimize adhesion formation, and preserve functional vasculature. If the laceration is complete, the tendon may have recoiled, requiring proximal and distal extension of the skin wound in an elongated S shape to locate both tendon ends (Figure 83-25). Flexing of the MCP/MTP joint may facilitate locating the distal tendon end. The wound and tendon ends should be débrided and lavaged. If the tendon ends can be apposed, tenorrhaphy can be performed using a monofilament absorbable suture (e.g., polydioxanone or polyglyconate). Nonabsorbable materials should be avoided, as they can result in shearing between the healed tissue and the suture material and may be responsible for persistent lameness. Two suture patterns are commonly used, the three-loop pulley and the interlocking loop. The three-loop pulley prevents distraction of the ends of the tendon under loading (i.e., gapping), whereas the interlocking loop has little suture material outside the tendon and is therefore the recommended technique for repair of intrathecal lacerations. The interlocking-loop pattern can be supplemented with simple-continuous sutures to reduce gap formation between the tendon ends. A single locking-loop suture is not as strong as a double- or triple-locking loop or a three-loop pulley pattern.[114]

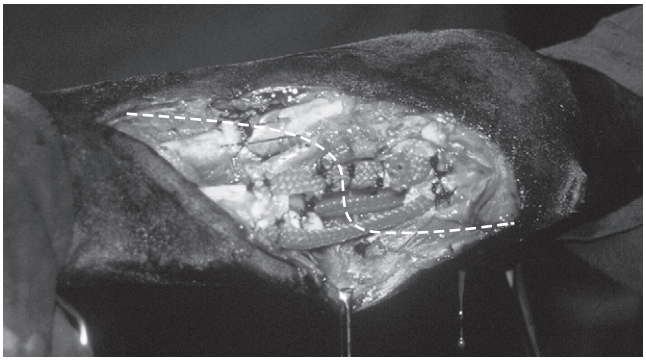

Figure 83-25. The use of poly-L-lactic acid (PLLA) to repair extensive defects in the digital flexor tendons. This horse suffered a transverse wound to the plantar aspect of the mid-metatarsal region, which transected both the superficial and deep digital flexor tendons and the lateral branch of the suspensory ligament. The wound margins were resected and the area exposed by using an S-shaped incision *(dotted line)*. After débridement of the tendons, a 5-cm defect was present in both tendons, which was bridged with two PLLA implants sutured to each end of the tendons. The lateral branch of the suspensory ligament was apposed using a three-loop pulley suture.

Frequently the injury is associated with significant blunt trauma to the tendon ends, which precludes direct apposition of the tendon ends. In this situation, the tendons ends can be left after débridement, the wound closed, and the limb cast, or an implant can be used to maintain the alignment of the tendon ends. The ideal tendon implant material would have biomechanical properties similar to those of normal tendon. Various implant materials have been used to repair lacerated flexor tendons, including carbon fiber, terylene (polyester), autologous extensor tendon grafts, and poly-L-lactic acid (PLLA).[115] Carbon fiber implants were associated with persistent lameness postoperatively, which may have been caused by tenalgia resulting from shear forces between inelastic carbon fibers and the healed tendon tissue. The use of autologous grafts using extensor tendons to bridge the deficit between two ends of a lacerated tendon has never gained popularity. The advantage of PLLA is

that it supports fibroblast growth on its surface and loses its strength over several months, thereby being able to match its mechanical properties with the tendon. Implants are anchored in each end of the lacerated tendon by fixing the ends in V-shaped incisions created in the tendon ends with sutures of monofilament absorbable sutures. Implants are not recommended as a treatment for strain-induced tendinopathy.

Partial lacerations involving less than 50% of the tendon may need only local débridement. Lacerations involving greater than 50% are probably best sutured, as this can prevent the generation of longitudinal splits between loaded and unloaded parts of the tendon.

Flexor tendon lacerations require a protracted rehabilitation period. A distal limb cast should be placed with forelimb lacerations postoperatively. In the hindlimb, a full-limb cast is required after flexor tendon laceration to immobilize the forces of the reciprocal apparatus. However, distal limb casts can be used in the hindlimb to avoid the increased risk of complications of full-limb casts, unless an implant has been placed, in which case the result is usually that one end is pulled out of the tendon. Casting is required for a minimum of 6 to 8 weeks and no more than 12 weeks, thereby often necessitating one cast change under general anesthesia, as studies have shown that the breaking strength of the tendon repair at 6 weeks approximates the body weight of the horse.[116]

Adequate support to the MCP/MTP joint in an adult horse cannot be provided by a bandage alone. A palmar/plantar splint applied over a modified Robert Jones bandage and caudal shoe extensions (for DDFT lacerations) provide protection of the repair after cast removal. Alternatively, a specifically designed boot has been shown to provide sufficient support.[65] This device has the advantage that it can be used during exercise because it is jointed. Continued stall rest is necessary for a further 2 to 3 months, after which walking exercise followed by an ascending exercise regimen can be initiated. Ultrasonographic monitoring of tendon healing is useful to assess the integrity of the tendon repair. A minimum of 8 to 12 months is usually required before full athletic function can be resumed.

The prognosis for flexor tendon injuries is guarded, with approximately 45% of horses returning to athletic function in one study[116]; in another study, the prognosis was 59% for flexor tendon lacerations.[117] In the second study, the prognosis for return to soundness was not increased if the DDFT and SDFT were simultaneously lacerated compared with involvement of only a single structure.[117] Complications of tendon lacerations include necrotic tendinopathy (which occurs as a result of infection or damage to the vascular supply), concurrent synovial sepsis, cast complications, adhesions resulting in continued pain and lameness, exuberant granulation tissue formation, flexural deformity, and reinjury.

EXTENSOR TENDONS

Extensor tendon lacerations heal remarkably well without tenorrhaphy and respond well to conservative management. The wound should be débrided and the primary wound closed if appropriate. If the extensor tendon has been lacerated within the confines of a tendon sheath (e.g., for extensor lacerations over the dorsal aspect of the carpus), the area should be lavaged, and elimination of sepsis from the tendon sheath will need to be addressed. Fibrous scar tissue gradually forms an attachment between the two tendon ends. A period of 4 to 6 weeks of stall rest until the wound has healed is usually all that is required. A cast can be used to assist soft tissue healing if contusion has been extensive. The prognosis for extensor tendon lacerations is good (with 72% to 80% of horses returning to athletic function).[117,118] The prognosis for extensor tendon lacerations is better than for flexor tendon lacerations, because the extensor tendon bears considerably less load and has a minimal effect on the gait, with most protraction of the limb and digit arising from the upper limb and momentum of the foot. Indeed, a publication reported the successful management of septic common digital extensor tenosynovitis by complete resection of the tendon.[119] Stumbling may be evident at the walk until the tendon has healed, but this can be reduced by shortening the toe of the hoof and rolling the toe of the shoe or by fitting a Natural Balance type of shoe. If extension cannot be maintained spontaneously, a toe extension can be fitted to the shoe to prevent secondary contracture, although this may make stumbling more severe.

The most common complications associated with extensor tendon lacerations include exuberant granulation tissue and sequestrum formation associated with underlying damage to the bone, although acquired flexural deformities can occur.

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CHAPTER

84 Biology and Management of Muscle Disorders and Diseases

John G. Peloso

STRUCTURE AND FUNCTION OF MUSCLES

Of the three types of muscle in the body—skeletal, smooth, and cardiac—skeletal muscle makes up about 40% of the body weight.[1,2] All movement is the result of contraction of skeletal muscles, and most patients with neuromuscular disease exhibit abnormal movement. *Skeletal muscle* consists of a central fleshy muscle belly and a tendon at each end. The tendon at one end of the muscle originates on one bone, and the tendon at the other end inserts on a different bone, with at least one movable joint interposed. When activated by a motor neuron, a skeletal muscle can only contract (or shorten). Movement occurs when the central muscle belly shortens, causing the two tendons and their bones of attachment to move with respect to each other around a central joint. A muscle returns to its original longer length by the pull of its antagonistic counterpart, a muscle that traverses the joint on the opposite side. In some locations, it may be more than one muscle. Some muscles increase joint angle (extensors), and some muscles decrease joint angles (flexors).

Anatomy

Skeletal muscle has several levels of organization (Figure 84-1). Each muscle belly is made up of thousands of muscle cells (also called muscle fibers). Each muscle fiber contains thousands of myofibrils that are arranged in parallel along its length, and each myofibril is made up of a series of repeating sarcomeres. The *sarcomere* is the basic contractile unit of the muscle fiber.[1,2]

Myofibrils have an arrangement of alternating thick and thin filaments. The thick filaments are composed of the protein *myosin*, and the thin filaments are composed of the proteins *actin*, *tropomyosin*, and *troponin*.[1,2] Myofibrils are surrounded by membranes in the form of tubules. These tubules are composed of a transverse tubular system and the sarcoplasmic reticulum, and collectively they are called the *sarcotubular system*. Each muscle fiber is innervated by a terminal branch of a motor nerve fiber. The junction between the nerve and the muscle fiber is the neuromuscular junction. All muscle fibers that are innervated by a single nerve fiber are called a *motor unit* because all of the muscle fibers within a unit are excited simultaneously and contract in unison.

Physiology of Contraction

It is at the neuromuscular junction that action potentials are generated. The resting membrane potential of the sarcolemma is excited by a synaptic transmission at the neuromuscular junction. Via transverse tubules, the action potential reaches the sarcoplasmic reticulum throughout the muscle fiber.

Skeletal muscle cells (muscle fibers) convert chemical energy into mechanical energy. At rest, calcium ions are maintained within the sarcoplasmic reticulum of the muscle cell. The arrival of an action potential to the sarcoplasmic reticulum causes the release of calcium ions. These calcium ions perfuse the sarcomere and bind with troponin. This alters the actin filament, allowing it to react with the myosin filament. Although a detailed molecular explanation of the sliding filament mechanism is not known, the altered actin molecule is now able to slide along the myosin molecule, causing a physical shortening of the sarcomere (Figure 84-2). As the action potential passes, calcium ions are returned to the sarcoplasmic reticulum, allowing the sarcomere to relax. Therefore, in the presence of calcium ions and adenosine triphospate (ATP), actin and myosin molecules slide over one another. This results in a physical shortening of the sarcomere and ultimately of the myofibril and the muscle belly. The cumulative effect of a shortened sarcomere is a reduction in the distance between the two tendon ends, and their bones of attachment move with respect to each other around a central joint.

ATP is needed for this contraction, and creatinine phosphate can be used to replenish ATP stores from adenosine diphosphate (ADP). The enzyme creatine phosphokinase (CPK) catalyzes this conversion and is found in high concentrations within muscle cells. During muscle cell damage, the CPK that

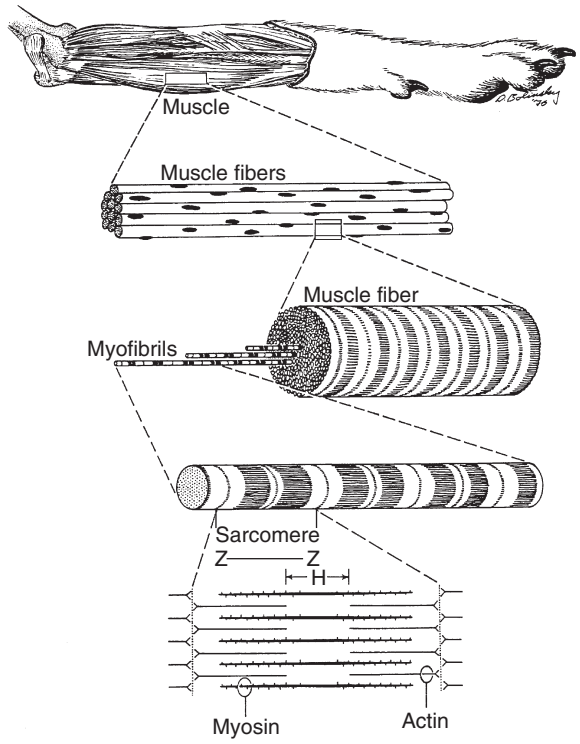

Figure 84-1. Levels of organization within a typical skeletal muscle. (From Cunningham JG: Textbook of Veterinary Physiology. 3rd Ed. Saunders, Philadelphia, 2002.)

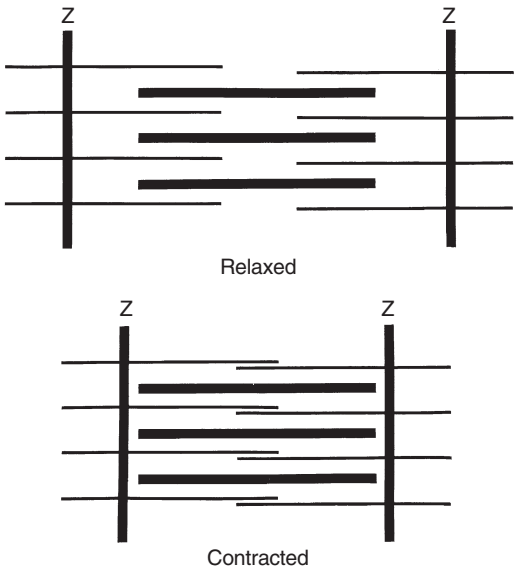

Figure 84-2. The sliding of actin along the myosin molecule results in the physical shortening (contraction) of the sarcomere. (From Cunningham JG: Textbook of Veterinary Physiology. 3rd Ed. Saunders, Philadelphia, 2002.)

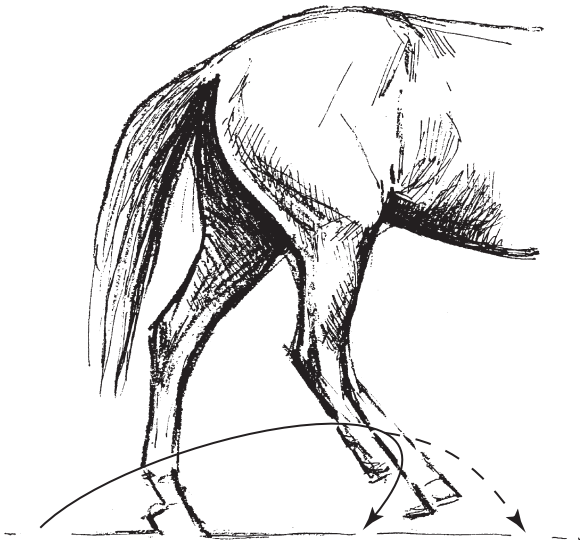

Figure 84-3. Motion of the rear limb of a horse affected with fibrotic or ossifying myopathy. The limb is jerked backward just before hitting the ground.

is normally contained within intact muscle cells can leak into the bloodstream. Therefore identification of an abnormally high concentration of serum CPK is used routinely to assess skeletal muscle necrosis. Muscle contraction is only about 40% efficient in converting chemical energy to mechanical energy, and most of the energy is lost as heat. This phenomenon is exploited during shivering to raise core body temperatures. During shivering, antagonistic muscle groups are activated so that they produce no useful work. The chemical energy lost as heat is transferred to the body core to increase temperature.

Muscle Repair

Muscle has a very limited capacity for regeneration. In some muscles, stellate cells have been identified that can form new muscle fibers. For practical purposes, however, new muscle fibers are not formed after birth. Enlargement and growth of muscle occur via enlargement of fibers caused by formation of additional myofibrils. Repair of damaged muscle occurs by replacement with connective tissue. Muscle has a good blood supply and a ready source of fibroblasts. Healing of damaged muscle with scar tissue is usually rapid, but replacement of damaged muscle with large amounts of scar tissue can restrict muscle length and function.

SPECIFIC MUSCLE DISORDERS

Fibrotic Myopathy

The classic description of fibrotic myopathy is that of a unilateral hindlimb gait abnormality, in which the cranial phase of the stride is shortened and there is a rapid caudal movement of the limb during the caudal phase of the stride as the foot slaps the ground (Figure 84-3). This condition is most evident when the horse is observed at the walk from a lateral perspective.[2-12] The overall prevalence of the disease is low: of the 167,132 horses examined by 22 university veterinary hospitals in North America over the 10 years from 1979 to 1988, 102 horses (0.06%) were diagnosed with fibrotic myopathy.[2]

Pathophysiology

In most horses, traumatic injuries that cause adhesions and fibrosis of the *semitendinosus muscle* are responsible for the condition. Less commonly, injury to the semimembranosus, biceps femoris, and gracilis muscles has been identified.[7,8] Trauma may result from lacerations, slipping while exercising, and entrapment of a foot in halters, lead shanks, or fences. In one study, 5 of 18 horses with fibrotic myopathy developed the condition secondary to intramuscular injections.[6] Tearing the insertion of the semitendinosus muscle during activities that create extreme tension on the caudal aspect of the hindlimb (barrel racing, the slide of a reining horse) has also been incriminated.[8] The characteristic gait of horses with fibrotic myopathy can occur temporarily after transportation in a trailer; in this case, it is caused by a localized myositis of the semitendinosus muscle because of prolonged pressure by the rear rope or bar on the muscle. Fibrotic myopathy in the neonate is speculated to be caused by trauma at or soon after birth, although the exact etiology is unknown in congenital cases.[9] Horses born with the gait characteristic of fibrotic myopathy have a taut semitendinosus muscle and tendon, but they do not have palpable fibrosis of the muscle. In a small series of horses, traumatic or degenerative neuropathy causing denervation of the distal semitendinous muscle resulted in fibrotic myopathy.[13] The underlying neuropathy is the most likely cause in horses when the condition progresses to involve both limbs. If bone forms in the affected tissue, the condition is termed *ossifying myopathy.*

Diagnosis

The diagnosis of fibrotic myopathy is made by observing the abnormal gait and by palpating the fibrotic or ossified muscle areas. Lameness is usually most apparent at the walk and is characterized by an abrupt cessation of the cranial phase of the

stride of the affected limb, with the foot suddenly jerked caudad just before it hits the ground (see Figure 84-3). This gait abnormality is not painful and is caused by mechanical restriction (either fibrous or osseous) of the affected thigh muscles. The gait abnormality is evident on every stride, is nonresponsive to analgesics, and is not altered by manipulative tests.

The fibrotic or ossified areas of the affected muscles may be palpated on the caudal aspect of the thigh at the level of the stifle joint or immediately above it. Ultrasonography may be helpful in defining areas of fibrosis, whereas radiography is more useful to demonstrate areas of ossification.

Treatment

Several surgical procedures for the correction of this disorder have been described. These techniques include semitendinosus myotomy, semitendinosus myotenectomy, or semitendinosus tenotomy at the tibial insertion of the muscle.[3,5,6,9-12] Earlier treatments described for these conditions include resection of the fibrous or ossified part of the semitendinosus muscle and removal of adhesions to surrounding muscles.[3,6] Myotomy and myotenectomy are no longer recommended because of the relatively high rate of postoperative complications, including protracted hemorrhage, wound dehiscence, high recurrence rate, and noncosmetic outcomes.

Semitendinosus tenotomy is presently the treatment of choice for fibrotic myopathy of the semitendinosus muscle.[9,11] This procedure is performed under general anesthesia in lateral recumbency with the affected limb down, providing access to the medial aspect of the tibia. Landmarks for surgery include the tibial insertion of the muscle on the caudomedial aspect of the tibia just distal to the medial femorotibial joint and caudal to the saphenous vein overlying the gastrocnemius muscle. An 8-cm vertical skin incision is made directly over the palpable tendon and through the subcutaneous and crural fascia until the tendon is exposed. A large forceps, such as a curved Kelly or Crile, is passed under the tendon to isolate it from the muscle, and the tendon is transected (Figure 84-4). Resection of a 3-cm segment of the tendon obviates or delays recurrence of the gait abnormality. Fascial layers are closed with interrupted or continuous synthetic absorbable sutures, and the skin is closed with interrupted or continuous nonabsorbable suture material.

When the affected limb is pulled forward during surgery, the tendon of insertion of the semitendinosus muscle onto the calcaneal tuber may become very taut in some horses.[2] An additional 3- to 4-cm-long incision has been recommended directly over the taut tendon, which is then isolated and transected. This incision is caudal and slightly distal to the first incision. Tenotomy is technically easier than the other myotomy procedures and eliminates an incision into diseased muscle.

Prognosis

With myectomy and myotomy, improvement is usually noticed immediately, but it may take up to 7 days for maximal improvement. Standing myotomy resulted in partial recurrence of the gait abnormality after surgery in approximately one third of 39 horses treated.[12] The tenotomy procedure alone results in fewer complications than the other techniques, including less predisposition for re-formation of the fibrous band, but it may not result in complete resolution of the gait abnormality.[9] If a degenerative neuropathy is the cause of the condition, recurrence of clinical signs is likely, regardless of the surgical technique employed.

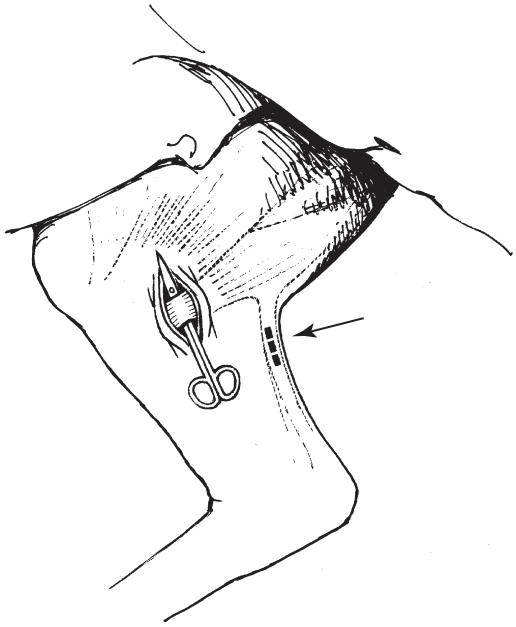

Figure 84-4. Semitendinosus tenotomy. The tendon of the tibial insertion has been isolated on the medial side of the proximal tibia and is ready to be severed. The *dashed line (arrow)* indicates the location for the second incision, which is performed if the insertion of the tendon on the tuber calcis is taut.

Stringhalt (Equine Reflex Hypertonia)

Stringhalt, recently renamed *equine reflex hypertonia*,[14] is a hindlimb gait abnormality characterized by involuntary and exaggerated flexion of one or both limbs.[14-26] During the cranial phase of the stride, the limb is jerked toward the abdomen (Figure 84-5). There may be a wide variation in the severity of the signs that occur during every walk stride. The gait abnormality has a classic appearance, but it should be differentiated from mild forms of intermittent upward fixation of the patella. The patella catches on the medial trochlea of the distal femur in intermittent upward fixation of the patella, and when it releases it gives the appearance of a horse with stringhalt.

Pathophysiology

Two clinical presentations of stringhalt have been described: an idiopathic (sporadic or conventional) form and an acquired (Australian or outbreak) form. The *idiopathic form* has been reported worldwide; it usually involves a single hindlimb, and clinical signs can progress over years.[16,20,26,27] Idiopathic stringhalt is rarely seen as a bilateral condition. The cause has been attributed to an overextension injury of the tarsus and metatarsus; metatarsal, tarsal, and stifle injuries; foot pain; spinal cord disease; and tendon adhesions.[16,20,26,27]Although the true etiology for the condition is unknown, idiopathic stringhalt is suggested to be caused by an underlying neuropathy.[19]

Figure 84-5. Exaggerated flexion of the rear limb is a characteristic sign in horses with stringhalt.

One author reports that all muscle or peripheral nerve isolates from horses with idiopathic stringhalt had histologic evidence of neuropathy or degenerative myopathy.[19] Idiopathic stringhalt has not been associated with the spontaneous recovery that is seen in the acquired form.

The *acquired form* is usually observed in both hindlimbs and occurs epidemically.[14-26] Presenting as outbreaks during the late summer or fall in horses on pasture, it often follows drier-than-normal months and is related to the presence of toxic plants. Outbreaks are usually related to one of the following plants: *Hypochaeris radicata* (Australian dandelion), *Taraxacum officinale* (European dandelion), or *Malva* (mallow), with outbreaks being reported in Australia, New Zealand, North America, Brazil, Italy, Chile, and Japan.[21-25] Its etiology has been clearly shown to involve an underlying neuropathy.[19,25] A definitive diagnosis of distal axonopathy has been obtained after postmortem examination of selected muscles and nerves[15,16] and antemortem by histologic examination of nerve and muscle biopsy specimens.[25] Evaluation of limb nerve samples from horses with acquired stringhalt has demonstrated widespread neuropathy. Lesions also occur in the left recurrent laryngeal nerve in approximately 60% of these horses.[17] The lameness in acquired stringhalt is caused by a peripheral neuropathy and neurogenic muscle atrophy of the long digital extensor, lateral digital extensor, and gastrocnemius muscles. Many horses recover without treatment.[14-19,21-26]

Diagnosis

The diagnosis is based on observation of the characteristic gait caused by exaggerated flexion of one or both hindlimbs. The abnormalities are usually bilateral in acquired stringhalt. The onset is often sudden. Some horses show a very mild flexion of the hock during walking, and others show a marked jerking of the foot toward the abdomen, with the foot actually hitting the abdomen in severe cases (see Figure 84-5). The abnormal gait is usually evident with each step. Rest and cold weather tend to accentuate the gait, and in some horses, the gait may return to normal with exercise. All breeds can be affected.

Treatment

Horses with *acquired stringhalt* often recover spontaneously. Removal from the incriminated pasture may be curative, although recovery can take from months to years and may not be complete. In one study, 78% of the horses recovered over a period ranging from a few days to more than 18 months.[17] Although surgical treatment is not recommended for horses with acquired stringhalt, good results were recently reported in 13 horses.[18] Five horses were able to walk normally immediately after the surgery (described later) in both hindlimbs, whereas only partial improvement was noted immediately postoperatively in the remaining six horses. Five of them resumed a normal gait in 4 to 12 weeks postoperatively. The last horse suffered a relapse several weeks postoperatively and eventually recovered spontaneously 9 months after surgery.[18]

Because *idiopathic stringhalt* is rarely associated with spontaneous recovery, lateral digital extensor tenectomy and partial myectomy is the treatment option for these horses.[20,28]

The objective of *lateral digital extensor (LDE) tenectomy and partial myectomy* is to remove the distal 2 to 10 cm of the LDE muscle, and the entire tendon, from the muscle to its attachment to the long digital extensor tendon. The procedure may be performed using local anesthesia when removing small portions of muscle belly, or under general anesthesia when removing large portions of the muscle belly. For the standing procedure, a 2-cm line of local anesthetic is injected over the LDE tendon just proximal to its junction with the long digital extensor tendon on the lateral aspect of the metatarsus. This site is easily palpable just below the tarsometatarsal joint. Local anesthetic also is infiltrated directly into the muscle belly of the LDE, about 2 cm above the lateral malleolus of the tibia.

The distal incision is made directly over the tendon just proximal to its junction with the long digital extensor tendon (Figure 84-6). The tendon is subsequently exposed by bluntly dissecting beneath the tendon with a curved Kelly or Ochsner forceps. The proximal incision is located on the lateral aspect of the limb approximately 6 cm above the lateral malleolus of the tibia. It extends through the skin, subcutaneous tissue, and fascia directly over the lateral digital muscle in a vertical direction, parallel with the muscle fibers. The muscle belly is exposed using blunt dissection, and a heavy curved instrument is placed underneath it (see Figure 84-6). Before severing the tendon, the surgeon should ensure that the tendon in the distal incision corresponds to the muscle belly in the proximal incision. The tendon is severed in the distal incision and is pulled through the tendon sheath by traction on the proximal portion with a curved Ochsner forceps or Mayo scissors. Removing the tendon from its sheath is a maneuver that requires substantial force, particularly if adhesions are present. After the entire length of the tendon has been exteriorized, the muscle is severed at the proximal aspect of the incision, thereby ensuring that at least 2 cm of the muscle is removed.

The heavy fascia of the proximal incision is closed with a simple-interrupted or continuous pattern of No. 0 synthetic absorbable suture material. The subcutaneous tissue is closed with 2-0 synthetic absorbable suture using a simple-continuous pattern, and the skin is closed with nonabsorbable suture material in a simple-continuous pattern. The distal incision is closed with skin sutures only. The wounds are covered with a sterile dressing, and the entire limb is bandaged for 10 to 14 days to

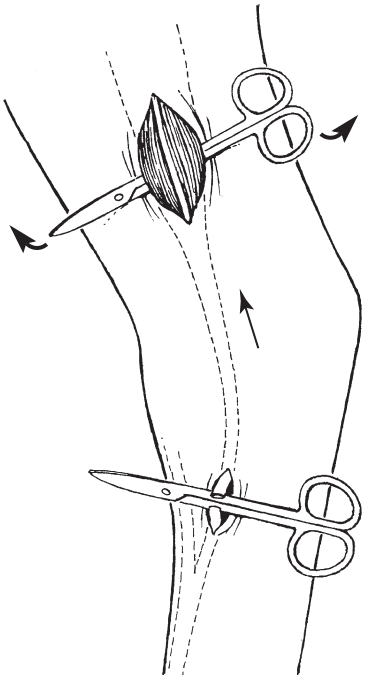

Figure 84-6. Lateral digital extensor tenectomy and partial myectomy. The muscle belly and tendon of the lateral digital extensor muscle have been isolated through two separate incisions. The distal part of the muscle and adjacent tendon are removed between these two incisions by transecting the tendon at the distal incision and pulling it out through the proximal incision *(arrows)*. The distal 2 cm of the muscle with the attached tendon are subsequently severed, and the incisions are closed.

control seroma formation. The horse is confined to a stall for this time, after which the sutures can be removed. The horse is then given 1 week of controlled hand-walking, and normal exercise can resume 3 to 4 weeks after surgery.

Complications of surgery include dehiscence; infection, which may enter the open tendon sheath of the LDE tendon; and failure of the lameness to improve.

Prognosis

The prognosis for acquired stringhalt is fair. Many horses recover spontaneously, although a few have stringhalt of such severity that they are euthanized. The prognosis for idiopathic stringhalt is guarded to favorable, because surgery is unpredictable and can result in partial to complete recovery. Many horses show improvement after surgery, but the degree of improvement varies from slight to a complete remission.[16,20,26] Improvement may occur immediately or take several months.

Peroneus Tertius Disorders

The peroneus tertius (PT) is an entirely tendinous muscle. It has a common tendon proximally with the long digital extensor muscle and originates in the extensor fossa on the cranial lateral aspect of the distal femur. Then it courses distad over the cranial aspect of the tibia and ultimately attaches to the distal tarsus and proximal metatarsus.[29,30] At the level of the proximal lateral aspect of the trochlear ridge of the talus the PT forms a sleeve through which the tibialis cranialis muscle passes.[29,30] At the dorsal aspect of the tarsus and just distal to this sleeve, the tibialis cranialis tendon divides into medial and dorsal branches and the peroneus tertius tendon divides into four portions (dorsal, medial, deep and superficial lateral). The dorsal portion attaches with the tibialis cranialis tendon on the central and third tarsal bone and the third metatarsal bone. The medial portion attaches medially on the central and third tarsal bones and the proximal aspect of the third metatarsal bone. The superficial lateral portion attaches to the calcaneal and fourth metatarsal bones, and the deep lateral portion attaches to the middle extensor retinaculum.[29,30]

The PT tendon is an important part of the reciprocal apparatus that coordinates flexion and extension of the stifle and hock (see Figures 97-2 and 97-3). The tarsocrural joint is flexed by the active contraction of the tibialis cranialis muscle and the passive pull of the tendinous peroneus tertius muscle.

Injury to the PT tendon is a relatively rare condition and has been reported to occur in three locations.[29-35] In a retrospective of 27 cases of rupture of the peronius tertius, the location of the rupture was identified in 24 cases: the origin (within 5 cm of its attachment to the distal femur; 2 cases), the insertion (just proximal to the sleeve formed by the peroneus tertius; 11 cases), and midbody (located between the origin and the insertion; 11 cases).[29]

Rupture at the origion of the PT tendon is typically associated with an avulsion fracture of the distal femur. Given their anatomic relationship, the long digital extensor muscle must also be involved. Ruptures at the origin of the PT are usually seen in foals,[29,31-33] but they have also been reported in adult horses.[29] It has been speculated that foals may be more likely to suffer avulsion injuries as a result of inherent weakness in their bones.[31]

Pathophysiology

Trauma was the most common cause for rupture to the PT.[29,31,34] In a retrospective of 27 cases,[29] blunt trauma resulting in a closed injury was caused by falling down (10 cases), followed by slipping (4 cases), caught in bars (3 cases), or running into a solid object (2 cases). In this same report, the PT was also disrupted in 6 of 27 cases because of a laceration resulting in an open injury.

By virtue of the anatomy described earlier, flexion and extension of the tarsus and stifle must occur simultaneously if the PT is intact. Therefore, rupture of the PT has also been surmised to be caused by an overextension of the hock while the stifle is held in flexion. This may occur when the horse slips with a hindlimb extended backward, during the exertion of a rapid start, or when the limb is trapped and the horse struggles to free it.[29,34] A full-limb cast extending to the proximal tibia may cause rupture if the horse slips with the leg extended behind or struggles against the cast.[34]

Diagnosis

Although the PT tendon may rupture anywhere along its length, it does so most commonly at the midbody of the tendon and the site of insertion. The classic sign of a complete rupture is the ability to extend the hock while the stifle is flexed.[29] A characteristic dimple forms in the common calcaneal tendon on the caudal lateral aspect of the crus, just proximal to the calcaneal tuber (Figure 84-7). When standing, the horse may bear weight

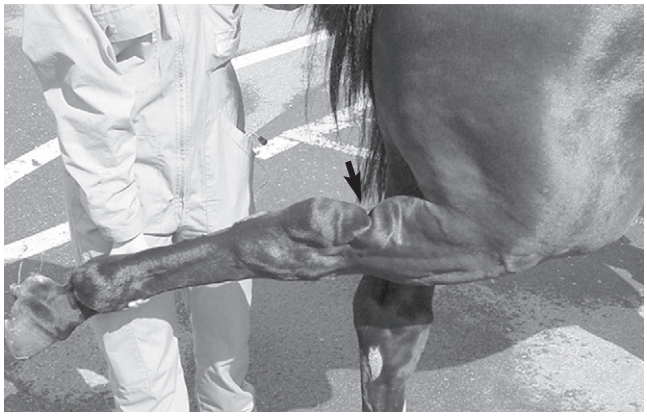

Figure 84-7. A horse with rupture of the peroneus tertius. The hock is extended while the stifle is flexed. Note the characteristic dimple in the contour of the caudal distal aspect of the crus *(arrow).*

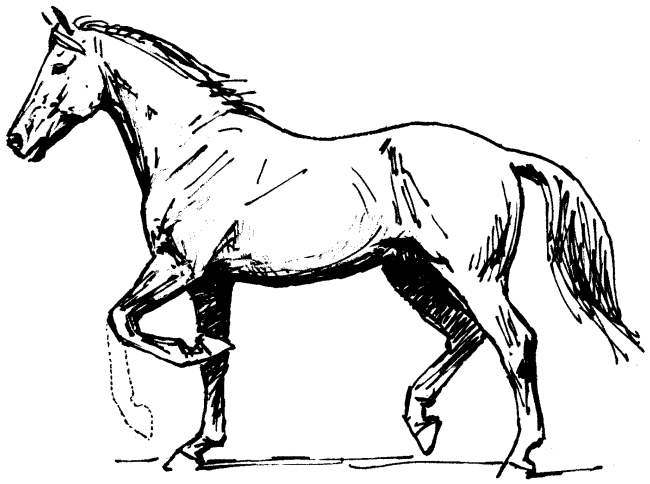

Figure 84-8. Drawing of a horse demonstrating the exaggerated flexion and forward movement of the left forelimb as a result of a traumatic rupture of the extensor carpi radialis tendon. The dotted outline of normal forelimb flexion is shown for comparison.

normally, but during the caudal phase of the stride during the walk or trot, the distal limb appears abnormally flaccid. With avulsion injuries, horses can be severely lame initially, and there may be accompanying distention of the femorotibial and femoropatellar joints.[31-33] In the acute phases of a rupture, digital palpation may reveal some swelling and pain in the craniolateral aspect of the tibia, but typically there is little evidence of pain on physical examination.

When the horse is walked, the tarsus may be noted to extend more than usual, and at the trot lameness was reported as a grade 1 out of 5 (3 horses), grade 2 (6 horses), grade 3 (10 horses), and grade 4 (2 horses).[29] A delay in the cranial phase of the stride can be easily identified because of an inappropriate degree of extension in the tarsus. Recognizing that the PT tendon is the most echogenic structure on the craniolateral aspect of the crus, it is readily identified by ultrasonography initially during the diagnosis and serially during the recovery period.[29] Radiography is necessary for diagnosing avulsion fractures.

Treatment

Horses with avulsion injuries of the origin of the PT tendon are best evaluated arthroscopically.[31-33] Small bone fragments can be removed, but large bone fragments that are encapsulated by the joint capsule are better left in place. Six weeks of stall rest followed by controlled exercise for 3 months is recommended. Ruptures of the midbody and insertion are treated conservatively using stall rest and a controlled return to exercise.

In a retrospective of 27 cases,[29] rupture of the PT was treated with stall rest for a median time of 16 weeks (range, 0 to 52 weeks), with or without hand-walking. After stall rest, horses were turned out to pasture for a median of 12 weeks (range, 4 to 48 weeks). Twenty-two of 27 horses were gradually returned to their previous level of exercise over a median time period of 8 weeks (range, 4 to 12 weeks). Overall, 18 of 23 horses (78%) returned to their previous level of exercise, and 5 of 23 (21.7%) horses were euthanized because of persistent lameness.[29] Premature return to exercise was implicated as the cause for reinjury. The value of serial ultrasonographic examinations to monitor the healing of the PT tendon before returning a horse to exercise was emphasized.[29]

Prognosis

Horses with avulsion injuries have a guarded prognosis for full soundness, but successful outcomes have been reported.[29,31-34] Age, an open or closed injury, lameness at presentation, ultrasonographic size at presentation, location of the rupture, and duration of rehabilitation had no significant influence on a horse's return to exercise.[29] A horse was significantly less likely to return to its intended use if the horse was racing at the time of injury (13 times less likely) or if a structure in addition to the PT tendon was damaged at the time of injury (8 times less likely).[29]

Rupture of the Extensor Carpi Radialis Tendon

Rupture of the extensor carpi radialis tendon is usually the result of a traumatic insult, such as a kick or laceration at the distal cranial aspect of the radius, stumbling and falling on a road (coronation), or contact between a forelimb and a sharp object.

Diagnosis

Horses suffering from a rupture of the extensor carpi radialis tendon have a peculiar gait. Protraction of the involved forelimb results in hypermetric elevation, because the flexor muscles are not opposed by their main antagonist, the extensor carpi radialis muscle (Figure 84-8). Palpation of the cranial aspect of the carpal and distal radial region reveals a defect in the area of the missing tendon. In cases with a laceration, the distal tendon stump is often seen in the injury site.

Treatment

Lacerations should be carefully débrided and closed, and a drain should be placed for the initial few days. If possible, the tendon should be repaired with a three-loop pulley or a locking-loop technique. The laceration should be sutured using routine wound repair techniques and the limb placed in a splint bandage that prohibits carpal flexion for 6 weeks. The bandage should be changed after 3 days and the drain removed. The

horse is restricted to a box stall and the splint bandage is changed at regular intervals. If no open wound is present or the wound does not involve the sheath of the extensor carpi radialis tendon, the tendon is not sutured, but the limb is kept in a splint bandage for 6 weeks, followed by bandaging the limb for an additional 3 weeks. Hand-walking is initially the only exercise, and it is gradually increased. Local wound management should be applied to superficial wounds.

Prognosis

Generally, a good prognosis for return to a successful athletic career is given for rupture of the extensor carpi radialis tendon. Complications include infection of the tendon sheath and dehiscence of the sutured tendon or sutured laceration. In such cases, conservative management involving daily wound care and bandage changes is required. The prognosis in complicated cases is guarded for return to an athletic career.

Cribbing and Aerophagia

Etiology

Cribbing or crib biting in the horse is a vice characterized by the placement of the upper incisors on a solid object, arching the neck, depressing the tongue, elevating the larynx, and pulling backward.[36-52] If the horse swallows air, the process is called *aerophagia* or *wind sucking,* and an audible grunt can be heard. The sequelae to this habit are poor performance, weight loss, erosion of the incisors, and chronic colic. It has been demonstrated that cribbing and aerophagia significantly increased the risk for simple colonic obstruction and distention in horses,[36] significantly more ulcers and inflammation of the stomach of foals,[37] and epiploic foramen entrapment colic.[38,39] The principal reason that horses with this vice are presented to a veterinarian is because the behavior is objectionable and irritating to owners, and it results in an unacceptable level of property destruction.[40] Studies suggest that limiting this behavior would reduce the incidence of certain types of gastrointestinal distress.[36-39] The habit is learned or acquired and is usually associated with stabled horses, although once learned it can continue in the pasture. Another reason patients are presented is that pressure is exerted on the horse's owner by owners of horses stabled in the same barn, who are afraid their horses will acquire the vice as well. It is not known what causes horses to initiate this activity, but a common explanation is boredom, stress, frustration, and habit or addiction.[40] In a 4-year prospective study of 225 young Thoroughbreds, abnormal behavior affected 34.7% of the population.[41] Foals of dominant mares versus low-ranking mares, weaning by confinement in a stable or barn versus paddock-weaning, housing in barns versus grass after weaning, and feeding concentrates after weaning were significantly associated with an increased rate of development of abnormal behavior.[41] The results of this study suggest that simple changes in feeding, housing, and weaning practices could substantially lower the incidence of abnormal behavior in young horses.[41] The results of a postal survey on 2341 Swiss horses from 622 stables led to the hypothesis that preventing sterotypic disorders like crib biting should be based on housing and management pratices that allow tactile contact with other horses, daily free movement (paddock exercise), and high amounts of roughage with little or no concentrates.[42] Aerophagia may also be a pleasurable sensation to horses experiencing gastrointestinal discomfort.[27] Cribbing and wind sucking may be acquired vices or may be learned from other horses, although learning the vice has not been proved.[27,40] It has also been associated with genetic factors.[46]

Treatment

NONSURGICAL MANAGEMENT

Horses that are stalled continuously may limit or discontinue the behavior when they are moved to pasture.[41-44] Removing fixed objects that are grasped while cribbing may deter horses that have just learned the vice. Management changes rarely deter horses with established habits; some horses are capable of aerophagia without grasping a fixed object. Cribbing straps are perhaps the most common nonsurgical means used to control cribbing or windsucking. Leather straps with or without a ventrally located piece of articulating metal are placed snugly around the throatlatch of the horse to discourage contraction of the ventral neck muscles and arching of the neck. More severe straps have metal prongs that pierce the skin when the neck is flexed. Cribbing straps are adjusted to allow the horse to eat and breathe normally and are worn at all times except during exercise. Acupuncture may be useful for eliminating or decreasing cribbing and aerophagia.[47] Aversion (shock) therapy has also been used to treat horses that crib.[48]

SURGICAL MANAGEMENT

Three surgical procedures are described for the control of cribbing and aerophagia: the Forssell procedure, a modified Forssell procedure, and bilateral neurectomy of the ventral branch of the spinal accessory nerve.[43-45,49-53] The original procedure was developed by Dr. Forssell in 1920 and involves the surgical removal of portions of the sternomandibularis, sternothyrohyoideus, and omohyoideus muscles.[43-45,49-53] A modification of Dr. Forssell's original surgery, developed to produce a better cosmetic appearance, is the treatment of choice for wind sucking when used in combination with neurectomy.[43-45,49-53] The technique of bilateral neurectomy of the ventral branches of the spinal accessory nerves (eleventh cranial nerves) is modified from the technique described by Hamm.[51-55] The ventral branches provide motor innervation to the sternomandibularis muscles, which are the major muscles used by the horse to flex its neck. Because of the poor results achieved with this procedure alone, it is now described as a part of the modified Forssell procedure.[51-53]

Modified Forssell procedure

The horse under general anesthesia is placed in dorsal recumbency with the head tilted at a 30-degree angle to the horizon.[45,50-53] After surgical preparation, a 30-cm incision is made on the ventral midline of the neck through the skin, the edges of which are retracted laterally, exposing the ventral surface of the paired bellies of the omohyoideus and the sternothyrohyoideus and the cranial aspect of the sternomandibularis muscle. Careful attention to hemostasis minimizes staining of the areolar tissue and thus simplifies identification of the nerve. A plane of dissection is created on the medial aspect of the sternomandibularis muscle about 5 cm caudal to the musculotendinous junction. The ventral branch of the spinal accessory nerve is located on the dorsomedial aspect of this muscle

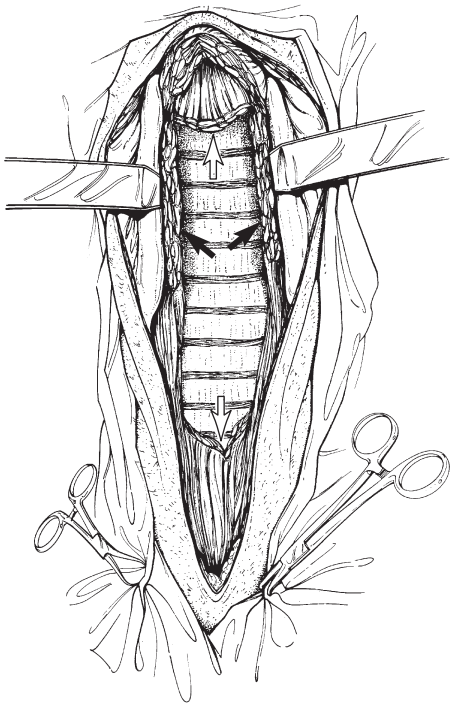

Figure 84-9. The modified Forssell procedure for cribbing is performed by a myectomy of the omohyoideus and sternothyrohyoideus muscles and by a neurectomy of the ventral branch of the spinal accessory nerve. *Black arrows,* Omohyoideus muscle stumps; *white arrows,* stumps of the sternothyrohyoideus muscles.

by carefully rolling the muscle belly laterally. Curved hemostatic forceps are placed under the nerve, which is elevated until a sizable portion of it can be exteriorized. Contraction of the sternomandibularis muscle after the nerve is pinched with hemostat forceps confirms isolation of the correct nerve. A large portion (6 to 12 cm) of nerve is removed. This same procedure is then repeated on the opposite side of the neck.

After the bilateral neurectomy, the myectomy is performed. A 30-cm (12-inch) section of the combined bellies of the omohyoideus and sternothyrohyoid muscles is removed (Figure 84-9). These muscles are identified in the cranial aspect of the incision, just ventral to the larynx, and they are freed from the larynx and fascial attachments to the linguofacial vein and thyroid gland. Curved scissors or other curved instruments are passed under the muscles to ensure inclusion of all parts of the muscle bellies, which are then transected. These muscles bellies are then grasped and "peeled" caudad. The areolar connective tissue connecting them to the trachea is sharply dissected. The omohyoideus muscles are sectioned obliquely and the sternohyoid bands transversely at the caudal aspect of the incision using scissors. The sternothyroid bands of the sternothyrohyoideus muscles are elevated from the trachea and removed. Special attention is given to hemostasis of the muscle stumps to prevent hematoma formation.

A Penrose drain is inserted in the wound and brought out through separate stab incisions on both ends of the incision. The subcutaneous tissues are closed with a simple-continuous suture pattern using No. 0 synthetic absorbable suture material, and the skin is closed with any suitable pattern and material. To help eliminate dead space, a stent bandage is sutured over the incision to provide constant pressure to the wound and to protect it during the initial phase of healing.

After surgery, the horse is confined to a stall or small paddock for 2 weeks. Unless it is soiled earlier, the stent bandage is removed 2 or 3 days postoperatively. The Penrose drain is removed 4 to 6 days later, followed by the skin sutures 10 to 14 days postoperatively. Exercise can be resumed after 21 days. Hematomas or seromas, although rarely encountered, can be managed conservatively or by drainage. Antibiotic therapy initiated preoperatively can be applied until the drain is removed. In a retrospective on 10 horses, there were slight modifications in the technique just described.[53] The sternothyrohyoideus and the omohyoideus muscles were transected rostral to the ventral aspect of the larynx at the basihyoid bone, and an Nd:YAG laser was used to perform the neurectomy.[53]

Prognosis

The modified Forssell procedure produces a good cosmetic appearance after surgery and rapid healing of the wound.[45] A review of 35 horses that underwent the modified Forssell procedure revealed that 57% had complete remission, 31% had improvement, and 12% had no improvement.[45] In a retrospective of 10 horses that had cribbing behavior and received surgery, none of the horses had cribbing behavior after surgery, and all horses had returned to their previous use with long-term outcome of 7 to 72 months.[53]

Calcinosis Circumscripta

Pathophysiology

Calcinosis circumscripta (tumoral calcinosis) is an uncommon condition that is identified more commonly in the young horse.[54-59] In a retrospective study on cutaneous nodular and proliferative lesions submitted to a veterinary diagnostic lab on horses, donkeys, and mules over a 3½-year period, 116 non-neoplastic nodular and proliferative lesions were identified and comprised 18% of all lesions submitted. Calcinosis circumscripta was identified as the most common non-neoplastic nodular lesion in horses up to 10 years of age.[59] Although the etiology of these lesions is unknown, it has been suggested that prolonged repeated trauma may be the cause.[55] These horses have normal serum calcium and phosphorus concentrations.[58] Histologically, these masses are calcified granular amorphous material that induces a fibrous reaction.[54-59] They are surrounded by a thick fibrous capsule that contains deposits of gritty material. They are typically located in the subcutaneous tissue close to joints or tendon sheaths. The lateral aspect of the stifle adjacent to the fibula is an area of predilection, although they also can be found on the dorsolateral aspect of the tarsus and around the neck and shoulder.[54-56] In 14 affected horses, the characteristic feature was a 3- to 12-cm dense subcutaneous mass located at the lateral aspect of the gaskin, adjacent to the stifle.[54-56]

Diagnosis

Rarely is lameness or pain associated with the condition. Typically, these horses present for cosmetic reasons and do not have any lameness.[58] Owners notice an unsightly lesion that may be increasing in size. Lesions are usually hard, are occasionally bilateral, are well circumscribed, do not involve the overlying

skin, and are tightly adherent to the underlying tissue. Radiographs demonstrate a circumscribed mineralized tissue lying close to the lateral aspect of the femorotibial joint with a density that is similar to that of cortical bone.

Treatment

Treatment is indicated only if the cosmetic appearance is unacceptable to the owner, or if there is lameness caused by the mass. When required, surgical excision is the recommended treatment option. Attention to aseptic technique is imperative, since some masses attach to the femorotibial joint capsule, which may be accidentally entered during surgery. Closing dead space during surgery and applying snug limb bandages or stent bandages will prevent postoperative seroma formation, because wound dehiscence and septic arthritis are potential complications.

Prognosis

For horses that are not treated, the mass remains and may not cause complications. A good result can be expected after surgical excision, and regrowth has not been reported. Wound healing is important if the joint capsule is entered during surgery.

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CHAPTER 85

Synovial and Osseous Infections

Dean W. Richardson and Benjamin J. Ahern

Musculoskeletal system infection remains one of the most challenging, frustrating, and expensive orthopedic problems commonly seen in the horse. Infections can involve bone and synovial structures such as joints, tendon sheaths, and bursae. Orthopedic infections can be separated into those that are acquired "naturally," such as from hematogenous or traumatic causes, and those that are induced iatrogenically, such as from surgical infections and therapeutic or diagnostic injections. Traumatic injuries of the lower limb in horses lead to infection of synovial structures and bones because there is so little protective soft tissue (muscle) over them. Fracture fixation in horses has a high incidence of infection because of the poor soft tissue coverage, meager vascularity of the lower limb, and the size and number of metal implants needed for stable fixation. Infections secondary to injections of synovial structures are common not only because of the frequency of the procedure performed but also because of the agents that are injected. For example, glucocorticoids are often administered and they impair the host's immune response.

The major change in therapy for musculoskeletal infection in the last 20 years or so has been the widespread acceptance of the principle that intensive local treatment is the key to success. This includes vigorous lavage, various means of delivering antimicrobials locally, and effective drainage.

SEPTIC ARTHRITIS/OSTEOMYELITIS IN FOALS

Neonatal foals with a failure of passive transfer of immunity are at high risk of bacteremia that can lead to localization of pathogens in a bone or synovial structure.[1-5] The anatomic locations where these infections are most likely to occur have been established.[6]

Types of Infections

S-Type Infections

The synovial membrane and fluid are the only structures involved in S-type infections. Foals with S-type arthritis tend to be the very young (less than 1 week old). The larger joints, such as the stifle or tarsocrural joints, tend to be affected most frequently, but any articular structure can be involved and multiple joints are commonly affected. The interphalangeal joints, distal tarsal joints, and axial skeleton joints are underrepresented.

Because these foals are neonates, the acute lameness exhibited is frequently presumed to be because they were stepped on by the mare. However, rarely is this a direct observation, and education of the owner and manager is essential to avoid delays in treatment.

E-Type Infections

The bone subjacent to the articular cartilage, technically the articular epiphyseal complex, which is a source of growth for the expanding epiphysis in the growing animal, is involved in E-type infections. These foals are weeks old and may have multiple joints involved. However, monoarticular E-type infections are common. These foals usually have a history of failure of passive transfer of immunity or development of some other disease, such as pneumonia or diarrhea.

These foals frequently have a history of very mild or intermittent lameness, with or without a fever, before an acute exacerbation coupled with noticeable joint effusion. Any joint can be infected, but the distal femur, talus, radius (proximal and distal), and tibia (proximal and distal) are commonly affected.

P-Type Infections

Primary infection of a long bone physis is termed a P-type infection. It may occur without joint involvement, but the infection can extend into the nearest joint. In most cases, this extension occurs at the attachment of the joint capsule. The peripheral portions of the physis are usually involved and at much more risk of dissecting into the adjacent joint. Foals with P-type osteomyelitis range in age from weeks to a few months. They may be seemingly healthy foals without a history of previous disease.

Clinical Signs

The clinical signs of S- and E-type infections are similar; lameness and effusion are consistent and fever is common. Radiographs of foals with E-type infection will reveal subchondral lysis, although lesions may be difficult to detect in some locations. Ultrasonographic evaluation may confirm both hypercellular synovial fluid and possibly an area of subchondral irregularity, but the combination of plain radiographs and synovial fluid analysis is usually adequate. Nucleated cell counts in

synovial fluid may be extremely high (greater than 50,000/μL), but lower cell counts in the single thousands do not preclude infection. Protein is less than 2 g/dL in the normal joint. In the septic joint, the total protein is elevated and may equal or even exceed serum values. The viscosity of synovial fluid from septic joints is usually reduced. The color of the fluid may vary from nearly normal to dark orange to red, or occasionally opaque pink or yellow. Cytology of septic joint fluid reveals more than 90%[7-9] neutrophils, the majority being degenerated. Fluid should be examined for the presence of intracellular bacteria, but they are only present in a minority of cases.[8]

Foals may have a neutrophilic leukocytosis, but the only consistent clinical laboratory abnormality is a hyperfibrinogenemia in E- and P-type infections. With bone involvement, the plasma fibrinogen is usually at least 900 mg/dL.[10]

Unlike in S- and E-type infections, foals with P-type infections present without synovial effusion but with periarticular edematous swelling. They are usually point sensitive to palpation over the affected area. These foals nearly always have a history of premonitory lameness, followed by acute severe lameness with a noticeable swelling if it involves the extremity. Although any growth plate in the body, including the vertebrae and ribs, may be involved, the distal physes of the third metacarpal/metatarsal bone (MCIII/MTIII), radius, and tibia are common sites. The P-type infection commonly involves only one site in an individual. The bacterial isolates from P-type osteomyelitis can include nearly any pathogen, but common organisms include *Streptococcus, Rhodococcus, Actinobacillus,* and Enterobacteriaceae (e.g., *Escherichia coli, Salmonella*).

SEPTIC ARTHRITIS/TENOSYNOVITIS/BURSITIS IN ADULT HORSES

The large majority of synovial infections in adult horses develop following an injection or a penetrating traumatic injury. Post–joint injection sepsis is predominantly caused by *Staphylococcus* spp. Penetrating wounds may have an enormous range of potential infecting organisms, including *Staphylococcus, Pseudomonas,* members of the Enterobacteriaceae family, and yeast and other fungi. The clinical signs are similar to those in foals, but early bone involvement is much less common. Exceptions to this are palmar/plantar and axial sesamoid osteitis in which hematogenous localization may lead to a bone abscess that eventually opens into the adjacent synovial structure, such as a joint or digital sheath.[11-14] Apparently because of hematogenous extension, septic bursitis has also been recognized in many sites, including the bicipital, calcaneal, atlanto-occipital, supraspinatus, olecranon, and other bursae.[15-22] In many of these septic bursae, the subsynovial bone appears to be the site of bacterial localization because radiographs often reveal bone lysis.

Diagnosis

Microbiology

Microbial culture is the gold standard for the diagnosis of sepsis. All synovial structures that are suspected of having an infection should have a culture and sensitivity performed before treatment with antimicrobials. It is important to emphasize, however, that valuable positive culture information is still commonly obtained when samples are taken after initiation of antimicrobials.[9] Culture of synovial fluid is more effective than a biopsy of the synovial lining.[23] Results of bacterial culture can be significantly improved when enrichment media or blood culture media are used.[7-9] In typical blood culture vials (Septi-Chek) an effort should be made to retrieve and culture 8 to 10 mL of fluid to maximize the chances of a positive culture. It is important to submit samples for fungal culture, especially if there is a history of intrasynovial injections or wounds, and to repeat culture submissions if results from the initial sample are negative. In foals with cases of hematogenous spread of infection it can be useful to culture a sample from the primary site of infection, or directly aspirate a suspected physis, because the synovial infection will likely result from the same pathogen and can guide antimicrobial choice.

The most common isolate from a retrospective study of 206 adult horses (older than 6 months) with synovial infection was *Staphylococcus aureus* (34.3%).[9] Earlier studies recognized that *S. aureus* is more common in cases of sepsis that develop after intra-articular injection or after surgery, and Enterobacteriaceae species are more common after traumatic wounds.[8]

Diagnostic Imaging

In S-type infections, the only radiographic evidence of infection will be soft tissue density associated with the marked effusion. In E-type infections, a subchondral lytic defect is generally apparent, although this may be subtle, especially in very young foals with incomplete endochondral ossification (Figure 85-1) P-type osteomyelitis is readily apparent through diagnostic imaging once the adjacent joint becomes septic, because there is generally a large lytic lesion extending to the outer cortex (Figure 85-2). Ultrasonographic guidance allows the accurate aspiration of a suspected site or evaluation of difficult to palpate articulations, such as the coxofemoral or scapulohumeral joints. In foals, the umbilical structures also should be examined. Scintigraphy may be useful for localization of a lesion, especially in the axial skeleton and proximal parts of the appendicular skeleton. It is important to remember that decreased rather than increased radiopharmaceutical uptake (photopenia) may be an indicator of a septic osteitis.[24] Labeled leukocytes[25] or other inflammation-seeking radiopharmaceuticals may prove to be of value.

Treatment

Initial Management

An essential component common to the successful treatment of any synovial infection is rapid recognition and aggressive therapy. It is imperative that articular cartilage be returned to a normal environment or there will be loss of matrix and irreversible loss of cartilage structure. If a significant amount of cartilage is lost, the chances of returning the horse to full athletic soundness drops drastically. Once a synovial sample has been obtained to confirm the diagnosis and for culture and sensitivity testing, treatment should be initiated. In a study of foals with osteomyelitis, positive results from synovial fluid cultures had 57.2% gram-positive and 42.8% gram-negative infectious agents.[26] Therefore, until culture results are available, suspected synovial infections should be treated with broad-spectrum antibiotics, such as a combination of penicillin and an aminoglycoside. In adult horses with postinjection sepsis, the probability of *Staphylococcus* infection is high, but even knowing that, broad-spectrum initial therapy is justified before culture results are available.

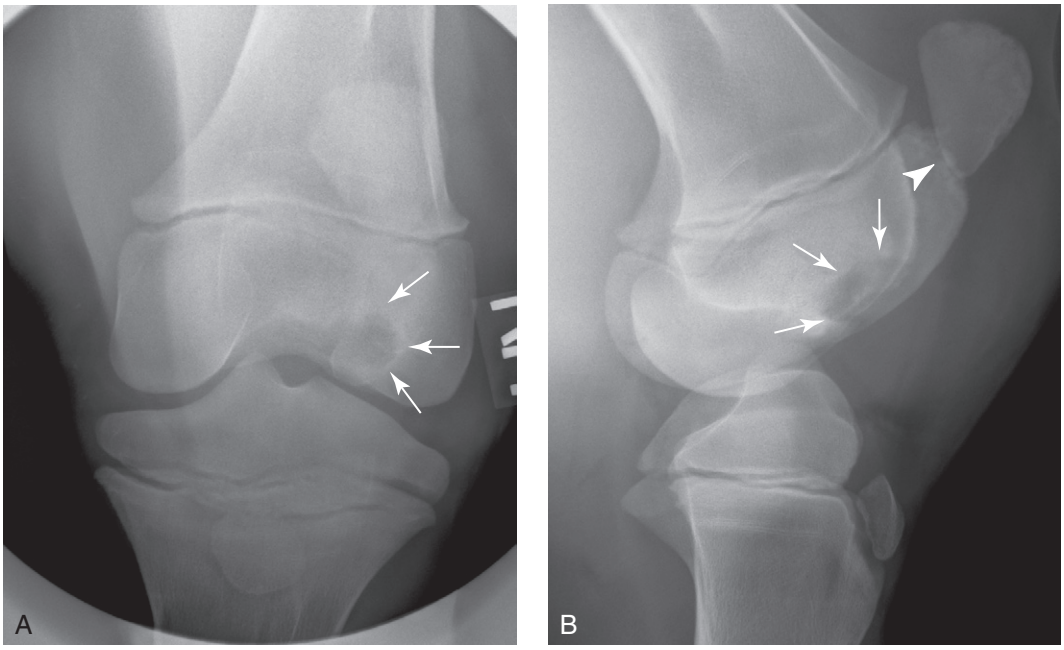

Figure 85-1. Craniocaudal **(A)** and lateromedial **(B)** projections of the stifle showing a large E-type osteomyelitis in the axial articular margin of the lateral femoral condyle *(arrows).* Note the normal irregular radiographic appearance of endochondral ossification of the femoral trochlear ridge *(arrowhead).*

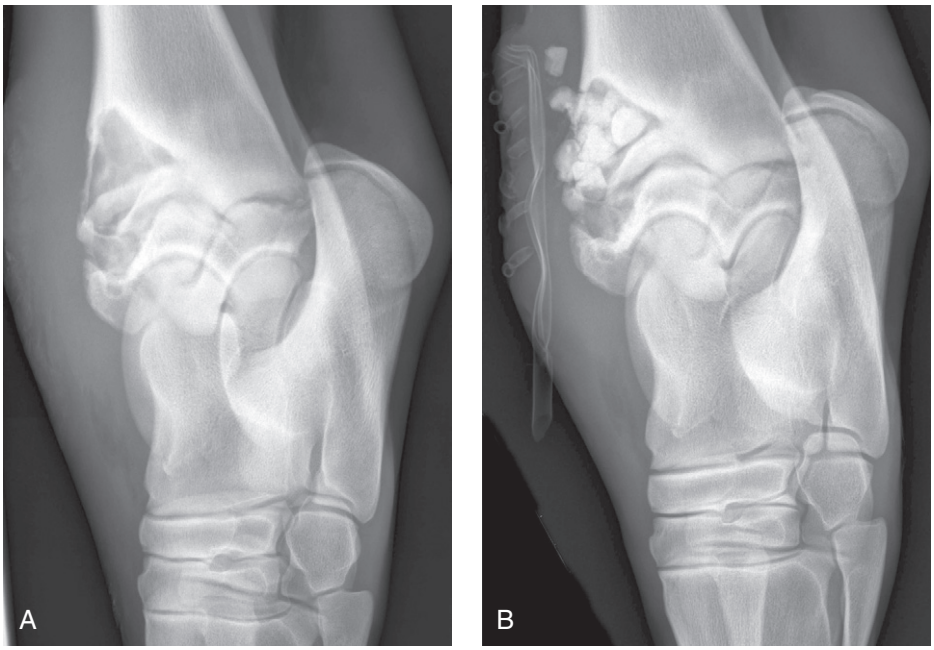

Figure 85-2. Dorsolateral plantaromedial oblique projections of the hock showing a large P-type osteomyelitis involving the distal medial tibial physis **(A).** The infected bone was débrided and packed with antibiotic-impregnated plaster of Paris beads **(B).** The foal healed well and became athletically sound.

Synovial Lavage

There is now widespread acceptance of the principle that septic synovial structures should be aggressively lavaged, early and often.[3,27-31] The physical removal of bacteria and inflammatory cells and mediators is still considered a mainstay of therapy, especially in the acute phase. There are many techniques in use by clinicians and little available proof that one technique or another is truly superior. The only likely truth is that early lavage is most valuable, and large volumes are superior. Probably the ideal technique for effective large-volume lavage is under anesthesia with the help of an arthroscope.[32] This allows not only rapid, accurate delivery of fluids but also the opportunity to remove fibrin clots, débride any unstable cartilage or bone, and evaluate the condition of the joint surfaces. If an arthroscopic examination is not possible for practical or economic reasons, a thorough lavage can still be performed under local or general anesthesia. If done standing, which is usually the case, sedation and local anesthesia facilitate the procedure. Local anesthesia can be achieved either by a regional

nerve block applied proximal to the joint, easy in a metacarpophalangeal (MCP) or interphalangeal joints, or by distending the joint with 2% mepivacaine and allowing it to sit for 5 to 10 minutes, followed by local infiltration of skin and subcutaneous tissues at the sites of needle insertion and drainage. Dean Richardson's preference is to use a large (18 to 16 gauge) needle for an ingress and a stab incision equivalent to an arthroscopic instrument portal for an egress. Another advantage of this approach is that an experienced arthroscopist can blindly remove fibrin clots through the stab incision with Schlesinger forceps or a similar instrument. Being able to unclog the egress site allows a much larger volume of fluid to be effectively flushed through the synovial cavity. With egress needles, synovial villi or fibrin obstruction can be a big annoyance. The stab incision can be left open and covered by a sterile bandage, or the skin can be closed with simple skin sutures if bandaging is difficult, such as in the stifle or shoulder.

Lavage should be performed with a balanced electrolyte solution. The addition of antiseptics such as povidone-iodine and chlorhexidine causes inflammatory changes at known bactericidal concentrations and has not been shown to have additional benefit for septic synovitis.[33-35]

Dimethyl sulfoxide (DMSO) scavenges free radicals and suppresses prostaglandin activity; it appears to be safe when added to lavage solutions.[36,37] However, there is no clear evidence of any advantage from using DMSO. Antibiotics should be injected into the synovial cavity following lavage to ensure maximum concentration.

Lavage of an infected synovial structure is initially performed daily in cases that do not fully respond immediately. One of most difficult decisions in the management of a septic synovial structure is when to discontinue lavage. Cell counts are of little value because the numbers are often unreliable in the frequently lavaged joint, and the lavage itself is moderately irritating, so cell numbers will not be normal. Improvement of lameness and swelling coupled with discontinuation of analgesic and anti-inflammatory drugs are the best indicators of when to discontinue regular lavage.

Antimicrobials

Antimicrobial therapy is ideally directed by culture and susceptibility results, but antibiotics with the broadest range of activity should be selected before sensitivity results are available. The route of administration of the antibiotics has evolved considerably and has shifted to focus on methods of local drug delivery. Systemic antibiotics are still commonly used, especially where there is a concurrent systemic infectious process or when local treatment is not possible. The systemic administration of a β-lactam and an aminoglycoside provide a broad spectrum of activity. Metronidazole can be added if an anerobic infection is suspected (e.g., *Bacteroides*). Local administration of antibiotics can achieve extremely high concentrations at the site of the infection and are a vital component of modern treatment of synovial sepsis.

LOCAL ANTIBIOTIC DELIVERY

Various techniques involving pumps have been used to deliver constant (or intermittent) infusion of antibiotics into joints.[38,39] The results of using constant infusion techniques seem to be similar to traditional intermittent dosing regimens using other techniques.

The choice of local antibiotic delivery method also may be influenced by the culture and susceptibility results. If an organism is susceptible to an aminoglycoside, the use of a technique yielding intermittent enormously high peak concentrations (i.e., perfusions or injections distal to a tourniquet) should be selected because the aminoglycosides are a clear example of a concentration-dependent antibiotic. A need to deliver time-dependent drugs suggests that the best methods to use would be indwelling pumps and other local infusion techniques.

The delivery of locally administered antimicrobials by intraosseous (IO), regional limb perfusion (RLP), intra-articular (IA), or antimicrobial infused biomaterials (e.g., collagen sponges, polymethyl methacrylate [PMMA], plaster of Paris, hydrogels, dextran gels) results in high concentrations of the antimicrobial at the site of infection. Intra-articular administration results in higher synovial concentrations than RLP, but there is no difference in the two techniques with regard to bone concentration of gentamicin.[40]

Regional limb perfusion

RLP with antimicrobials is possible in situations where a peripheral vessel is accessible and an effective tourniquet can be applied to isolate the infected region. The major disadvantage of regional perfusion in postoperative cases is the condition of the tissues and the need to prevent vascular damage near the surgical site. Some antibiotics, notably enrofloxacin, will induce significant vasculitis when used for regional perfusions.[41] Under heavy sedation, a tourniquet is placed above and, if feasible, below the area to be treated. The key is to have an accessible peripheral vein distal to the proximal tourniquet. If necessary, regional analgesia is used but many horses will stand with sedation alone. Although there are reports of success with indwelling catheters, the use of 25- to 27-gauge butterfly catheters seems to cause the least vascular trauma (Figure 85-3). Because treatments are usually repeated, it is imperative to keep the vessel in the best possible condition. The application of topical anti-inflammatories (e.g., liposomal encapsulated diclofenac) has been shown to decrease postinjection swelling and makes repeated treatments easier to perform.[42] Dosage varies, but around one third of a systemic dose diluted to about 30 mL is typical for the distal limb, 60 mL if the tourniquet is above the carpus or tarsus. With aminoglycosides, it is possible to use the entire daily systemic dose for regional perfusions. The injection is performed slowly over a period of about 5 to 10 minutes and the tourniquet is left in place for approximately 30 minutes, if possible. Subjectively, these very high doses given regionally seem to result in the best clinical responses, but more vascular irritation can be a complication with the more-concentrated doses of antimicrobials. The efficacy of RLP depends upon the function of the tourniquet, so it is essential to use a tough broad rubber tourniquet (see Figure 85-3) or a pneumatic tourniquet.[43] It is equally important to have the horse well sedated, and occasionally locally anesthetized, to prevent motion of the treated limb.[43] Care must be taken to ensure that the systemic dose of antibiotic is not exceeded in cases of polysynovial infections, especially in foals.

Intraosseous and intra-articular administration

Intraosseous and intra-articular antibiotic administration are used to maximize antibiotic levels at the desired site of action. Commercial intraosseous catheters as well as

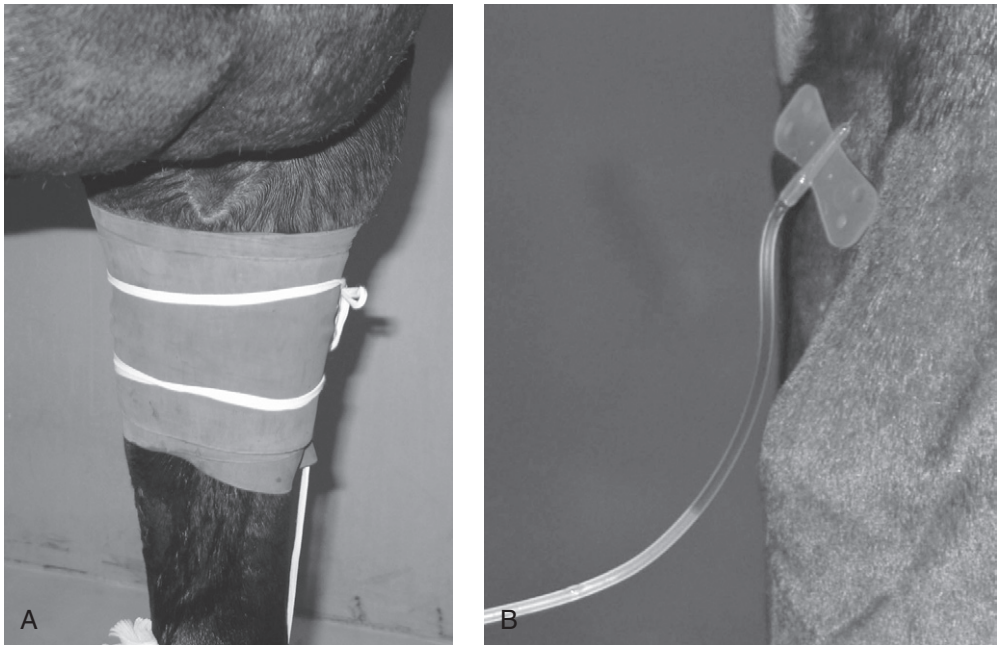

Figure 85-3. Two important technique tips for successful repeated regional limb perfusions are to use a wide rubber tourniquet **(A)** and a small-gauge butterfly catheter in a large vessel **(B).**

homemade cannulated screws can be inserted for repeated intraosseous treatments. A simple technique is to drill a 4-mm hole into the medullary cavity at the desired location. The male end of a Luer-tipped extension set will fit snugly into the hole, allowing direct injection. Recently, a severe complication was reported, where intraosseous perfusions of gentamicin caused toxic osteonecrosis and resulted in pathologic fracture of the perfused proximal phalanx.[44] Both intraosseous and intra-articular perfusions should ideally be performed with the region under tourniquet for 30 minutes to maximize tissue levels.

Antibiotic-impregnated biomaterials

Antibiotic-impregnated biomaterials (collagen sponges, PMMA, plaster of Paris, hydrogels) have not been used as commonly in septic synovial structures as they are in orthopedic infections, because synovial structures generally have motion and there is the risk of the implant moving and damaging adjacent structures such as cartilage. Collagen sponges have been successfully used in the tarsocrural joint in healthy horses without eliciting mechanical trauma or an inflammatory response[45] and have been used clinically with some success.[46] One potential disadvantage of collagen sponges is that they release the antibiotic very rapidly.[45] For P-type osteomyelitis or septic arthritis in foals, antibiotic plaster of Paris beads can deliver antibiotics efficiently[47,48] and have the distinct advantage of being absorbable, unlike PMMA beads (see Figure 85-2).

Débridement and Drainage

Débridement of infected synovial structures is often essential for the complete resolution of an infection. This is especially evident in cases with subchondral bone involvement or chronically infected structures. In these cases, mechanical débridement either arthroscopically or via open incision is used to reduce bacterial numbers to a point that antibiotics can be effective. Endoscopic procedures allow synovial débridement with motorized resectors, but it should be emphasized that motorized equipment must be used prudently. It is possible to damage the joint capsule with overly aggressive resection. Even severely thickened synovial tissue is usually soft and easily resected. If the surgeon is having to apply a lot of pressure to the handpiece, the resection may be too extensive. Open drainage can be achieved by performing multiple arthrotomies and subsequent daily lavage. However, experimentally, the success of arthroscopic lavage or arthrotomy with lavage were both similarly high.[49] Open drainage is probably best reserved for chronically infected structures that are resistant to initial vigorous attempts to treat with closed techniques. Endoscopic débridement would have to be considered the most appropriate technique for the great majority of infected synovial structures that require it.[32]

Analgesia

Septic synovial structures in horses can cause excruciating pain, which in turn can lead to contralateral limb laminitis or prolonged recumbency with attendant potential for decubital sores, weight loss, and respiratory and gastrointestinal problems. The response of individual horses to typical nonsteroidal anti-inflammatory drugs (NSAIDs), such as phenylbutazone and flunixin, can be remarkably variable. Adverse responses to NSAIDs seem equally inconsistent, but the clinician must always be looking for signs of NSAID toxicity in these patients. Oral ulcers, soft manure, and low plasma protein may be early signs. Increasing creatinine due to renal toxicity and colic because of gastric ulcers or right dorsal colitis are even more serious problems that can arise in horses receiving high doses of NSAIDs for extended periods. Horses that are debilitated by pain and that are neither drinking nor eating normally also are probably more vulnerable to NSAID-related problems. Omeprazole and sucralfate are usually both given in stressed, high-risk horses receiving long-term NSAIDs.

In horses that need more analgesia, opiates can be used judiciously. In a hospital setting, constant infusions of lidocaine or ketamine can be helpful. If the septic process is in the hind limbs, an epidural catheter for administration of opiates or detomidine, or both, can be extremely effective and reasonably economical.

Because clinical lameness and pain are the most reliable (and *important*) measures clinicians can use to assess the response of an individual horse to treatment, it is absolutely critical to remember that NSAIDs and other treatments could be masking that pain and giving a false impression of clinical success. A clinician should never discontinue any antimicrobials or efforts at drainage until the status of the horse is more fairly evaluated after use of analgesics and anti-inflammatories has been sharply reduced or discontinued.

Other Intrasynovial Treatments

Restoration of the synovial lining back to normalcy is the secondary goal after eradication of infection. Injections with hyaluronan, autologous conditioned serum, or platelet-rich plasma are all reasonable adjuncts to consider, even though there is little information regarding their efficacy. Hyaluronan is arguably the most plausible candidate since it is an essential moiety in normal synovial fluid and has few potential deleterious effects on host resistance and healing. The most difficult decision would be to inject a glucocorticoid to help resolve a persistent post-septic synovitis because of concern that a subdued, but not eradicated, infection might recrudesce under the potent anti-inflammatory actions of the steroid.

OSSEOUS INFECTIONS

Sequestrum

The equine limb is vulnerable to trauma because of its anatomy and how it is used. Kicks, lacerations, punctures, and any other penetrating wounds that infect bone are quite common, especially in horses at pasture. The poor soft tissue coverage over most of their limbs allows easy, direct contamination of bone, and the lack of muscle tissue eliminates a source of healthy blood supply. The consequence is often sequestrum formation. The stripping of periosteum and loss of blood supply leads to necrosis of a segment of bone that in turn is gradually walled off (i.e., sequestered) from the healthy intact bone. Reactive bone forms around the necrotic portion (the sequestrum) in a somewhat conical form termed the *involucrum*. The draining tract to the skin that is usually present connects with an opening to the sequestrum that is termed the *cloaca* (Figure 85-4).

The diagnosis of a sequestrum is usually only made after a wound over a bone has failed to heal normally or when it opens and drains following sutured closure. Radiographs may be unremarkable until the lysis around the sequestrum becomes advanced (Figure 85-5).

Treatment of bone sequestra does not always require surgical intervention. If there is minimal tissue reaction, minor discomfort, and a relatively small piece of sequestered bone, patience and several weeks of appropriate antimicrobials may result in complete resolution. However, it is always prudent to discuss with the owner or trainer that earlier surgical intervention might actually be less expensive. A sequestrectomy with accurate débridement can completely eliminate the infection quickly with minimal supportive therapy with antimicrobials. If possible, an Esmarch bandage or tourniquet should be placed proximal to the surgical site because it will make it much easier to identify normal and abnormal tissues during débridement. If the sequestrum and the tract leading to it are not obvious, it may be helpful to inject the tract with diluted methylene blue dye. This will make is easier to follow the tract and to completely excise the dyed tissues. It is not necessary to remove the entire involucrum unless cosmetic "debulking" of the area is an issue. It is necessary, however, to fully open the cloaca to expose the sequestrum and thoroughly scrape out all infected and reactive bone and exudate. Local antimicrobial delivery in conjunction with a cancellous bone graft, plaster of Paris, or other

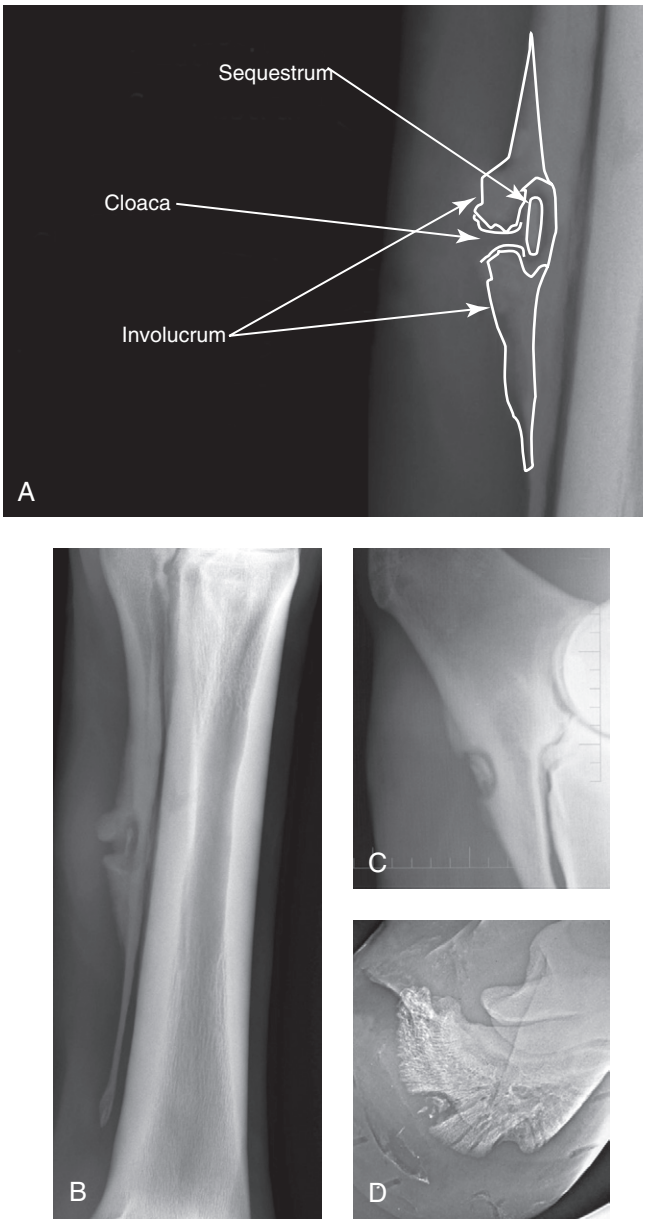

Figure 85-4. A-C, Infected sequestra have typical radiographic appearances including surrounding reactive bone (involucrum) and an opening that typically forms a tract to the skin surface (cloaca). **D,** In some anatomic locations like the distal phalanx, the reactive bone is less likely to be evident.

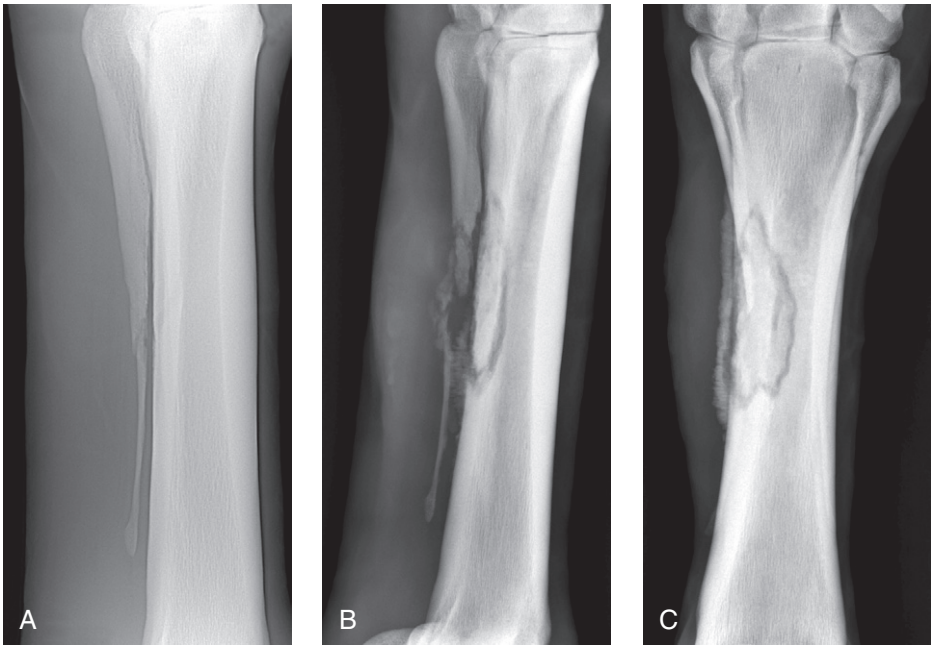

Figure 85-5. A, Radiograph taken 1 day after a kick wound to the metacarpus. There is clearly a minimally displaced small metacarpal fracture, but nothing more is evident. **B** and **C,** Approximately 2 weeks later the sequestrum of MCIII is evident.

degradable delivery device may all be used in cases where there is a large postdébridement defect. With large sequestra of the small metacarpal or metatarsal bones, an antibiotic-soaked collagen sponge is placed axially to separate the bone defect from the suspensory ligament and flexor tendons before filling the defect created by the débridement (Figure 85-6). Simple drainage with a Penrose drain also should be strongly considered if a significant dead space is present. With proper débridement, neither intensive local nor long-term antimicrobials are usually necessary.

Postoperative Infection

With the advent of arthroscopy completely replacing traditional open arthrotomies, infections following joint surgery are rare. Unfortunately, the same cannot be said of major orthopedic surgical interventions in horses, where infection has proved to be the most important and disappointingly common complication following internal fixation.[50]

Etiology

A surgical infection occurs when bacteria (or much more rarely, fungi) overwhelm the host's immune response. It has long been established that any foreign material in a surgical wound greatly increases the risk for a clinical infection, primarily because the bacteria can form biofilms on their surfaces that protect them from host defense. In equine fracture repair, surgeons are often forced to use numerous and large implants for mechanical reasons, enhancing the chances of infection considerably. The number of bacteria that enter the wound also affect the probability of surgical infection. In horses, plating procedures usually involve extensive open surgical approaches that expose the site and implants to the ambient air for long periods. Efforts have been made to develop minimally invasive plate fixation techniques in horses,[51] but not all fractures are amenable to these types of repair. There is no doubt that the risks of common joint surgeries have been diminished by the ubiquitous use of arthroscopy. Presumably this is because of the minimally invasive nature of arthroscopy combined with the constant mechanical cleaning (fluid delivery) during the procedure. It still remains to be proved if minimally invasive bone plating techniques reduce the incidence of surgical infection.

Prevention

Optimal aseptic technique should be the standard for major orthopedic procedures in any species because of the inherent risks. Even with excellent aseptic technique, however, the risks are high enough that antimicrobial prophylaxis is justified, usually using multiple delivery methods in the same patient. Preoperative antimicrobials ideally should be administered intravenously shortly before (or during) the beginning of the procedure to maximize plasma concentrations. The choice for most surgeons is a broad-spectrum combination of a penicillin and an aminoglycoside. During the procedure, tissues should regularly be rinsed with antibiotic-containing sterile fluids. Because of the volume used, a relatively inexpensive selection of drugs that are not commonly used systemically (e.g., bacitracin, polymyxin B, neomycin) are often chosen for lavage solutions. The use of hyperimmune plasma for intraoperative lavage has also been proposed.[45] In plating procedures, the voids in the surface of the plate next to the screw heads can be filled with antibiotic-impregnated PMMA (AIPMMA) as can the voids in the undercuts of specific plates—limited-contact dynamic compression plate (LC-DCP), locking compression plate (LCP)—if open reduction and internal fixation is performed. A half-dose (20 g) of a sterile prepackaged PMMA (e.g., Surgical Simplex P) can be mixed with 5 mL of 250 mg/mL amikacin sulfate solution. The components should be mixed quickly in a sterile

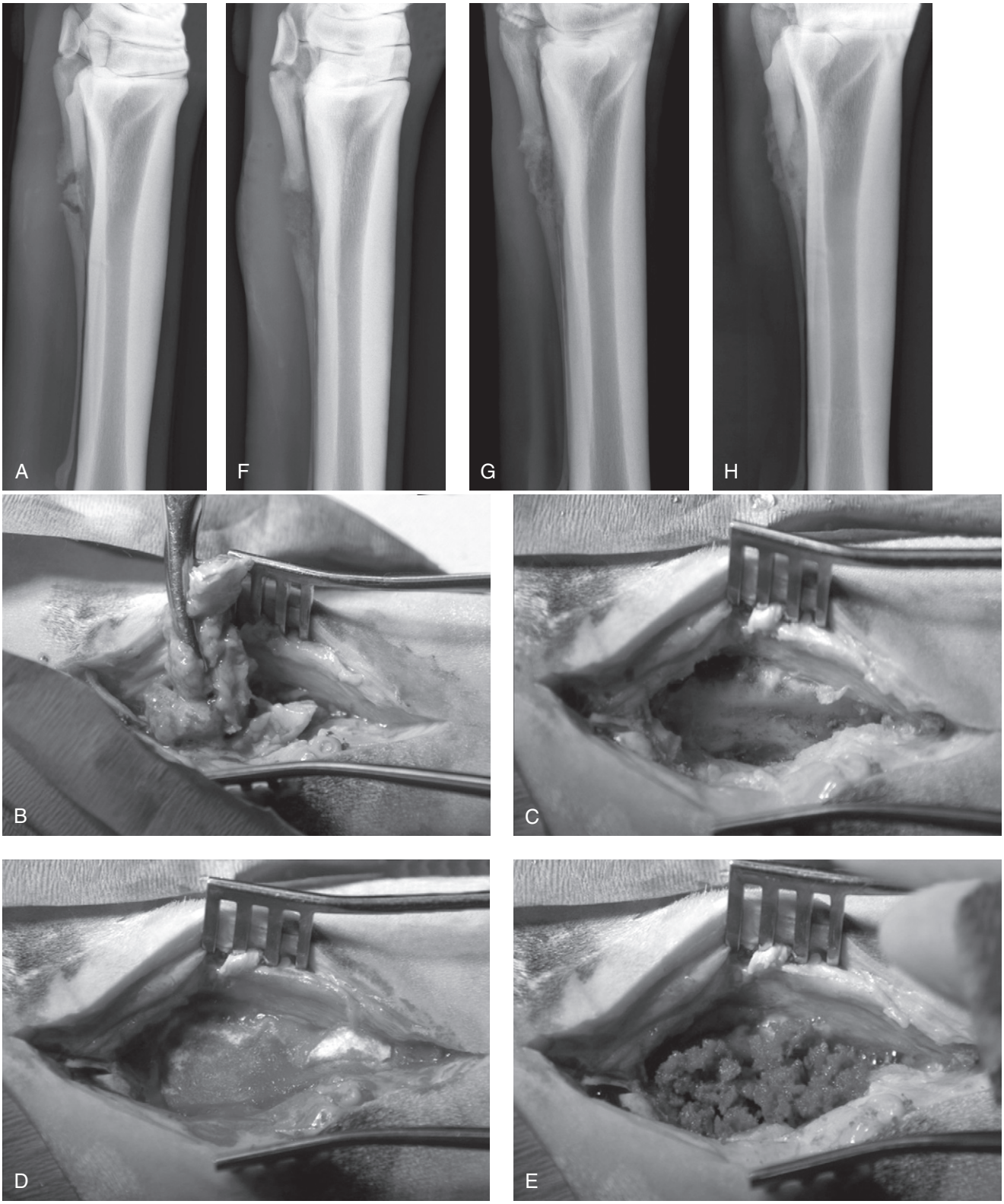

Figure 85-6. A, An infected large sequestrum of the proximal MTII. **B,** A simple linear incision made over the lesion allows débridement of the infected tissue to the point where it is visibly clean **(C). D,** An antibiotic-soaked collagen sponge is placed axially. **E,** A combination of cancellous bone and small chips of antibiotic soaked plaster of Paris fill the defect. **F,** Follow-up radiographs at 1 month. **G,** Follow-up radiographs at 3 months. **H,** Follow-up radiographs at 6 months.

container and aspirated into a 60 mL dose syringe while it is still very liquid. As soon as the mass becomes slightly doughy, it can be squeezed out of the dose syringe into the screw hole voids and around the edges of the plate (Figure 85-7). Again, this must be done quickly, especially if the ambient temperature is high, because the AIPMMA will harden rapidly. An injection needle or a small pin is used to remove any cement that gets into the screw head recesses. Meticulous attention should be given to avoid getting AIPMMA into any fracture gaps. A premade AIPMMA with tobramycin is also available. Additional useful antibiotics that have been used include gentamicin, imipenem, vancomycin, ceftiofur, cefazolin, daptomycin, teicoplanin, enrofloxacin, ticarcillin/clavulanic acid, and metronidazole. Heat-sensitive drugs such as chloramphenicol should not be used, and most penicillins are not ideal. A large range of antibiotic concentrations have been used, but clinical success has been seen with weight/weight ratios of 1:10 to 1:5 (antimicrobial/substrate) (Table 85-1). A larger proportion of antimicrobial placed in the cement presumably has more effect

Figure 85-7. Antibiotic-impregnated PMMA can be mixed, placed in a 60-mL dose syringe, and injected while still soft and doughy into any "open" spaces in a bone plate.

TABLE 85-1. A Partial Listing of Commonly Used Biomaterials for Local Antimicrobial Delivery

Biomaterial	Brand Name	Antibiotic	Concentration	Notes
BIODEGRADABLE				
Collagen sponge	Ultrafoam Collagen Sponge; Davol Inc., Warwick, RI	Gentamicin	130 mg/joint[43,44]	Short elution profile, quickly resorbed. Commercially available. Easy to use.
Plaster of Paris (calcium sulfate hemihydrate)		Gentamicin Tobramycin	25-60 mg/g[46] 100 mg/g	*Very* inexpensive. Beads are best made before surgery; slow to harden. No reasonably priced commercial product ("homemade," requires sterilization)
Hydroxyapatite	BoneSource; Stryker Corp., Kalamazoo, MI	Gentamicin	50 mg/g[52]	Expensive commercial bone replacement product.
		Amikacin Vancomycin	62.5-125 mg/g[52] 100 mg/g[53]	
		Ceftiofur	50-100 mg/g[52]	Poor kinetics for cefazolin.
Dextran gel	R-Gel; Royer Animal Health, Frederick, MD	Amikacin Amikacin + clindamycin Vancomycin	60 mg/mL 50 mg/mL + 25 mg/mL 25 mg/mL	Easy to use gel formulation. Elution 7-10 days. Gel degraded in 3-4 weeks. Commercial product.
NONBIODEGRADABLE				
Polymethyl methacrylate	Simplex P; Stryker Corp., Kalamazoo MI Palacos R, Zimmer Inc., Warsaw IN	Gentamicin	50-100 mg/g[54]	Easy to use sterile commercial product. Very exothermic curing so only heat stable antimicrobials can be used. Decades of successful clinical use.
		Ceftiofur Vancomycin Amikacin Cefazolin Tobramycin	50 mg/g 25 mg/g 50-125 mg/g 50-200 mg/g 100-250 mg/g	

on the mechanical properties of the resulting cement, but that is not a concern when it is used for drug delivery.

Finally, during or following wound closure of major internal fixation in the distal limb, a regional limb perfusion with amikacin should be considered. The "extra" 20 to 30 minutes is a minimal concern, because the perfusion can be done while a lower limb cast is being applied. Time is also often spent applying a special shoe to the contralateral foot.

The postoperative duration of prophylactic antimicrobial therapy depends on multiple factors, including the presence of drains, difficulty encountered in closing the incision, concerns about incisional integrity, and the condition of the adjacent skin or nearby wounds. In closed fractures without complicating factors, however, there is little evidence to support antimicrobials being given for more than 48 to 72 hours.

Meticulous management of the incision, especially in the first few days following surgery should include changing bandages in clean areas and keeping the wound covered with sterile dressings. Skin sutures should be removed at the appropriate time (usually 10 to 14 days postoperatively) after carefully cleaning the sutures and skin surface. It is particularly important in incisions with little underlying soft tissues covering a plate that sutures be correctly removed, not leaving fragments of potentially contaminated material beneath the skin. If a cast is going to be left in place over an incision for more than 2 weeks, a monofilament absorbable material for skin closure should be strongly considered, especially if skin sutures are being placed under tension or in complex patterns (mattress, near-far-far-near) that will be very difficult to remove when they pull through the skin surface.

Diagnosis

Early subtle signs are invariably present on every orthopedic case that develops an infection. Early recognition of the clinical signs of a developing septic process can prove essential to successful management. Experience with the degree of postoperative comfort to be expected from each orthopedic procedure is useful because an early sign of a developing infection is lameness not in line with case expectations. An adequately stabilized fracture or arthrodesis without infection is nearly always fully weight bearing and comfortable to palpation within 36 to 48 hours. Any prolonged discomfort that is resistant to reasonable analgesic efforts should raise the suspicion of infection. The noninfected surgical site should heal evenly, rapidly, and cleanly. If expected postsurgical swelling persists or increases, it should be further investigated, as should any type of drainage or a fluid pocket palpable at the surgical site. Although a cast precludes direct palpation of a surgical incision, careful assessment of the cast can detect heat, and drainage can be seen or possibly smelled if there is a problem. A regular inspection routine of any horse wearing a cast should include picking up the contralateral limb and walking the horse a short distance each day. Any early decrease in comfort is a reason to consider early removal of the cast and inspection of the incision.

During the postoperative period an easy and reliable indicator of potential infection is pyrexia. Any elevated temperature (higher than 38.6° C [101.5° F]) that cannot be readily and clearly attributed to another cause should be assumed to be a surgical site infection until proved otherwise (see Chapter 7). Even minor fevers should be considered important in a horse receiving substantial NSAID therapy. All clinical signs inconsistent with expectations should be investigated aggressively. It is vastly more cost-effective to aggressively investigate and treat a suspected case of infection than it is to wait for overwhelming proof and commence treatment at that stage. Monitoring of plasma fibrinogen is a useful means of monitoring case progression. The absolute number is often not conclusive; however, serial monitoring can be very useful in the early detection of infection. Leukocytosis is generally an unreliable indicator of infection in horses because they can have normal white cell counts. Diagnostic aids are very useful in detecting orthopedic infections. Ultrasonography performed by an experienced operator is a sensitive means of detecting infection. The accumulation of exudate adjacent to a bone or implant can be identified and early accurate aspirates obtained for culture and sensitivity testing. Radiography can be useful; however, the information gained from the images in the acute stages is often limited to increased soft tissue swelling and separation of tissue planes. Later signs are bony lysis developing adjacent to metallic implants and periosteal proliferation unassociated with fracture healing (Figure 85-8). A major benefit of early radiographic evaluation of the operated site is to ensure the structural stability of the construct. Culture and sensitivity testing of any subcutaneous fluid pocket or a draining tract is mandatory for directed treatment of an orthopedic infection. All sampling should be performed aseptically and using ultrasound guidance if necessary. Aspirates collected into culture media or swabs of draining tracts should be performed using previously outlined techniques to maximize culture efficiency. Culturing the deeper portions of the infected region may prevent erroneous culture results. If any implants are removed, they should also be submitted for culture.

Treatment

A major factor in the improved handling of orthopedic cases over recent years has been a shift to concentrated local delivery of antibiotics to the surgical site. Obvious benefits of these local techniques over systemic administration include extremely high levels of antimicrobials at the site of infection, avoidance of potential side effects, and significant financial savings. Many of these techniques are the same as those used for prophylaxis, including local antibiotic elution devices and regional vascular perfusions. Once an infection has become established, in addition to the use of local antibiotics, basic principles of treating infection must still be followed. If possible, drainage of the infected tissues should always be considered. This may involve an ultrasound-guided incision into a fluid pocket or opening of the most dependent portion of the incision.

Local techniques to treat an established infection can be used to control an infection until there is sufficient healing of the fracture/arthrodesis that implants can be safely removed. Removal of an infected implant will help resolve even the most persistent infections.

If there is the unfortunate combination of an inadequately stable internal fixation and infection, the best options, if finances allow, is usually to either replace the implants or remove them and manage the fracture/arthrodesis with a transfixation cast (or external fixator). It is feasible to remove an infected plate and vigorously débride the underlying bone, adjacent tissues, and fracture site. While the débridement is being performed, the plate(s) can be cleaned and autoclaved and reimplanted with fresh screws in new holes. If the situation

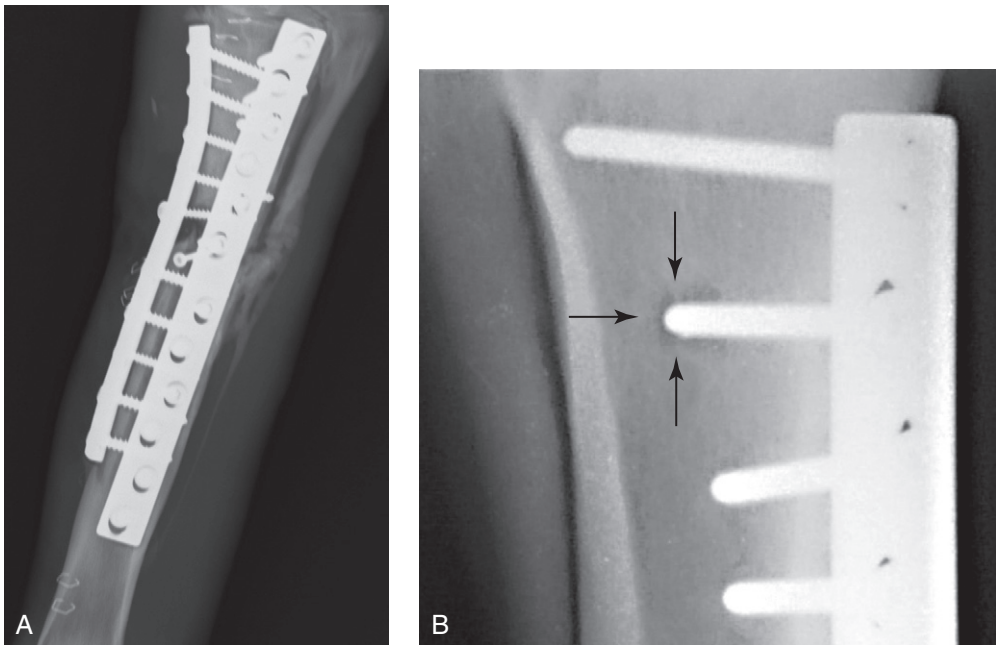

Figure 85-8. A, Typical radiographic signs of infected implants include lysis and proliferation at the fracture site; **B,** or more subtle signs such as lysis along a single screw *(arrows)*.

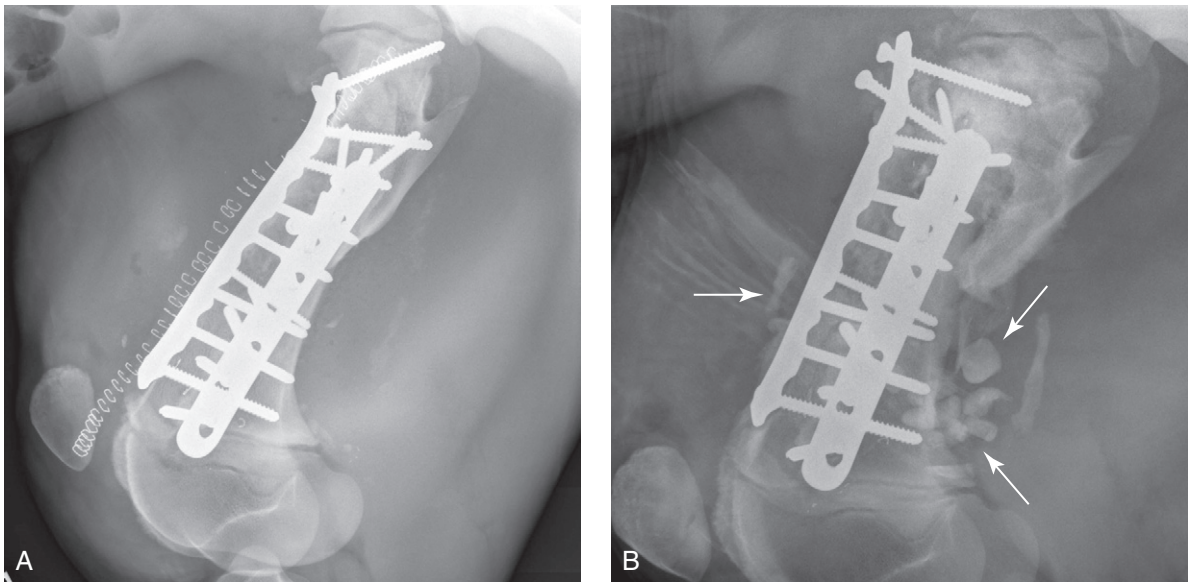

Figure 85-9. A, A severely contaminated open 4-day-old fracture of the femur repaired with two locking plates and abundant antibiotic-impregnated polymethyl methacrylate (AIPMMA) along the plates. As expected, some infection did develop. **B,** The wound was drained and packed multiple times with AIPMMA beads *(arrows)*. The fracture healed. Normal femoral longitudinal growth followed, and the implants are still in place 4 years later.

allows it, dynamic compression or a tension device can be used to restabilize the site. The empty screw holes can be filled with antibiotic-impregnated collagen, antibiotic-impregnated dextran gel (R-gel), antibiotic-impregnated plaster of Paris or other absorbable antibiotic-eluting materials. Drainage should always be considered; leaving a few centimeters open at the bottom of an incision or even using surgical drains may help.

Parenteral antimicrobial therapy alone nearly always fails in seriously infected internal fixation cases. If the internal fixation is stable, the treatment strategy involves drainage, local instillation of correctly selected antimicrobial beads if feasible (Figure 85-9), and repeated regional limb perfusions. The implantation of eluting materials can usually be performed in the standing horse under local anesthesia. Although AIPMMA has the disadvantage of being nonabsorbable, PMMA has very good elution properties and excellent biocompatibility, and it is very easy to form beads of appropriate shape for insertion. AIPMMA beads have widely been used on a "string" to facilitate later removal, but individually placed fusiform beads are easier to insert in a standing patient alongside an infected plate or in pockets surrounding an infected implant. The beads can usually be removed without too much difficulty when the plate is removed later.

Although it is possible for the depleted beads themselves to later serve as a nidus for an infection, many horses have lived for a decade or more with depleted AIPMMA beads inertly in place.

The regional limb perfusions ideally should be performed daily for as long as possible, using the highest possible dose, which is the systemic daily dose. Using very small gauge (25- to 27-gauge) butterfly needles for injection, large vessels (e.g., accessory cephalic and saphenous veins), immediate compression of the injection site to minimize bleeding and extravasation, application of topical NSAIDs and bandaging, it may be practical to treat with maximum vigor for weeks. Regional limb perfusions can be safely and repeatedly performed in a horse wearing a lower limb cast. As emphasized earlier in this chapter, an appropriate tourniquet and heavy sedation to prevent movement of the perfused limb both improve efficacy.

Prognosis

The prognosis for horses with orthopedic infections has substantially improved with the routine use of intensive local therapy. But the cost of treatment is enormous, and there may still be significant long-term adverse consequences for a horse, even with successful clinical resolution of the infection.

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CHAPTER 86

Angular Limb Deformities

Jörg A. Auer

The surgical techniques used for the management of angular limb deformities (ALDs) in foals are well established.[1-10] However, which technique to use and when remains controversial. At one time, most foals showing some degree of angular deviation were surgically corrected, but a more differentiated approach is presently applied.[11]

DEFINITION

Animals with ALD present with either a valgus deformity (lateral deviation of the limb distal to the location of the deformity) or a varus deformity (medial deviation of the limb distal to the location of the problem) (Figure 86-1). Either type of deviation is usually associated with a certain degree of axial rotation. In foals with valgus deformity, this is displayed as an outward rotation (splay foot), and in cases of varus deformity, as a medial rotation of the feet (pigeon toes). In most cases, these deformities are initially merely postural, through a rotation of the limb axis toward either the outside or the inside, respectively. With time, however, the bone adapts to abnormal loading, according to Wolff's law, and differential metaphyseal growth results in the development of a permanent rotational deformity.[8,12]

ANATOMY

Maturation of the skeleton from the primordial or precursor cartilage to bone is a very complicated process and occurs mainly during the later stages of gestation. Radiographs taken of the carpal and tarsal regions of aborted fetuses at 230 days of gestation reveal only partial ossification of the diaphyses and metaphyses of the long bones as well as of the calcaneus.[10] Other bones are not ossified at all at that time. For instance, intra-articular injections of contrast medium provide outlines of the bone templates in their normal shapes, although they consist of precursor cartilage only. Ossification of these structures begins in the center and expands spherically in all directions to the periphery.[10,13] At about 260 days of gestation, ossification centers are noticed in the distal radial epiphysis, distal tibial epiphysis, talus, and accessory carpal bone. Later,

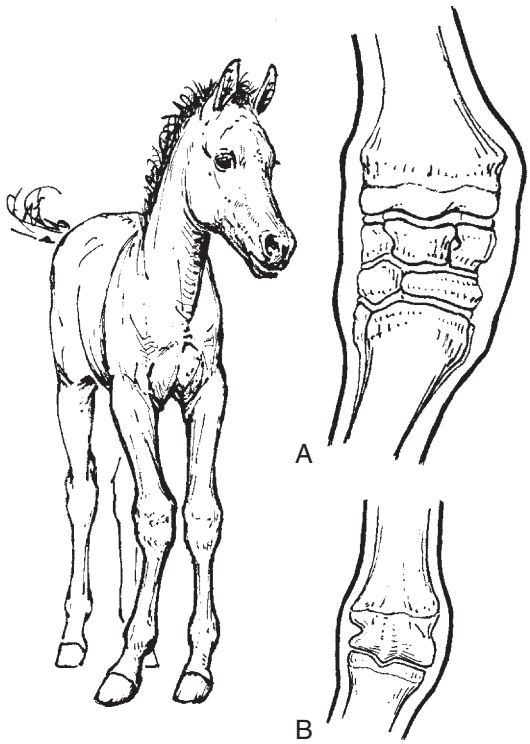

Figure 86-1. Drawing of a foal with a valgus deformity in the right carpal region **(A)** and a varus deformity in the left metacarpophalangeal region **(B).**

ossification centers in the intermediate, radial, and third carpal bones, and the central, third, and fourth tarsal bones develop. At around 290 days, the proximal epiphyses of the third metacarpus/third metatarsus (MCIII/MTIII) appear, and they unite shortly thereafter with the metaphyses of these bones (Figure 86-2). At about 300 days of gestation, all bones of the carpus and tarsus are visible radiographically. The ulnar styloid process, which is the last ossification center to appear, is still not seen at this time. During the remaining days of gestation, ossification progresses toward the periphery, and the bones acquire their final shape. At birth, the edges of these bones are still somewhat rounded, but the "radiographic joint spaces" are within normal limits (Figure 86-3). These spaces consist of two layers of cartilage in addition to the actual joint space.[6,14,15]

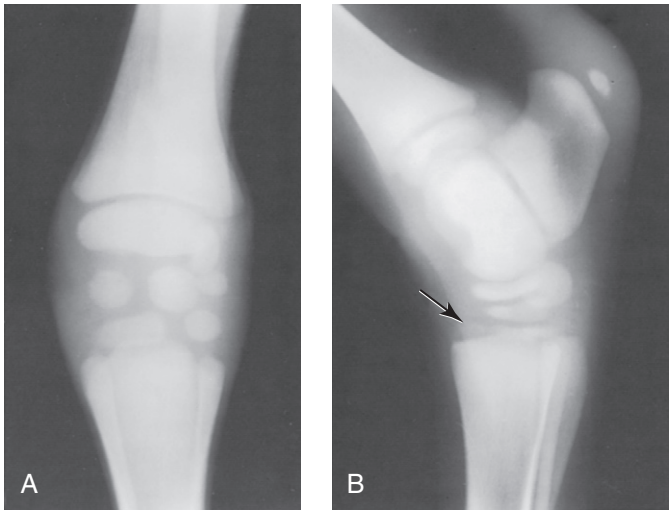

Figure 86-2. Dorsopalmar (DP) radiograph of a carpal region and lateromedial (LM) radiograph of a tarsal region of a premature foal. **A,** The DP view of the carpal region at 2 days of age shows incomplete ossification of the carpal bones. **B,** The LM view of the tarsal region shows inadequate ossification of the tarsal bones, especially the third and the central tarsal bones. Note the ununited proximal epiphysis of MTIII *(arrow)*.

Longitudinal Bone Growth

The vast majority of longitudinal growth in the long bones occurs at the level of the physis. Some growth is attributed to the epiphysis, growing toward both the articular cartilage and the physis. However, the vast majority of growth occurs in the metaphyseal region of the physis. The exact mechanisms responsible for this bone growth are not completely understood, but strides toward solving this mystery are being made with the help of molecular biology techniques.[16,17] In mice, it was found that a feedback loop exists in the growth plate centered on the following molecules: parathyroid hormone–related protein (PTHrP), PTH/PTHrP receptors (PTHrPR) and Indian hedgehog (Ihh).[18] The feedback loop is regulated via the periosteum/perichondrium through the signaling molecules Patched1 (Ptc1), Gli, and Smoothend (Smo). If Ihh secretion is increased, PTHrP molecules are upregulated and inhibit differentiation of proliferating chondrocytes into hypertrophic chondrocytes, thus promoting longitudinal growth. Since Ihh and PTHrP are not expressed in the same vicinity, and molecule diffusion through the extracellular matrix is limited, intermediates, such as Ptc1, transforming growth factor-β_2 (TGF-β_2), bone morphogenetic proteins (BMPs), and fibroblast growth factor (FGF), that are located in the perichondrium participate in this regulatory process.[18] Once these mechanisms are understood in the horse, an explanation for the disproportionate long bone growth across a physis may become clear and specific molecular therapy may be developed.

The following is clear. Ideally, bone growth should occur evenly across the entire physis; immediately after birth, growth is most active; and each physeal region has an approximate time span during which new bone growth occurs. Closure of the physes of the different long bones occurs at predictable time points. Therefore, if manipulations to correct growth are to be effective, the closure time of the physes has to be considered.

ETIOLOGY

A variety of etiologies are responsible for the development of ALD and can be grouped into two main categories: perinatal factors, which include conditions that are present either during

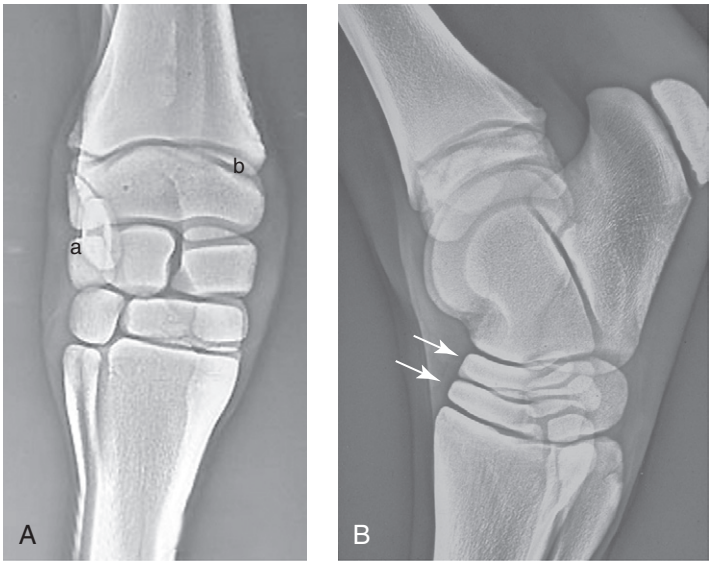

Figure 86-3. Normal ossification of the carpal and tarsal regions at the time of birth. **A,** Dorsopalmar radiograph of the carpal region. All the bones are ossified adequately, and the ulnar styloid process *(a)* is visible. The rough surface at the medial distal metaphysis of the radius *(b)* represents active endochondral ossification and is normal at that age. **B,** Lateromedial radiograph of the tarsal region showing adequate ossification of the central and third tarsal bones *(arrows)*. It is important that the ossification process proceed to the level of the proximal aspect of MTIII. These two radiographic views are the most relevant to evaluate ossification at the time of birth.

the later phases of gestation or in the immediate perinatal period, and developmental factors, which influence limb axes at a later stage.

ALD may originate at different locations of the appendicular skeleton: within the cuboidal bones of the carpus or tarsus, in the epiphyseal region of the long bones (epiphyses, physes, and metaphyses), and occasionally in diaphyses of the long bones. Most frequently, the deviation develops through disproportionate growth at the level of the metaphyseal growth plates. Physeal trauma, such as Salter-Harris type V or VI fractures, can cause local retardation of growth at the medial or lateral aspect of the bone and result in the development of ALD.[18,19] Compensatory ALD can occur in remote sites such as the proximal phalanx of the affected limb because of prolonged disproportionate loading of a growth plate distad to the deviation. Such compensatory deviations may straighten the limb axis, but when the foal ambulates, the joint involved rotates, because the joint is not oriented at a right angle to the long axis of the limb and the ground.

Perinatal Factors

Incomplete Ossification

After a normal gestation period, a foal should be born with adequate ossification of the carpal and tarsal bones (see Figure 86-3). A variety of events affecting the mare, including placentitis during pregnancy, severe metabolic disease over a prolonged period, heavy parasite infestation, and colic, may jeopardize the intrauterine environment of the foal and result in incomplete ossification at the time of birth.[10] Premature birth is usually associated with the same problem. Because the placenta of a mare does not provide as intricate a blood exchange between the fetus and the mare as it does in humans and dogs, normal intrauterine development of more than one fetus at the same time is not possible. Twin foals always exhibit incomplete ossification at birth. These immature foals do not mature properly and have a poor prognosis as athletes. Therefore abortion of one of the twins during the early embryonic stages is standard.

Because most foals are born with a certain degree of angular limb deformity, they initially load their joints unevenly. The uneven pressure on the relatively thick layer of articular and precursor cartilage may deform these soft structures, especially in a dysmature foal. Incomplete ossification per se does not represent an ALD, but the uneven loading of the dysmature skeleton leads to it. Once ossification reaches the periphery, the precursor cartilage ossifies in an abnormal configuration, resulting in a permanent ALD. In severely dysmature foals, osteochondral fractures may occur, which render the foal permanently lame because of osteoarthritis (Figure 86-4).[8] This is especially true in the rear limb, where weight-bearing forces must change direction in the tarsal region from a cranioproximal-caudodistal direction to a vertical one. The most affected region is the dorsal aspect of the small tarsal bones. If these bones are incompletely ossified, loading causes thinning of the soft precursor cartilage and eventually leads to a pathologic fracture and dislocation of a dorsal fragment.[6,8] This problem results in a partial collapse of the dorsal aspect of the tarsus and tilting of the proximal limb in a cranial direction (Figure 86-5). Affected foals do not trot but rather "bunny hop" with their rear limbs. In chronic cases, a secondary flexural deformity of the forelimbs may result because of chronic overload.

If collapse of the cartilage template of cuboidal bones in dysmature foals is left untreated, the deformations worsen and

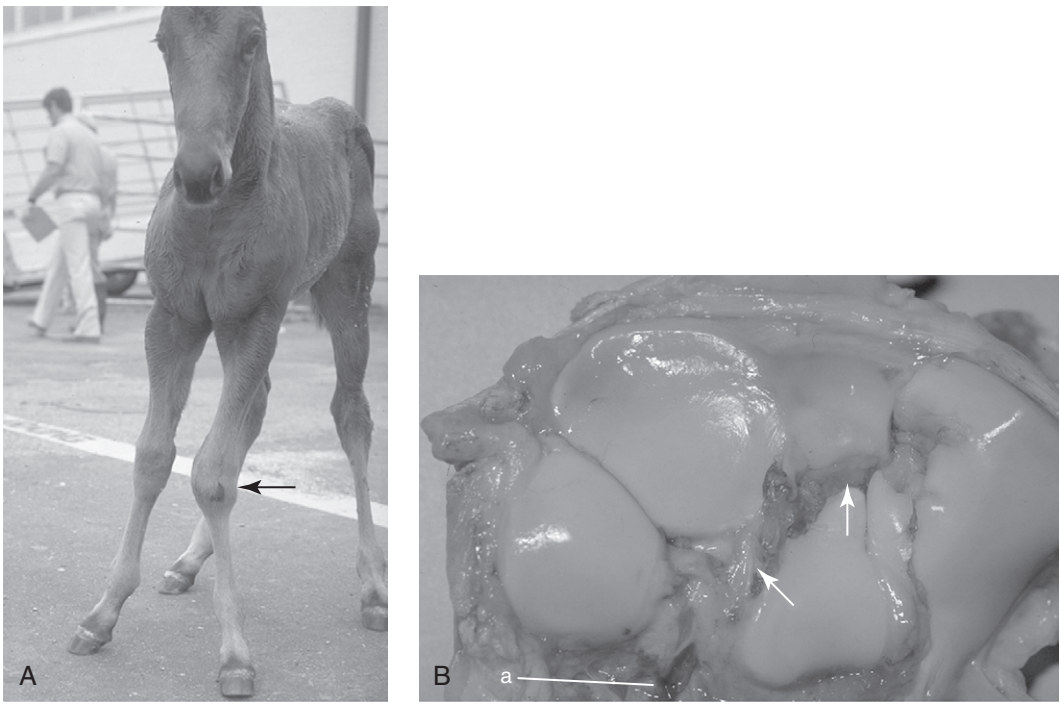

Figure 86-4. A, A 6-week-old foal with a marked carpal valgus deformity in the left limb. There is swelling *(arrow)* and the animal is lame. **B,** Postmortem picture of the articular surface of the third carpal bone, showing an osteochondral fracture *(arrows).* There is also marked discoloration of the synovial membrane caused by intra-articular hemorrhage.

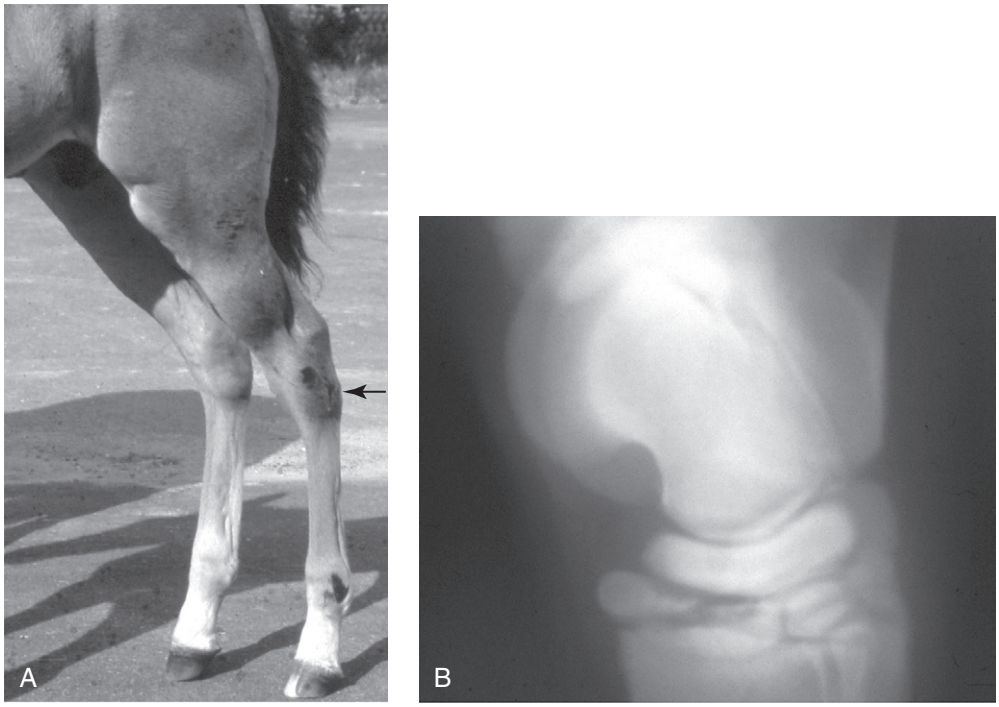

Figure 86-5. A, The hind limbs of a 1-month-old foal with a marked dorsal angulation at the level of the tarsus *(arrow)*. The foal showed the typical gait of a foal with collapsed third tarsal bones: it trotted with the forelimbs and "bunny-hopped" with the hindlimbs. **B,** Lateromedial radiograph showing the collapsed third tarsal bone, which is displaced dorsad.

eventually the small tarsal bones fuse to a single irregularly-shaped, uncorrectable bone. Therefore, early intervention and aggressive treatment are necessary to ensure normal development.

Laxity of Periarticular Structures

Some foals are born with severe ALDs in several regions, allowing oscillation between valgus and varus deviations depending on limb position during weight-bearing. Rotational deformities of various degrees also are encountered during the first few days after birth (Figure 86-6). These foals probably suffer from laxity of the periarticular supporting structures or from soft tissue trauma, both of which result in unstable joints. In most cases, several joints are involved at the same time. These deformities lead to abnormal loading of the articular surfaces and may, in the presence of incompletely ossified cuboidal bones, induce a severe ALD. It is unknown exactly how these problems develop. In some cases, the aberrant development of the long bones relative to the soft tissue structures may be caused by a hormonal imbalance or by intrauterine positioning.[19]

Aberrant Intrauterine Ossification

Occasionally a foal is born with a deformed long bone. The etiology is unknown. Ossification of the third metatarsal bone starts at approximally 150 days of gestation.[20] The influence of a mechanical factor, leading to passive deformation of the precursor cartilage of the bone involved during this gestation period, represents the most likely explanation of the presence of this deformation. A teratogenic factor influencing intrauterine ossification abnormally can be dismissed because in most cases only one bone is involved.

In the majority of these cases, the distal physeal region of the third metatarsal bone is involved and forms a triangular epiphysis. Interestingly, a varus deformity is most frequently diagnosed (Figure 86-7, *A* and *B*). Because there is aberrant ossification, the deformity cannot be manually corrected. On rare occasions a multiplanar deformity is encountered in the diaphyseal region of the third metatarsal bone (Figure 86-7, *C*).

Developmental Factors

Unbalanced Nutrition

The common practice of "crib feeding" often leads to excessive grain intake. It is important that the nutrition be balanced, especially with regard to trace minerals.[21] If the diet is not balanced, developmental orthopedic disease may occur and jeopardize future athletic potential.[22,23] Intake of trace minerals is further compromised by the exhaustion of grasslands and a lack of appropriate fertilization. A soil analysis of the local pasture may not be relevant, because forage (hay) is usually brought in from a distant source. Problems associated with unbalanced nutrition include osteochondrosis, flexural deformities, and ALD, the latter being represented mainly by disproportionate growth at the level of the physes.[22,23] Severe generalized osteochondrosis resulting from zinc toxicity or copper deficiency may cause ALD. On farms with a high occurrence of ALD, feeding practices should be evaluated through a complete feed and water analysis. If abnormalities are encountered, they should be corrected at once.[21]

Excessive Exercise and Trauma

Excessive exercise and trauma cause ALD. This trauma induces microfractures in and actual crushing of the proliferative zone

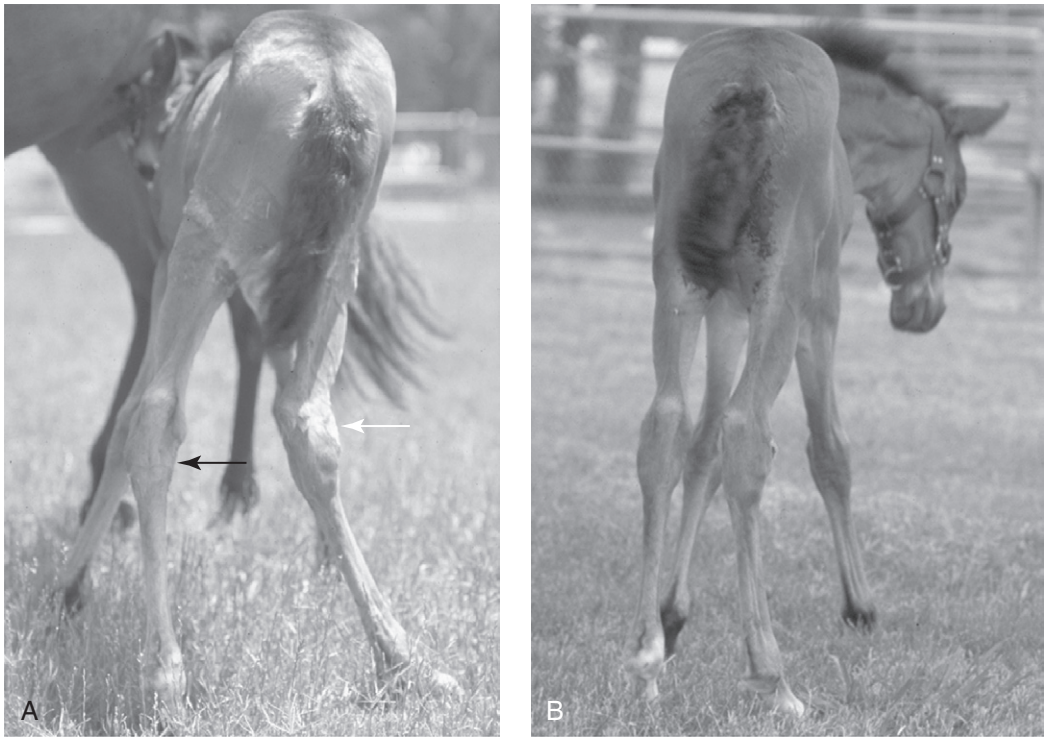

Figure 86-6. A, Photograph of a 1-week-old foal with a marked valgus deformity of the right tarsus *(white arrow)* and a varus deformity of the left tarsal region *(black arrow).* The degree of ossification was normal. **B,** After 2 weeks of light hand-walking and no treatment, the deviations had corrected.

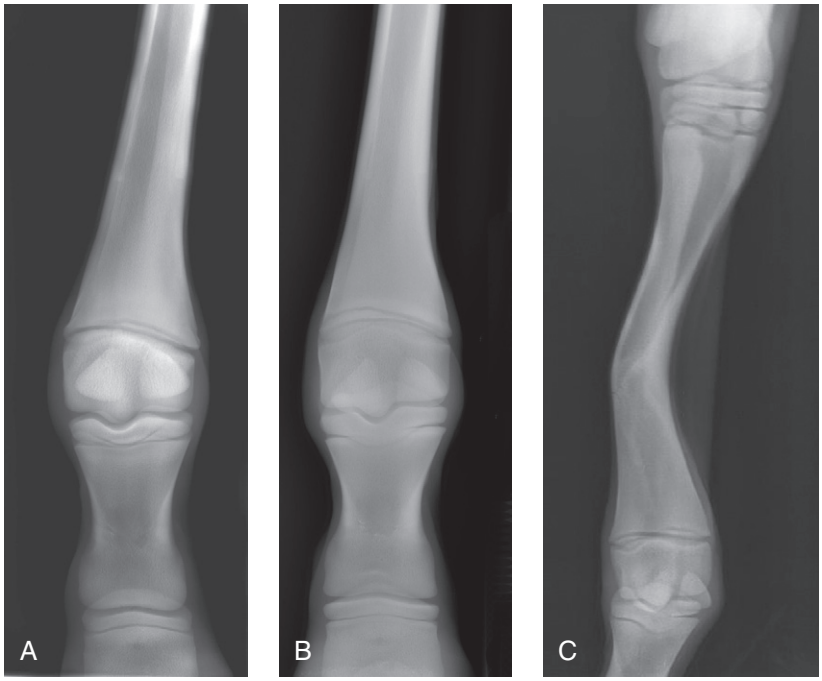

Figure 86-7. A, Dorsoplantar radiographic view of the distal limb in a 2-day-old Warmblood foal suffering from a marked varus deformity in the fetlock region. Note the triangular shape of the epiphysis. **B,** The same foal after 2 weeks of stall rest. The deformity is partially corrected because of the development of a compensatory deformity in the proximal phalanx. **C,** Dorsoplantar radiographic view of severely deformed MTIII in a 1-week-old Thoroughbredd foal. Note the rotational deformity of the distal half relative to the proximal half of the bone. (**B,** Courtesy R. Smith, London.)

at the base of the physis, altering cell proliferation and maturation, and in severe cases it leads to early local closure. This type of injury has been classified as a Salter-Harris type V fracture.[24] Additionally, epiphyseal fractures caused by external trauma such as kicks from another mare may cause ALD.[24] Most physeal fractures heal with residual ALD because it is almost impossible to maintain viability of the entire physeal plate.[25]

DIAGNOSIS

Diagnosis is based on inspection, manipulation, and diagnostic imaging techniques. The foal is observed from several angles, most importantly from the front and back. To evaluate the limb, the clinician should be positioned perpendicular to the frontal plane of the examined limb. Splay-footed foals, with or without

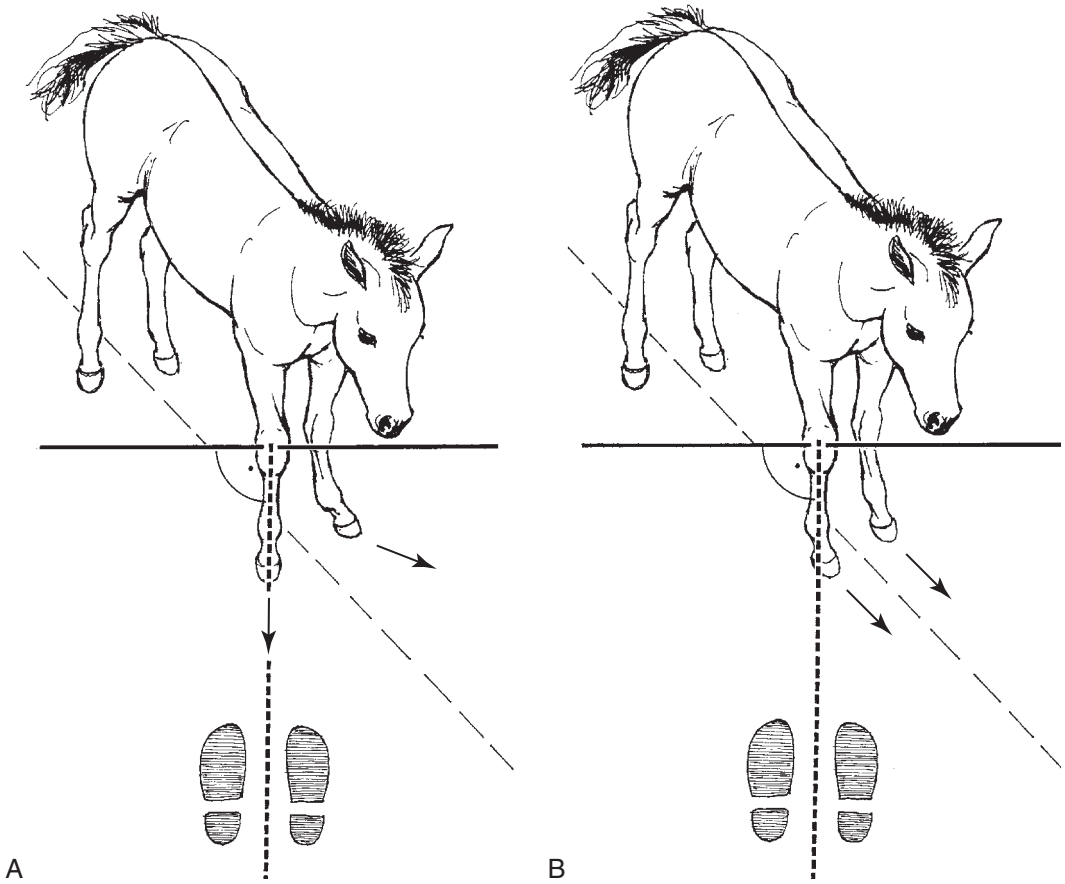

Figure 86-8. A, A valgus deformity of the carpal region is evaluated perpendicular to the frontal plane of the outward-rotated carpus. If the toe points in the same direction, the entire limb is rotated out and surgery can possibly be postponed. **B,** If the toes point forward and the carpus points outward, surgical intervention is indicated to prevent the development of a varus deformity in the fetlock region.

ALD, are therefore evaluated from a craniolateral position, allowing observation for proper alignment of the toe and carpus or tarsus, respectively (Figure 86-8, *A*). It is very important that the toe point in the same direction as the carpus. Newly born foals are weak, thin-chested, and, in relation to their size, long-legged. To provide some support to the forelimbs, which are connected to the chest by only seven muscles, the elbow region is pressed to the side of the chest. This results in an outward rotation of the entire limb and a toed-out posture. With increasing strength and age, the chest fills out and the connecting muscles become stronger, resulting in an inward rotation of the limb and correction of the toed-out posture. However, if the carpus points outward but the toes point straight forward (Figure 86-8, *B*), a varus deformity of the distal limb is present, which may go unnoticed for a few months. Once the limb rotates into a normal position, the feet attain a toed-in posture. Unfortunately, the distal MCIII/MTIII experiences longitudinal growth only for 3 to 4 months,[26] after which the physis closes. Frequently, this is when the problem is noticed.

Palpation and manipulation of the limb help differentiate between most perinatal and developmental deformities. If application of manual pressure to the medial aspect of the carpal region straightens out a valgus deformity (Figure 86-9), the cause of the deformity is either incomplete ossification or flaccidity of the periarticular supporting structures. If the limb cannot be straightened with manipulation, changes of the osseous structures of the region are involved.

Observation of the foal as it walks toward and away from the clinician also provides valuable information. If the joints are aligned parallel to the ground, all the joint movements occur in the same planes and no outward or inward rotation of a joint is noticed. However, pigeon-toed foals frequently rotate their metacarpophalangeal/metatarsophalangeal (MCP/MTP) joints outward while advancing the limb. This is caused by the joints not being oriented orthogonally relative to the long axis of the long bones in the frontal plane or parallel to the ground.

The only diagnostic aid that allows exact determination of the location and degree of the deformity is radiography. It is important to use long, narrow cassettes for the radiographs and to include as much of the bones proximal and distal to the deformity as possible. The dorsopalmar/dorsoplantar views are most important, except in the tarsus, where the lateromedial view is preferred. The radiographs should be taken at a right angle to the frontal plane of the limb (the sagittal plane of the tarsus). The bones constituting the distal MCIII/MTIII and phalangeal region should be aligned in one plane for the radiographs, allowing interpretation of the articular orientation and differentiation of deformities.

The need for early diagnosis cannot be overemphasized, especially in foals with incomplete ossification. The soft precursor cartilage is deformed through the uneven axial loads and, combined with the rapid progression of endochondral ossification, may result in a permanent deformity within 2 weeks after birth.

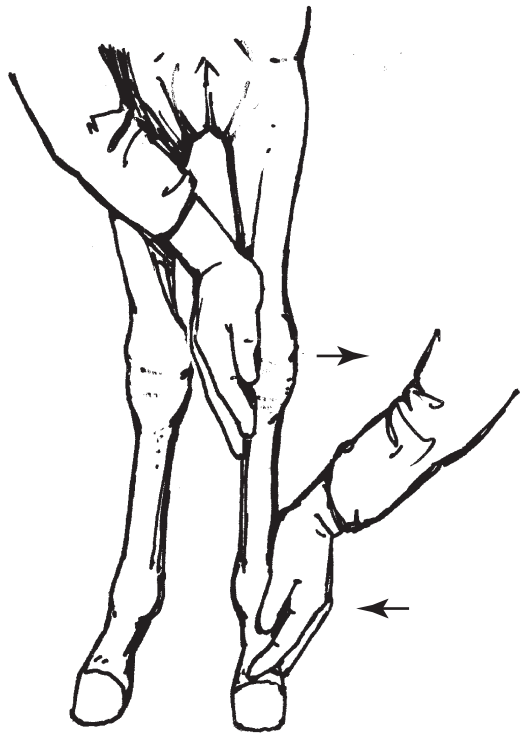

Figure 86-9. Manual pressure to the medial aspect of the carpal region of a foal suffering from bilateral valgus deformity corrects the deformity temporarily, indicating that conservative treatment is possible. Counterpressure is applied to the fetlock region of the same limb.

Compound and noncorrectable deformities present an added challenge. It is sometimes important to accept a compensatory deformity, if it aids the overall athletic soundness of the animal. A foal with a varus deformity in the metacarpophalangeal region would profit from a slight valgus deformity in the carpal region to balance the horse's weight-bearing axis over the foot. This necessitates the visual evaluation of the entire limb at the time of the diagnosis and caution in using radiographs as the sole diagnostic aid.

TREATMENT

Two studies conducted in Thoroughbred horses have significantly changed the philosophy of the management of ALDs.[27,28] One study documented conformational changes with age.[27] The changes occurring from weanling age to 3 years in a population of racing Thoroughbreds were determined by recording specific body measurements. There was a strong relationship between long bone lengths and wither height in all age groups, supporting the theory that horses grow proportionally. Longitudinal bone growth in the distal limb increased only 5% to 7% from weanling age to 3 years and was presumably completed before the yearling year. This study provided objective information regarding conformation and skeletal growth in the Thoroughbred, which can be used for selection and recognition of significant conformational abnormalities.[27]

The other study investigated the relationship of conformation to injury.[28] It found that offset carpi (offset ratio) contributed to MCP joint problems. Long pasterns increased the odds of a fracture in the front limb. A certain degree of carpal valgus deformity exhibited a protective mechanism, because the odds for a carpal fracture and carpal effusion decreased with an increase in the carpal angle.[28]

Based on these studies, the goals of effective management of ALD have changed. Severe deformities that do not correct on their own (see Figure 86-4) need to be distinguished from deviations that are a variation of the normal.[11] The art of management of ALD involves deciding which conditions require immediate aggressive management and which can tolerate a wait-and-see approach.

The following treatment methods are divided into nonsurgical and surgical procedures, according to the appearance and severity of the problem.

Nonsurgical Techniques

Stall Rest

Stall rest is an effective treatment in newborn foals with the following types of ALD:

- *Foals with incomplete ossification and straight limbs at the time of birth.* Under no circumstances should these animals be turned out in a field.[6,8,9,10] Exercise on the weakened, partially ossified carpal and tarsal bones may result in abnormal ossification of the bones with subsequent development of osteoarthritis.[6,8,9] The foals (and mares) should be restricted to stall rest for a maximum of 1 month. During this period, radiographs should be repeated at 2-week intervals to evaluate the progress of ossification. Longer restriction of activity is undesirable because of the normal development of the foal as a potential athlete.
- *Foals with adequate ossification and ALD because of disproportionate growth at the level of the physis (greater than 10 degrees) and foals with diaphyseal deformities.* Stall rest may be continued for 4 to 6 weeks. If correction does not occur during that time, surgical treatment should be implemented.
- *Foals with laxity of the periarticular supporting structures and a normal degree of ossification.* Such animals should be exercised daily for 10 to 20 minutes through walking the mare, to stimulate muscle work and strengthening of the involved soft tissue structures. Three to 5 minutes of swimming daily is the best exercise if facilities are available (see Chapter 87). Growth of the foal usually resolves the problem.

Predicting whether the problem will resolve with stall rest alone is not possible, and valuable time can be lost by waiting too long. This is the main reason for not prolonging stall rest treatment beyond 4 to 6 weeks. It is important to critically observe the foal continually during the stall rest period.

Splints and Casts

Foals with incomplete ossification of the carpal and tarsal bones may be treated effectively with splints or casts (Figure 86-10, *A*).[6,8] The purpose of these devices is to maintain the limb in proper alignment and to allow the inadequately ossified cartilage template to ossify sufficiently and facilitate weight-bearing without detrimental consequences.[9] It is of paramount importance that the cast or splint end at the fetlock. If the foot is incorporated in the splint, the musculotendinous flexor and extensor units weaken, resulting in a dropped fetlock and osteopenia. In most cases, this problem is temporary, but it should

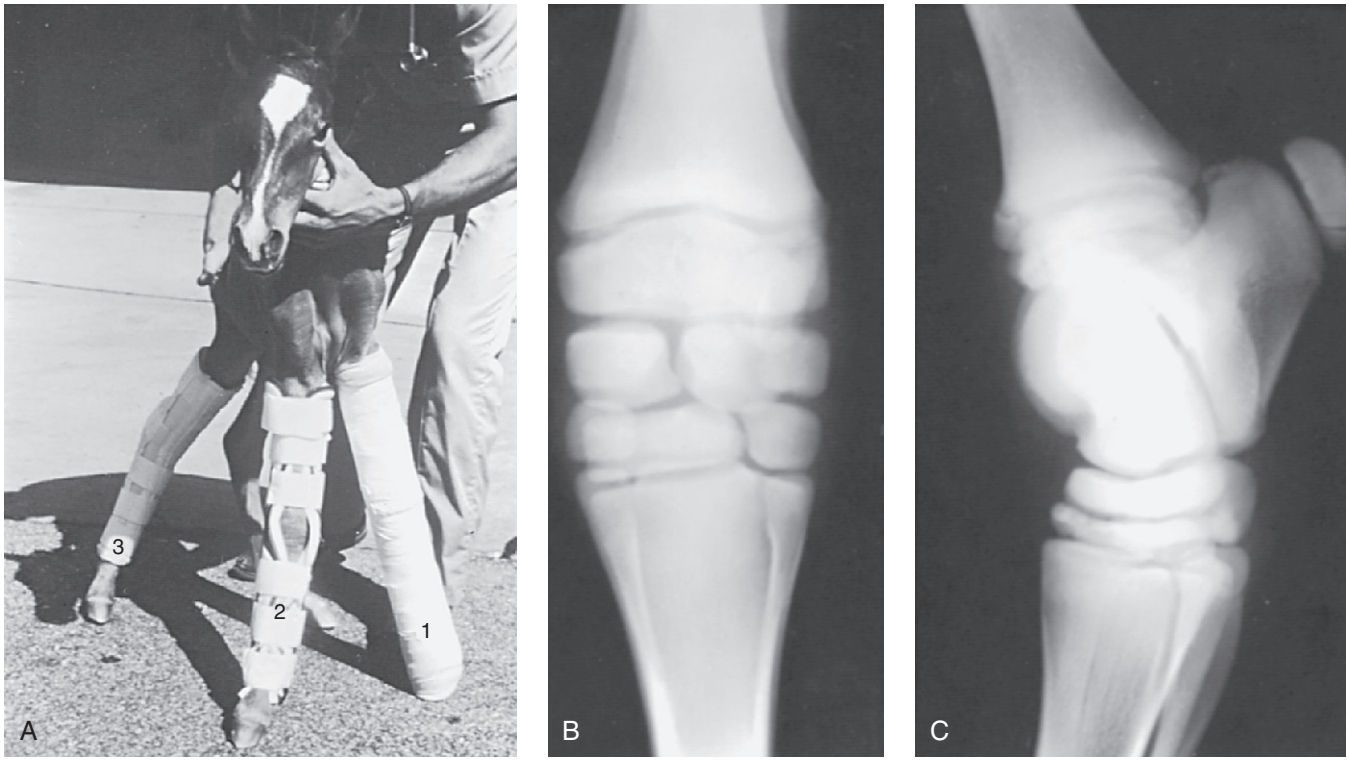

Figure 86-10. The foal mentioned in Figure 86-2 at 2 days of age. **A,** Splints are applied to three limbs. A polyvinylchloride (PVC) pipe splint is applied to the left forelimb (*1*). The PVC pipe is centered over the carpal region and reaches distad to the hoof because the animal also suffered from a ruptured common digital extensor tendon in that limb. The total limb is incorporated in the bandage. A commercial splint (*2*) is applied to the right forelimb. The splint is fastened with Velcro straps and contains medial and lateral metal strips for support. The right tarsal region is also splinted with a commercial splint (*3*). This splint is of the same material as the forelimb splint (*2*). Note that the commercial splints do not incorporate the phalanges. **B** and **C,** Radiographs carried out 30 days after the original radiographs. The dorsopalmar view of the carpal **(B)** and lateromedial view of the tarsal **(C)** regions show increased ossification compared with the previous radiographs. Ossification was sufficient to allow the foal to go without splints. The foal was maintained in bandages, kept in a stall for an additional week, and then turned out.

be prevented because it may lead to additional orthopedic problems. The limb should be well padded before splint or cast application. The splint is changed every 3 to 4 days, and dry padding is placed next to the limb. Casts should be changed between 10 and 14 days after application. Leaving the cast on for too long results in skin damage because of the rapid growth of young foals. The radiographic evaluation is repeated at 2-week intervals and the change in degree of ossification is determined.

Casts and splints should be maintained as long as incomplete ossification is present. Depending on the degree of ossification at the time of birth, this may take 2 to 4 weeks (Figure 86-10, *B* and *C*). In twin foals, it may be continued for up to 2 months of age.

While the limb is under a cast or splint, the flexor carpi ulnaris and the ulnaris lateralis muscles are immobilized and become flaccid. This results in a temporary calf-kneed conformation immediately after the cast is removed (see Chapter 87). However, with time, these defects correct as strength returns to the muscles. Daily swim therapy is beneficial and helps in rapidly overcoming this condition. It is important to keep the limb bandaged for an additional 4 to 5 days after the cast or splints are removed. Care must be taken to prevent development of pressure necrosis of the skin over the accessory carpal bone.

If radiographs demonstrate that the deformity is located within the distal radius or tibia and is not caused by laxity of the periarticular supporting structures or incomplete ossification, splints and casts are ineffective and, in fact, contraindicated. Application of such devices almost invariably results in pressure sores without correction of the deformity.

Aside from somewhat labor-intensive management with the help of polyvinyl chloride (PVC) half-shell splints, custom-fitted snap-on splints made of padded fiberglass (EnduraSplint 2) can be used.[29] These splints have enough strength for foals younger than 6 weeks of age. The foal is sedated and placed in lateral recumbency, and a rectal sleeve is placed over the limb to protect it from contact with water and polyurethane resin. The splint material is immersed in warm water (21° C to 23° C [69.8° F to 73.4° F]) until it is soft and pliable. Excess water is squeezed out and the splint is swiftly applied to the limb with the felt positioned against the skin and covering half of the circumference of the limb. It is secured to the limb with gauze and allowed to dry, with care taken not to flex the limb during the setting time. The splint is removed and allowed to set for 5 to 7 minutes before reapplying it to the limb. Because the splint is padded and molded to the contour of the limb, it is easy to place, and only a single roll of self-adhesive elastic tape is needed to secure it, making it convenient for daily use. In a hospital setting, it is recommended that the splint be left on for

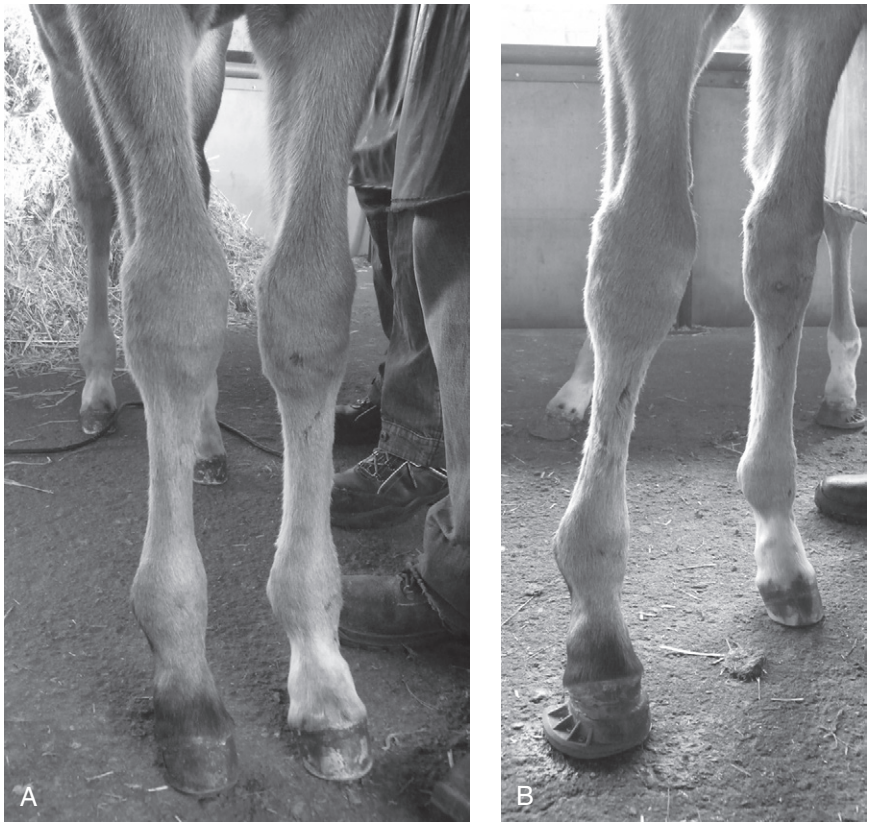

Figure 86-11. A, A 1-day old foal with a slight varus deformity in the fetlock region. **B,** The same foal after application of a lateral toe plate.

12 hours at a time to prevent pressure sores; it should be applied at night while the foal is less active.[10] This is especially important if all four limbs are splinted, making it cumbersome for the foal to lie down and get up. Splinting two limbs for 12 hours at a time and alternating limbs during the following 12 hours may help the foal to cope with this situation. Assisting the foal manually in lying down and getting up several times a day is another option.

One study showed that foals with incomplete ossification of the tarsal bones and greater than 30% collapse of the third and central tarsal bones had a poorer outcome than did similar foals with less than 30% collapse, thereby stressing the importance of early recognition and treatment.[30]

Special custom-fitted hinged braces, which allow the foal to move the carpal and tarsal regions while maintaining correct axial alignment, have been introduced to the market (Farley brace, Redden brace).[31] Foals with incomplete ossification or laxity of the periarticular supporting structures can be successfully treated with these devices. Because these braces allow movement of the joints, this type of treatment is theoretically preferred over stiff splints or casts but is considerably more expensive. However, this is the only application of such braces in foals with ALD.

Hoof Manipulation

Corrective hoof trimming is a frequent conservative treatment of ALD. In valgus deformities, the outside half of the hoof wall is slightly shortened. This causes the inside half of the foot to contact the ground first, and during the process of placing weight on the entire foot, it rotates medially. This type of hoof trimming is carried out with a rasp in young foals. In contrast, for varus deformities and pigeon-toe conformation, the inside wall is slightly lowered. In young foals, this type of treatment may be beneficial as an adjunct therapy to a surgical technique and should be maintained for only a few weeks because of the danger of the development of an abnormal hoof shape and of interphalangeal joint osteoarthritis.

The application of foot plates or shoes with an extension to the inside or outside may assist in correcting the deformity (Figure 86-11). Additionally, these devices prevent excessive wear-down at certain areas of the foot. For valgus deformities, extensions are placed on the medial side of the foot, and for varus deformities, on the lateral side. It is important to fill the extensions placed on the medial aspect of the hoof with acrylic to prevent the foal from stepping onto the plate with the other foot.

Corrective hoof trimming should not be used as the sole treatment for significant ALD distant from the phalangeal region in older foals. By forcing the foot into an uncomfortable, abnormal position, torsional and stress forces are created that cause early degenerative changes of cartilage and periarticular structures. Therefore it is better to opt for surgical procedures at the location of the deformity than to try to bring about correction through altering the loading conditions of the entire limb up to the location of the deformity.

Surgical Techniques

The surgical techniques for correcting ALD are reviewed for the carpal region. As the various techniques are discussed, consideration is given to the other anatomic regions where these surgical procedures may be used.

Growth Acceleration

Since its introduction into equine surgery, periosteal transection and elevation (stripping) has gained universal acceptance.[5,7,8,9,32-34] Periosteal manipulation is performed on the concave aspect of the limb (e.g., in an animal with a valgus deformity in the carpal region, on the lateral side). The original technique described a 3-cm vertical skin incision between the common and lateral digital extensor tendons, starting from a point 4 to 5 cm proximal to the distal physis of the radius and continuing in a proximal direction (Figure 86-12, *A*). The incision is carried down to the periosteum. With a curved hemostatic forceps, the subcutaneous tissues and tendons are separated from the periosteum parallel to the physis and perpendicular to the skin incision at its distal border. The forceps is abducted, with its slightly spread tips pressing onto the bone. Under this protection, a curved scalpel blade (No. 12) is inserted craniad between the tips of the hemostatic forceps. Using moderate pressure, the scalpel blade is pulled back toward the level of the skin incision, transecting the periosteum. Once the blade has reached the lateral aspect of the bone, the incision is stopped. The same procedures are repeated on the caudal aspect of the distal radius. The hemostat is first advanced to separate the lateral digital extensor tendon from the rudimentary ulna and then is redirected to a frontal plane to reach the caudal aspect of the distal radius. The scalpel blade is inserted under the protection of the forceps, and the periosteum, including the rudimentary ulna, is transected as described earlier. The cranial and caudal incisions are connected. Because this transection severs the rete carpi volaris, marked bleeding occurs. The periosteum is then incised parallel to the skin incision in a proximal direction over a length of 2 cm, starting at the horizontal periosteal incision, to create an inverted T. The periosteal elevator is advanced at a 45-degree angle to the periosteal incisions underneath the periosteum to elevate two triangular flaps (Figure 86-12, *B*). Once elevated, the periosteum is gently laid back onto the bone to prevent the ends from curling, which could result in abnormal new bone formation. The subcutaneous tissues are closed in a simple-continuous suture pattern, using 2-0 absorbable suture material, followed by a simple-continuous intradermal suture line. This results in an acceptable cosmetic appearance of the surgical site.

In about 20% of all cases, the rudimentary ulna is ossified and therefore has to be removed with rongeurs. It is important that the rudimentary ulna be transected, because it can act as a tethering mechanism and slow down growth at the concave aspect of the radius.

Postoperatively, a bandage, consisting of a nonadherent dressing (Telfa) and unfolded 4 × 4 gauze sponges, covered by an adhesive elastic bandage (Elastikon), is applied. The elastic tape is attached directly to the skin for a width of 2 cm proximal and distal to the gauze sponges. The bandage is replaced 3 days postoperatively. The second bandage may stay in place for an additional week before it is removed. Because the skin sutures are placed below the surface (intradermally), they do not have to be removed. It is advisable to keep the animal in a stall for 2 to 3 weeks and to allow minimal exercise during that time.

Postoperatively, the outside of the feet (in a valgus deformity) are rasped slightly every 2 weeks to assist in correction of the outward rotation of the feet and straightening of the limbs.

The surgery can be carried out in a foal as young as 2 weeks of age. The earlier the surgery is performed, the faster the correction occurs (Figure 86-13). However, opting for surgical intervention at such an early age may include foals in which the deformity would have corrected spontaneously if the animal was allowed stall rest. Therefore foals should be selected for this surgery after 4 weeks of age, unless the ALD is severe (greater than 10 degrees).

The landmarks for periosteal transection at the distal MCIII/MTIII are the distalmost aspect of the metaphysis of MCIII/MTIII or MCIV/MTIV and, depending on the location of the concavity of the deformed bone, the medial or lateral aspect of the bone (see Figure 86-12, *B*). Care should be taken not to enter the palmar or plantar outpouching of the MCP/MTP joint. These surgical interventions must be performed before 3 months of age, because after that there is only limited growth at the distal physis of MCIII/MTIII.[26] The periosteum at the surgical site of MCIII/MTIII is markedly thinner than that of the distal radius or tibia.[8]

Surgery at the proximal phalanx should be carried out at the level where the extensor branch of the suspensory ligament curves over the lateral or medial aspect of the bone (see Figure 86-12, *B*).[34] On this bone, the periosteal incisions are T-shaped, with the horizontal incision about 1 cm distal to the physis and the vertical incision in a distal direction. At the palmar or plantar aspect of the horizontal incision, some attachments of the oblique distal sesamoidean ligaments will be transected. This appears to present no serious consequences but can result in minor new bone formation that resolves with time.

The surgical approach for the distal tibia is either cranial or caudal to the lateral digital extensor tendon (see Figure 86-12, *C*). The rest of the procedure is carried out in a fashion analogous to that described for the distal radius. It is interesting to note that the periosteum in that area is thicker than that at any other surgical site discussed earlier.

"Bench knees" are a frequently encountered conformational defect that may continue to develop after 1 year of age.[31,35,36] These conformational defects are the result of two opposing ALDs—a valgus deformity at the distal radius and a varus deformity of the proximal third of MCIII (Figure 86-14, *A*).[8,31,36] The limb has a straight appearance in the presence of bench-kneed (off-set) conformation. If this conformational defect is diagnosed within the first 2 months of life, the deformities can be successfully treated with periosteal transection at the distal lateral aspect of the radius and the medial aspect of the distal metaphysis of MCIII (Figure 86-14, *B*).[31,36] Periosteal stripping over the total length of MCIII/MTIII using an I-shaped incision (see Figure 86-12, *D*) is also effective in correcting diaphyseal or metaphyseal deformities of that bone in foals younger than 2 months.[32]

Periosteal transection has its effect for approximately 2 months, which corresponds to the time needed to fill in the defect between the transected ends of the periosteum.[31,36] A prolonged effect is achieved through surgical excision of the elevated triangular flaps. Periosteal transection can be repeated if complete correction is not achieved. Overcorrection of the deformity does not occur.

It has been shown that periosteal transection carried out when the ALD is located within the carpus also corrects malformation of these bones to some degree.[37,38] Nevertheless, such problems should be recognized earlier and the incomplete ossification treated during the immediate postnatal period, as described previously. However, surgery accelerates correction even in these cases, allowing the foals pasture exercise at an

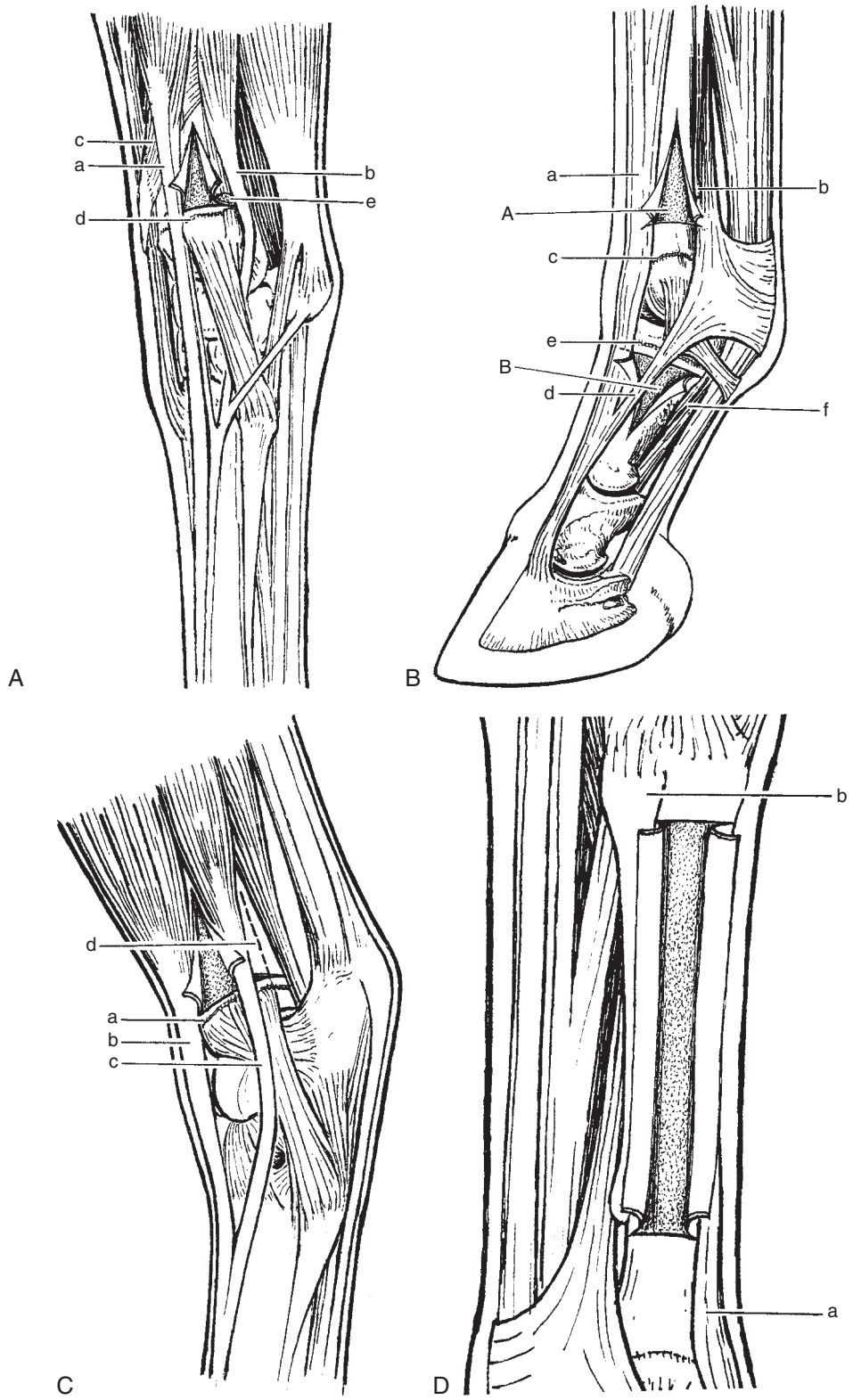

Figure 86-12. Locations at which growth acceleration may be carried out. In each location the T incision together with the elevated triangular flaps is drawn. **A,** Anatomic relationship of the landmarks for the surgical approach at the distal carpus. *a*, Common digital extensor tendon; *b*, lateral digital extensor tendon; *c*, abductor pollicis longus; *d*, distal radial physis; *e*, transected rudimentary ulna. **B,** Location of growth acceleration at the distal MCIII/MTIII (*A*), and proximal phalanx (*B*). *a*, Common digital extensor tendon and lateral to it the lateral digital extensor tendon; *b*, distal end of MCII/MTII; *c*, distal physis of MCIII/MTIII; *d*, extensor branch of suspesory ligament; *e*, proximal epiphysis of the proximal phalanx; *f*, oblique distal sesamoidean ligaments. **C,** Anatomic relationship of the landmarks for the surgical approach at the distal tibia. *a*, Distal physis of the tibia; *b*, long digital extensor tendon; *c*, lateral digital extensor tendon; *d*, vertical periosteal incision caudal to the lateral digital extensor tendon is an alternate site *(dotted line).* **D,** Location of the procedure carried out over the total length of the MCIII/MTIII. *a*, Common digital extensor tendon; *b*, proximal aspect of the MCII/MTII. The procedure was performed and the two periosteal (barn-door) flaps were elevated.

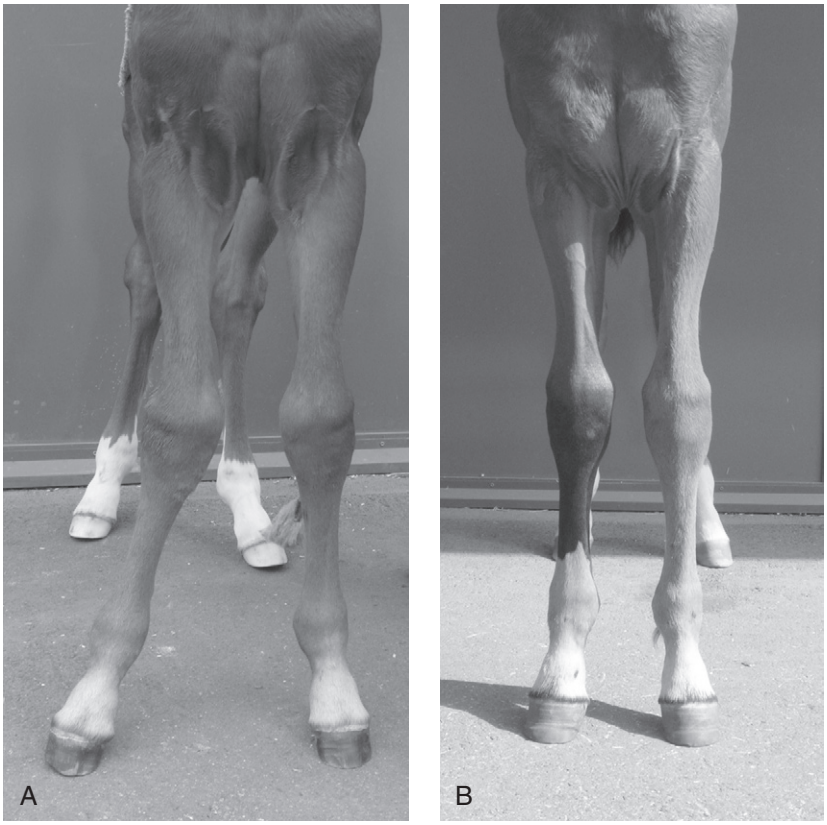

Figure 86-13. A, A 2-week-old Warmblood foal with a marked carpal valgus deformity in the left forelimb. **B,** The same foal 2 months following periosteal transection and elevation at the lateral aspect of the left distal radius.

earlier age while preventing the development of additional deformities farther distad (e.g., digit, hoof).

Periosteal transection and elevation are routinely carried out on an outpatient basis, with the mare and foal returning home immediately after recovery from surgery. The most critical location for early diagnosis and surgery is the distal MCIII/MTIII, because longitudinal growth of MCIII/MTIII occurs mainly during the first 3 months after birth.[26] Later on, growth occurs at a very slow pace, and delaying surgery leads to incomplete correction. Additionally, the prolonged abnormal loading of the metacarpophalangeal joints leads to the development of compensatory deformities in the proximal phalanx.[31,32] Thus the limb may appear to be straight, but when the foal walks, an outward rotation is noted. This is caused by the orientation of the articular surfaces, which are not parallel to the ground (Figure 86-15).

During the first weeks of life, affected foals show an outward rotation of the carpal region while their feet point straight forward (see Figure 86-8, *B*). At that point, an actual varus deformity is hard to detect, especially for an inexperienced clinician. With time, the deformity is aggravated, making diagnosis easy but surgery difficult.

The effectiveness of hemi-circumferential periosteal transection and elevation in treating ALD was questioned after reviewing the results of a controlled study, where temporary transphyseal bridging was performed on the lateral aspect of the distal radius to induce a carpal valgus ALD of 15 degrees in normal young foals.[39,40] At the time of implant removal, the foals were divided into two groups. One group was subjected to an immediate hemi-circumferential periosteal transection and elevation on the same lateral aspect of the distal radius, whereas the other group underwent a sham surgical procedure at the same site to mimic the hemi-circumferential periosteal transection and elevation procedure. In both groups, the deformity corrected, leading to the erroneous conclusion that periosteal transection and elevation was ineffective. Three facts might explain the results: (1) the deformity was induced artificially through manipulation of the physis, (2) a clinical case with a carpal valgus deformity of 15 degrees does not correct without surgery, and (3) physeal growth is influenced by mechanical load, which in turn triggers signal molecules in a feedback loop between the cells of the different cartilage zones and the perichondrium or periosteum of the limb involved.[41] This regulatory mechanism of signaling molecules was triggered at the time of initial surgery, when the periosteum was disturbed, and it was accentuated by the second surgery, performed to remove the implants. Periosteal transection could not be expected to further trigger this cascade. Therefore the assumption that differences should be seen between groups was wrong.

The results of a recent study indicate that periosteal stripping caused an upregulation of Ihh in the early prehypertrophic and hypertrophic zone of the growth plate, followed by an increase of PTHrP, mainly in the perichondrium. In contrast, an increase of parathyroid hormone receptors (PTHR) was noticed in all zones, although it was highest in the perichondrium and the hypertrophic zones.[16,38] FGF and TGFs were upregulated in all zones, but FGF in response to periosteal stripping was more intensely expressed in the proliferatve zone, and the highest peak of TGF was found in the perichondrium. Length measurements of the various zones revealed significant negative correlations between the proliferative and prehypertrophic and hypertrophic zones, an indication that indeed a negative

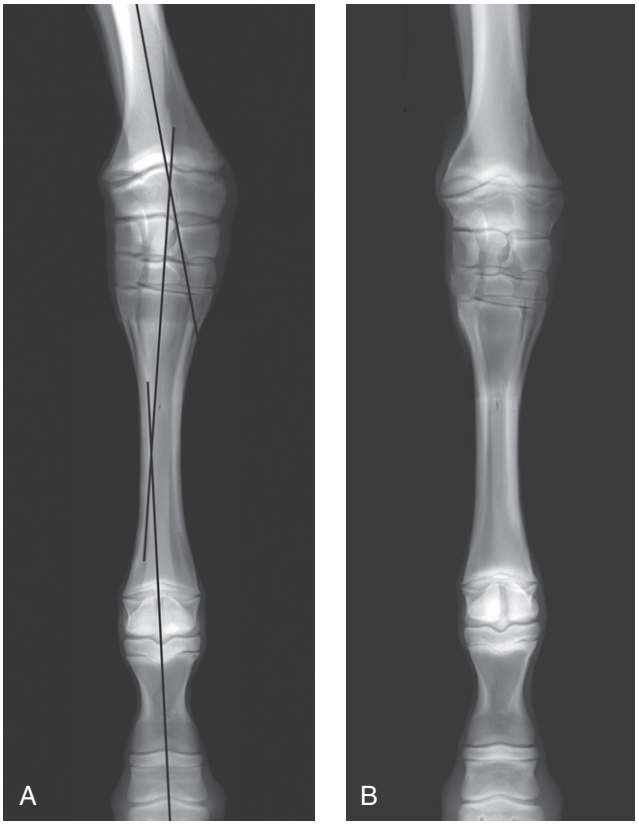

Figure 86-14. A, Composite cranicaudal/dorsopalmar radiographic view of a 2-month-old Warmblood foal with bench-knee conformation. The bisecting lines drawn along the bone axes show the composition of the different deformities, distal radial valgus and MCIII varus deformity. **B,** Composite radiographic view 2 months following periosteal transection and hemi-circumferential stripping at the lateral aspect of the distal radius and medial aspect of the distal MCIII. A slight compensatory deformity developed in the proximal phalanx.

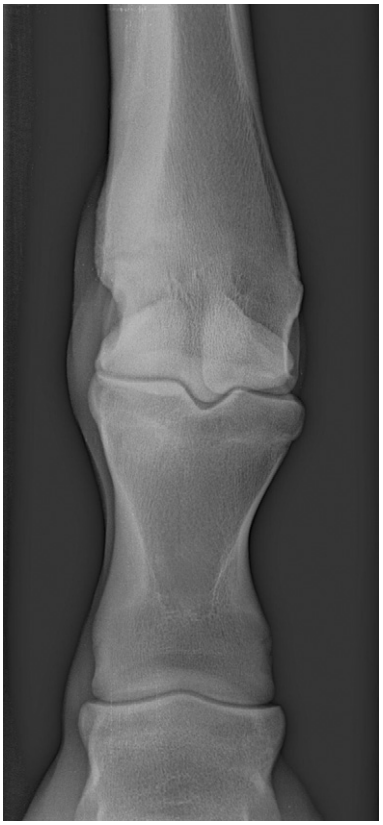

Figure 86-15. Dorsopalmar radiograph of the metatarsophalangeal region of a foal with a relatively straight limb. The proximal phalanx and distal MTIII have opposing deformities, resulting in an oblique orientation of the joint surface relative to the ground.

feedback loop after periosteal stripping exists, coupled with the Ihh/PTHrP/PTHR cascade. The hypothesis that periosteal stripping had an effect on the Ihh/PTHrP/PTHR–related feedback loop in epiphyseal growth was confirmed in this experimental study in lambs.[16] Since these mechanisms are very basic and similar in most species, it can be safely assumed that the effects in foals are similar. In fact, the asymmetric mechanical load in animals suffering from axial limb deformities may even increase the enhancing effect of length correction.[16] Additional work is needed on foals to lay to rest the discussion on effectiveness once and for all.

Modifications of the technique have been described.[42-44] One modification involves minimization of the surgical approach at the same location described originally.[42] The skin incision is performed horizontally down to the bone over 1 cm, followed by a vertical 1-cm incision. This reduction in incision size improves the cosmetic appearance of the surgical intervention. In one study, different approaches to periosteal manipulations in addition to hoof manipulations were evaluated in young foals on a large beeding farm. Ten foals with up to three deformities, mainly of the distal MCIII/MTIII, were randomly assigned to one of eight groups.[42] Each group was treated with a different technique, varying from the technique described earlier,[42] to a transcutaneous horizontal incision conducted by rolling the skin from the dorsomedial aspect of the bone to the palmarolateral/plantarolateral aspect while simultaneously cutting the periosteum with a No. 12 scalpel blade (Figure 86-16), to ligation of an afferent epiphyseal vessel, and juxtaepiphyseal injection of PTH-containing hydrogel. All deformities corrected clinically within 2 months.[43] Another study revealed that multiple insertions of a hypodermic needle into the physis at the concave side lead to correction of the deformity.[44]

These results further verify that periosteal manipulations are effective in correcting angular limb deformities and that a less-traumatic approach leads to good correction as well.[43] I currently use the transcutaneous approach exclusively in young foals. PTH gel may also have interesting applications in human surgery. It represents the least-invasive technique but has two drawbacks: high expense and limited shelf life of the PTH gel.[43]

Growth Retardation

Growth retardation is performed either in young foals (less than 3 months of age) with severe ALD or in foals with significant ALD in a bone after the rapid growth phase is over (MCIII/MTIII and proximal phalanx after 2 months, tibia after 4 months, radius after 6 months).[8,26] Most of the techniques described for growth retardation use the same principle. Implants, applied on the convex aspect of the bone, bridge the physis temporarily,

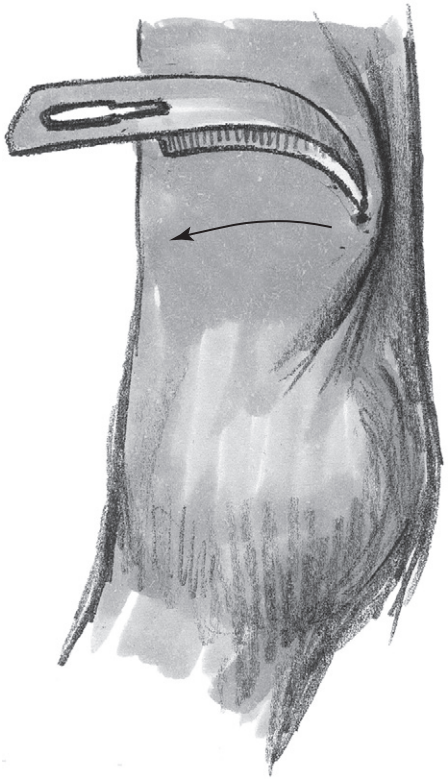

Figure 86-16. Transcutaneous periosteal transection technique. The skin over the distal medial MCIII/MTIII is rolled with the thumb palmarly. The pointed scalpel blade is inserted through the skin down onto the bone surface, and both the scalpel and skin are pulled in a dorsal direction. This technique allows horizintal transection of the metaphyseal periosteum and afferent physeal vessel through a 1- to 2-mm skin incision that does not have to be sutured.

allowing the shorter aspect of the bone to continue to grow, eventually correcting the deformity.

Stapling was the first technique of growth retardation described in the foal.[45] The surgery is performed in some clinics with favorable results but is not discussed here because it has fallen into disfavor. Complications include implant and correction failures. Despite these drawbacks, a report on how to prepare, place, and remove self-made staples was published.[46]

Screws and cerclage wires are the most frequently applied implants.* Implants are inserted through two stab incisions, one in the center of the epiphysis and the other proximal to the physis (Figure 86-17, *A*). The soft tissues between these incisions are elevated with a hemostat (Figure 86-17, *B*). A 4.5-mm cortex screw is inserted through each incision but not completely tightened (Figure 86-17, *C*). A wire loop is inserted through the proximal incision and hooked over the distal screw head (Figure 86-17, *D*). The two wire ends are twisted together and tightened over the proximal screw head (Figure 86-17, *E*). Twisting the wire ends over the proximal screw rather than the distal screw results in less irritation of the surrounding soft tissues and produces a better cosmetic result. If deemed necessary, a second figure-of-eight wire loop can be applied. The screws are tightened completely, which increases wire tension as the wires ride up on the shoulder of the screw heads. The stab incisions are then closed with two simple-interrupted skin sutures, and the area is protected with a light bandage for approximately 10 days. Sutures can be removed by the referring veterinarian. In foals with minor ALDs and laterally rotated forelimbs, surgery may be delayed for some time. However, foals

*References 1-3, 5, 8, 9, 13, 31, and 36.

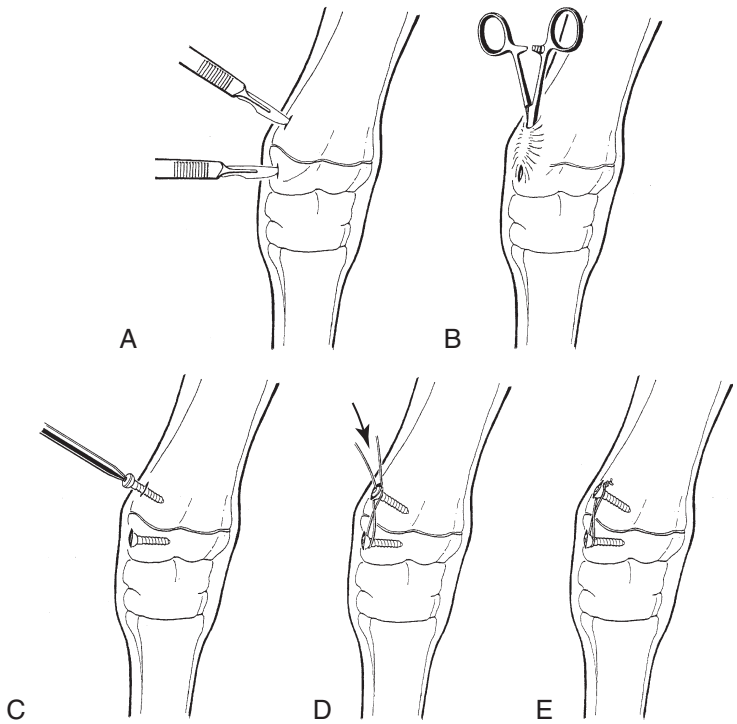

Figure 86-17. Growth retardation. **A,** A stab incision to the bone is carried out at the convex aspect of the epiphysis and the distal metaphysis. **B,** The soft tissue between the two incisions is elevated. **C,** A 4.5-mm screw is implanted through each incision but not tightened. **D,** A cerclage wire loop is introduced through the proximal incision, hooked over the distal screw head, and tightened in figure-of-eight fashion over the proximal screw **(E).** The screws are subsequently completely inserted, tightening the wire further.

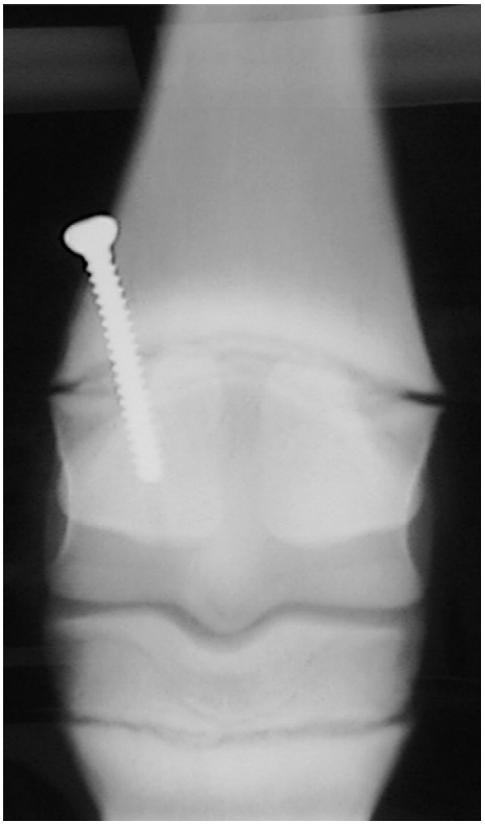

Figure 86-18. Dorsopalmar radiographic view of the distal MCIII. A single screw is inserted in an oblique proximodistal direction across the physis at the medial aspect of the bone. (Courtesy L. Bramlage, Lexington, KY.)

with carpal valgus deformity and feet pointing straight forward (fetlock varus deformity) should be treated immediately by applying a growth retardation procedure to the lateral aspect of the distal MCIII/MTIII to prevent development of a toed-in conformation. Implant insertion through two stab incisions improves the overall cosmetic result considerably, compared with the large incision initially used.

Implantation of a transphyseal screw has been described as a method of retarding growth (Figure 86-18).[47] The cortex screw is inserted using a position screw technique from the metaphysis across the physis into the epiphysis to arrest growth in that region. The major application is the distal MCIII/MTIII. The screws are removed when the desired conformation is reached. Some caution is indicated when the screw is applied in the distal radius and tibia, because of the potential development of a physitis. The reason the distal MCIII/MTIII is not affected by the potential development of physitis is attributed to the early closure of their physis (approximately 4 months). The advantages of the single transphyseal screw technique are ease of insertion and the improved cosmesis. A study comparing the results of transphyseal bridging using screws and wires (253 foals in 2003) to the use of a single transphyseal screw (315 foals in 2005) revealed that both techniques represent viable options. However, foals treated with a single transphyseal screw had a significantly higher risk of developing physitis or metaphyseal collapse compared to the ones treated with screws and wire.[48] Another study revealed no deleterious effects on sales or racing performance of 2- and 3-year-old Throroughbreds after the use of a single transarticular screw in the lateral aspect of the distal radius for the treatment of carpal varus deformities.[49] Varus deformities are usually recognized later in life, when the growth potential is already reduced, decreasing the risk for developing a physitis. I still prefer the screw and wire technique for growth retardation of a carpal valgus deformity.

The use of an absorbable screw has been described for MCIII/MTIII.[50] Because this screw was meant to stay in place, a slightly different implantation technique was applied. The periosteum was elevated at 15 mm proximad to the lateral physis. A 5.5-mm drill bit was used to create a glide hole of 5-mm depth. A 3.5-mm drill bit was subsequently seated in the glide hole at a 70-degree angle relative to the physis to prepare the thread hole of 40- to 45-mm depth. A countersink was used to ensure that the screw could be placed without bending forces developing during insertion of the screw head. After the length of the hole was determined and the prepared hole was tapped, a slightly shorter screw than measured (Smart Screw) was inserted and carefully tightened. The periosteum, subcutaneous tissues, and skin were closed in routine manner. ALD was corrected in all six foals treated and none developed complications, such as foreign body reaction or infection. The fact that the screw does not need to be removed is viewed as an advantage.[50] The price for the single screw is considerably higher than for a cortex screw, but because screw removal is not necessary, it adds up to approximately the same overall cost.

A small, 2.7-mm bone plate can be used to correct ALD in older foals. The last hole on each side of the plate is enlarged to accept a 3.5-mm cortex screw.[8,9] This technique provides an excellent cosmetic appearance of the limb. The surgical approach consists of either a slightly curved incision centered over the physis or a stab incision on either side of the physis. The distal stab incision is carried out parallel to the fibers of the collateral ligament. A 2.5-mm hole is drilled and tapped into the physis parallel to the joint surface, which is readily palpable. A 2.7-mm bone plate of adequate length is manually contoured to the shape of the bone and slid through the proximal stab incision distad, where the 3.5-mm screw of about 26 mm in length is inserted through the distal hole of the plate. An additional hole of the same diameter is drilled about 1 mm proximal to the last hole of the plate into the radial metaphysis. The hole is tapped, and the second screw is inserted into the hole. Both screws are tightened alternately until they are completely inserted into the bone (Figure 86-19). It is important that the distal screw be tightened completely to prevent it from protruding into the collateral ligament. The skin incisions are closed with simple-interrupted sutures. Radiographic evaluations should be carried out at monthly intervals to determine the correct time of implant removal and to prevent overcorrection of the deformity.

Growth retardation procedures also may be performed in the distal MCIII/MTIII, the proximal phalanx, and the distal tibia.[5,8,9] It is important to evaluate the anatomic situation at each location. This is especially important for the distal tibia, where the epiphysis has an undulating configuration and the distal screw has to be inserted in a mediodistal to lateroproximal direction to avoid penetration of the joint.[5,8,9] A short screw is used to prevent penetration of the physis. However, the screw is at such a favorable angle, relative to the direction of the tension, that a short screw (22 mm) has adequate holding power. The proximal screw is easily inserted as previously described. A retrospective study on 39 foals evaluating the response to various

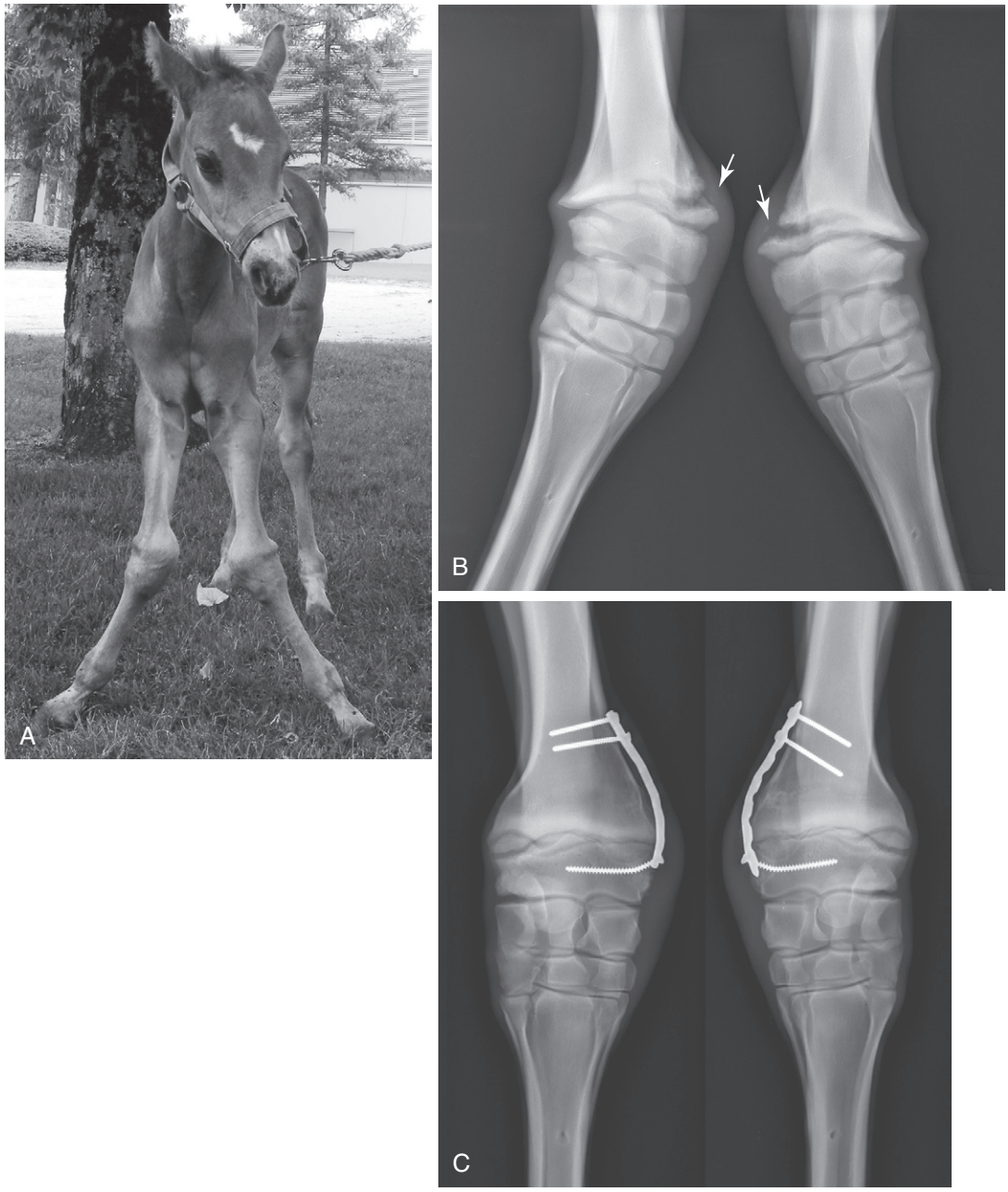

Figure 86-19. A, A 2-week-old Warmblood foal with a severe bilateral carpus valgus deformity. **B,** Preoperative craniocaudal radiographic view of the carpal region. Note the physeal ectasia located at the medial aspect of the distal radial metaphysis *(arrows).* **C,** Ten-week follow-up craniocaudal radiographic views of both carpal regions showing correction of the deformity. Note the severe bending of the cancellous 3.5-mm screws in the epiphysis. The implants were removed, and the animal was discharged. The lateral aspect of the distal radius shows some degenerative changes.

treatments and long-term outcome of foals with tarsal valgus deformities revealed that 22 foals (56%) had concomitant incomplete ossification of the tarsal bones.[51] Eight of 19 foals with tarsal valgus deformities that were treated with periosteal stripping responded favorably. Younger foals (60 days or younger) were significantly more likely to respond to periosteal stripping than older foals. Eleven of 21 foals with long-term follow-up performed as intended. Because foals with more-severe incomplete ossification of the tarsal bones respond poorly to periosteal stripping alone, treatment by growth retardation was recommended.[51]

Once the correction is complete and verified radiographically, the implants must be removed through stab incisions over the screw heads, with the horse under short-term anesthesia.[8,9] The exact location of the screw heads is easily determined radiographically. It is important to be sure that the screwdriver is correctly seated in the hexagonal indentation in the screw head. Otherwise, the screw head can be stripped, making removal with a screwdriver difficult or impossible.[8,9] In that case, a screw-removal device (see Figure 76-16) should be used to remove the screws. The plate or wire is then removed through the proximal stab incision by introducing a curved hemostatic forceps into

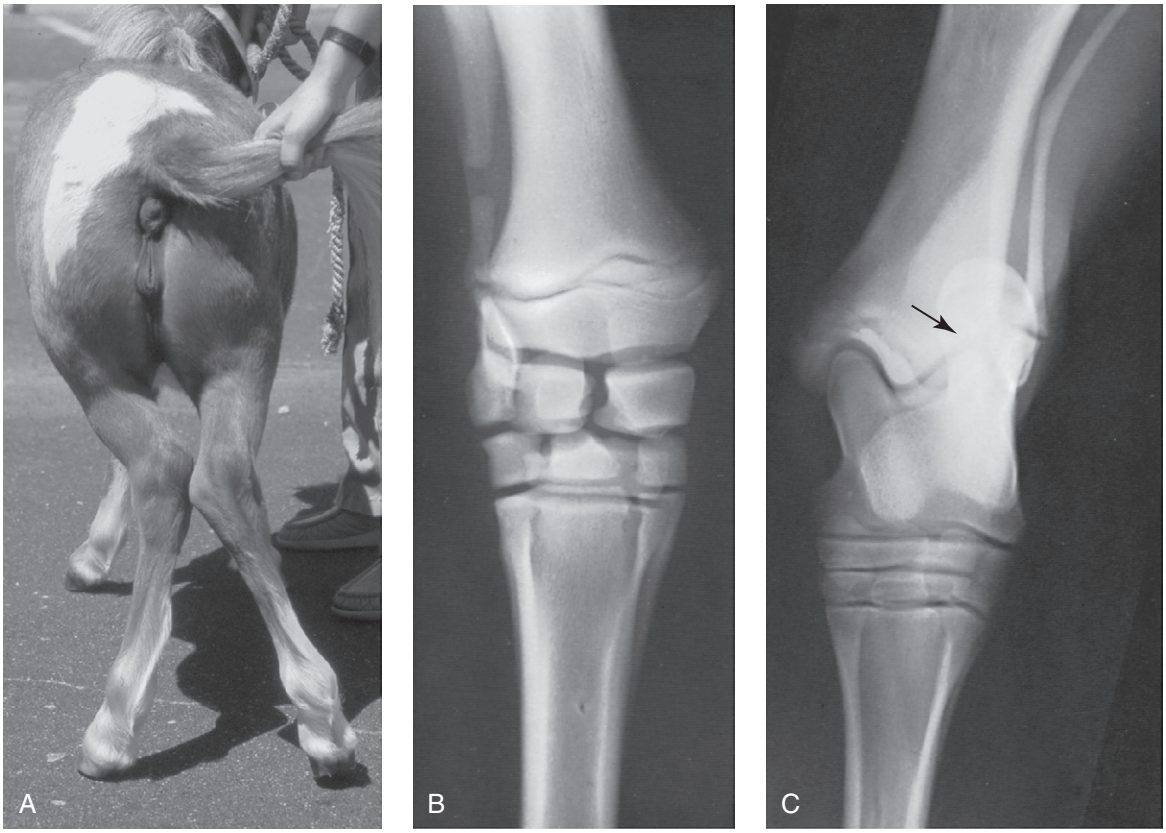

Figure 86-20. A, A 5-month-old Miniature Horse with severe tarsal valgus deformities in both rear limbs. Dorsoplantar radiographic views of the left carpal region show a completely developed ulna **(B)** and of the left tibia show a completely developed fibula **(C).** Note the acute angle of the lateral trochlear ridge of talus, representing early degenerative changes *(arrow).*

the hole and walking it off the edge of the hole in the bone, engaging the implant and pulling it through the incision. The stab incisions are closed using 2-0 absorbable suture material in a simple-interrupted pattern.

If implants are inserted in both forelimbs, it is important to remove the implants when each limb is straight. Waiting until both limbs are straight invariably results in overcorrection of one or undercorrection of the other limb. Implant removal can be carried out on an outpatient basis. Antibiotics are not routinely used.

As with growth acceleration, signal molecules from the proliferating and prehypertrophic zone in combination with the perichondrium are involved in retardation of the growth plate. It was shown in rats that compressing the growth plate using surgical staples changed the feedback loop of PTHrP and Ihh, so that the prehypertrophic chondrocytes were pushed toward the osteogenic lineage, enhancing osteochondral ossification.[52] After stapling, the Ihh and Ptc1 molecules were modestly upregulated at 3 days, whereas BMP-2 and BMP-6 were inhibited.

An alternative nonsurgical approach of local growth retardation has been proposed.[53] Using a radial shock wave generator at 3 bar, 15 Hz, and 2000 cycles with a 15-mm applicator, weekly treatment sessions were conducted at the convex aspect of the deformed bone on the sedated foals. Straightening of the limbs occurred between 15 and 76 days with a median of 25 days.[53] No explanation of mechanism of action has been offered.

Combination of Growth Acceleration and Retardation

In severe ALD, growth acceleration and growth retardation procedures are combined for a faster and more complete correction.[8,9] By combining the two techniques, there is a possibility that the deformity can be completely corrected. The advantage of using implants is that they can stay in place as long as they are needed, in contrast to periosteal transection, which has a time-limited effect.

In Miniature Horses, ALD is frequently diagnosed (Figure 86-20, *A*). The presence of complete ulnas and fibulas is the predominant cause of ALD in this species and can be judged as an atavism (Figure 86-20, *B* and *C*). Early recognition of the problem is important in these animals. Miniature Horses have less growth potential than regular horses; therefore, a 5-month-old foal with a moderate ALD will not achieve correction, even if growth acceleration and retardation procedures are combined. It is important to remove a portion of both the ulna and the fibula at the time of periosteal transection. However, the distal epiphyses of the complete fibula and the tibia are not united, and the continuous movement at their junction may result in abnormal development of the lateral trochlear ridge of the talus and associated osteoarthritis (see Figure 86-20).[54] This problem is not as severe in the carpal region because of the architecture of the distal radius. In selected cases, transfixation of the distal fibular styloid process to the distal tibial epiphysis or metaphysis, or both, is needed to stop movement at the level of the joint. To insert the screws through the distal fibular

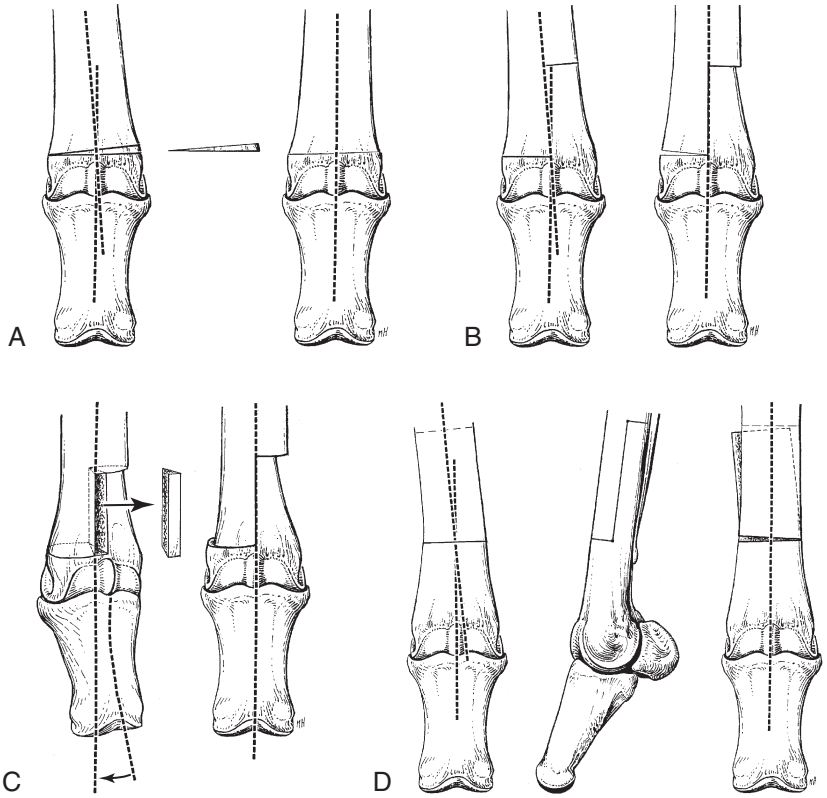

Figure 86-21. A, Closing wedge ostectomy. The bone wedge is removed from the horizontal plane near the physeal region. **B,** Step ostectomy. The wedge is removed from the center of MCIII in the sagittal plane. **C,** Derotational step ostectomy. By removing a vertical wedge with its wide aspect located either dorsally or in a palmar direction, a rotational deformity can be corrected. **D,** Step osteotomy. The Z-plasty is performed in the frontal plane, and the fragments are rotated into alignment.

metaphysis into the tibia, position screw technique (not lag technique) (see Chapter 76) should be applied to prevent alteration of the articular geometry.

Corrective Osteotomy/Ostectomy

Two types of ostectomy have been described in the literature: the closing wedge and the step. Closing wedge ostectomies (Figure 86-21, *A*) have been used for correction of diaphyseal and metaphyseal/epiphyseal ALD in foals with closed growth plates.[55,56] However, the step ostectomy carried out in the sagittal plane and the step osteotomy carried out in the frontal plane are presently the preferred techniques and are discussed here.[57,58]

The skin incision is carried out over the lateral digital extensor tendon from the top of the MCIII/MTIII to the distal physis. At that point, the incision is continued medially (in a varus deformity) or laterally (in a valgus deformity) toward the MCP joint. The lateral digital extensor tendon together with the periosteum is split longitudinally. Subperiosteal dissection is carried out to gain access to the dorsal aspect of MCIII/MTIII. At the level of the proposed Z-shaped osteotomy, the periosteum is elevated circumferentially around the bone.

The step ostectomy in the sagittal plane removes a vertical wedge from the center of the bone (Figure 86-21, B).[57,58]

The pivot point, which was previously established on radiographs (Figure 86-22, *A*) by drawing the longitudinal bisecting lines through MCIII/MTIII and the phalanges, respectively, is identified relative to proper landmarks, and a 3.2-mm hole is drilled across the bone. A second hole is prepared parallel to the first one, approximately 4 cm farther proximad. The oscillating saw is used to cut between the two drill holes across the bone. The previously prepared and sterilized aluminum template with the appropriate angle of the wedge to be removed is laid with one limb parallel to the saw cut and compared with the bisecting lines on the radiograph. A second saw cut is then made along the second limb of the template and parallel to the first one in the sagittal plane. The Z-shaped osteotomy is completed with the proximal cut through the thinner portion of MCIII/ MTIII (lateral in a varus deformity) and the distal cut across the opposite side of the bone in a horizontal plane, parallel to the physis. The wedge is removed, and the two bone fragments are rotated into alignment. Final adjustments are made to ensure complete correction of the deformity and bone-on-bone contact at the palmar/plantar aspect of the bone. If an additional rotational deformity is present, it can be corrected at that point by removing an additional bone wedge from the palmar or dorsal aspect of the bone (see Figure 86-21, *C*). Cortex screws are applied in lag fashion across the vertical section of the cut to reunite the two fragments of the bone. A bone plate is subsequently applied medially (in a varus deformity) or laterally (in a valgus deformity) to the bone, and along the vertical cut the screws are inserted in lag fashion (see Figure 86-22, *B* and *C*). The gap at the distal aspect of the osteotomy may be filled with some bone from the removed wedge as a cortical bone graft. The first row of sutures is placed into the split lateral digital extensor tendon using 2-0 absorbable suture material in a simple-continuous pattern. The subcutaneous tissues are closed, followed by intradermal closure of the skin, as previously described. In selected cases, skin sutures are applied.

Postoperatively, the limb is bandaged and supported by a splint or a cast for a few weeks. A cast would be applied in an adult horse, an excitable horse, or a foal whose step osteotomy

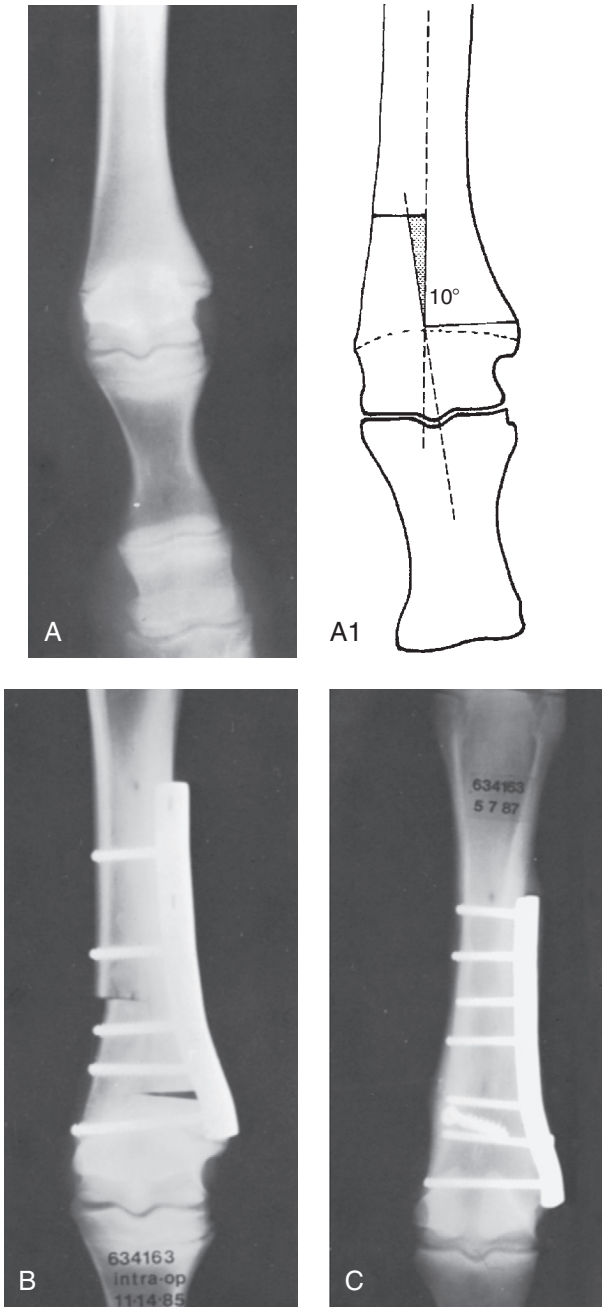

Figure 86-22. A, Dorsopalmar radiograph and diagram of the distal MCIII region with a varus deformity in an Arabian foal. **A1,** The preoperative drawing shows the 10-degree wedge to be removed from the center of MCIII. **B,** Intraoperative radiograph of the corrected deviation showing a 7-hole dynamic compression plate applied to MCIII with five screws inserted. The second and third screws from the bottom were inserted in lag fashion across the vertical aspect of the step osteotomy. The gap underneath the plate was filled with bone obtained from the wedge that was removed. The remaining screws were inserted through the plate, and an additional lag screw was placed across the vertical saw cut. **C,** Six-month follow-up radiograph of the corrected deformity. Note the excellent cosmetic result. The implants were subsequently removed. (From Auer JA: Step Osteotomy/Ostectomy for Angular Limb Deformities. p. 329. In White NA II, Moore JN (eds): Current Practice of Equine Surgery. Saunders, Philadelphia, 1990.)

was carried out near the physis, allowing only a limited number of screws in the distal fragment.

The holes drilled at the proximal and distal ends of the vertical saw cut reduce the concentration of stress at these locations and prevent postoperative fracture development.

The step osteotomy in the frontal plane is carried out similarly (see Figure 86-21, *D*).[57,58] Again, a Z-shaped osteotomy is performed across the bone, but this time the vertical cut is placed in the frontal plane. In this type of osteotomy, no bone wedge is actually removed. Once the Z-shaped osteotomy is completed, the two fragments are rotated in alignment. At that point, lag screws are inserted and a plate is applied to the bone's dorsal surface. Performing the saw cuts is somewhat more difficult with this technique; nevertheless, careful handling of the periosteum ensures no inadvertent damage to other structures.

Step osteotomies are most frequently carried out in MCIII and MTIII. However, in selected cases, they can be performed in the proximal phalanx or the radius.[56,57] The major advantages of a step ostectomy over a closing wedge ostectomy are maintenance of the bone length and the ability to create a lag effect across the vertical aspect of the saw cut and achieve good interfragmentary compression.[56,57] This cannot be accomplished in a closing wedge ostectomy. Also, the easy correction of rotational deformities is an advantage.

Step ostectomies are gaining in popularity, and a good prognosis can be given in many cases. The use of 5.5-mm bone screws is encouraged for these procedures.[57,58]

PROGNOSIS

Several studies have reported the response to both nonsurgical and surgical treatment of foals with angular limb deformities. One study reported that in 81.5% of foals treated with hemicircumferential periosteal transection and elevation (HCPTE) to correct angular deformities, total limb straightening was achieved and 60% of foals went on to be used at their intended performance level.[37,38] In a study that reported the results of transphyseal bridging, 80% of foals with carpal deformities and 27.3% of foals with metacarpophalangeal or metatarsophalangeal deformities went on to a form of athletic use.[26] The poor results obtained in the foals treated for metacarpophalangeal or metatarsophalangeal deformities were attributed to the fact that several of these foals were operated on near or after the end of the rapid growth phase of the distal third metacarpal or metatarsal growth plate. Racing performance after HCPTE in Thoroughbreds has been reported; treated foals had fewer starts at 2 years of age and lower start percentile ranks.[59] However, it is difficult to rely heavily on these comparison numbers, because many foals that have undergone surgery for ALDs are not declared as such in yearling sales. A study reporting the results of treatment in foals with tarsal valgus demonstrated that only 52.4% met the expectations of their intended use, and these authors concluded that foals with tarsal valgus have a poorer prognosis for future athletic use than do foals with carpal deformities.[34] Another study showed that foals with incomplete ossification of the tarsal bones and greater than 30% collapse of the third and central tarsal bones had a poorer outcome than did similar foals with less than 30% collapse, thereby stressing the importance of early recognition and treatment.[30]

Conflicting results regarding the significance of the location of the pivot point and the presence of radiographic abnormalities in cases of carpal deformities have been published.

According to one study, the more radiographic abnormalities that are seen distal to the distal radial physis and the more distal the location of the pivot point is, the poorer is the prognosis.[37] In another study, no such correlation could be made.[38] Also, it has been demonstrated that surgical manipulation of distal radial physeal growth can cause changes in the angles of all the carpal joints.[38] Thus surgical manipulation of physeal growth may be successful in correcting ALDs originating distal to the growth plate (e.g., epiphyseal deformities).

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CHAPTER 87

Flexural Limb Deformities

Jessica A. Kidd

The complex problem of flexural limb deformities appears often in the equine veterinary literature,[1-5] and its treatment was documented as early as the fifth century AD in the *Mulomedicina* by Vegetius.[6] Flexural deformities are a common condition of growing horses in which a joint is held in an abnormally flexed or extended position. They affect soft tissue structures and occur in the sagittal plane, as opposed to angular limb deformities, which primarily affect osseous structures and occur in the frontal plane. Persistent hyperflexion has been termed *contracted tendons*, even though in most cases the tendon units are not actually contracted but are just functionally too short relative to the associated osseous structures.[7] *Contracted tendons* implies a defect in the tendon itself and is incorrect in light of the proposed pathogeneses; this term should consequently be avoided. Tendon contractions can occur as a result of scarring secondary to tendon injury; this is sometimes seen in adult horses but is rare in foals.[8] More broadly, hyperextension deformities are considered as part of the flexural deformity complex in this chapter.

By convention, a deformity is named according to the joint involved and not the tendon. Most often an animal suffers from one type of deformity only, but several areas can be affected in severe congenital flexural deformities. The forelimbs are more commonly affected, and the problem can be encountered in more than one limb at the same time. Flexural deformities present at birth are referred to as congenital deformities. Acquired flexural deformities develop during the remainder of the animal's life, although in typical age groups. Congenital flexural deformities most commonly affect the metacarpophalangeal (MCP) joint[9] or the carpal region. Much less commonly the tarsal region, metatarsophalangeal (MTP) joint, and distal and proximal interphalangeal (PIP) joints are affected. Congenital lateral luxation of the patella can also create a functional flexural deformity of the stifle (Figure 87-1).[10] Acquired flexural deformities affect the distal interphalangeal (DIP) joint and MCP joints most frequently, with the MTP and PIP joints less frequently affected.

CONGENITAL FLEXURAL DEFORMITIES

Pathogenesis

Factors responsible for the development of congenital flexural deformities are shown in Figure 87-2. Some causes mentioned are speculative and lack scientific evidence of their existence. Work is needed to understand the intricate details that lead to the development of such deformities.

Intrauterine malpositioning is a commonly mentioned cause of the problem.[11-13] This could actually be the case in an abnormally large foal relative to the size of the mare, where intrauterine crowding leads to development of the problem. This, however, is the exception to the rule. More likely, congenital flexural deformities are multifactorial in their origin and therefore difficult to explain.

Diseases acquired by the mare during pregnancy can lead to the development of flexural deformities in the foal *in utero*.[1] A multitude of agents and causes have been associated with the problem, including ingestion of locoweed and hybrid Sudan grass during pregnancy, a dominant gene mutation in a sire, equine goiter, an influenza outbreak, neuromuscular disorders, and defects in cross-linking of elastin and collagen caused by lathyrism.[1,14-20] A glycogen branching enzyme deficiency was recently documented in Quarter Horse foals, which caused transient flexural deformities.[21] The evidence in these cases may be only circumstantial, accentuating the need for further investigations into the development of congenital deformities.

Figure 87-1. A congenital flexural deformity of the stifle caused by luxation of the patella secondary to hypoplasia of the trochlear groove.

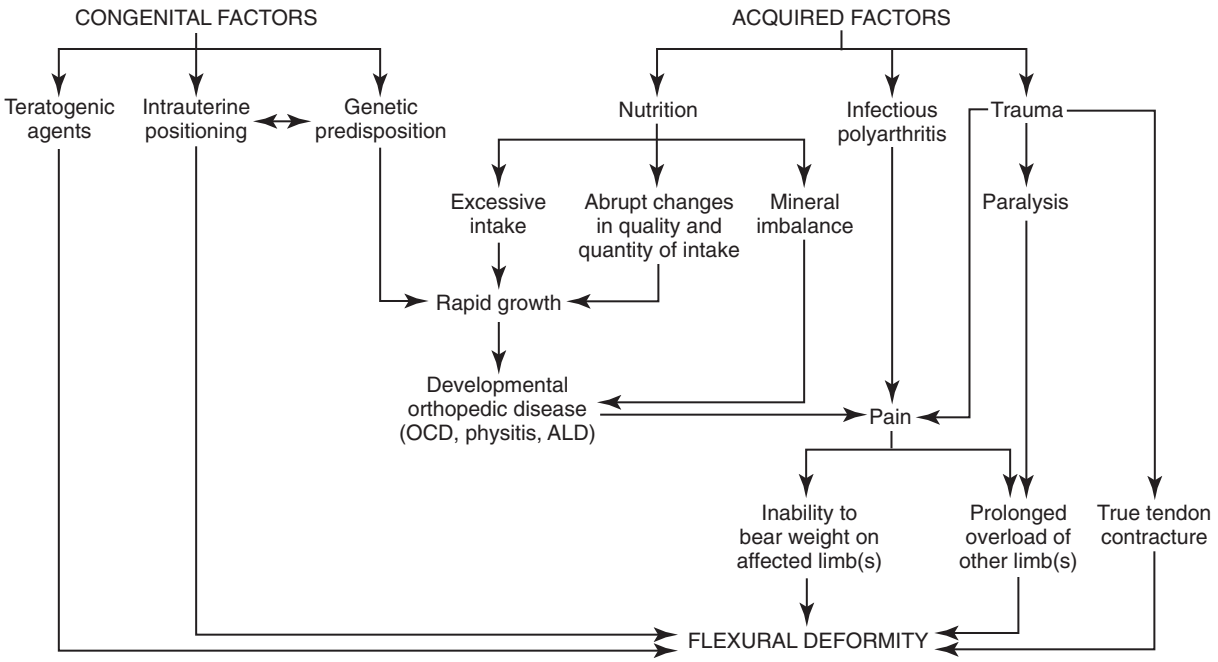

Figure 87-2. Interrelationship among the various congenital and acquired factors and their involvement in the development of flexural deformities. *ALD,* Angular limb deformities; *OCD,* osteochondrosis.

An early report stated that 20% of 608 fetuses and newborn foals submitted for necropsy suffered from miscellaneous limb contractures, which underscores the trend noted by clinicians that the incidence of flexural limb deformities is increasing.[22-24]

Diagnosis

A flexural limb deformity, whether a flexural or digital hyperextension deformity, is considered congenital when it is present at birth. The problem is easily recognized in most cases and should be evaluated by a veterinarian at that time.

Digital Hyperextension Deformities

Newborn foals can be presented with a mild degree of digital hyperextension (Figure 87-3). Such foals might be unable to maintain their toes on the ground; while standing, their MCP or MTP joints are angled more acutely than normal, and the animals are reluctant to ambulate. The problem is caused by flaccidity of the flexor muscles and usually corrects itself within a few weeks because of negative allometric growth of the tendons relative to the bones and increased muscle tone. In more severe cases, the foal walks on the palmar or plantar aspect of the phalangeal region (Figure 87-4), where skin lesions rapidly develop as a result of abnormal loading. These severe hyperextension deformities have to be distinguished from the milder forms, and the skin must be protected accordingly. Radiographic and ultrasonographic evaluations should not be necessary to diagnose the deformity because in most cases, no abnormalities will be found.

Flexural Deformities

Congenital flexural deformities may cause dystocia if they are severe enough[25] or the owner may report that the foal is unable to stand. Some flexural deformities may be overlooked in recumbent foals if the joints can be manually straightened, but they should become apparent once the foal is assisted to stand. Joints should be manipulated to see if manual straightening is possible.

Figure 87-3. A newborn foal with digital hyperextension in all four feet. Additionally a mild flexural deformity is present in both carpi.

DISTAL INTERPHALANGEAL REGION

Flexural deformities of the DIP joint are more common as an acquired deformity. A flexural deformity of the DIP joint seen at birth is usually associated with a flexural deformity of the fetlock.[10]

Figure 87-4. A premature foal showing severe digital hyperextension. The foal responded quickly to exercise restriction.

METACARPOPHALANGEAL/METATARSOPHALANGEAL REGION

Mild cases of MCP/MTP flexural deformities can be overlooked if the foal is examined in grass or deep bedding, whereas patients with more severe deformities may be unable to stand unassisted. Mild flexural deformities usually resolve spontaneously in 4 to 5 days with limited exercise. Patients that do not respond to conservative therapy can be splinted, treated medically, or both (Figure 87-5). One author reports that if there are no osseous changes to the joint or the joint can be manually straightened, the prognosis is good for all degrees of severity of MCP/MTP flexural deformities.[26,27] If the deformity is very severe, radiographs should be taken first to rule out abnormally formed bones, which decreases the prognosis to poor.[26]

CARPAL REGION

Carpal flexural deformities are usually bilateral, and in mild cases, the foal can stand but cannot completely straighten the carpi (Figure 87-6). Mild cases are usually self-correcting in 4 to 5 days with limited exercise, but if the deformity does not resolve spontaneously, other treatments may be instituted (see later). More severely affected patients may be unable to stand, and the common digital extensor tendon may rupture secondary to the flexural deformity (see later). If the limbs can be manually straightened (which may require sedation), the chance of resolution using splints or casts is good, but the prognosis is guarded if the carpus cannot be manually straightened.[26,28]

PROXIMAL INTERPHALANGEAL REGION

Congenital PIP joint flexural deformities are rarely reported and often involve both osseous and soft tissue abnormalities.[26] Cases may show subluxation of the PIP joint or ankylosis of the PIP joint in more severe cases.

TARSAL REGION

Congenital flexural deformities of the tarsal region are rare and are most often the result of incomplete ossification of the tarsal bones, resulting in a secondary flexural deformity. Cases must be identified before the bones are irretrievably damaged, after which time there is no effective treatment (see Chapter 86).

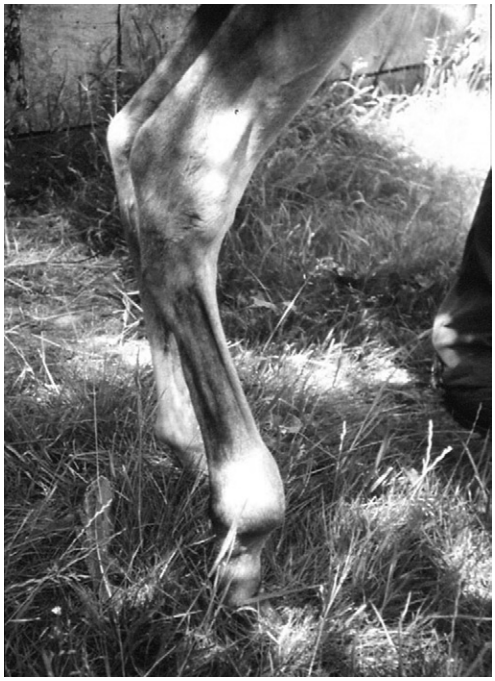

Figure 87-5. A congenital flexural deformity of the metatarsophalangeal joint. The foal responded to splinting, oxytetracycline administration, and analgesics.

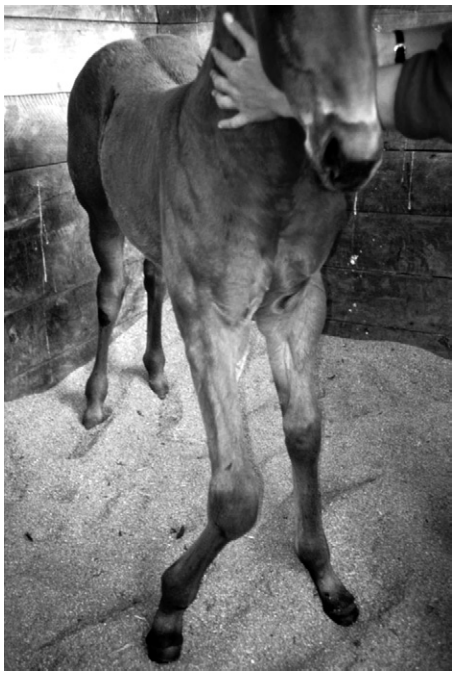

Figure 87-6. A flexural deformity of the carpus. The foal is unable to completely straighten the limb.

Radiography is indicated in these cases for this reason (Figure 87-7).

RUPTURED COMMON DIGITAL EXTENSOR TENDON

This relatively common congenital disorder has some pathognomonic features that simplify the diagnosis.[4,5,29] Affected foals display a characteristic swelling in the tendon sheath at the

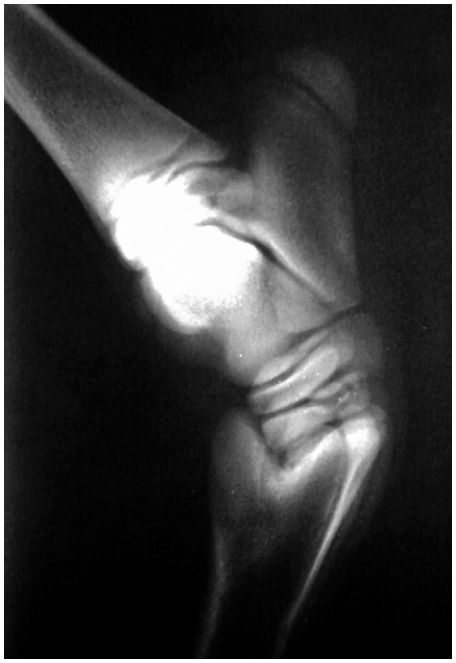

Figure 87-7. A lateromedial radiographic view of a newborn foal with an anomaly of the tarsometatarsal joint. The small tarsal bones have collapsed through the subchondral bone plate of the proximal third metatarsal bone. Surgical correction in such a case is not possible or at least not feasible.

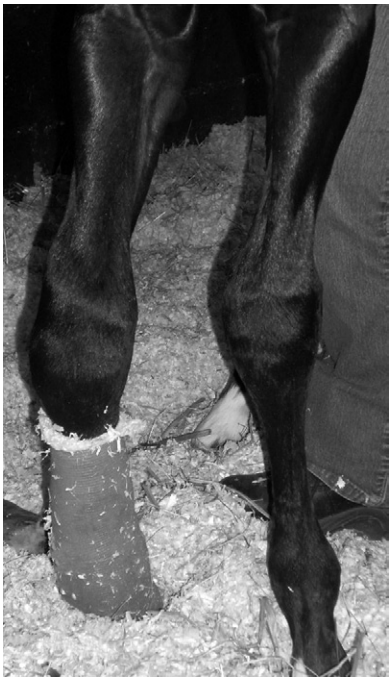

Figure 87-8. Rupture of the common digital extensor tendon in the right forelimb of a young foal, which can create the appearance of a flexural deformity. Note the characteristic bulge over the dorsolateral aspect of the carpus. The foal also has a moderate carpal valgus deformity in the left forelimb.

dorsolateral aspect of the carpus (Figure 87-8), which is recognized soon after birth, but in rare cases it is not evident for up to 3 weeks. Rupture of the common digital extensor tendon is often seen secondary to a flexural deformity and less commonly can lead to a flexural deformity.[30] The foal often has a slightly bowlegged and an over-at-the-knees stance. This stance is caused by the lack of support at the dorsolateral aspect of the carpus, which is normally conferred by the intact common digital extensor tendon. Therefore it is not truly a flexural deformity, but it clinically appears as one. The two ends of the partially or completely ruptured tendon can be palpated in the tendon sheath. With time, these ends proliferate, making their detection during palpation even easier.

During walking, affected animals throw their forelimbs forward, extend them completely, and retract them slightly before contacting the ground. A foal with a ruptured common digital extensor tendon often knuckles at the MCP joint during walking and might buckle in the carpal region while standing. In severe cases the foal may not be able to stand straight without knuckling forward (Figure 87-9).

Radiography for the Diagnosis

Flexural deformities, which can be diagnosed on the basis of clinical signs, do not need to be radiographed, but is useful to identify abnormalities that may alter the prognosis for correction of the deformity. Radiographs of carpal flexural deformities that cannot be manually straightened may reveal incomplete ossification or wedging of the carpal bones, decreasing the prognosis for correction. The same applies for the less common tarsal flexural deformities.

Figure 87-9. The same foal as in Figure 87-8. Because of a lack of extensor support, the foal was unable to walk without stumbling. Application of a distal limb splint improved the foal's ability to walk and support weight on the limb.

Treatment

Digital Hyperextension Deformities

Most foals with mild digital hyperextension do not need treatment other than minimal attention and trimming of the feet; the condition is self correcting.[31] Moderate exercise is indicated to strengthen the musculotendinous unit; therefore access to pasture is allowed. Excessive exercise is contraindicated because fatigue often aggravates the problem. The animals should be carefully observed, and if the problem worsens, further treatment should be promptly initiated. Severe digital hyperextension problems must be treated immediately, because neglect soon leads to necrosis and traumatization of the skin in the palmar or plantar phalangeal region and jeopardizes treatment.

SWIMMING

Swimming has been advocated as excellent controlled physiotherapy.[4] The animal is supported in a swimming pool or pond by one or two helpers or a rescue net (Figure 87-10). The paddling action of the foal in the water is carried out against the resistance of the water and without placing weight on the limbs. The resultant increase in muscle tone brings about rapid amelioration of symptoms.

FARRIERY

Trimming is often unnecessary, but the toe can be shortened with rasping and the palmar/plantar half of the foot also lightly rasped for increased contact with the ground.[31] In more severely affected foals in which the toe is lifted off the ground and the foal is ambulating on the palmar/plantar pastern, application of glue-on shoes or similar devices with palmar or plantar extensions helps maintain the hoof sole on the ground (Figure 87-11).[32] Ready-made or custom-made extensions are used; a cuff type of shoe is available (Dallmer cuff shoes), or the extension can be made from light aluminum, which is cut to shape, curved over the toe for contact, and attached with acrylic adhesive (Equilox) (Figure 87-12). Some farriers feel that any shoe should be attached with adhesive tape only in foals younger than 3 weeks to avoid heat trauma to the delicate foot and contracture of the heels.[31] Extensions are changed at 10 day intervals, although some foals dislodge the extensions before this. These devices constrict the foot and can deform the foot if left in place too long.

BANDAGING

Light bandaging of the phalangeal region is indicated to minimize skin trauma in foals that walk on the back of their pasterns, but the bandages should not be substantial enough to

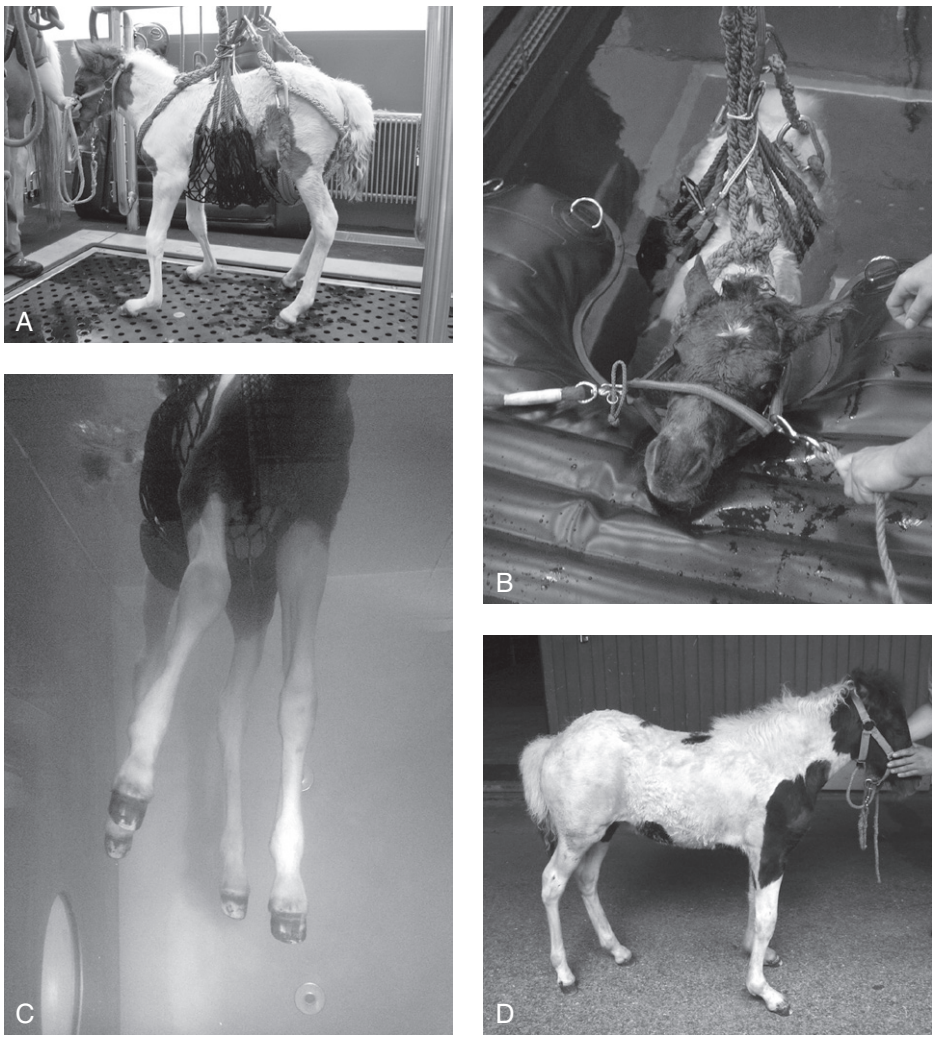

Figure 87-10. A, A 2-week-old Icelandic pony is shown with marked digital hyperextension in both forelimbs. The foal wears a rescue net and stands on a scissor table. **B,** The table was lowered into warm water so that the foal was immersed in it, protected by the rescue net, which was attached to a hoist. **C,** The foal is shown swimming and in doing so, effectively exercises its weak limb muscles. **D,** The same foal 4 weeks later. There is some improvement, but the fetlock angle is still hyperextended. (Courtesy J. Auer, Zurich, Switzerland.)

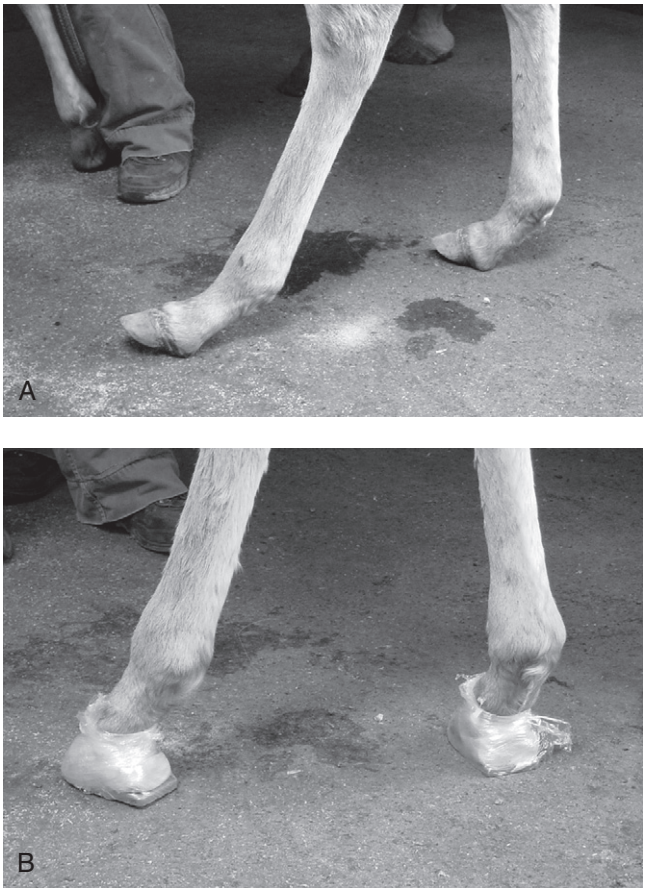

Figure 87-11. A, A newborn foal with marked digital hyperextension in the rear limbs. **B,** The same foal immediately after application of a pair of Dalric glue-on shoes with the clear plastic wrap ensuring good contact between the shoe and the hoof wall during the hardening process of the polymethyl methacrylate (PMMA) still in place. Note the ability of the foal to bear weight on the soles after shoe application. (Courtesy J. Auer, Zurich, Switzerland.)

completely support the distal limb. Splint bandages and casts incorporating the foot are contraindicated because they totally support the distal limb, leading to a further loss of tone of the already hypotonic flexor tendon units. Additionally, development of pressure sores on the delicate skin is a common untoward sequela.

Application of some padding over the elongated braces attached to the foot will protect the palmar or plantar aspect of the phalangeal region, decrease the excessive hyperextension angle, and decrease the likelihood that the extensions will be dislodged by the mare or the foal (Figure 87-13). In addition to benefiting the cardiopulmonary system, daily swimming would strengthen the muscles, tendons, and ligaments, and in doing so they help to correct the deformity. Carefully dosed anti-inflammatory drugs are indicated to keep the foal comfortable.

SURGICAL MANAGEMENT

Tenoplasty has been described as a possible surgical management technique for severe digital hyperextension problems in small or miniature foal patients, but it is currently not recommended.[33]

Figure 87-12. Application of metal palmar extensions in a standing sedated foal with a severe digital hyperextension in the hind feet. The metal strips are curved over the toe and secured to the foot with acrylic.

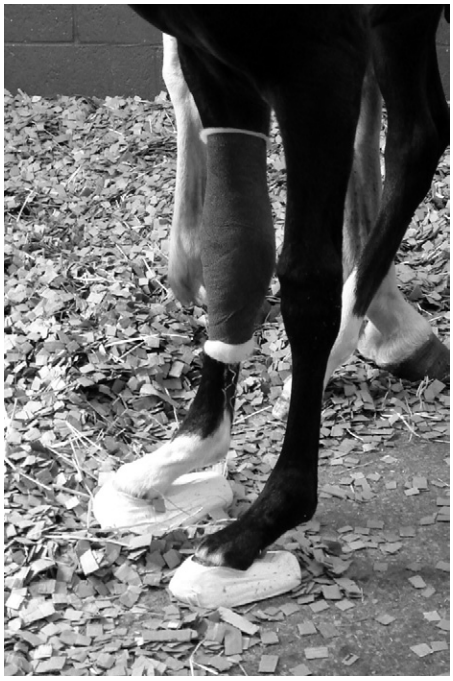

Figure 87-13. A foal with marked digital hyperextension shod with metal palmar extensions, which have been covered with padding and elastic bandage to provide additional protection to the palmar pasterns and to prevent injury to the mare and foal from the metal extensions.

Flexural Deformities

NONSURGICAL MANAGEMENT

There are many available treatment modalities for the treatment of congenital flexural deformities and they can all be used in conjunction with one another. It is important to ascertain

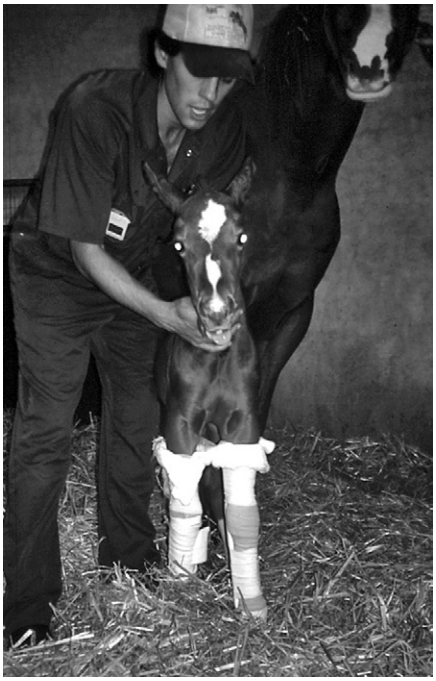

Figure 87-14. A foal with flexural deformities of both carpi, which were treated with application of splints, oxytetracycline, and analgesics. Because both forelimbs were splinted, the foal required assistance to stand but could walk in the splints unaided once it was upright.

whether the foal can stand without assistance, because in these cases, specific treatment is often not required.[30]

EXERCISE

Congenital flexural deformities are best treated with moderate exercise. In many cases, the foal is unwilling to stand and walk and needs regular encouragement to do so, especially if a cast or a splint is applied (Figure 87-14). The treatments discussed later can then be used singly or in combination.

ANALGESICS

Both the primary cause and the effects of treatment of flexural deformities may be painful, and affected foals should be treated with nonsteroidal anti-inflammatory drugs (NSAIDs). However, these drugs should be given judiciously because of the potentially detrimental side effects, including gastric ulceration and nephrotoxicity. Phenylbutazone at 1.1 mg/kg body weight IV or orally once a day or flunixin meglumine at 1.1 mg/kg IV or orally once daily are typically prescribed. Concurrent treatment with omeprazole (4 mg/kg orally once daily) or ranitidine (6.6 mg/kg orally three times a day, or 1.5 mg/kg IV three times a day) has been proposed as gastric protectants.[34] Ideally, biochemical monitoring of the foal's renal parameters (blood urea nitrogen, creatinine, and total protein) should be performed in these cases.

INTRAVENOUS OXYTETRACYCLINE

Administration of oxytetracycline has become popular as an initial treatment for congenital flexural deformities.[35] A single dose of 3 g oxytetracycline in 250 to 500 mL of physiologic saline is administered slowly by the intravenous route. The treatment may be repeated once or twice within the first weeks of life if necessary. Previously, it was thought to act by chelation of calcium and inhibition of muscle contraction, although intuitively, if this were the mechanism of action a more widespread effect on the body would be noted. A more recent *in vitro* study showed that oxytetracycline induced a dose-dependent inhibition of collagen gel contraction by equine myofibroblasts. Oxytetracycline also induced a dose-dependent decrease in matrix metalloproteinase 1 (MMP-1) mRNA expression by equine myofibroblasts.[36] Results of this study indicate that oxytetracycline inhibits tractional structuring of collagen fibrils by equine myofibroblasts through an MMP-1–mediated mechanism.[36]

In young foals, oxytetracycline administration can make the developing ligaments and tendons more susceptible to elongation during normal weight bearing, resulting in correction of the deformity within 24 to 48 hours. In a comparative study, the clinical effect of a single 44 mg/kg dose of intravenous oxytetracycline in normal foals and foals with flexural deformities was examined.[37] Treatment with oxytetracycline resulted in a significant decrease in the MCP joint angle as measured radiographically in both affected and unaffected foals. Joints returned to their pretreatment angles by 4 days after treatment. Oxytetracycline therapy had no significant effect on the DIP joint angle and therefore is unlikely to be of use for flexural deformities of the DIP joint based on these data. No alterations in renal biochemical parameters were detected after a single dose. However, oxytetracycline induced acute renal failure in a foal, which was successfully treated by hemodialysis.[38] This underscores the importance of biochemical monitoring in these cases, particularly in systemically ill foals.

Mild cases of flexural deformities can respond to this treatment with complete correction. In more severe cases, the response is minimal and other treatment modalities must be used.

FARRIERY

Toe extensions

Application of a dorsal hoof extension using acrylic alone or in combination with a lightweight foot plate protects the toe from excessive wear and increases the tensile forces in the deep digital flexor tendons during ambulation (Figure 87-15). This facilitates a delay in breakover in foals suffering from flexural deformities of the DIP and MCP/MTP joints. In severe deformities, however, toe extensions that are not braced back along the dorsal hoof wall can exert significant forces at the toe, leading to distraction and separation of the dorsal hoof wall, infection of the white line, and pain.

To help maintain these devices on the feet, numerous holes of approximately 2 mm in diameter and depth can be drilled into the dorsal hoof wall. The hoof acrylic used to achieve the dorsal extension is spread over the dorsal hoof wall and worked into the holes to add stability to the device. The acrylic, interdigitating with the dorsal hoof wall, serves as an anchoring device and prolongs the life of the extensions. Filling in the space between the extension and the dorsal hoof wall decreases the risk of the foal's stepping on the extensions or stumbling over them.

These devices may be successfully applied in newborn foals with mild flexural deformities of the DIP or MCP joints, when the foal cannot bear weight without knuckling over. Extending the dorsal hoof wall adds the necessary elongation to compensate for the lack of extension in the phalangeal region and allows the foal to stand without knuckling. Once the foal is able

to ambulate, correction of the deformity is usually achieved within 2 weeks, at which time the extensions are removed or typically become detached.

The toes must be protected from excessive wear as the foal is allowed free exercise. A special half-round glue-on shoe has been developed for this purpose, with a toe extension built into its design (Figure 87-16).[39]

SPLINTS AND CASTS

Splints and casts are effective for treating flexural deformities. Splints and casts are useful in cases of MCP/MTP, carpal, and some tarsal flexural deformities. The PIP and DIP joints are difficult to immobilize without casting. Foals suffering from flexural deformities of the DIP joint that are unresponsive to hoof extensions can be treated with half-limb casts, incorporating the feet. This treatment relaxes the muscle–tendon units and corrects the problem.[3,4,40]

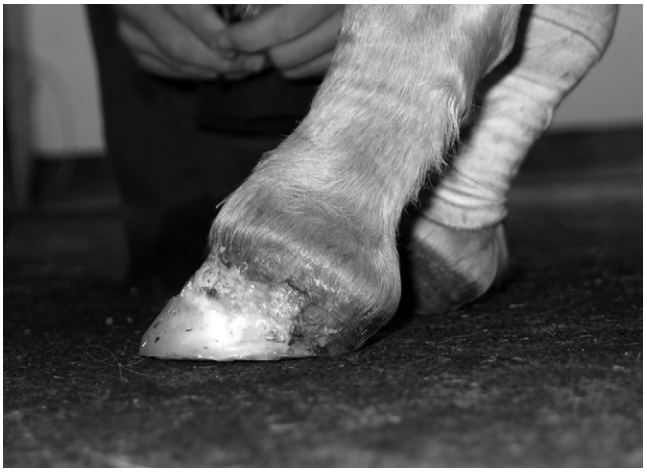

Figure 87-15. A polymethyl methacrylate toe extension applied to a foal following a desmotomy of the accessory ligament of the deep digital flexural deformity for treatment of a flexural deformity of the distal interphalangeal joint. The toe extension stretched the deep digital flexor musculotendinous unit, and by filling the gap between the toe extension and the dorsal hoof wall with acrylic, the distraction forces between the hoof wall and the underlying laminae are neutralized.

Splints have several advantages over casts in that they can be removed regularly, are easily reapplied, can be applied under sedation, and are more cost-effective than repeated casting. Casts cannot be reset regularly (unless they have been bivalved) without added expense, often require general anesthesia to apply, and do not allow inspection of the flexural deformity and the skin under the cast. Because the forced extension of the affected limb in a cast or a splint is painful and requires analgesia, some clinicians avoid the use of casts, preferring instead to use splints, which can be placed on the leg for 12 hours and then left off for 12 hours.[34] Splints however, can move out of position on the limb, creating pressure sores. Some clinicians favor the use of casts for these reasons.[41] Casts have to be changed at least every 2 weeks to keep pace with the growth of the foal.

Splints can be made from a variety of materials, including PVC pipe, wood, and fiberglass, and can be custom-made to the limb.[42] Splints made from PVC pipe or wood have the disadvantage of a lack of conformation to the limb and the potential to cause pressure sores. More severe problems such as distal limb necrosis as a result of improperly applied splints have been reported.[43] Splints should be placed over enough padding that the skin is protected from excoriation, but little enough that the splints do not shift out of position. The splints often rotate around the leg, and it is often quite difficult to keep them in the desired position.

Splints that have been custom fit to the contour of the limb are very useful and decrease the likelihood of complications because of their custom-made nature. In foals with a single limb affected, the custom splint can be made from the contralateral unaffected leg. The splint is made from a splinting material consisting of a double layer of seven strips of fiberglass encased in a synthetic padding (Dynacast Prelude) using the unaffected limb as a template. The same construct can be made from strips

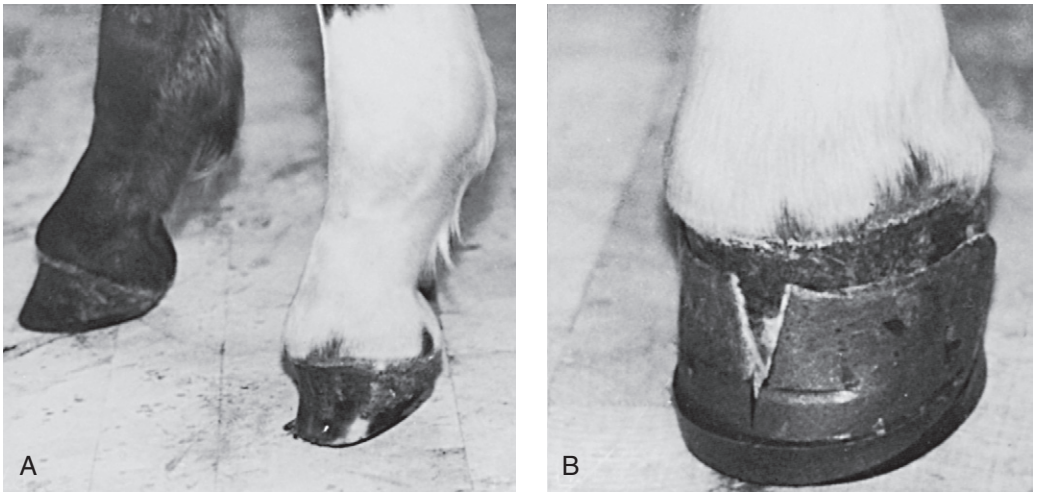

Figure 87-16. A, A 4-month-old foal with a stage I flexural deformity in the distal interphalangeal joint of the left forelimb. Note the dished appearance of the dorsal hoof wall. Allowing the foal to walk around like this would result in excessive wear at the toe and possible development of a painful condition or infection. **B,** A Dalric glue-on shoe was applied to protect the toe and to extend the footing surface dorsad, facilitating delayed breakover during walking. Note the spread cut in the region of the dorsal hoof wall. This allows better fit of the shoe to the foot because of the limited sizes available. (Courtesy H. Dallmer, Salzhausen-Putensen, Germany.)

of 10-cm casting material stacked and encased in padding. The technique is detailed in Chapter 17. The disparity between the affected limb and the splint created from the unaffected limb can be substantial (Figure 87-17). The affected leg is then padded with the same amount of padding used when preparing the splint. The splint is placed on the affected limb and forced into the splint configuration under bandage pressure. The splint is reset daily, and the leg is examined for any evidence that the splint is rubbing. Each day the limb should be able to be pulled further into the splint.

Figure 87-17. A custom-made splint for a foal with a flexural deformity of the metacarpophalangeal region. The splint has been made using the normal metacarpophalangeal region as a template and is now ready to be placed on the limb with a flexural deformity. The extent of the deformity can be seen as the disparity between the splint and the affected limb.

Some foals require sedation to apply the splints to facilitate maximal extension of the affected joint and to allow the splint to be placed on the limb properly. α_2-Adrenoreceptor agonists such as xylazine or detomidine are useful for this purpose and have the added benefit of providing analgesia.

Splints are preferably used in flexural deformities of the MCP and carpal region, especially in mild carpal flexural deformities. Foals that buckle forward on their MCP/MTP joints have lax flexor tendons and tight extensor tendons; these foals should be splinted to allow loading of the flexor tendons.[30] In these cases, the phalangeal region should not be incorporated into the splint.[40] Splints have to be well padded and changed regularly. It is important to use new, dry padding at each bandage change to minimize development of pressure sores (Figure 87-18).

Foals that are unable to rise and nurse must be assisted many times daily. Stretching of tendons and associated contracted soft-tissue structures (e.g., joint capsules) is painful; therefore administration of low doses of anti-inflammatory drugs is necessary (see "Analgesics," earlier).

SURGICAL MANAGEMENT

Surgical intervention is seldom necessary with congenital flexural deformities and is used in severe cases or those that do not respond to medical therapy. Surgical treatment is most commonly carried out for carpal flexural deformities, and transection of the flexor carpi ulnaris and the ulnaris lateralis tendons 2 cm proximal to the accessory carpal bone can correct mild carpal deformities.[44] A recent report of 135 procedures on 72

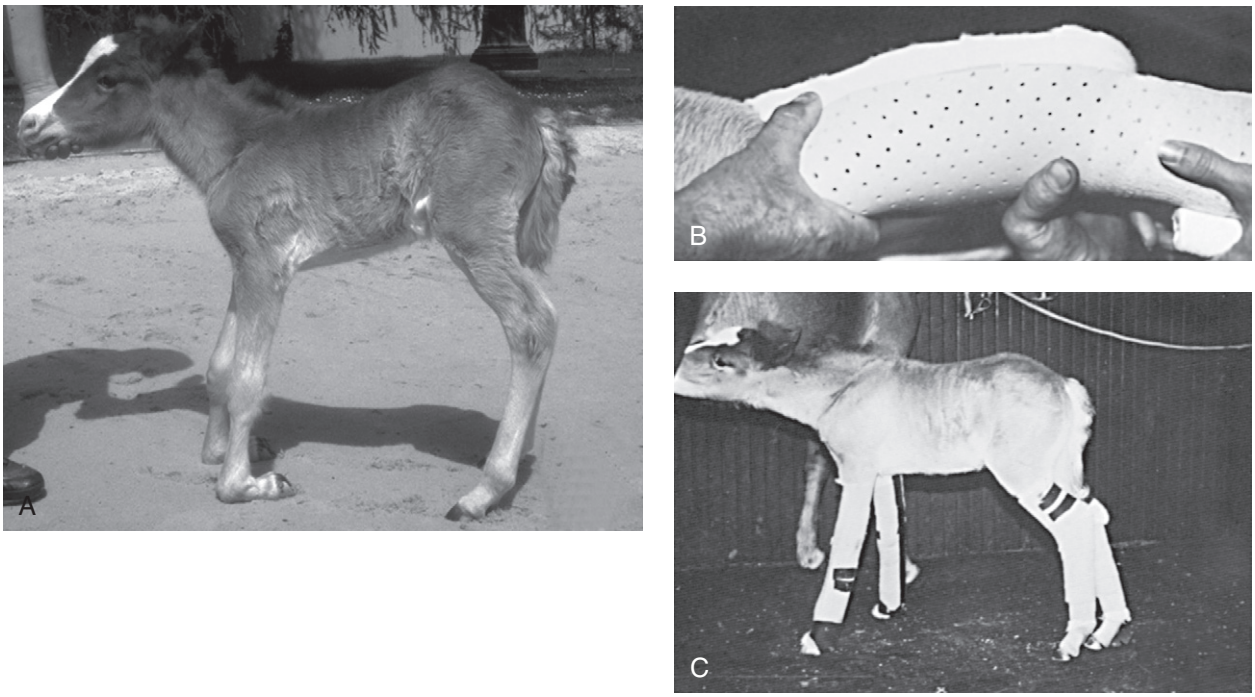

Figure 87-18. Splint application to a foal suffering from rupture of the common digital extensor tendon and incomplete ossification of the carpal and tarsal bones. **A,** The foal knuckling over on both front feet. **B,** A piece of Plastazote is applied to the dorsal and cranial aspect of MTIII and distal tibia, respectively, to manage incomplete ossification. This padding material is suited to application to the tarsal region. **C,** The foal with all four splints applied. Note that the splints were applied without incorporating the feet. In the forelimbs the splints are located palmarly and caudally, and in the hindlimbs they are located dorsally and cranially.

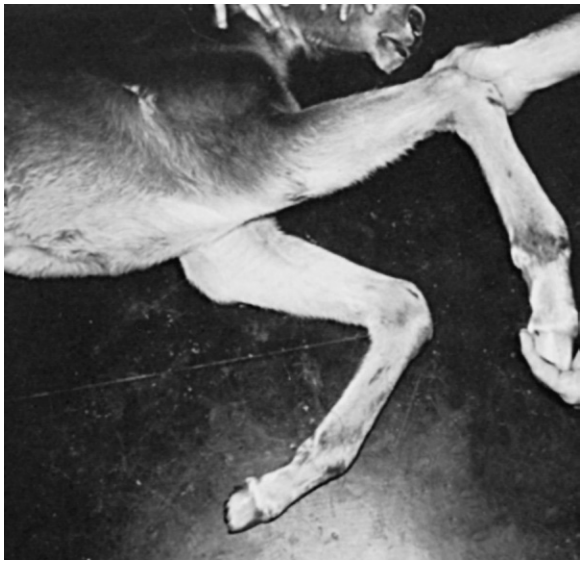

Figure 87-19. A foal with a severe flexural deformity in the carpal region, which was unresponsive to treatment.

horses documented a successful outcome, defined as a straight palmar carpal angle, in 82% of the cases.[27] Cases were graded as 1, 2, or 3, in order of increasing severity. Lower grades with less than 40 degrees of flexion carried the best prognosis, and the success rate in grade 3 cases fell to 57%. The surgery is performed under general anesthesia; a vertical incision is made over the lateral aspect of the accessory carpal bone and the tendons identified deep to the fascia. It is advisable to manipulate the limb immediately before surgery, while the foal is under anesthesia, to ascertain if these tendons tighten when forceful carpal extension is applied. If these tendons are the structures preventing extension of the carpus, once they have been transected surgically, the limb can be manually straightened. After surgery, box confinement is recommended for a few days, and then access to a small paddock or yard is allowed. The limb is kept bandaged until the sutures are removed.

Occasionally, surgical transection of the flexor tendons and palmar capsule of the middle carpal and antebrachiocarpal joint is required to allow the limb to be straightened, but these cases carry a worse prognosis (Figure 87-19).[45] The carpal canal is opened through a medial approach; the joints are identified and subsequently opened through a horizontal incision.

Flexural deformities of the MCP/MTP joint that do not respond to medical treatment can be treated surgically by transection of the flexor tendons or suspensory ligament, but these salvage procedures are not recommended for animals intended for an athletic future.[30] Severe MCP/MTP flexural deformities secondary to abnormally formed bones have been treated using an arthrodesis and resulted in pasture-sound horses.[46]

PIP joint flexural deformities resulting in subluxation may be treated by means of an arthrodesis (see Chapter 81).

Ruptured Common Digital Extensor Tendon

NONSURGICAL MANAGEMENT

Foals with ruptured common digital extensor tendons are best confined to a box stall, because they tend to stumble frequently. Within a few weeks, locomotion normalizes without any other treatment, and more exercise can be allowed.

Application of a well-padded splint bandage to stabilize the MCP joint and allow the foal to walk without stumbling is the treatment of choice when treatment is required.[29] The use of a thermoplastic splinting material (Plastazote) or a custom-made fiberglass splint has had excellent results (see "Splints and Casts," earlier). The splints must be monitored carefully for slipping or causing sores, and they are usually required for several weeks until the ruptured ends have fibrosed and allow a return to a normal ambulation. The prognosis for recovery is excellent, but some foals have a persistent thickening over the dorsal carpus for 6 to 12 months.[47] Aspiration of the synovial fluid from the tendon sheath is discouraged because of the risk of infection.

SURGICAL MANAGEMENT

It is generally accepted that ruptured common digital extensor tendons should not be treated surgically because of the favorable prognosis with conservative management.[29] The risk of complications secondary to surgery outweighs any improvement of the prognosis. However, thorough removal of all fibrin clots from the tendon sheath through a surgical incision, followed by installation of a suction drain and cast immobilization, has resulted in excellent cosmetic appearance of the limbs.[44]

ACQUIRED FLEXURAL DEFORMITIES

Pathogenesis

There are several pathways for the development of acquired deformities (see Figure 87-2). It has been suggested that acquired flexural deformities are part of the developmental orthopedic disease (DOD) complex, which also includes angular limb deformities, osteochondrosis, physitis, and cervical vertebral malarticulations or malformations.[24] Although they are a condition of the developing horse, the DOD complex initially was defined as conditions resulting from a failure of the conversion of cartilage to bone and in the context of this definition, acquired flexural deformities were not included.[24] However, it is likely that acquired flexural deformities occur as a result of pain and may therefore be a sequela to the conditions listed earlier. It is likely that the etiopathogenesis of acquired flexural deformities is multifactorial and complex, but several theories have been proposed for its occurrence. The two main theories are a mismatch in bone and tendon/ligament growth and contraction of the musculotendinous unit in response to pain.

It has been postulated that in rapidly growing foals, the longitudinal growth of the bone is greater than the potential of the tendon unit to elongate passively at a corresponding rate.[8] Most acquired flexural deformities are encountered between 4 weeks and 4 months of age and again at the yearling age, an observation that supports the theory.[3-5,7] Rapid bone growth occurring between 4 weeks and 4 months can induce a flexural deformity in the distal interphalangeal joint. Passive elongation of the tendon might be limited because of the relatively unyielding accessory ligament of the deep digital flexor tendon, which originates at the proximal and palmar aspects of the third metacarpal bone (MCIII) and joins the tendon in the midmetacarpal region. Passive elongation proximal to that region, therefore, has little influence on preventing the problem. Because of the

functional shortening of the deep digital flexor tendon unit, excessive tension is exerted on the distal phalanx, which results in palmar rotation of the entire foot and the development of the typical clubfooted stance.

The rate of bone growth is determined by genetics and nutrition. Foals can be overfed either by heavily lactating mares or by excessive supplementation with concentrates.[48] An abrupt change from inadequate—both in quality and quantity—nutrition to abundant nutrition also can induce the problem in yearlings.[49]

At 3 months of age, growth at the distal MCIII has ceased but continues to occur at the distal radius. The accessory ligament of the superficial digital flexor tendon originates just proximal to the distal radial growth plate from the caudal aspect of that bone. Rapid growth of the radius around 1 year of age can result in a functional shortening of the superficial flexor tendon unit and development of a flexural MCP deformity.[50] This theory was validated experimentally when foals previously kept on a poor ration were fed free-choice high-quality feed at the yearling age and subsequently developed flexural deformity.[51] However, the development of the problem might be more related to nutritional imbalances than excessive high-energy intake.[49,52]

Other authors have theorized that longitudinal bone growth is insufficient at any age to create a relative shortening of the flexor tendons.[34] They postulated that rapid growth increased tension within the flexor tendons and that this could induce pain, ultimately leading to the development of flexural deformities. Flexural deformities often have an acute onset of 24 to 48 hours, which also supports this theory; though muscle contractions can develop in such a short period, bone lengthening would take longer.

A study of skeletally normal foals revealed that most of the cells in the deep digital flexor tendon and its accessory ligament are myofibroblasts.[53] These cells have contractile ability and therefore might play a role in the development of flexural deformities.

This leads to pain as the primary inciting factor associated with acquired flexural deformities.[12,31] Any painful condition could be responsible for the *flexion withdrawal reflex* and the resultant muscle contraction, leading to an altered stance, and it is common for lameness in the affected limb to precede development of an acquired flexural deformity.[30] Osteochondrosis, osteoarthritis, infectious joint disease, acute severe trauma to osseous and soft-tissue structures, bruised feet as a result of prolonged exercise on hard ground,[54] or overzealous foot trimming leading to solar bruising[31] and hoof problems are just a few conditions that may precipitate an acquired flexural deformity.[5,54-56] Such painful processes can induce muscle contractions, leading to the development of flexural deformities. Although pain can be the inciting factor in acute-onset flexural deformities, more permanent states of flexion of the musculotendinous unit can occur with contracture of the flexor aspect of the joint capsule and maintain the deformity, underscoring the need for early diagnosis and effective management.[55,56]

Diagnosis

Acquired flexural deformities are seen more often than acquired hyperextension deformities. Continuous overload of certain limbs can, in selected cases, induce hyperextension deformities. Acquired flexural deformities can also be encountered after ruptures of flexor tendons. The onset of acquired deformities can be divided into two periods of the foal's growth (Table 87-1). Early diagnosis is important to increase the chance of complete resolution, but diagnosis may be made difficult by the foal's being out at pasture with the mare, especially for DIP joint deformities, which may be hidden by the grass. Regular examination on a hard, level surface will aid early detection of acquired deformities. The MCP and DIP joints are most commonly affected.

TABLE 87-1. Age of Onset of the Most Commonly Encountered Flexural Deformities

Age of Onset	Deformity
CONGENITAL DEFORMITIES	
Birth up to 1 month	Carpus
	Metacarpophalangeal region
Less commonly	Metatarsophalangeal region
	Proximal interphalangeal region
	Distal interphalangeal region
Rarely	Tarsal region
ACQUIRED DEFORMITIES	
1 to 4 months	Distal interphalangeal region
1 to 6 months	Metacarpophalangeal region
	Proximal interphalangeal region
Less commonly	Metatarsophalangeal region

Distal Interphalangeal Region

Flexural deformities of the DIP joint occur primarily in foals between 1 and 4 months of age and almost always affect the forelimbs. The condition is usually bilateral, although one limb may be more severely affected. Deformities of the DIP joint involve the deep digital flexor tendon, because this tendon inserts on the solar surface of the distal phalanx and is responsible for flexion of the DIP joint. Initially, the dorsal hoof wall assumes a more vertical angle and the heels may not contact the ground if the condition has occurred acutely. Two sequelae are associated with a foot in this conformation. First, the heels overgrow because of the lack of ground contact, and the foot appears "boxy" as the heels approach the length of the toe. Second, the toe is under greater stress and wear, which can widen the white line and lead to flaring of the distal hoof wall. In more slowly developing cases, the heels may maintain contact with the ground and overgrow.[30] These changes in the conformation of the foot are the result of the deformity and not a cause.

The deformities are divided into stages I and II. Stage I deformities (Figure 87-20) have a more upright dorsal hoof wall where the angle described by the dorsal hoof wall and sole is greater then 60 degrees but less than 90 degrees, and the wall has not passed the vertical plane. In stage II deformities (Figure 87-21), the dorsal hoof wall has passed 90 degrees and is beyond the vertical plane. Stage II deformities have a worse prognosis for resolution than stage I deformities,[57] and the prognosis declines with increased time before treatment.[28] This classification scheme is useful for description of the deformity and formulation of a prognosis, but it does not always dictate the preferred method of treatment.

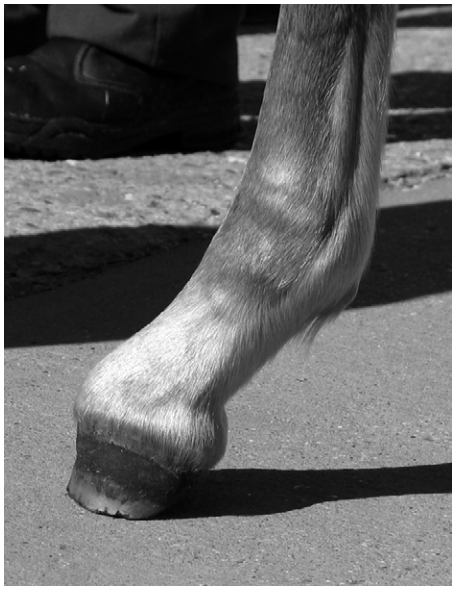

Figure 87-20. A stage I flexural deformity of the distal interphalangeal joint. Note that the dorsal hoof wall has not passed the vertical and that the distodorsal tip of the hoof wall has flared from the abnormal forces caused by the abnormal foot position.

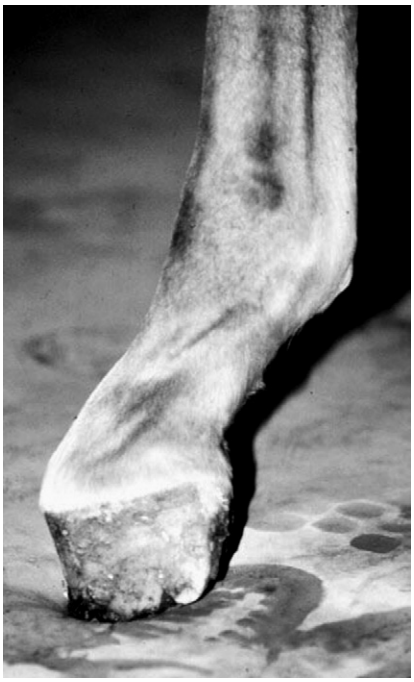

Figure 87-21. A stage II flexural deformity of the distal interphalangeal joint in which the dorsal hoof wall has passed vertical. (Courtesy Alistair Barr, University of Bristol, UK.)

The longer the condition is neglected, the worse the deformity becomes. Permanent changes of the associated soft-tissue structures occur, and the abnormally loaded bones remodel according to Wolff's law.[5] The deformation and distraction of the dorsal hoof wall can lead to seedy toe or subsolar abscesses, in turn causing more pain.

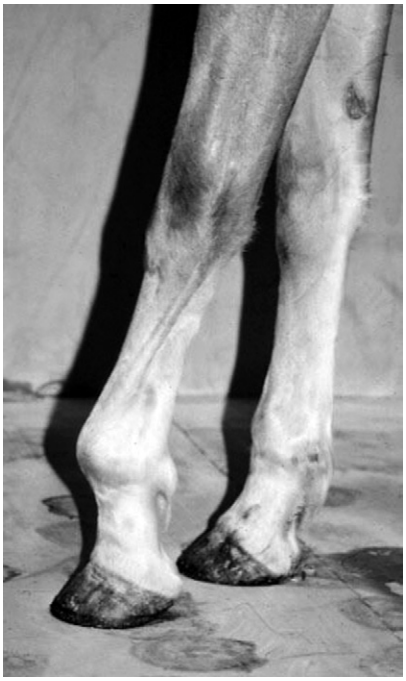

Figure 87-22. An acquired flexural deformity of the metacarpophalangeal joint in which the fetlock joint knuckles forward. The animal is bearing full weight, but the metacarpophalangeal region does not extend fully and there is no loading on the flexor tendons. (Courtesy Alistair Barr, University of Bristol, UK.)

Metacarpophalangeal/Metatarsophalangeal Region

These flexural deformities can be congenital but are also an acquired flexural deformity in animals from 10 to 18 months of age. Both front and back limbs can be affected, but acquired MTP joint flexural deformities are much less common than those affecting the MCP joint.[58] Acquired flexural deformities of the MCP joint are initially characterized by a straighter MCP angle. The foot usually appears normal and is in contact with the ground, but the pastern assumes a more upright position, and if the deformity is left untreated, the MCP joint knuckles forward. Knuckling over in the MCP region is seen in more severe cases (Figure 87-22) and in cases in which treatment has been neglected for a long time.

Three grades of severity of acquired MCP/MTP flexural deformities have been described.[59] *Mild deformities* show a straight MCP/MTP region and rarely flex to greater than 180 degrees; in other words the joint remains caudal to the foot at all times, albeit with a straighter angle than normal. *Moderate deformities* have greater than 180 degrees of flexion, which causes the MCP/MTP joint to be dorsal to the foot, but when walking, these horses can extend their joints to a position caudal to the foot. *Severe deformities* have greater than 180 degrees of flexion at all times, and in these cases, the flexor tendons and suspensory ligament are lax and the extensor tendons prevent further flexion of the joint. In these cases, the extensor tendons are prominent on the dorsal aspect of the leg. The speed at which such a deformity develops greatly depends on the degree of pain present, the pain threshold of the patient, the growth rate, the amount of exercise allowed, and possibly the foot-trimming techniques employed. It is important to diagnose a flexural

deformity as early as possible, and promoting client awareness can be helpful in this regard.

Deformities of the MCP/MTP joint can involve one or both of the deep and superficial digital flexor tendons (and less frequently the suspensory ligament), because these structures all support the palmar/plantar surface of the joint. The choice of surgical treatment, should surgery be required, is affected by which tendon is considered to be involved in the deformity. Identification of the primarily affected tendon is not always straightforward. Palpation of the flexor tendons should allow determination of which structure is tightest: the deep digital flexor tendon unit, the superficial digital flexor tendon unit, or the suspensory ligament. Application of pressure to the dorsal joint region in a palmar direction causes tension in the tendons,[30] and the structure with the most tension is the first released at surgery. Palpation and passive manipulation of the involved region should be carried out with the limb in a non–weight-bearing position to recognize potential adhesions between the tendinous structures.

Flexural deformities also develop after prolonged periods of not bearing weight, such as that occasioned by radial paralysis or following conservative fracture treatment (classically olecranon fractures). Depending on their management, deformities can become permanent and debilitating despite healing of the original ailment. The joint region involved with the flexural deformity can be fixed by the contracture, without osseous ankylosis present (fibrodesis).

Acquired flexural deformities of the MCP joint have also been reported in mature horses secondary to desmitis of the accessory ligament of the deep digital flexor tendon[60] and in a case of nonresponsive digital sheath sepsis, which led to rupture of the flexor tendons within the sheath.[61]

Cases detected and treated early carry a good prognosis for correction with conservative treatment.[14,58] One author reported that mild cases requiring corrective shoeing only had a good prognosis for resolution.[62] Cases requiring surgical intervention carried a guarded[62] to poor[28] prognosis.

Proximal Interphalangeal Region

Acquired flexural deformities of the PIP joint have been primarily diagnosed bilaterally in the hindlimbs of rapidly growing weanlings.[58] It occurs in foals with straight hindlimb conformation in the same time period as acquired flexural deformities of the MCP joint. The biomechanical basis of the condition has been postulated as shortening of the deep digital flexor musculotendinous unit and a concurrent laxity in the superficial digital flexor tendon, which inserts adjacent to the PIP joint.[63] Usually a dorsal subluxation is diagnosed and is accompanied by an audible click as the foal walks. Radiographs might show osteoarthritis in chronic cases.

Radiography for the Diagnosis

Radiography is not required for the diagnosis of congenital flexural deformities, which can be made on the basis of clinical signs. However, radiography is useful to identify abnormalities that may alter the prognosis for correction of the deformity. Secondary radiographic changes can occur as a consequence of the deformity; in relation to the DIP joint, these include modeling of the dorsodistal aspect of the distal phalanx, rotation of the distal phalanx in the hoof capsule, or osteoarthritis of the DIP joint, and all of these worsen the prognosis.[28] MCP/MTP joint flexural deformities may show evidence of osteoarthritis in the MCP/MTP or PIP joints secondary to chronic subluxation.[64]

Treatment

Early recognition and treatment of flexural deformities improves the prognosis, especially if pain-mediated flexion is involved, because these will become worse with time if left untreated. If an underlying cause of the pain can be identified, it should be addressed first. Medical or nonsurgical treatment is indicated initially for all but severe cases. If nonsurgical treatment does not improve the flexural deformity, surgery should be considered. Surgery should be considered as the initial treatment of severely affected patients.

Common Treatment Principles

NUTRITION

Nutrition and genetics control the growth rate of the foal. Overfeeding of foals can occur either by heavily lactating mares or excessive supplementation with concentrates. Alterations in the growth rate may also occur when the nutritional plane increases suddenly, especially after a period of relative deficiency, such as after weaning or after a change of ownership. When a flexural deformity occurs in an unweaned foal, the energy content of the diet should be reduced either by early weaning of the foal or a decrease in the mare's concentrate ration. Weaning at 10 weeks of age did not usually affect the foal's size as a yearling in one hospital.[41] The mineral balance of the ration for both the mare and the foal should also be balanced, primarily with respect to calcium and phosphorus, because research has shown that calcium/phosphorus imbalances are also implicated in developmental diseases.[23]

Older foals and yearlings should have the concentrate portion of the diet reduced to a minimum and be fed hay and a balanced mineral supplement. Presumably, if contraction of the musculotendinous unit in response to pain is the more accepted etiopathogenesis for acquired flexural deformities, then prevention should be aimed at monitoring growth and weight and adjustment of the diet accordingly.

Additionally, it is advisable to evaluate soil and drinking water samples for their mineral and trace mineral composition; adjustments should be implemented immediately after identifying inadequate levels.

PHYSIOTHERAPY AND EXERCISE

Opinions differ on the role of exercise in the treatment of flexural deformities. If the deformity is secondary to a painful condition, then exercise limitation and analgesics would be beneficial. Uncontrolled exercise may exacerbate the painful stimuli and the deleterious loading of the contralateral limb.

ANALGESICS

Both the inciting cause and treatment of acquired flexural deformities is a painful process. To aid the foal in standing and ambulating, NSAIDs are given at low doses as for congenital flexural deformities. Foals with painful limbs tend to lie down for longer periods, which can aggravate the flexural problem

and underscores the need for analgesics. (See "Analgesics," earlier, for a fuller discussion.)

Distal Interphalangeal Region

NONSURGICAL MANAGEMENT

DIP joint flexural deformities in which the foal is bearing weight on the toe generally benefit from controlled exercise on a firm surface to allow stretching of the deep digital flexor musculotendinous unit combined with analgesics; however, it is important that the toe region is protected to prevent excessive wear and possible development of a septic process. Turnout in a small yard that is sufficiently small to prevent uncontrolled exercise is probably a reasonable choice.

FARRIERY

The overgrown heels of DIP flexural deformities giving the typical "boxy" conformation to the foot may prevent proper realignment of the hoof–pastern axis if the heel contacts the ground. The heels should be rasped back gradually. Radical trimming of the heel only serves to increase strain forces on the dorsal toe because of the smaller sole surface making contact with the ground. Similarly, if the heel has been lifted off the ground by the deformity, the heels should not be rasped because this only applies greater forces and leverage to the dorsal laminae and the distodorsal aspect of the distal phalanx. However, judicious trimming of the heel combined with application of a toe extension and protection can be an effective treatment (see Figures 87-15 and 87-16).

Application of a toe extension or a glue-on rubber shoe is effective for many flexural deformities of the DIP joint by increasing tension in the deep digital flexor tendon,[39,47] although some clinicians and farriers do not agree that their use is always beneficial.[30,41] Another useful purpose of toe extensions is protection of the toe and prevention of excessive wear in that region. Simple application of a shoe with a toe extension, without filling or bracing the extension against the dorsal hoof wall with hoof acrylic, can cause frequent stumbling and abnormal flaring of the dorsal hoof wall.

The argument against toe extensions is that if the flexural deformity is secondary to a painful stimulus, further tension on the deep digital flexor tendon (DDFT) will exacerbate this painful element and some clinicians support elevation of the heel to reduce pain, encourage relaxation in the DDFT, and change the weight-bearing surface of the foot from the toe to the entire sole; however, other clinicians feel that elevating the heel leads to a worsening of the deformity. There are likely a subset of cases of DIP flexural deformities that respond to a toe extension and another subset that respond to heel elevation. (See also "Toe Extensions," earlier.)

CAST APPLICATION

Cast application in foals causes temporary weakening of the tendons. The distal limbs of affected foals may be covered with a fiberglass cast that incorporates the feet for 10 days to a maximum of 14 days. After cast removal, the weakened tendons allow correction of the problem. Despite the fact that the weakening persists for only a few days, one author has achieved permanent correction of the problem with this technique.[65] However, because of the potential complications associated with this type of treatment, cast application is rarely used.

SURGICAL MANAGEMENT

Surgical intervention is indicated in foals unresponsive to conservative treatment and in severely affected foals. Corrective trimming can be carried out when the foal is anesthetized.

Desmotomy of the accessory (check) ligament of the deep digital flexor tendon

The treatment of choice for stage I flexural deformities is desmotomy of the accessory ligament of the deep digital flexor tendon (ALDDFT) (inferior check ligament) (Figure 87-23).[4,5,57,66-68] The ligament may be approached from the lateral or medial aspect of the limb. If both limbs require surgery, they can both be approached with the horse in dorsal recumbency, the horse can be turned halfway through surgery from left to right lateral recumbency, or one limb can be approached medially and one laterally with the horse in lateral recumbency. The advantages of a lateral approach are that the major neurovascular bundle, located medially at this level, can be avoided, and the ligament is in a more lateral position. The major advantage to the medial approach is cosmetic, although the procedure is more difficult.

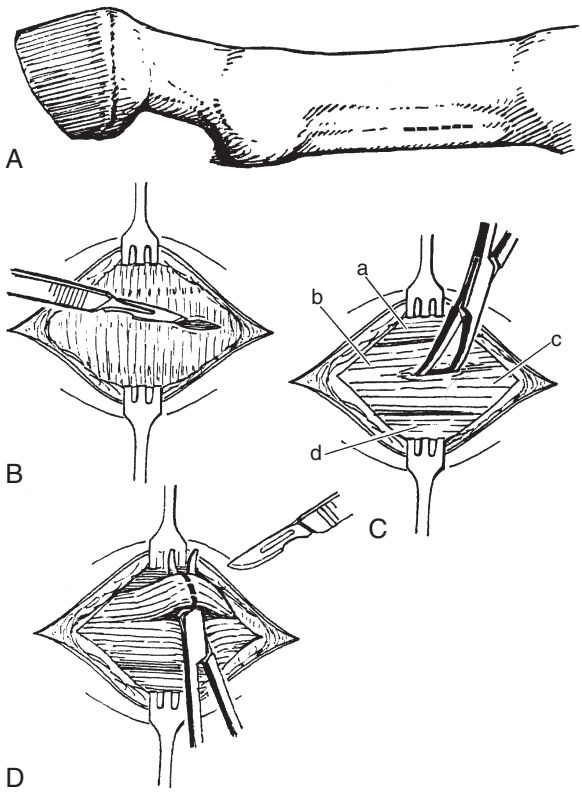

Figure 87-23. Surgical technique of desmotomy of the accessory ligament of the deep digital flexor tendon (inferior check ligament desmotomy). **A,** Location of the surgical site on the medial, or in most cases the lateral, side of the limb *(dotted line).* **B,** The paratenon enclosing the deep flexor tendon and the accessory ligament is incised. **C,** The accessory ligament is isolated along its division plane with the deep digital flexor tendon. *a,* Musculus interosseus (suspensory ligament); *b,* accessory ligament of the deep digital flexor tendon; *c,* deep digital flexor tendon; *d,* superficial digital flexor tendon. **D,** The isolated and elevated accessory ligament is transected along the dotted line. (From Turner AS, McIlwraith CW: Techniques in Large Animal Surgery. 2nd Ed. Blackwell, Oxford, UK, 1989.)

A 5-cm skin incision, centered at the junction between the proximal and middle third of the MCIII, is made over the deep digital flexor tendon. The subcutaneous tissues are bluntly separated and the tendinous structures are identified. With the medial approach, the neurovascular bundle overlying the deep digital flexor tendon and its accessory ligament are identified and reflected away from deeper structures. Palpation of the paratenon surrounding the deep digital flexor tendon and its accessory ligament allows separation between the two structures.

A curved hemostatic forceps is introduced and advanced following the slightly curved surface of the tendon to the opposite side, where the forceps is spread and turned. The ALDDFT lying dorsal to the tendon is elevated to the level of the skin incision. Manipulation of the foot in a dorsal direction tightens the ligament and ensures the isolation of the correct structure. Once the ligament is positively identified, it is sharply transected with a scalpel blade. Dorsal rotation of the DIP joint produces at least a 1-cm gap between the transected ends of the ligament. The gap is inspected and palpated, and any remaining fiber strands of the ALDDFT are transected. The paratenon, subcutaneous tissues, and skin are closed using 2-0 or 3-0 absorbable suture materials in a continuous pattern. Intradermal placement of the skin suture is advised for a better cosmetic result. Postoperatively, a pressure bandage is applied and maintained for 2 to 3 weeks and changed every 3 to 4 days.

Correction or improvement of the deformity is usually observed immediately. In some cases, however, it takes a few days until it is complete. Occasionally, application of a toe protection or extension is needed. Young foals and those without long-standing contracture are allowed controlled exercise within 3 to 6 days after the surgery. Free pasture exercise is encouraged after 2 weeks. In older foals and those with chronic or severe contracture, limiting exercise for a period of months might prevent excessive fibroplasia at the surgery site. To relieve potential pain, NSAIDs may be administered at low doses.

An ultrasound-guided tenotomy of the ALDDFT has been described in standing horses.[69] The surgery applying this technique was more successful in restoring a normal hoof conformation in the younger horses treated (median age, 6 months) compared to the older group of horses (median age, 12 months). The age of surgery did not affect the cosmetic outcome.

Postoperative fibroplasia occurring at the surgery site reunites the transected ends of the ligament with time. In selected cases, fibroplasia in that region is excessive and results in a cosmetically undesirable appearance. Bandaging for 3 weeks after surgery improves the cosmetic outcome and lessens scarring at the surgical site.

Cosmetically unacceptable results can also occur from tendinitis that can be induced from exercise when the tendon has been protected by the contracted accessory ligament for a long time. Tendinitis can be avoided with longer periods of controlled exercise. Daily massage of the swelling can reduce the size.

In horses intended for showing or pleasure riding, the prognosis for athletic function is good; in one study 86% of horses treated before 1 year of age were subsequently used for their intended purpose.[67] Horses treated after 1 year of age had a lower success rate of 78%. One author reported a good prognosis for surgical correction of mild cases but a poor response of severely affected cases to an desmotomy of the ALDDFT.[9] Other authors reported that Standardbred foals treated for DIP joint flexural deformities could reach their athletic potential but that the prognosis was better if the foal was treated at a younger age.[70] In this series, no foals treated after 8 months of age had a favorable outcome.

Tenotomy of the deep digital flexor tendon

Stage II flexural deformities might not correct after desmotomy of the ALDDFT, although this intervention is commonly used as the first surgical approach. Tenotomy of the deep digital flexor tendon can then be used successfully for correction,[71,72] but the deformity is reported to reappear in many cases within months of surgery.[10] Some clinicians feel that cases with a hoof–ground angle of greater than 115 degrees usually require a deep digital flexor tenotomy.[30] Initially, such a tenotomy had been viewed at as a salvage procedure; however, many animals have developed into sound riding horses, although the prognosis for return to function is usually guarded.[30]

Two main sites for the surgical procedure have been proposed: midmetacarpus or midpastern. The distal approach centers at the palmar and median aspect of the pastern region and enters through the digital flexor tendon sheath just distal to the bifurcation of the superficial digital flexor tendon.[72] The deep digital flexor tendon is identified, exteriorized, and transected with a scalpel blade. Immediate retraction of the proximal stump into the tendon sheath is noted. The tendon sheath may or may not be sutured using routine technique in addition to the subcutaneous tissue and the skin.[68,72] While the animal is under anesthesia, the feet are trimmed to a shape that is as normal as possible.

Postoperative correction is often immediate but can be associated with substantial pain. Administration of NSAIDs is therefore routine. Because of the time it takes for some stage II flexural deformities to develop, the soft-tissue structures, such as the joint capsule, ligaments, and periarticular tissues at the palmar aspect of the phalanges, may be significantly contracted. Transection of the deep digital flexor tendon at the level of the PIP joint does not often result in a marked elevation of the toe during weight-bearing, which it does after rupture of this tendon in the region of the navicular bone. Nevertheless, in selected cases, application of a shoe with a heel extension is necessary.

Transection of the deep digital flexor tendon at the midmetacarpal level is also advocated.[73] The procedure is carried out through a medial or lateral approach. The advantage of this approach is the greater distance from the feet and the fact that a tendon sheath is not invaded. Additionally, the procedure is easier to perform at the midmetacarpal level and can be performed as a standing procedure. However, scarring associated with marked disfigurement of the tenotomy site can be an undesirable sequela. Postoperative management is identical to that used with the other technique. Recurrence of the deformity is a potential problem following this technique, because the tendon ends may reunite, initially with scar tissue; this is followed by its contracture.

Metacarpophalangeal Region

NONSURGICAL MANAGEMENT

Nonsurgical management includes proper nutrition, physiotherapy, analgesics, corrective shoeing, and application of splints.

PHYSIOTHERAPY

Hopping an animal is a further attempt to bring about correction of the deformity without surgery.[4] One limb of a foal is elevated and held in that position while a helper leads the animal at a walk. During the support phase of the elevated limb, most of the weight is transmitted to the person holding the limb. The opposite limb is quickly advanced and all the weight brought to bear on it. By repeating this type of physiotherapy several times daily, a potential stretching of the musculotendinous unit can be achieved. Exercise is an important type of therapy, but it should be carried out in a controlled manner.[74] Excessive exercise leads to fatigue, which should be avoided.

FARRIERY

Corrective shoeing can successfully eliminate flexural deformities of the MCP joint. Raising the heel with wedge pads results in a more acute angle of the MCP joint from partial relaxation of the deep digital flexor tendon.[4,50,73] Raising the heels by the use of a wedge pad or shoe has been suggested to decrease the strain in the deep digital flexor tendon while increasing the strain on the superficial digital flexor tendon and the palmar joint structures.[9,75] Some authors have questioned the effectiveness of such treatment, but clinically, an improvement is possible.[5] Conversely, some clinicians[48] recommend lowering the heel to invoke a reverse myotactic reflex. Toe extensions have been advocated and have proved very effective, especially when the condition was diagnosed early (Figure 87-24).[3,5,74] As the animal walks, breakover occurs later, and through this, greater tensile stress is exerted on the flexor tendons.

SPLINTS

Splint application can bring about some correction, especially in cases recognized early. Care must be taken to prevent development of pressure sores. Splints that position the MCP joint caudal to the foot, loading the horse's weight onto the flexor tendons during weight-bearing, can be useful.[30]

Figure 87-24. A toe extension incorporated into a shoe for treatment of an acquired flexural deformity of the metacarpophalangeal joint.

SURGICAL MANAGEMENT

Horses that are unresponsive to conservative management are candidates for surgical correction. Some clinicians feel that any horse with an MCP angle of greater than 180 degrees (forward of the hoof) are immediate surgical candidates.[30]

Because both the superficial and deep digital flexor tendons cross the palmar/plantar surface of the MCP/MTP joint, either or both can contribute to flexural deformities of this region. Distinguishing which tendon is primarily affected through palpation is not necessarily straightforward. Both desmotomies of the ALDDFT and accessory ligament of the superficial digital flexor tendon (ALSDFT) have been used for flexural deformities of this joint and can be used together for the treatment of severe deformities. Using the angle of the MCP region when the joint is in forced extension, guidelines exist for selection of the correct surgical procedure. Flexural deformities with an MCP angle of less than 180 degrees usually respond to desmotomies of the ALDDFT or ALSDFT.[76] For MCP angles of 180 degrees, the surgical procedures mentioned earlier are used in conjunction with a splint. Severe cases with an MCP angle of greater than 180 degrees benefit from both desmotomies plus a tenotomy of the superficial digital flexor tendon. The aim of all surgical procedures for MCP/MTP flexural deformities is to return the regional angle to less than 180 degrees, which facilitates loading of the flexor tendons.[30]

Desmotomy of the accessory (check) ligament of the deep digital flexor tendon

If the deep digital flexor tendon was tighter during palpation and manipulation, its accessory ligament should be transected using the technique described earlier.[76] Application of a preshaped polyvinyl chloride (PVC) half-pipe splint is necessary for 2 to 3 weeks to maintain the metacarpophalangeal angle in the normal range.

Desmotomy of the accessory (check) ligament of the superficial digital flexor tendon

Transection of the ALSDFT should be performed when this tendon is tighter during manipulation.[4,77] Two surgical approaches have been developed. The surgical landmarks for both approaches are the same and consist of the medial distal physis of the radius, the chestnut, and the cephalic vein. An approximately 10-cm skin incision is centered along the chestnut and made craniad to the cephalic vein. The subcutaneous tissue is bluntly separated, and communicating branches to the cephalic vein are isolated, double ligated, and transected between ligatures.

One surgical technique approaches the accessory ligament craniad to the flexor carpi radialis muscle.[50,59,78] The oval foramen in the carpal fascia can serve as the distal border of the desmotomy incision.[66] The carpal fascia is transected carefully, and the ensheathed flexor carpi radialis muscle is identified. Both the cephalic vein and the flexor carpi radialis muscle are displaced using a self-retaining retractor. The desmotomy of the fan-shaped accessory ligament is performed, taking care to avoid inadvertent injury to the palmar carpal rete. After transection of the ligament, the radial head of the deep digital flexor muscle appears and the carpal sheath becomes visible. Hemostasis is established, and the carpal fascia, subcutaneous tissues, and skin are closed routinely.

An alternative approach involves invasion of the sheath of the flexor carpi radialis tendon (Figure 87-25).[79,80] After reflecting

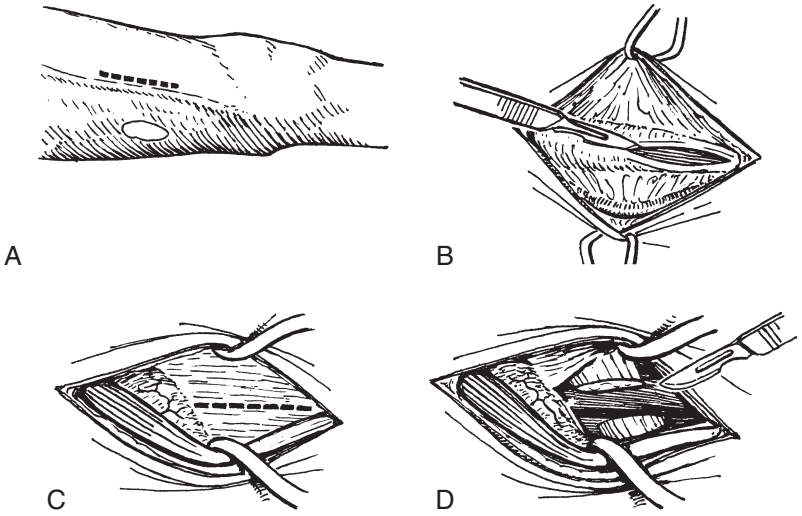

Figure 87-25. Surgical technique of accessory ligament desmotomy of the superficial digital flexor tendon (superior check ligament desmotomy). **A,** Location of the surgical site relative to the anatomic landmarks: cephalic vein, distal radial physis, and chestnut. **B,** The tendon sheath of the flexor carpi radialis muscle is incised. **C,** The flexor carpi radialis tendon is retracted, allowing visualization of the craniolateral tendon sheath wall. The site of the desmotomy incision is marked with a *dotted line* over the accessory ligament, which is at this location part of the craniolateral tendon sheath wall. **D,** The accessory ligament desmotomy is completed, allowing digital access to the radial head of the deep digital flexor muscle. (From Turner AS, McIlwraith CW: Techniques in Large Animal Surgery. 2nd Ed. Blackwell, Oxford, UK, 1989.)

the flexor carpi radialis tendon in the sheath with a self-retaining retractor, the craniolateral wall and accessory ligament are identified. A curved Kelly forceps is placed under the distal border and spread. The ligament is then transected in a distal to proximal direction. Care is taken to avoid injury to the nutrient artery of the superficial flexor tendon, which enters along the proximal border of the accessory ligament. After transection, the same anatomic structures are visible as mentioned with the first technique. Inadvertent incision of the carpal sheath is of no consequence. The tendon sheath is closed using a continuous suture pattern with 2-0 or 3-0 synthetic absorbable material. The rest of the closure is routine.

Additionally, a tenoscopic surgical approach has been developed, which allows a shorter convalescent period and fewer incisional complications.[81]

An *in vitro* study revealed that desmotomy of the ALSDFT was associated with significantly increased strains on the superficial digital flexor tendon and the suspensory ligament and significant alterations in the angles of the MCP and carpal joints.[80] Lengthening of the superficial digital flexor musculotendinous unit after desmotomy of the ALSDFT can be associated with increased strain on the suspensory ligament.[80]

Postoperatively, a sterile pressure bandage is applied and maintained for 2 to 3 weeks. The bandage is changed every 3 to 4 days. A shoe with an elevated heel may be used postoperatively to preferentially load the superficial digital flexor tendon.

In severe cases, the accessory ligaments of both the deep and the superficial digital flexor tendons are transected.[67]

Transection of the suspensory branches

As an additional alternative procedure, desmotomy of the medial and lateral branch of the suspensory ligament may be performed for salvage purposes in persistent cases.[50] However, subluxation of the PIP joint is to be expected.

A stab incision is made directly over the suspensory branch, the subcutaneous tissues are separated with a Kelly forceps, and a curved tenotome is introduced. By applying pressure with a sawing motion, the previously undermined suspensory branch is transected. Care should be taken to prevent inadvertent injury to the palmar artery and vein and the MCP joint capsule. The skin is closed using a few simple interrupted sutures. The same procedure is performed on the other side of the limb.

Horses with chronic or severe flexural deformities respond poorly to any treatment, including surgery, and superior check ligament desmotomies do not carry as good a prognosis as inferior check ligament desmotomies.[9,59] Severe flexural deformities following prolonged non–weight-bearing lameness do not respond to desmotomy of either of the two accessory ligaments. Such cases should be treated either with a tenoplasty as the lengthening procedure in both flexor tendons, followed by long-term cast application, or with an osteotomy plus an MCP arthrodesis (see Chapter 81), although euthanasia is a valid consideration in these cases.

Carpal Region

NONSURGICAL MANAGEMENT

Foals with long-term debilitating injuries that prevent weight-bearing on the limb should be treated with splints early in convalescence to prevent development of a flexural deformity. Passive stretching exercises are indicated. These two therapies are combined with controlled exercise if the original injury allows it. A contracture typically occurs suddenly over a few days after several weeks of not bearing weight on the limb. Therefore, physiotherapy should not be delayed. Care should be taken to prevent development of pressure sores under the splint.

SURGICAL MANAGEMENT

In cases resistant to conservative treatment, tenotomy of the ulnaris lateralis and flexor carpi ulnaris tendons can be performed with good results.[27] The same surgical procedure is performed as described earlier. Passive manipulation of the carpal region at that time demonstrates the greater mobility gained through this procedure.

Postoperatively, a well-padded pressure bandage is applied and a splint centered over the carpal region is incorporated. While the horse is under anesthesia, the limb is forced into a straight position. The splint bandage is changed regularly and maintained for about 2 weeks. Hand-walking exercise is important for the first 3 to 4 weeks. As soon as the animal easily bears weight on the limb, the splint is removed.

Proximal Interphalangeal Region

NONSURGICAL MANAGEMENT

Rest is not usually an effective management tool in these cases. The periarticular tissues may fibrose, and eventually the clicking noise is no longer heard. However, a marked bend at the joint level will be visible. Osteoarthritis could develop, necessitating an arthrodesis of the PIP joint to render the animal pain free again.

The clicking sound associated with dorsal snapping of the PIP and MCP area could also be observed in foals with persistent foal hoof at the tip of the hooves, resulting in an elongated hoof configuration. In one case, trimming of the hoof was successful in eliminating the clicking sound and the snapping of the proximal interphalangeal and metacarpophalangeal area.[65] This approach should be tried before surgical intervention is performed.

SURGICAL MANAGEMENT

Affected horses may respond to exercise restriction and analgesics, but in cases unresponsive to medical treatment, surgical transection of the accessory ligament of the deep digital flexor tendon and the tendon of the medial head of the deep digital flexor tendon at the level of the chestnut has been reported.[63] Three horses treated surgically with this method had resolution of the subluxation, and the one horse for whom follow-up information was available was sound after 10 months.[63]

COMPLICATIONS

Nonsurgical Management

Splint-associated pressure sores are the most often encountered complication and septic arthritis following injudicious use of splints has been reported.[82] To avoid such complications, padding is placed around sites of predilection. Once necrosis is present, local pressure has to be avoided in that area. Application of a donut-shaped pad around the periphery of the lesion can assist in that effort. Alternate application or temporary removal of the splint may be attempted. Daily topical wound care is necessary, especially in the initial period.

Surgical Management

Persistent hematoma formation, especially at the site of transection of the ALSDFT, wound dehiscence, and infections are the most common complications encountered after surgical treatment. Whenever fever, leukocytosis, warmth at the surgery site, or increased pain are noted, the incision should be evaluated and the necessary steps taken immediately. For additional information on wound dehiscence, please review Chapter 26.

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CHAPTER
88

Osteochondrosis

P. René van Weeren

The term *osteochondritis dissecans* was coined by König in 1887 and used to describe loose or semiloose bodies in joints of young persons that could have three causes: very severe trauma, lesser trauma and necrosis, or minimal trauma acting on an underlying lesion.[1] Although the name and concept have led to substantial confusion from the onset, this categorization is still valid today.[2]

Osteochondrosis (OC) is the last of König's categories described in the equine veterinary literature, and it is the most difficult to understand. OC represents a disturbance of the

process of endochondral ossification without a clearly understood etiology. This disturbance can eventually lead to the formation of semiloose or even completely loose fragments within a joint.

THE PROCESS OF ENDOCHONDRAL OSSIFICATION

In all mammals, the primordial skeleton is laid down first as a cartilaginous structure that, during the entire period of early development of the animal, is coupled in a process of simultaneous growth and transformation into bone. It is important to note that unlike mature articular cartilage, these fetal cartilaginous structures are well vascularized by vessels running through cartilage canals. Ossification of the primary centers of ossification in the diaphyses of the long bones starts early in fetal life, and at the time of birth, all the diaphyses are bony structures. This does not occur in many secondary centers of ossification located in the epiphyses of the long bones and in other sites such as apophyses and cuboidal bones in complex joints, which remain partly cartilaginous at the time of birth.

After birth, *longitudinal* growth of long bones results from the growth plates or physes where, from a germinal layer of cells (resting cells), chondrocytes proliferate and lay down a scaffold of extracellular matrix. These cells initially hypertrophy and later undergo apoptosis. The scaffold is used for the apposition of primary bone by osteoblasts originating from the metaphysis (Figure 88-1). This primary spongiosa then undergoes continuous remodeling under the influence of biomechanical loading according to Wolff's law during the entire growth period of the foal. This remodeling continues in the adult when biomechanical loading changes, for instance, when the skeleton is exposed to athletic challenges.[3] The entire process of cartilage remodeling, followed by calcification of cartilage, deposition of primary bone, and successive remodeling into bony trabeculae, is known as *endochondral ossification*.[4]

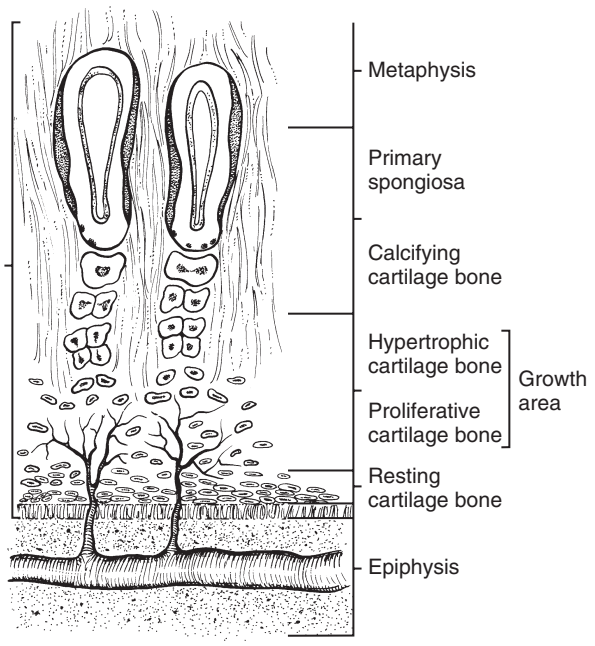

Figure 88-1. The relationship of the cartilage zones of the growth cartilage of a physis. (Redrawn from Watkins JW, Auer JA: Learning Systems 6:S227, 1984.)

The increase in *diameter* of the long bone during growth is the product of a different, but simultaneous and coordinated process, which is by appositional growth from the periosteum. This process results in the formation of compact cortical bone, characterized by the haversian canals.

In the epiphyses of the long bones a growth process similar to that of the physis takes place, but it is not as completely developed as in the diaphyses at birth. This leads to large differences between joints in the times when ossification occurs. In some joints at birth, there is a complete ring of cartilage around the ossification center, connecting articular cartilage with the growth plate. Ossification of this cartilage ring takes place first at the border of the physis and at the perimeter of the epiphysis. The thick cartilage mass at the articular side of the epiphysis functions as a type of growth plate where the simultaneous processes of growth, remodeling, and ossification take place that finally result in a considerably thinner layer of articular cartilage in the mature animal. It is at this level that the characteristic lesions of equine OC develop.

PATHOPHYSIOLOGY

There is little controversy about the gross pathogenetic mechanism of OC. Disturbances of the process of endochondral ossification result in irregularities in thickness of the epiphyseal cartilage. These create areas of focal weakness, which are exacerbated because the cartilage canals regress with increasing age and have disappeared in the horse by the age of 7 months.[5] This affects the nutrition of the deeper layers of the retained cartilage plugs that cannot be sufficiently nourished by diffusion from the articular surface, possibly leading to necrosis. Biomechanical influences, mainly shearing forces, then lead to the formation of fissures and produce cartilage flaps, or detachment of cartilage or fragments of cartilage and subchondral bone. In some locations where biomechanical forces are mainly compressive, infolding of cartilage in these weakened areas can lead to the formation of subchondral bone cysts as another manifestation of OC.[6]

Many caveats concerning this seemingly straightforward mechanism have been made. In an extensive review of pathogenesis and possible etiologic factors of OC, the view that defective ossification lies at the base of all lesions commonly qualified as OC in the horse has been questioned based on clinical and pathologic observations.[4] One of the major arguments was the observation that after injury, bone and cartilage can manifest only a very limited reparative response. This makes it very difficult, if not impossible, to judge the stage of the process (recently originated or already in some phase of repair) and more so the origin of the lesion (delayed ossification or trauma, for example).[7] Things become even more difficult if one realizes that OC is generally assumed to be a multifactorial disease in which the problem is not to single out a specific causative factor but to determine to what extent, and in which order, a variety of factors play a role. These considerations are discussed in more detail later, under "Etiologic Factors."

Along similar lines, comparisons between species should be dealt with cautiously. In the late 1970s, in-depth studies on the phenomenon of OC in a multitude of species (including horses, poultry, dogs, and cattle, but with the emphasis on swine) concluded that because of the striking similarities in manifestation

across the species, including humans, OC should have a common pathogenesis and etiology, the latter being principally growth rate that itself was determined by nutrition and hereditary factors.[8] Ever since, this paradigm of a "seemingly unified hypothesis"[4] has hovered over OC research, and the ease with which conclusions for the horse have been drawn based on research in entirely different species has been remarkable in some instances. It is questionable if this simple explanation of the pathophysiology of OC is justifiable, given many findings from more recent research that are discussed later.

DIAGNOSIS: CLINICAL AND RADIOGRAPHIC SIGNS

The typical OC patient is a yearling that is presented with effusion of the tarsocrural or femoropatellar joint that has been noted recently by the owner. The horse usually is not lame, and radiographic examination shows a fragment at the cranial end of the distal intermediate ridge of the tibia (Figure 88-2) or irregularities at the lateral trochlear ridge of the distal femur (Figure 88-3). However, as with any typical presentation of a disease, many variations on this theme are possible. OC in cervical facet joints has been related to cervical stenotic myelopathy, being a cause of wobbler syndrome in young horses. However, the relationship does not seem to be straightforward, although common pathogenetic pathways may exist.[9]

The age at which OC becomes clinically manifest varies, although in the great majority of cases, OC patients are juvenile animals. In severe cases of OC, which are more common in the femoropatellar (FP) joint than in the tarsus, signs can be seen in foals as young as 6 months. OC can also manifest itself at

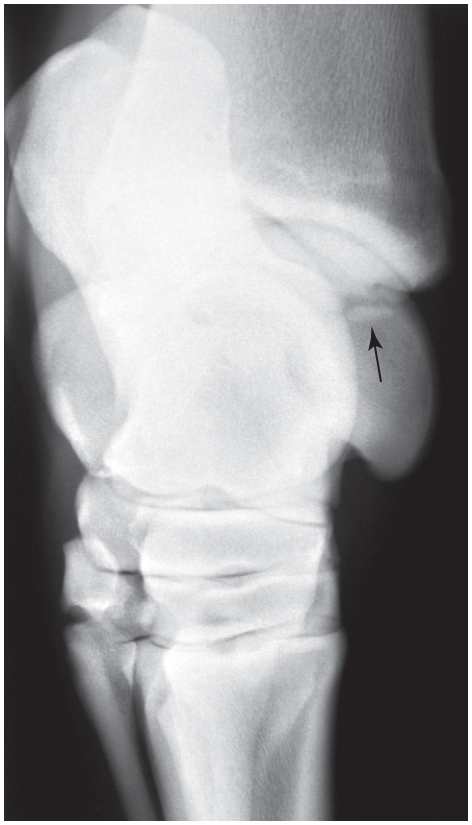

Figure 88-2. Radiograph showing a typical osteochondritic lesion of the dorsal aspect of the distal intermediate ridge of the tibia *(arrow).* (Courtesy Dr. AJM van den Belt, University of Utrecht, Netherlands.)

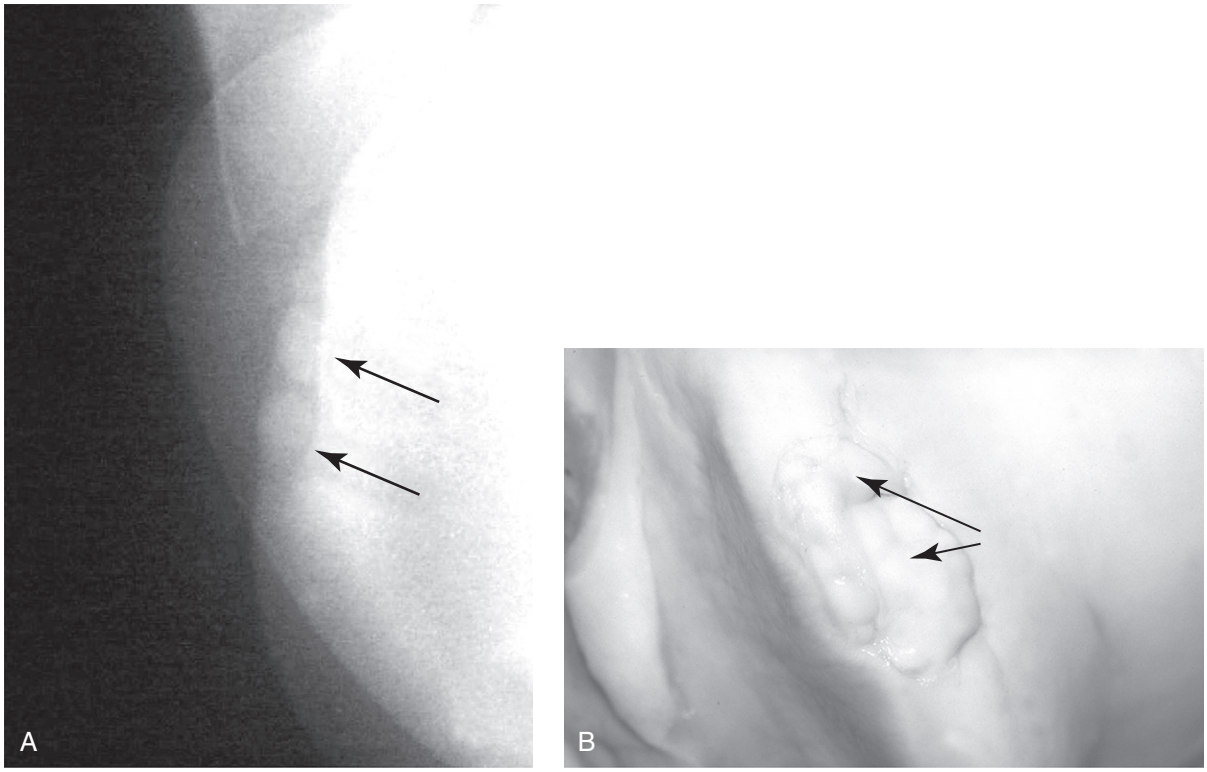

Figure 88-3. A, Radiographic view showing osteochondritic lesions on the lateral trochlear ridge of the distal femur *(arrows).* **B,** Postmortem view of an osteochondritic lesion of the lateral trochlear ridge of the distal femur *(arrows)*.

the age the animals are put into training and the joints become challenged by athletic activity. The age at which this occurs varies with the branch of equestrian industry. For example, Warmblood horses commonly are presented at about 3 years of age or older, whereas racing Thoroughbreds and Standardbreds manifest signs much earlier when they enter training.

Radiography is the gold standard for diagnosing OC but is not capable of detecting very subtle lesions. For this reason, other approaches using a variety of markers (see later) have been tried with varying success. An interesting development is the use of infrared absorption spectral characterization of synovial fluid using Fourier transform infrared (FTIR) spectroscopy that was able to discriminate between samples from OC-affected and normal animals with 50% specificity and 73% sensitivity.[10]

Distribution of Lesions

OC is most commonly diagnosed in tarsal, FP, and metacarpophalangeal/metatarsophalangeal (MCP/MTP) joints, but it has been described in almost every diarthrodial joint. In an experimental study, 43 Warmblood foals were selected out of pairing OC-positive sires and partially OC-positive mares to produce offspring with a high prevalence of OC. Twenty-four foals were sacrificed at 5 months, all joints were inspected at necropsy, and macroscopic lesions were confirmed by microscopy.[11] Lesions were most numerous in the tarsocrural (TC) joint (average of two lesions per animal), followed by the FP and the cervical intervertebral (facet) joints (one lesion per animal), the MTP joint (0.6), the MCP and carpal joints (0.4), humeroradial joint (0.2), and scapulohumeral joint (0.04).[12]

Although the prevalence of OC in this study was artificially high, the relative distribution is in agreement with clinical experience in the Warmblood. Breed differences occur to a certain extent with regard to lesion distribution and relative clinical importance. OC in the FP joint is common in the racing Thoroughbred,[13] but in Warmbloods and Standardbreds, TC OC is seen more often.[14-18]

A hallmark of OC is that lesions almost always occur at certain predilection sites within a joint. In the TC joint, the most common site is the cranial end of the distal intermediate ridge of the tibia (see Figure 88-2), followed by the distal end of the lateral trochlea of the talus and the medial malleolus of the tibia.[19] In the FP joint, the most common predilection site is the lateral trochlear ridge of the femur. Less common sites are the medial trochlear ridge of the femur, the trochlear groove, and the distal end of the patella.[6] Subchondral cysts that occur in the medial femoral condyle are a manifestation of OC as well and are discussed in Chapter 89. The predilection site in the MCP/MTP joints is the dorsal end of the sagittal ridge of the metacarpus and metatarsus. Not all osteochondral fragments are osteochondrotic in origin. Opinions on the nature of the fragments seen at the dorsal margin of the proximal phalanx differ, and the palmar or plantar osteochondral fragments (POFs) that were originally reported as being part of the OC complex[20] are now considered traumatic in origin.[21,22] In a histological study on osteochondral fragments that were harvested from mature (average age 6 years) horses it was shown that even in long-present intra-articular fragments, histology may give an indication about the original etiology with more indications of osteoarthritis (higher Mankin score) in fragments that were not of osteochondrotic nature.[23] Recently, it was shown that POF had a significant linear relationship with grades of wear lines, cartilage ulceration, and dorsal impact injuries in a cohort of Thoroughbred racehorses, and hence could be classified as a manifestation of traumatic overload arthrosis.[24] In the shoulder joint, OC is commonly located on the glenoid and the humeral head.[25]

Lesions are often encountered bilaterally in the TC and FP joints and quadrilaterally in the MCP/MTP joints.[26] Bilateral presence, often with unilateral clinical manifestation only, can occur in the TC and FP joints in more than 50% of clinical cases.[6] Therefore, in horses with unilateral clinical signs it is advisable to radiograph the contralateral joint. In contrast, concomitant occurrence in other joints or joint pairs is much less common. In a study of 225 horses with TC OC, lesions were found in other joints in only eight cases.[26] Therefore, joints other than the contralateral one do not need to be radiographed, except when clinical signs exist.

Joint effusion is by far the most common clinical sign, but lameness can occur, especially when large radiographic lesions exist. Lameness is seen more in FP OC than in TC OC and most likely when a loose or semiloose fragment is observed radiographically, thus when the osteochondritis dissecans (OCD) form is present. Other, less severe radiographic signs include irregularities in the articular contour of the subchondral bone and sometimes only a flattening of this contour.

Minor radiographic aberrations can be reliably classified as OC by an experienced radiologist. In one study, there was a correlation of 0.87 ($P < 0.001$) between radiographic classification of OC of the distal intermediate ridge of the tibia on a 0 to 4 scale[27] and histology.[12]

Nevertheless, the lesion's severity as determined radiographically does not always correspond with arthroscopic or necropsy findings. In many cases, cartilage lesions are more severe than radiographic appearance suggests, or cartilaginous lesions are present without changes in the subchondral bone and hence do not show up on radiographs (Figures 88-4 and 88-5). Severity of lesions can be assessed more accurately with more sophisticated imaging modalities such as magnetic resonance imaging (MRI). However, economic and physical constraints severely limit the wide clinical use of this kind of equipment, especially in the FP joint.

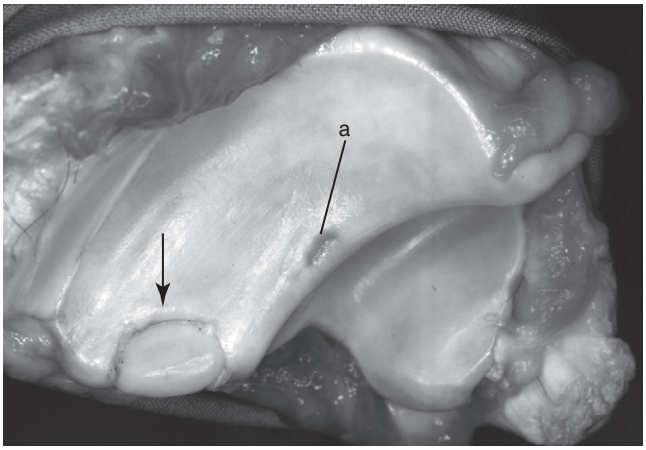

Figure 88-4. Typical osteochondrotic lesion of the distal intermediate ridge of the tibia in a cadaveric specimen *(arrow)*. *a*, Synovial groove.

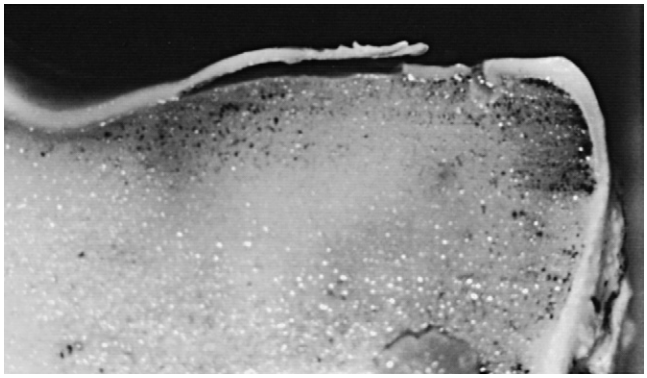

Figure 88-5. Example of an osteochondrotic lesion of the humeral condyle showing the formation of a large cartilage flap but relatively little damage to the subchondral bone.

Breed Predilection

OC is common in many breeds of horses. In Swedish Standardbreds, an incidence of 10.5% was found in the tarsal joints,[17] which was comparable to the 12% found by other authors[15] but less than the 26% found earlier by yet other researchers,[28] also in Standardbreds. One group found an incidence of 35% in the FP and MCP/MTP joints of Standardbreds in a Canadian population.[29] There are fewer studies in Thoroughbreds, but the incidence in that breed is reportedly relatively high as well.[25,30,31] In a study using repository films taken at a yearling sale in New Zealand, relatively low figures of 4% OC in the TC joint and 3% in the FP joint were found, but these figures have limited value because the population at the yearling sale was strongly preselected.[32]

In the Warmblood horse, early Swedish data mentioned an incidence of 15%.[28] More recent figures in the Dutch Warmblood population are higher (25% in the TC joint and 15% in the FP joint).[27] In a large study of 1180 horses in France (mainly Selle Français and Anglo-Arabs, a minority of Thoroughbreds), an incidence of 13.3% was reported in the TC joint.[18] A large-scale field study in Germany in several Warmblood breeds yielded figures of 19.5% for the MCP/MTP joints, 11.1% for the TC joint, and 7.2% for the FP joint.[33] This supports the findings that Warmbloods are at higher risk to develop OC than Thoroughbreds.[34]

Statistics on the prevalence of OC must be considered with utmost caution, given that OC may occur in almost any diarthrodial joint, but above all because the disease is highly dynamic with many lesions disappearing before maturity (see later). Additionally, figures are heavily affected by the number of joints examined and by the age at which examinations are carried out. Further, any form of preselection by breeders (which is common) will affect prevalence figures. and the effects of these factors may be dramatic. In a study on 811 yearlings that had not been preselected, a staggering 67.5% prevalence was reported, when combining results from TC, FP, MCP, and MTP joints in Dutch Warmbloods.[35] When measured conventionally at the age of 3 years, the mean figure for this breed is approximately 30%. A similarly high prevalence for TC and MCP/MTP OC (61.7%) was found in a population of South German Coldbloods that contained many young animals.[36]

Overall, it has been estimated that in northwestern Europe alone, 20,000 to 25,000 foals are born annually that will develop some degree of OC.[11] OC lesions are only rarely encountered in ponies.[37] Further, in a survey of 80 feral horses, extremely low incidences of 2.5% were found in the TC joint and 0% in the FP joint.[38] Because OC is a relatively new disease (see next section), these observations strongly implicate breeding policies and possibly management aspects as key factors in this disease.

OC is a debilitating disease only in exceptional cases, where lesions are so extensive that no repair is possible. Nevertheless, the ailment has a strong impact on the economics of the equine industry, and to a certain extent on animal welfare, because tens of thousands of animals are operated upon each year. Apart from the direct economic loss, there is an even larger indirect cost of the disease, because many studbooks do not approve horses with (major or minor, depending on studbook policy) evidence of OC. It has been estimated that the strict breeding policy the Royal Dutch Warmblood Studbook (KWPN) has adhered to until recently precluded 30% of all potential male breeding stock from participating in the selection procedures for becoming an approved sire. Apart from direct financial loss for the breeders, this also eliminates a large part of the gene pool. A last, but not insignificant, economic impact of OC is the loss in value of animals showing radiographic evidence of OC, regardless of their athletic capacities.

EVOLVING CONCEPTS

The first description of what is retrospectively judged to have been OC appeared in 1947 when fragments in the FP joints of Ardenner horses were noted.[39] Although in the following decades some reports described intra-articular fragments that might have been osteochondrotic,[40] the real history of OC does not start before the classic publication by Birkeland and Haakenstad in 1968.[41] These authors described a series of seven cases of OC of the distal ridge of the tibia, but they did not use the term *osteochondrosis*. In the early 1970s gradually more publications appeared,[42,43] and in the mid-1970s a comprehensive study on OC in a multitude of species was published.[8,44-47]

OC was originally seen as a largely static condition, but this concept gradually changed after a report appeared on changes in the radiographic appearance of FP OC after repeated examinations.[48] Other researchers recognized, after sequentially radiographing a number of foals, that OC lesions of the distal femur could progress until the age of 9 months.[49] In the same year, another group published a study in which they followed a cohort of 77 horses radiographically and found that no major new OC lesions were identified after 8 months of age in the TC joint. Interestingly, the authors noted a regression of a number of minor lesions before the age of 8 months, which had been detected between ages 1 and 3 months.[16]

A group of researchers radiographed the TC and FP joints of 43 foals on a monthly basis from the age of 1 month until age 5 months and of 19 of them until age 11 months. The study showed that not only minor lesions but also radiographically visible larger fragments ceased to exist. The ages at which lesions originated and the ages at which they became undetectable varied for each joint. In the TC joint, lesions at the distal intermediate ridge of the tibia and at the distal aspect of the lateral trochlear ridge of the talus that were seen within the first few

months of life had become undetectable before the age of 5 months. Thereafter no major changes occurred and existing lesions remained visible. In the FP joint where the epiphyseal maturation is known to be late compared to other joints,[50,51] lesions originated later and peaked at approximately 6 months before declining in number until about 8 months, after which the lesions remained stable (Figure 88-6).[27]

In a follow-up study in another group of Warmblood foals that were tracked for 24 months, this general joint-specific pattern was confirmed.[52] In that study, very little change in radiographic appearance was noted from 12 to 24 months. Further confirmation of these patterns resulted from two additional studies. In one study, groups of horses were used to evaluate the relationship between liver copper content and OC,[53] and in the other study the influence of nutrition on OC was examined in the French Saddlebred.[54] The latter study also featured sequential radiographic examinations.

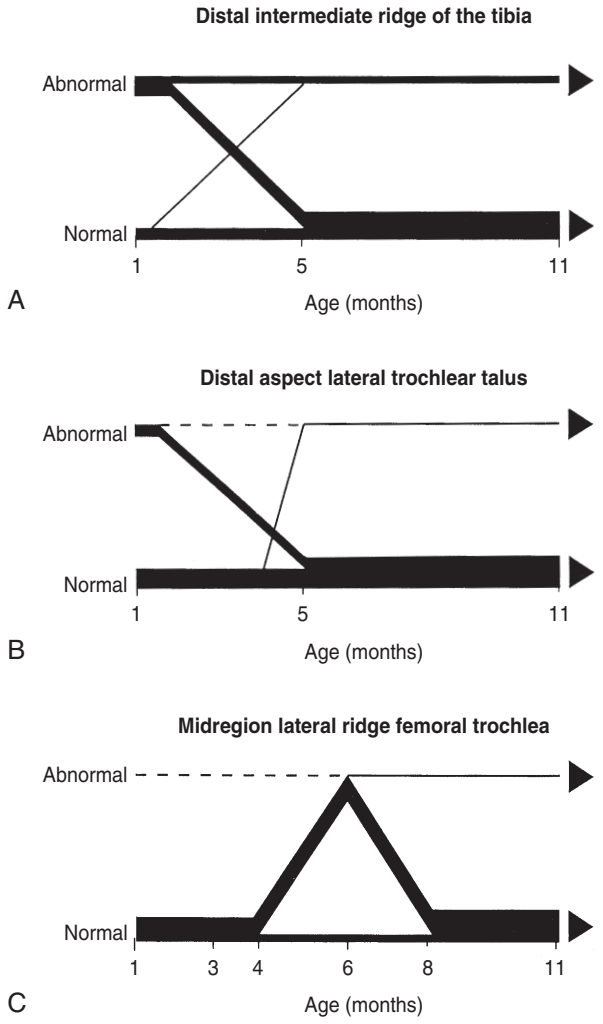

Figure 88-6. A, Schematic diagram of the early development of osteochondral lesions at the distal intermediate ridge of the tibia. At 1 month of age, several lesions can be identified that will heal *(thick line pointing down).* Only a few lesions originate after the age of 1 month *(thin line pointing up)* and, after the age of 5 months, the situation remains stable. **B,** Schematic diagram of the early development of osteochondral lesions at the distal aspect of the lateral trochlear ridge of the talus. The same general pattern as in **A** is seen, but healing potential is better. **C,** Schematic diagram of the early development of osteochondral lesions of the lateral femoral trochlear ridge. The pattern is distinctly different from that of the tarsus. Lesions develop only after the age of 3 months, peak at about 6 months, and have resolved at the age of 8 months, although some lesions will remain. (From Dik KJ, Enzerink EE, van Weeren PR: Radiographic development of osteochondral abnormalities, in the hock and stifle of Dutch Warmblood foals, from age 1 to 11 months. Equine Vet J Suppl 31:9, 1999 with permission from Equine Veterinary Journal Ltd.)

All these observations have led to the conclusion that OC is by no means a static condition, but in contrast is an extremely dynamic one in which lesions appear and apparently heal during the first months of life.[55] The acknowledgment of the dynamic character of OC has led to a profound change in the concept of this disease.

It is well known that the extracellular matrix of the articular cartilage goes through a phase of rapid remodeling in the fetal and neonatal animal. In a benchmark study on the early development of articular cartilage, the research group from Québec investigated very early changes in epiphyseal growth cartilage and articular cartilage (and the relationship between the two) in fetuses (6 to 11 months gestation length) and neonates (0 to 8 days) using histological techniques. They showed that the demarcation of articular cartilage from the much more voluminous growth cartilage occurs by 6 to 8 months of gestation and is followed by subsequent skeletal maturation. Importantly, they also showed areas in the growth cartilage, notably between the proliferative and hypertrophic zones near the ossification front where a dramatic change in collagen fiber organization occurred. These areas were interpreted as regions that were biomechanically very susceptible to induced trauma.[50]

It is in the juvenile period of ongoing endochondral ossification and rapid changes in growth that the topographic heterogeneity in biochemical composition that is necessary to withstand the locally different biomechanical challenges develops through the process of functional adaptation.[56,57] In this process, the collagen network of the articular cartilage is molded under biomechanical influences that induce both compositional and architectural changes[58] in a process not unlike that in bone.[3] However, whereas bone retains its capacity to remodel throughout the life of the horse, cartilage metabolism decreases quickly in the early juvenile period, and turnover times are very long in mature horses.[59,60]

The latter fact precludes any substantial remodeling or repair and makes the formation of the collagen network in the early juvenile phase a once-in-a-lifetime process that can have very important consequences for disease prevention in future life.[61] The sharp drop in collagen metabolism in the early juvenile period determines when the window for repair of lesions closes. Some lesions, either because they originate too late or are too large, do not have enough time for repair. These are the lesions that might eventually become clinically manifest. This means that in OC, a clear distinction should be made between the pathogenesis triggered by a number of etiologic factors, which can be supposed to be specific for OC on the one hand, and the probably largely unspecific repair process on the other.[62,63] A flow chart for the putative mechanism of OC based on this concept is presented in Figure 88-7.

The current concept is that OC is a highly dynamic disturbance of endochondral ossification that is intricately linked with the rapidly changing metabolic status of articular cartilage in the juvenile animal. This concept has led to some hypotheses and statements regarding the origin and implications of the

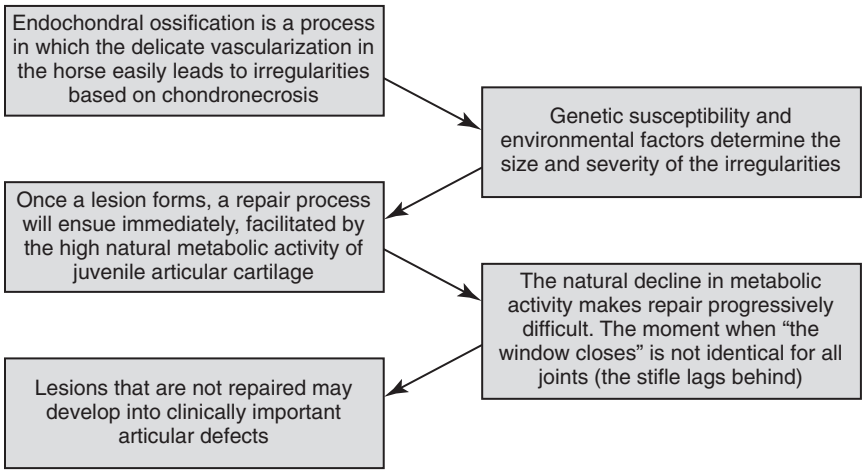

Figure 88-7. Flow chart outlining the supposed pathway along which clinical lesions eventually could become manifest in equine osteochondrosis.

disorder that were expressed at the Third International Workshop on Equine Osteochondrosis that was held in Stockholm in 2008. These assumptions and conclusions include that (1) "OC defined as irregularities in endochondral ossification is inherent to the equine species; modern management and breeding policies have caused the sharp rise in incidence," (2) "differences in collagen metabolism and/or gene expression between OC cartilage and normal cartilage may in many cases reflect the secondary repair process," and (3) "there may be a genetic correlation between susceptibility for OC and desired traits such as mature height, conformation and performance."[64]

ETIOLOGIC FACTORS

OC is a complex disease and multifactorial in origin. Biomechanical influences, exercise, failure of vascularization, nutrition imbalances, and genetic influences have all been incriminated as potential etiologic factors and are probably interrelated. Support for each etiologic factor is presented next.

Biomechanical Influences

Biomechanical loading plays a role in the pathogenesis of OC. This assertion is supported by the consistency of predilection sites within specific joints. It is probable that the dramatic changes in biomechanical loading that take place after birth are an important trigger for initiating lesions. In a pathologic study of the FP and TC joints of 9 fetuses and 10 foals aged 0 to 35 days, an OC lesion was detected in only one 3-day-old foal and none were found in any of the fetuses.[46] Although one group of researchers found many tiny areas of chondronecrosis in all 21 fetuses they studied, which they saw as a feature of normal development, they were not able to find specific changes in the collagen matrix compatible with early OC in any of them.[50] Other attempts to find OC lesions in fetuses have also failed.[65,66] Therefore biomechanical loading most likely plays a role in all proposed mechanisms for OC.

Biomechanical loading can exert an influence late in the process of OC development, where it causes the formation and loosening of cartilage flaps after faulty endochondral ossification, or early where it prematurely disrupts the vascular supply to neonatal cartilage (see next section). Biomechanical influences are strongly correlated with other etiologic factors such as exercise and genetics (through the determination of conformation of the animal). However, apart from direct blunt trauma that will cause an osteochondral fracture, biomechanical forces are considered a necessary additive factor rather than a sole cause of OC.

Failure of Vascularization

One report described the existence of cartilage canals in the epiphyseal cartilage long ago.[67] The author stated that "the primary function of cartilage canals is … the nutrition of cartilage too large to be supplied by diffusion of nutriments through their substance. … Their presence retards rather than hastens the end of ossification." The author also described the process of obliteration of these canals, called *chondrification*, which precedes ossification.

In the pig, extensive studies have been performed on the vascularization of juvenile cartilage, the physiologic process of regression of cartilage canals, and disturbances thereof as possible causes for OC. In this species, areas of chondronecrosis related to obliterated cartilage canals can be found, and they are much larger in commercial pig breeds than in miniature pigs from wild hog ancestry.[68] Artificial devascularization creates islands of cartilage without vascular supply, and these could develop into OC-like lesions.[69] These observations led to the hypothesis that premature interruption of the vascular supply of the growth cartilage of the articular–epiphyseal complex would lead to necrotic areas in the cartilage layer. These would later become engulfed in the ossification front and result in the typical irregularities and cartilage islands seen in OC.[70]

Later in-depth studies in this same area applied a more subtle and natural way of interrupting vascularization, by transecting only a limited number of cartilage canals, leading to the development of OC-like lesions.[71] No relationship between the overall regression pattern of cartilage canals and OC was shown. This led to the conclusion that OC is not the result of a general failure in endochondral ossification, and hence cannot be caused by a systemic factor such as growth rate, but is incited by local biomechanical factors.[72] This finding led, together with additional observations, to the hypothesis that OC was caused by local biomechanical damage to cartilage canals, especially to the anastomosing branches that run through the ossification

front from the bone marrow.[73] Based on their extensive work in pigs, some researchers suggested that a disturbance of the blood supply through cartilage canals to predilected areas of the growth cartilage, with ensuing formation of necrotic areas, was a pivotal element in the early pathogenesis of OC. The authors proposed a refinement of the existing nomenclature by introducing the terms *osteochondrosis latens* for focal areas of necrosis that are not clinically manifest, and *osteochondrosis manifesta* for where the necrotic area had developed into a delay in endochondral ossification. The term *osteochondrosis dissecans* would be retained in cases of cleft formation.[74]

In the horse, cartilage canals are present in the early juvenile period as well, and it could be assumed that a similar early pathogenesis seen in the pig would occur in the young foal. Much work in this area has been done. In the first cross-sectional study in random source foals (ranging from 191 days gestation to 153 days old), it was shown that lesions resembling those in pigs (i.e., characterized by chondrocyte necrosis and apparently caused by cartilage canal failure) were present in 9 out of 100 animals.[75] Interestingly, in all but the two youngest individuals (aged 12 and 18 days) an ongoing repair process was also noted. A highly interesting follow-up experimental study into the development of the vascularization of the tarsus of seven very young foals (0 to 7 days old) followed, showing in an elegant way how the advancing ossification front induces a change in the arterial supply of the cartilage canals in the growth cartilage from perichondral arteries to subchondral sources that are connected to the original cartilage canals through newly formed anastomoses that cross the ossification front (Figure 88-8). The authors found 12 lesions in these seven foals, all located where vessels crossed the ossification front to supply cartilage canals.[76] In a similar study focusing on the distal femur, another predilection site of OC, they could find in principle similar changes in vascularization, but the regression of blood vessels was much less extensive at this early age than in the tarsus, and no lesions could be found.[77] This may have to do with the fact that the FP joint lags behind in its development[50,51] and shows the peak of OC lesion development later than in most other joints.[27] In the MCP/MTP joint, again the pattern of changes in vascularization was similar; in this case one ("*latens*") lesion was found.[78] Of note, microcomputed tomography of the early lesions showed that the secondary repair process follows almost immediately after the formation of the lesion.[79]

As a result of recent studies, there now remains little doubt that, as in the pig, failure of vascularization through occlusion of cartilage canals (and not retention of these, as suggested earlier[80]) plays a crucial role in the early pathogenesis of OC in the horse.[76-79] Many features of equine OC, such as the joint-specific windows in time (related to joint-specific patterns in the progress of the ossification front and subsequent vascular rearrangements), and the frequent bilateral occurrence can be explained in this way. However, it should be realized that these

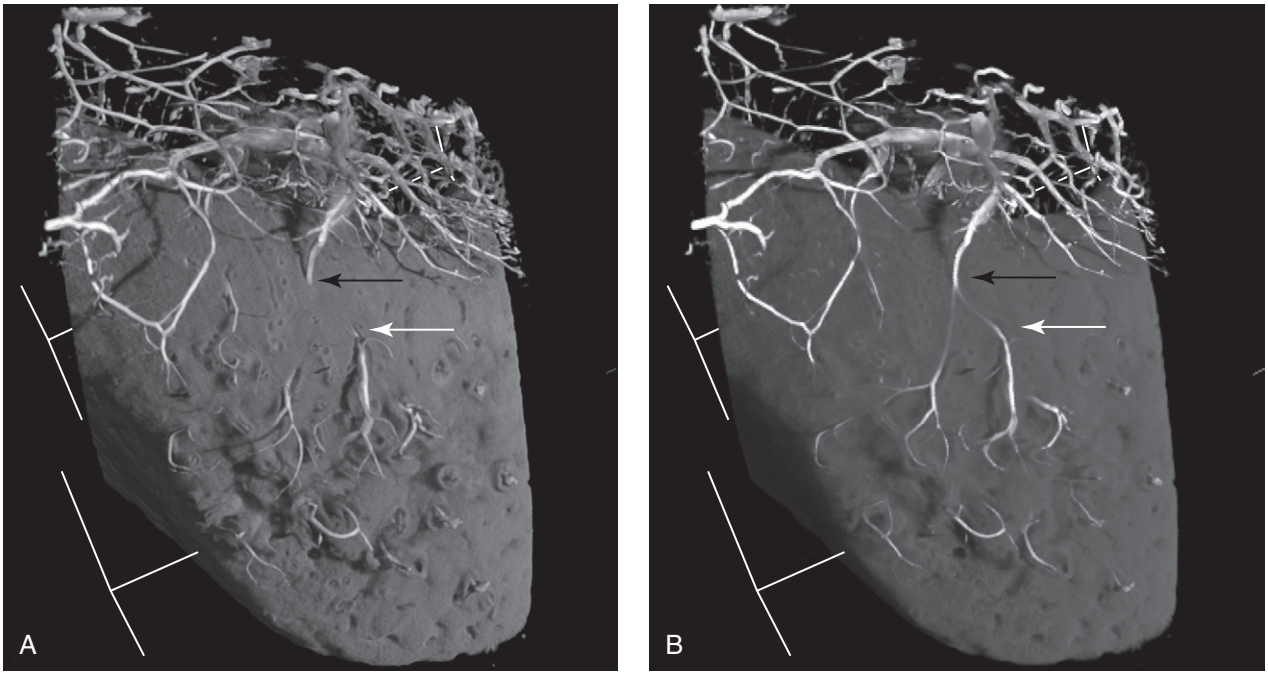

Figure 88-8. Images of three-dimensional volume-rendered models of micro-CT scans of a tissue block from the cranial part of the distal intermediate ridge of the tibia of a 3-week-old Standardbred foal. The block contained a permanent barium angiogram, and only the gray-scale segments representing barium and bone are shown. The block measured approximately 2 cm in all dimensions. **A,** A vessel originating from the perichondrial plexus on the cranial aspect of the distal tibia courses into the subchondral bone toward the cranial apex of the distal intermediate ridge *(black arrow).* Distal and caudal to this, toward the distal articular surface of the intermediate ridge, vessels emerge into the growth cartilage directly from subchondral bone *(white arrow).* **B,** In this model of the same tissue block, the gray-scale segment for bone has been rendered less opaque/more translucent than in model **A.** The vessels emerging from bone into growth cartilage *(white arrow)* are branches of the vessel entering bone on the cranial aspect of the intermediate ridge *(black arrow).* The model illustrates how the midsection of cartilage canal vessels is incorporated into the advancing ossification front during growth. This process, and in particular the requirement for vessels to traverse junctions between tissues of different qualities such as bone and cartilage, is believed to render vessels particularly vulnerable to failure. (Courtesy Dr. K. Olstad, Norwegian School of Veterinary Science.)

vascular events present an early mechanism and not the etiology, as still other factors (like biomechanical loading or even perhaps inferior quality of the cartilage extracellular matrix) are necessary to cause the lesion during the period of vulnerability. These factors will determine in the end why a given horse develops OC and another does not.

Exercise

Exercise is known to be the steering factor in the process of functional adaptation during which the biochemical composition of the extracellular matrix of articular cartilage takes on a topographically heterogeneous character.[57,81,82] This process takes place principally in the first year of life, with emphasis on the first few months. Given the important role of exercise in the juvenile period for cartilage development, it may be hypothesized that this factor would be of importance in the development of OC lesions too, because these originate in the same period that articular cartilage composition is molded.

Preliminary research in which controlled exercise was given to foals from 3 to 24 months of age using a high-speed horse walker yielded promising results. OC incidence was 6% in the high-exercise group and 20% in the low-exercise group, but results were flawed because of a concurrently running nutritional study employing different energy levels that were not consistently applied.[83,84] A study focusing on the effect of exercise alone, given from 10 days until 5 months of age, did not yield conclusive results, although there was a tendency toward a decrease in *severity* of OC lesions in the exercised groups.[12]

In a large field study, one researcher found fewer OC lesions on the distal sagittal ridge of the metacarpus and metatarsus in the MCP and MTP joints of foals that got more exercise during the first months of life. There was, however, no effect on the incidence of OC in the TC joint.[85] Another large field study conducted in Normandy (France) revealed an increased risk of OC in foals with irregular access to pasture, as well as in animals kept in very large plots. These results underline the somewhat ambivalent character of the effect of exercise.[34] Exercise might hence be a factor codetermining the final appearance of OC lesions, but it does not seem to be of primary pathogenetic importance.

Nutrition, Hormonal Factors, and Growth Rate

From the start of research into equine OC, much effort has been dedicated to nutritional factors. The studies can be roughly divided into research on minerals and trace elements, mainly copper and zinc, to a lesser extent calcium and phosphorus, and on dietary energy level. The latter factor is closely related to growth rate, a factor heavily incriminated in OC pathogenesis in a large number of species. However, growth rate is determined not only by energy intake but also by genetic predisposition.

Interest in trace elements was raised by a report on the relationship between low *copper* levels in serum and ceruloplasmin concentrations (a copper-transporting protein present in the blood) and OC.[86] Studies followed on the effect of deliberately feeding low-copper diets[87] and the possible role of zinc in inducing a relative copper deficiency.[88] The mechanism was thought to act via the enzyme lysyl oxidase, a copper-dependent enzyme that is essential for the formation of collagen cross-links. Zinc and cadmium can antagonize copper by displacing it from the sulfhydryl binding sites on metallothionein.[89]

The epidemiologic studies on dietary mineral and trace element levels and the occurrence of developmental orthopedic disease in Ohio and Kentucky seemed to give further evidence for a key role of copper.[90] The original National Research Council (NRC) recommendation[91] of 10 ppm copper in dry matter, which was based on a study with a limited number of horses,[92] was challenged, and supplementation studies were started. These yielded positive results to some extent,[93-95] but they certainly were not conclusive enough to pinpoint copper deficiency as a sole cause of OC.[96] Higher dietary copper levels of 20 to 25 ppm[95] or 50 ppm[97] were recommended, but they were later questioned following studies revealing that copper levels of 4.3 to 8.6 ppm in pasture were sufficient for a healthy development of bone and cartilage.[98] The evidence to support copper supplementation to pregnant mares is equivocal. Although one study showed an increase of liver copper concentration in newborn foals after prenatal supplementation of mares during late pregnancy[99] and noted a (minor) reduction in articular cartilage lesions at age 5 months,[98] another study conducted in the same country (New Zealand) failed to demonstrate both effects.[100] In both studies lesions were minimal, however, and no clinical OC was seen. This means that, if any effect on the initiation of lesions exists, this will be minor. It does not mean, however, that copper supplementation of pregnant mares or newborn foals, which may also raise liver copper content,[101] may have no clinical benefits, because another study showed that there was a relationship between neonatal liver copper concentration and the resolution of lesions, but there was no correlation with the initial occurrence.[53] It was concluded from that study that copper had a positive effect on the repair of OC lesions but not on their pathogenesis. This concept could explain many of the somewhat contradictory and inconclusive earlier findings. Therefore copper retains a clinically important role, because it is the outcome of both the pathogenetic process and the ensuing repair process that determines the eventual and clinically important lesions. Although the proposed mechanism of the effect of copper via lysyl oxidase has been challenged,[102] there is still mechanistic evidence for a role of copper, because *in vitro* research yielded additional information on the role of copper that appeared to have a chondroprotective effect through reduction of the activity of the proteinases cathepsin B and cathepsin D.[103]

High *calcium* levels did not influence the incidence of OC in foals, but high levels (four times the NRC recommendation) of phosphorus resulted in significantly more lesions.[104] The mechanism was supposed to be the induction of secondary hyperparathyroidism, which would lead to increased osteoporosis and subsequent weakening of the subchondral bone. The study is interesting because foals were studied in the period from 2.5 to 6.5 months of age, which is now known to be a period in which the dynamic process of OC is very active.

Dietary *energy levels* have been implicated in the pathogenesis of OC for decades.[105] This was mostly, but not solely, in relation to a high growth rate. In the early Scandinavian studies, it was stated that OC primarily occurred in large-framed, fast-growing animals, be they broilers, bulls, dogs, swine, or horses.[8,48] Later, the literature turned more ambivalent on the subject of growth rate. Several authors found no relation to growth rate,[14,106,107] but others reported a larger prevalence in horses taller at the withers[15,34] or in horses having a higher average daily weight gain.[108] Sometimes this was only true for a limited period of development.[109] In a study where a normal level of nutrition

was compared with an increase of 120% to 150% in all components of the diet, a fast growth rate was associated with a higher amount of OC lesions, regardless of whether the fast growth rate was determined by a higher nutritional level or by genetic predisposition.[54]

Although the data on the effect of growth rate are conflicting and the concept of a high growth rate putting pressure on the process of endochondral ossification and thus leading to irregularities might be too simplistic, there is substantial evidence for other mechanisms. Excessive levels of energy, especially when fed in the form of easily digestible carbohydrates,[110,111] result in a strong postprandial hyperinsulinemia. *Insulin* and its derivatives insulin-like growth factor-1 (IGF-1) and IGF-II have a direct effect on the process of endochondral ossification, acting as mitogens for chondrocytes and stimulating chondrocyte survival or suppressing apoptosis.[112] Many of today's horses are fed excessively and have low activity levels, which may, as in humans, lead to obesity and insulin resistance. The latter condition has been related to many pathological conditions, including laminitis and OC.[113]

Insulin also stimulates a rapid removal from the circulation of the *thyroid hormones* T_3 and T_4,[114] which are involved in the final stages of chondrocyte differentiation and in the metaphyseal invasion of growth cartilage by blood vessels.[115] In fact, OC-positive foals have a significantly lower IGF-1 activity than OC-negative foals.[116] In this way, high carbohydrate levels would induce a transient relative hypothyroidism and hence a retardation of the maturation of growth cartilage. Hypothyroidism in horses has been known to produce skeletal lesions, although they are not equivalent to those seen in OC.[117]

Interestingly, the effect of *carbohydrates* on thyroid hormone levels can be demonstrated in weanlings, but it is not present in yearlings.[118] It has been possible to induce cartilaginous lesions by administering diets with high levels of digestible energy.[104,119] Furthermore, horses with OC have been shown to have higher postprandial glucose and insulin responses to feeding high-grain ratios than did normal horses.[120]

Another indication of the importance of carbohydrate levels in the feed comes from a study in Kentucky on the influence of the season on the occurrence of OC. Early foals appeared to have a significantly higher incidence of OC in the TC joints, but late foals had a higher incidence in the FP joints. This seemingly contradictory effect of the season could be explained by the different windows of vulnerability of these joints, which appeared to coincide with the spring and autumn peaks in the high-energy value of the grass.[31]

There is hardly any doubt that the pathogenetic mechanism outlined here plays a certain role in the development of equine OC. However, it is unlikely that it is the sole mechanism, because many lesions provoked by the administration of high-carbohydrate diets were similar, but they were not always identical to those seen in clinical OC. Besides, many lesions were seen in the growth plate,[107] and in contrast to other species, clinical OC is rarely, if ever, seen in the growth plate in the horse.

Genetics

In many species, the incidence of OC varies considerably with breed. In wild boars, the disease seems to be nonexistent, whereas it is common in many commercial pig breeds.[70] In horses, lesions are rarely found in ponies and have a much lower incidence in feral horses.[37,38] This suggests a considerable genetic influence. Further evidence comes from studies showing large differences in incidence of OC between progeny groups of different stallions.[15,47,121]

There is no doubt that OC is a polygenic trait and that the method of inheritance is complex.[122] Some estimates on heritability have been made, but they differed widely. For the TC joint, heritabilities of 0.24,[123] 0.26,[15] and 0.52[124] have been reported. A large progeny study in the Netherlands succeeded in establishing reliable heritabilities for navicular disease and bone spavin, but it did not give unequivocal results with respect to TC OC, which finally was estimated to be approximately 0.25.[125]

Heritabilities of 0.25 and higher are high enough to warrant selection programs, which indeed have been implemented by many studbooks. However, progress has been disappointing. The Royal Dutch Warmblood Studbook (KWPN) started rejecting any candidate sire with even the slightest radiographic sign of TC OC in 1984 and any sign of FP OC in 1992, but no substantial decrease in incidence had been realized by mid-2010. This is in contrast to the conditions of navicular disease and bone spavin, where a similar selection policy has been followed. The number of potential stallions rejected for these disorders at selection events has fallen dramatically over the past decades.[126]

There are various explanations for this. OC is classically seen as an all-or-none trait with an underlying continuous scale.[122] The genetic background may, however be much more complicated than originally supposed. First, there is mounting evidence that different genes are involved in different joints. This translates to different heritabilities for different joints. In a study where OC was scored on a 5-point categorical scale in 811 yearlings, the h^2* at animal level was estimated at 0.23, which concurs well with the other studies mentioned earlier. However, the figures per joint strongly varied, being high (0.36) for the TC joint, intermediate (0.11) for the MCP/MTP joints, and very low (0.06), for the FP joint.[127] Heritabilities are largely breed dependent. A study on South German Coldbloods revealed heritabilities of 0.16 for the MCP/MTP joints, which is comparable to the figure found by another group, but of not more than 0.04 for the TC joint.[128] A second point complicating the genetic approach of OC is its dynamic nature. This feature allows many animals to be radiographically free of lesions at age 3 or 4 years when they enter the stallion selection procedures, yet they will have had evidence of the disease as a foal. Hence, these foals are genetically predisposed to OC and will pass this trait on. Realization of the complexity of the genetic background of OC has led to adaptations in selection policies in certain studbooks. The KWPN now uses a more differentiated approach in which lesions are classified and stallions with minor lesions still may pass. Approval is temporary, however, because data from offspring are now included in the selection procedure and are used to calculate breeding values. If a provisionally approved sire produces a high prevalence of OC in its offspring, it may still be eliminated.

Recently, studies have been undertaken to identify OC-related genes through modern molecular genetic techniques. The previously mentioned study on South German Coldbloods involved mapping of quantitative trait loci (QTL) for OC and

*h^2 is the broad-sense heritability and reflects all possible genetic contributions to a population's phenotypic variance.

POF in the MCP/MTP and OC in the TC joints. QTL with chromosome-wide significance on was found on 10 chromosomes.[129] The researchers later expanded the data from their whole-genome scan further and found an association of a single nucleotide polymorphism (SNP) located in the acyloxyacyl hydrolase (AOAH) gene on chromosome 4 with OC in the MCP/MTP joint[130] and of other SNPs located in the Xin-actin-binding repeat containing 2 (XIRP2) gene with OC in both MCP/MTP and TC joints on chromosome 18.[131] A similar genome-wide approach in Hanoverian horses identified QTLs with chromosome-wide significance on chromosomes 2, 3, 4, 5, 15, 16, 19, and 21.[132] Further investigations identified a relevant QTL on chromosome 5, indicating collagen type XXIV as a potential functional candidate gene.[133] A later study, in which marker density on chromosome 18 was increased based on the earlier observations by the same group in South German Coldbloods,[131] showed a new QTL on this chromosome that was associated with OC and located very close to the parathyroid hormone 2 receptor gene, qualifying the latter, which had earlier been implicated in familial early-onset osteoarthritis (OA) in humans,[134] as an interesting positional candidate gene.[135]

Another approach was taken by a group, who compared gene expression profiles of leukocytes from horses affected by OC and normal controls, and identified dysregulation of a number of pathways, among others Wnt,* Indian hedgehog (Ihh), and transforming growth factor-β (TGF-β) signaling, in OC-affected animals.[136] The problem with this approach, as with the earlier described genome-wide scans, is that material from horses with established OC is used so that no distinction can be made between primary and secondary processes. This applies to a much lesser extent to work performed on the very young foals from the experimental study[76] on vascular changes in the growth cartilage alluded to earlier, where the Tousler-like kinase 2 gene (TLK2) and an unknown gene were found to be upregulated in foals predisposed to OC.[136]

It is clear from the previous discussion that the genetic background of OC is complex and not very easy to unravel. This may have to do with the fact that most genetic studies try to link the final phenotype, and thus the end product of various processes that have most probably different genetic backgrounds, to certain genes. It is highly unlikely that in the near future OC can be selected against using a simple genetic test that detects one or two culprit genes.

MOLECULAR MECHANISMS

The need for a more fundamental approach to understanding and preventing OC is obvious,[137] and in recent years a large part of research on equine OC has focused on the molecular mechanisms involved. Both *chondrocyte behavior* and composition of the extracellular matrix have been targets of study. A study found that chondrocytes that were freshly harvested from fetal growth cartilage underwent hypertrophy and subsequently apoptosis when cultured in either the commonly used fetal calf serum or horse serum in a situation that strongly resembled the natural processes involved. Chondrocytes from animals older than 5 months did not undergo hypertrophy, and chondrocytes from neonates (7 days) showed intermediate behavior.[138] This research once more stresses the importance of age when studying OC. Failure to undergo hypertrophy as main cause for OC was discarded in a study in which OC was induced in 3- to 6-month-old foals by a high-energy diet and that investigated the expression of hypertrophy-related genes, such as collagen I, II, and X; matrix metalloproteinase (MMP)-13; and the transcription factor Runx2. The higher expression of collagen X and MMP-13 (the main matrix metalloproteinase involved in collagen degradation) indicated that failure of chondrocytes to undergo hypertrophy is not the cause of OC, as had been suggested earlier.[139] Another study found changes in proteoglycan composition of osteochondrotic fragments, but they could not tell whether this was primary or secondary.[140] This is a constant problem, because data from clinical cases and fragments collected at surgery are not representative of the initial phase of the disease.[7]

Studies have focused on normal and abnormal (dyschondroplastic) cartilage, on the expression of various *collagen types* that are represented in the extracellular cartilage matrix (collagen types II, VI, and X), and on the expression of growth factors (TGF-β, IGF-1, IGF-2) that are known to play a role in the development and maturation of cartilage.[112,141-145] There appeared to be distinct differences in expression patterns between normal and abnormal tissues, with notably high levels of activity around the chondrocyte clusters or chondrones in early cases of OC. This increase in activity is in line with the higher level of chondrocyte metabolism demonstrated by one research group[146] and could be the primary cause, but it is more likely a secondary event representing an attempt at repair.[147] The borderline between the OC lesion itself and repair tissue is vague, probably because the onset of repair is almost immediately after formation of a lesion and because events are taking place in juvenile cartilage that still has considerable regenerative capacity. In a study where OC tissue was compared with tissue from healing surgically created osteochondrotic fragments, no discrimination could be made between the two types of tissue. Both resembled an anabolic, reparative process when compared to age-matched controls. However, immunohistochemically, the OC tissue bed stained positive for chondroitin sulfate and collagen type II, which the fracture bed did not.[51]

Little work has been done on the *subchondral bone* underlying the cartilage defects. One group showed changes in bone morphogenic enzymes and in membrane lipid composition of the cellular components of the subchondral bone.[148] There are clear indications that the bone component is involved in lesion formation in OC too, as there is a strong correlation between serum osteocalcin levels as early as at 2 weeks of age with radiographically scored OC at 5.5 and 11 months and postmortem scores at 11 months (i.e., when lesions had become virtually immutable).[149] Osteocalcin might therefore be a potential marker for the susceptibility of individual animals to develop OC. The relationship between OC and osteocalcin may indicate very early events indeed, because in another study where serum from foals aged 1 to 49 days was used, no such correlation was found.[150]

Levels of MMPs were found to be elevated in copper-deficient horses with clinical OC lesions,[95] and there is increasing evidence that changes in collagen metabolism play an important role in the molecular mechanism of OC. The Cambridge group

*The Wnt (pronounced "wint") signaling pathway is an important pathway in intracellular signaling. The name comes from "wingless" (referring to a *Drosophila* mutant where all these basic pathways have been discovered).

performed in-depth studies on the distribution of cathepsins B, D, and L in normal and abnormal cartilage. They found physiologic differences in distribution[151-153] as well as a strong increase in cathepsin B activity in chondrocyte clonal clusters in OC.[154] The latter finding was confirmed in a study on the effect of copper supplementation.[155]

Another study revealed an increase in activity of *gelatinases* (MMP-2 and MMP-9) in osteochondrotic cartilage.[156] However, expression of membrane-type matrix metalloproteinase-3 (MT3-MMP) is not significantly altered in osteochondrotic cartilage.[157] The increased collagen turnover in osteochondrotic cartilage is reflected by an increase in collagen split products that can be detected in the synovial fluid. An increase in the C-propeptide of collagen type II was detected in synovial fluid from OC horses, but a decrease was found for the epitope 846 of aggrecan, demonstrating different alterations in aggrecan and collagen turnover in OC.[158] This finding was confirmed in a later *ex vivo* study where no evidence was found for an increase in proteoglycan metabolism, but an increase in the levels of the $col_{2\text{-}3/4\ short}$ epitope indicated increased collagen breakdown.[159] The different signature of osteochondrotic cartilage with respect to proteoglycan and collagen metabolism was further underlined by a study on expression patterns and chondrogenic potential of osteochondrotic cartilage (from mature animals) versus normal cartilage (from age-matched controls) where OC cartilage showed increased expression of collagen types I, II, III, and X; MMP-13; ADAMTS-4; and TIMP-1; and decreased expression of TIMP-2 and TIMP-3. Furthermore, pellet cultures produced from OC tissues contained significantly less glycosaminoglycans (GAGs).[160] *In vivo* increased collagen degradation has also been shown.[161]

One group was able to predict the presence of OC based on the detection of *collagen markers* in serum.[162] In line with this, a well-controlled experimental study revealed that the ratio of the markers for collagen anabolism and catabolism (CPII/CIIC) at 20 weeks had strong positive correlations to OC, as diagnosed radiographically at 5.5 months.[149] Additional evidence for the crucial role of collagen was provided by studies demonstrating a significant increase in the levels of MMP-1 (but not general MMP activity or specific activity of MMP-3), and in the levels of the collagen degradation product hydroxyproline in synovial fluid from horses with OC compared to OC-free animals.[163-165] Additionally, differences in posttranslational modifications of collagen type II have been demonstrated in samples from early lesions.[166] Serum levels of the collagen degradation marker Coll2-1 were lower in OC-affected horses, and levels of the nitrated form Coll2-1NO_2 were higher. These findings point to some degree at an inflammatory state and at the retention of cartilage, rather than at accelerated breakdown.[167] Also the inflammatory marker myeloperoxidase (MPO) was higher in serum from horses suffering from OC, compared to age-matched controls.[168] However, the subjects of both studies were (young) mature animals in which only secondary processes could be expected to occur. In a study on synovial fluid markers in another group of mature horses, an indication for the existence of low-grade inflammation was also found in the elevated leukotriene B_4 (LTB_4) level, which was putatively associated with the occurrence of joint distention.[169] Overall, there now seems to be conclusive evidence that changes in collagen metabolism play a crucial role in the molecular mechanism of OC. The question of whether this is a secondary or a primary event remains unanswered. There is also low-grade inflammation, which seems to be reactive in nature and is not a primary factor.

Parathyroid hormone related protein (PTHrP) and Ihh have a role in controlling cartilage differentiation and hypertrophy in the growth plate.[170] This influence is mediated by *bone morphogenetic proteins* as signaling peptides. This led to the hypothesis that these molecules would have a similar role in cartilage differentiation in the equine articular–epiphyseal complex and could be implicated in the pathogenesis of OC. There was indeed a significant increase of PTHrP and mRNA expression in chondrocytes from OC-affected cartilage and a trend in Ihh.[171] Expression of bone morphogenetic proteins 6 and 2 was not changed.[172] Also, expression of transcription factors Gli1 and Gli3 was not increased, suggesting either a different transcription factor in osteochondrotic tissue, a dysfunction in local receptor activation, or elevations in Ihh inhibitors. It should be noted that these investigations were performed on samples from horses aged 6 to 18 months, which makes their outcome likely to be representative for early repair rather than for the pathogenesis of lesions.[173] The same applies to the observation that serum IGF-1 levels were higher in a large group of OC-affected horses aged 15 months to 10 years compared to controls.[174]

TREATMENT

Nonsurgical Management

Treatment of small OC lesions might not always be necessary. Single small, radiographically obvious OC lesions in the TC joint without effusion did not influence performance in racing Standardbreds, in contrast to joints where there was significant effusion.[123] There is a negative effect on performance, however, if the lesions are more severe.[175] Trotters with OC lesions in the TC joint had a significantly lower number of starts and somewhat lower earnings compared to controls.[176]

Nonsurgical treatment consists principally of rest and controlled exercise. Systemic NSAIDs and intra-articular medication (corticosteroids to enhance resolution of joint effusion and certain disease-modifying osteoarthritic drugs such as hyaluronan, chondroitin sulfate, or pentosan sulfate) may or may not be administered, but they have not been reported to be of great value in OC.[177] Given the nature of the disease described earlier, nonsurgical management can only be expected to be successful in either very young animals, where there is still good capacity for regeneration, or in very mild cases.

A large study on the natural history of FP OC in three crops of Thoroughbred foals showed improvement and repair of several lesions, which was compatible with the age of the animals studied.[13] One group described a favorable outcome of nonsurgical treatment of TC OC in a group of Standardbreds, half of which were treated nonsurgically and half surgically. The authors mentioned, however, that these results were biased, because the more severe cases tended to be treated surgically.[178]

One study concluded that nonsurgical treatment of TC OC (consisting of simple box rest or a less intense training program with a low-energy diet) was a good option in Warmbloods (80% success rate), but not in Standardbreds (25%). It should be noted that in Warmblood horses, especially those destined for dressage, resolution of joint effusion is often a therapeutic goal in itself. This aspect was not classified as such in this study.[179]

Surgical Management

Surgical management is the treatment of choice in most cases.[180,181] Clinical experience has taught that arthroscopic surgery presents definite advantages over arthrotomy.[182] Soft-tissue trauma is less, the convalescent period is considerably shorter, and functional and cosmetic recovery are better. An additional advantage is that a more comprehensive examination of the joint is possible.[6] A direct comparison of arthroscopic treatment and treatment by arthrotomy demonstrated that hospitalization time was almost five times shorter after arthroscopy than after arthrotomy. Even more importantly, horses returned significantly more often to their intended use.[182]

Arthroscopic surgery is widely used in the FP, TC, MCP, and MTP joints to treat OC. In some other joints where OC is encountered less often but can present a serious clinical problem, such as the scapulohumeral joint, an arthroscopic approach is feasible as well, but the technique is considerably more difficult.[25,183] The surgical techniques applied are discussed in more detail in Chapter 13 and in the relevant chapters discussing the respective joints.

PROGNOSIS

The prognosis after surgical intervention varies among joints and depends on the amount and extent of the lesion. It further depends on the definition of "favorable outcome." In most racing Thoroughbreds and Standardbreds, a favorable outcome means a sound horse that can compete at its maximal athletic capacity. In many show horses, the cosmetic appearance is important as well. In general, prognosis for a return to athletic activity is fair to good for the majority of joints involved.

For the FP joint, a 64% success rate was reported,[184] but this figure might be too pessimistic because the study included many horses operated on at a young age before their first performance. Therefore it included a number of horses that would never have raced despite the OC lesions because attrition rates in Thoroughbred training are high and many young horses will never race. Jockey Club records indicate that only about 60% of all Thoroughbreds intended for racing ever reach the starting gate in the United States.

In another study, 19 of 25 arthroscopically treated horses (76%) were able to perform their intended use.[182] Racing performance in Thoroughbreds treated for FP OC was not different from that in unaffected siblings, but fewer horses raced as 2-year-olds and earnings were less, both at 2 and 3 years of age.[185] Also, 2-year-old Thoroughbreds and Standardbreds that were operated on for OC in the TC joint were less likely to race, compared to unaffected siblings at 2 years of age, but findings were not different between groups at 3 years of age.[186] The differences might be mainly the result of the delay in training caused by the surgery.

One group reported success rates of 73% and 83% for the TC joint in racehorses and non-racehorses, respectively. Synovial effusion resolved in 89% of racehorses and 74% of non-racehorses.[19] Of the other joints, prognosis is least favorable in the shoulder joint, with 46% success reported.[25] In one study a favorable outcome of only 15% was found in racehorses suffering from shoulder OC, regardless of whether treatment was surgical or nonsurgical,[187] emphasizing the poor prognosis of shoulder OC in animals that have to perform strenuous exercise.

In the MCP and MTP joints, there is some confusion about the extent to which radiographically detectable lesions are part of the OC complex. Fragments of the dorsal aspect of the distal metacarpus and metatarsus are commonly accepted to be osteochondrotic, and in these, 57% success has been reported after surgery. This figure is negatively biased because 18% of the cases were classified as unsuccessful for other reasons that made the horse unsuitable for use.[188] A more recent source mentions 90% return to athletic activity if the lesion is located in the more proximal part of the sagittal ridge but an unspecified lower rate for lesions in weight-bearing areas.[189]

Prognosis for arthroscopic removal of osteochondral fragments of the dorsal articular margin of the proximal phalanx, of which the osteochondrotic nature is still contentious, is given as nearly 100%.[188]

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CHAPTER
89

Subchondral Bone Cysts

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DEFINITION

Subchondral cystic lesions (SCLs) are commonly referred to as subchondral bone cysts and are considered by some to be part of the developmental joint disease complex. SCLs in bone were once considered a coincidental finding in horses and not necessarily related to lameness or disease.[1,2] Currently they are recognized as a serious cause of lameness that can be difficult to treat.[3] They are characterized by radiolucent areas of bone (from several millimeters up to and even larger than 3 cm) often accompanied by a thin, but well-demarcated, sclerotic rim (Figure 89-1). The center of the SCL is usually filled with myxomatous tissue, with or without fluid that resembles normal or slightly blood-tinged synovial fluid.[4] The SCL cavity was thought to be lined with a synovial-like membrane that was responsible for fluid production; however, this membrane cannot be verified in all instances.[4]

SCLs can be uniloculated or multiloculated and occur usually in the subchondral bone underlying the articular cartilage in a weight-bearing area of the joint.[3-6] Occasionally they are also found in the metaphysis or close to the growth plate (see Figure 89-1, *B*).[4,7]

ETIOLOGY AND PATHOGENESIS

In the past, SCLs were considered part of the osteochondrosis (OC) complex.[8,9] However, the location of OC lesions is different from that of SCLs. Normally, OC lesions occur at the transition from the weight-bearing to the non–weight-bearing articular surface,[10] whereas SCLs are found underneath the cartilage in a weight-bearing area of the joint.[4] All in all, many pathologic mechanisms leading to the development of SCLs have been proposed.[1,3-14] However, only two hypotheses have been supported experimentally in horses. The first hypothesis is based on a *hydraulic theory* and was supported by an experiment in which a slitlike lesion created in the articular cartilage led to the development of SCLs over a period of several months.[15,16] Other coincidental findings of SCLs in experimental studies using osteochondral grafting procedures support this theory, because SCLs developed at the host–graft interface

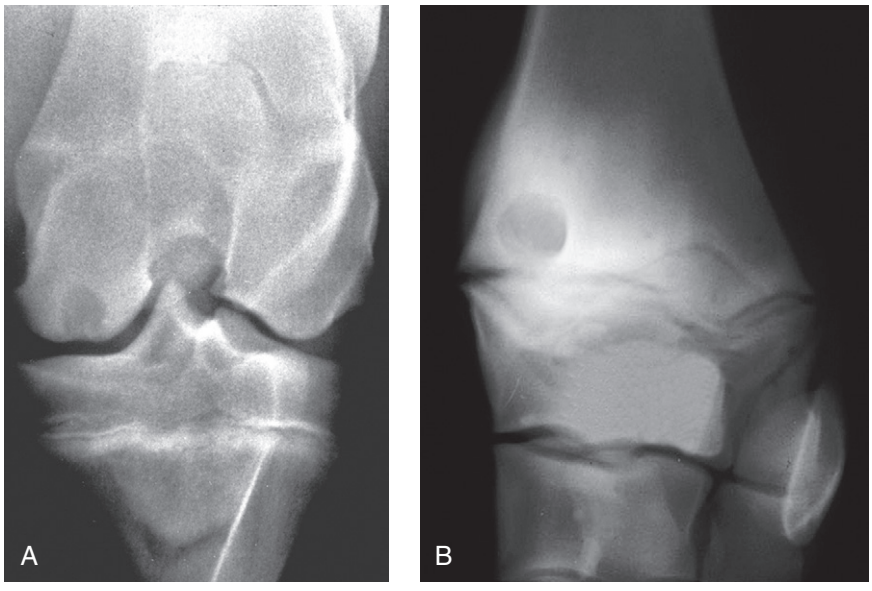

Figure 89-1. A, Radiographic view of a subchondral cystic lesion (SCL) in the medial femoral condyle of the stifle joint of a 3-year-old mare. The lesion is in the center of the weight-bearing area of the condyle and shows as a radiolucency, well demarcated from the surrounding normal bone by a thin sclerotic rim. **B,** Radiographic view of an SCL adjacent to the distal radial physis of a young foal. Note the periosteal new bone formation at the dorsomedial metaphysis.

between the osteochondral transplants and the host bone.[14,17,18] These experiments had a common denominator: there was primary cartilage damage followed by secondary intrusion of synovial fluid. The fluid was thought to place mechanical pressure on the subchondral bone through its hydraulic action during weight-bearing, resulting in necrosis of the subchondral bone plate. The second hypothesis (*inflammatory theory*) evolved around cellular and molecular mechanisms, when inflammation could be demonstrated in tissues and cystic fluid harvested from SCLs in horses.[7] The fibrous tissue harvested from the center of surgically managed lesions contained elevated concentrations of the local proinflammatory mediator prostaglandin E_2 (PGE_2), which also was found in high concentrations in the cystic fluid.[19] In addition, an upregulation of interleukin 1 (IL-1) and IL-6 was documented in the SCL. Using quantitative real-time polymerase chain reaction (qRT-PCR), an upregulation of IL-1 could be demonstrated at the periphery and upregulation of IL-6 at the center of the lesion.[20] Lastly, increased recruitment and activation of osteoclasts could be elicited *in vitro* when media of cultured fibrous, cystic tissue, and cystic fluid was incubated with osteoclasts isolated from 1-day-old rats (the pit resorption assay).[19] This was attributed to the combined effect of the cytokines IL-1 and IL-6 and the mediator PGE_2, which are normally elevated in clinical cases of pathologic bone resorption and inflammation.[21-23]

Other mechanisms have been suggested in the development of SCL in horses. These include primary subchondral bone damage and secondary collapse of overlying cartilage, disturbances of endochondral ossification, primary intraosseous fibroplasias or metaplasia, herniation or protrusion of synovial membrane through a small fissure in the articular surface, and vascular disturbances.[1,4,6,24] In contrast to the two theories presented earlier, none of these have been supported experimentally.

EPIDEMIOLOGY

An extensive literature review[4] involving 703 lesions in 619 horses (84 bilateral) revealed that SCLs occur mainly in the medial femoral condyle (MFC) (45.8%), followed by the phalanges (26.2%) (which included the navicular bone—18.5% of all phalanges), carpal bones (7.1%), metacarpal and metatarsal bones (6%), tibia, radius, talus, proximal sesamoid bones, humerus, patella, scapula, distal tarsal bones, and hemimandible (all less than 5%).[4] Sex distribution revealed that 62% of SCLs occur in males (41% stallions and 21% geldings). Thoroughbreds represented the majority of affected animals (39.5%), followed by Quarter Horses (14.1%), Crossbreds (12.2%), Standardbreds (9.9%), Arabian horses (8.5%), and Warmbloods (7.3%), with less than 5% in various other breeds. It seems that Warmbloods were overrepresented with cysts that occurred in the phalanges,[25] often accompanied by osteoarthritis.[3,26] Other sources report locations where lesions were encountered rarely, such as the proximal tibia and the patella.[12,27]

CLINICAL SYMPTOMS

Horses are often presented with lameness in the affected limb with or without joint effusion.[1] For example, when SCLs are located in the MFC, joint distention may be difficult to appreciate. Lameness is attributed to increased intracystic or intraosseous pressure, or both.[15] SCLs occur mostly in young horses between the ages of 1 and 3 years, when lameness commonly appears at the onset of training.[4] If SCLs are seen in older horses (Warmbloods), they often are accompanied by osteoarthritis (Figure 89-2) and associated with a poor prognosis.[1] Although the onset of lameness can be acute, the lesions are usually longstanding, having formed over time.

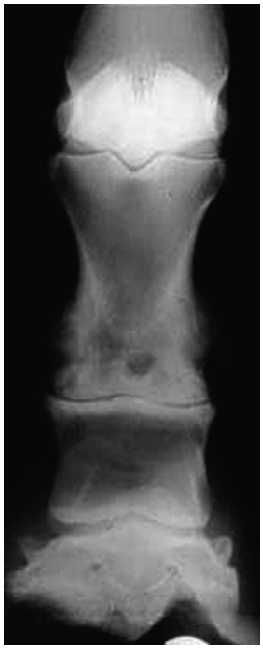

Figure 89-2. Radiographic view of a subchondral cystic lesion in the distal aspect of the proximal phalanx of a 9-year-old Warmblood gelding. The lesion is accompanied by osteoarthritis of the proximal interphalangeal joint.

SCLs usually can be localized using routine lameness examinations followed by standard radiographic views of the affected joints. In rare cases, when SCLs cannot be visualized radiographically,[1,5] computed tomography CT) has been of great value (Figure 89-3). Obviously, CT is applicable only from the carpus and tarsus distally, whereas scintigraphy can be used for other regions proximally. In one report, a variety of occult lesions in various bones of the tarsus, which were not radiographically visible, were localized using scintigraphy and confirmed with computed tomography.[28] SCLs also can be found unexpectedly during exploratory arthroscopic examination.

Communication between the SCL and the joint only occurs in 30% of affected animals[4] and often can only be localized during arthroscopic surgery (Figure 89-4) after insertion of an instrument across the overlying cartilage. The cartilage overlying an SCL is generally normal looking except close to the canal, where signs of matrix degradation can be detected (Figure 89-5). When SCLs are associated with osteoarthritis, signs pertinent to osteoarthritic cartilage lesions are present (see Chapter 78).

Tissues harvested from SCLs at the time of surgery or necropsy (Figure 89-6) reveal dense fibrous or myxomatous tissue containing necrotic bone, calcified or mineralized areas, or in some instances fibrocartilage.[4,8,24] The cystic wall consists of elongated fibroblasts aligned parallel to collagen bundles, scattered macrophages, and polymorphonuclear cells. Hypervascularity and thickened bone trabeculae can be noticed in the adjacent bone.[4]

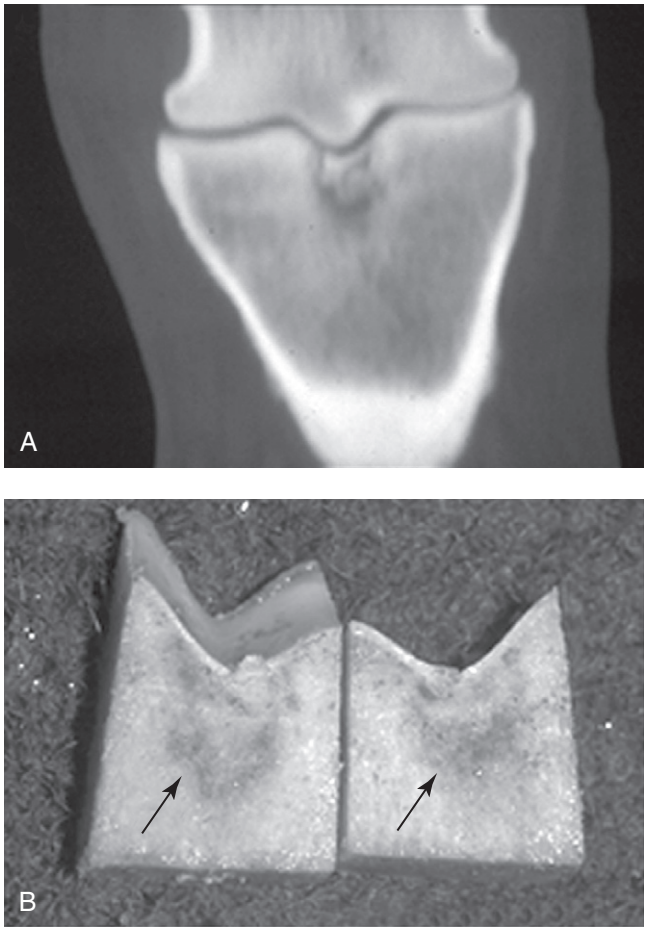

Figure 89-3. A, Computed tomographic image of a subchondral cystic lesion (SCL) in the metacarpophalangeal joint of a 4-year-old Warmblood mare, which was not apparent on radiographs. **B,** The postmortem specimens demonstrate that the SCL is not completely formed and that bone was still present within the SCL cavity *(arrows)*.

DIAGNOSIS

The diagnosis is made through a routine lameness examination. Flexion tests usually aggravate the lameness. Synovial distention is commonly seen, apart from joints proximal to the carpus and tarsus. One exception is in the stifle: when the medial femorotibial joint (with an SCL) communicates with the femoropatellar joint, it becomes effusive. Lameness usually improves with intra-articular anesthesia, and radiographic examination provides a definitive diagnosis in most instances.[5] The same joint in the contralateral limb should be evaluated radiographically to detect bilateral lesions. Other joints should be radiographed if effusion is present, because of the potentially multifocal nature of the disease. The size of the lesions on the radiographs can vary from a flattened condyle to a slight indentation in the subchondral bone surface to a round or oval lesion a few centimeters in height and width.

SCLs are graded using the following classification system[1,16]:

- Grade 1: Lesion less than 10 mm (4 inches) and usually dome shaped
- Grade 2a: Lesion more than 10 mm in depth with a narrow cloaca

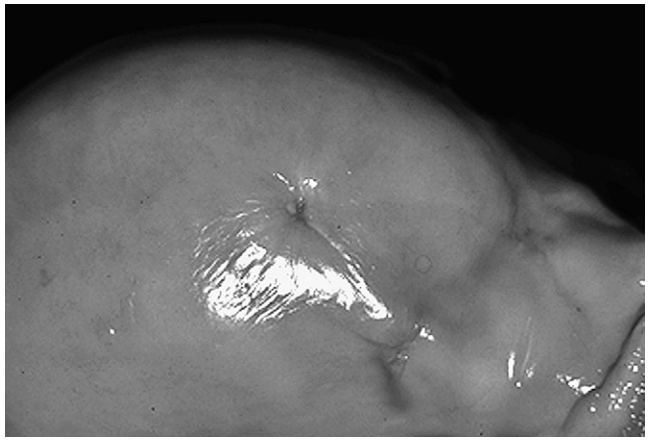

Figure 89-4. Postmortem specimen of a femoral condyle demonstrating a communication of a subchondral cystic lesion with the joint, which occurs in only 30% of the cases.

- Grade 2b: Lesion more than 10 mm in depth with a wide cloaca
- Grade 3: Condylar flattening or small defects in the subchondral bone
- Grade 4: Lucency in condyle seen with no radiographic evidence of a cloaca

The size of the lesion and the amount of articular cartilage involved has been correlated with clinical signs and prognosis in one study (70% returned to racing with less than 15 mm surface involvement after débridement versus 30% with more than 15 mm[29]), but this finding has not been confirmed in others.[1,16]

MANAGEMENT

Nonsurgical Management

Nonsurgical management of SCLs involves rest and the use of nonsteroidal anti-inflammatory drugs (NSAIDs), vitamin supplements, and anabolic drugs. This type of treatment is not very successful, because one study reported a failure rate of 66%.[4] Other reports on conservative management of this condition, with or without the use of nonsteroidal anti-inflammatory medication, listed success rates of 45% to 64%.[11,30] Interestingly, in one study, no significant differences were found in racing results between yearlings with radiographically diagnosed SBCs in the MFC and the unaffected horses, which led to the prediction that some lesions spontaneously resolve without treatment.[30] Nonsurgical management of older horses also suffering from osteoarthritis has a worse prognosis.[31] One author reported that horses resuming athletic activity following conservative management redevelop lesions.[32]

Intralesional corticosteroid injections do have a better success rate than NSAIDs and rest but are usually performed under arthroscopic guidance. This technique is described in the next section.

Surgical Management

SCL enucleation is the treatment of choice. The animal is anesthetized and, depending upon the location of the SCL, the preference of the surgeon, and the approach selected, the patient is

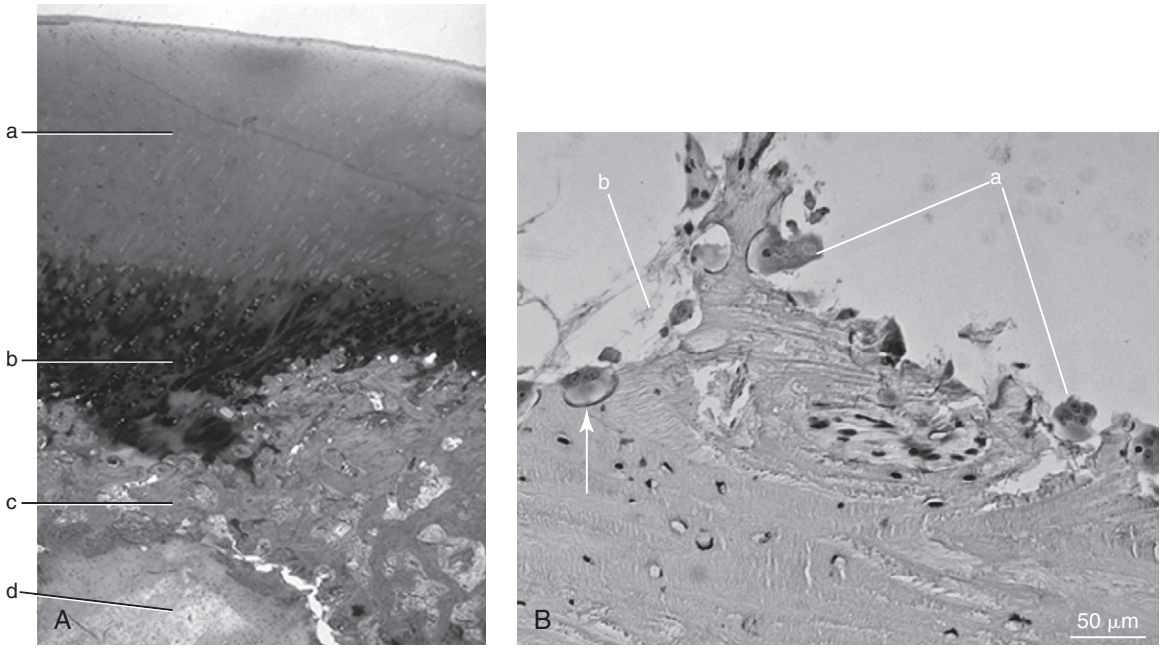

Figure 89-5. Histology specimen of a typical subchondral cystic lesion (SCL) of a horse. **A,** The cartilage overlying the SCL is normal except where it communicates with the joint. *a*, Normal articular cartilage; *b*, calcified cartilage layer; *c*, subchondral bone, *d*, cyst cavity. (Undecalcified 5-μm section, embedded in acrylic resin, stained with toluidine blue.) **B,** Osteoclasts *(a)* are lining the calcified bone *(b)* at the periphery of the SCL. The lacunae are easily recognizable. (Undecalcified 5-μm section, embedded in acrylic resin, Pentachrom Movat stain.)

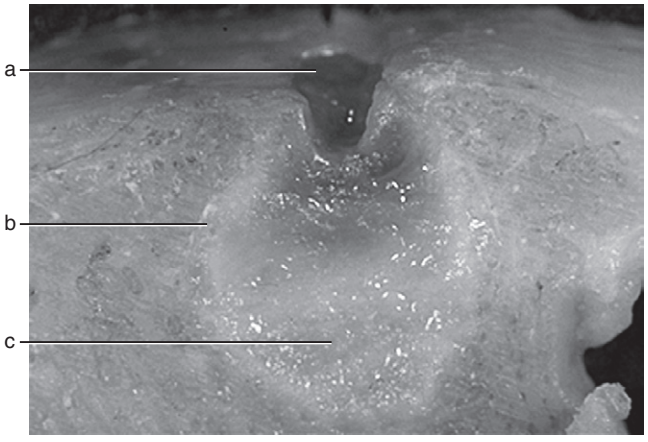

Figure 89-6. Postmortem specimen of a distal sesamoid bone showing a subchondral cystic lesion (SCL) in cross section. *a*, Communication into the joint; *b*, sclerotic border of the SCL; *c*, the contents of the SCL consist of a mixture of fibrous tissue, fibrocartilage, and necrotic bone and can be filled with synovial-like fluid.

positioned in lateral or dorsal recumbency on the surgery table. The surgical site is prepared for aseptic surgery and draped. Surgical débridement is performed using either an intra-articular approach[5] by arthroscopy or a transcortical approach.[33-35]

Approach

ARTHROSCOPIC APPROACH

Early studies used an arthrotomy approach for curettage with excellent results[36] (42 of 51 horses, 82% success rate), but this high rate of success could not be repeated. Despite this, with the rise in popularity of arthroscopy, arthrotomy approaches to joints with their accompanying high morbidity rates, have been abandoned. The arthroscope is introduced into the affected joint using routine technique. The lesion is identified either through the presence of a canal when the SCL has a communication into the joint or by a slight indentation or a "Mercedes star" irregularity. With the help of a hypodermic needle, the location for the instrument portal is identified. A No. 11 scalpel is used to make a small incision through the skin and into the joint along the needle. A blunt obturator or right angled probe is used to identify the cyst either by advancing it through the small articular cartilage opening or by pushing it through the soft, slightly movable cartilage into the cyst.

TRANSOSSEOUS APPROACH

Some SCLs are not accessible through an articular approach and have to be débrided transosseously. Careful preplanning of the surgery is necessary. The skin incision over the selected location is advanced down to the bone. Under fluoroscopic control, a 2.5-mm pilot hole is drilled through the bone into the cyst. If no fluoroscope is available, frequent radiographic images using multiple projections are needed to verify the correct drill direction. If the SCL communicates with the joint, distending it with saline solution may allow backflow through the drill hole when the drill penetrates the SCL. Once placement of the drill tip into the SCL has been verified, the drill hole is enlarged with a 5.5-mm drill bit so that it will accept a curet.

Surgical Curettage and Débridement

Surgical débridement of the lesion with a thorough curettage of the cystic tissue and lining has been considered the treatment of choice for several years because of early reports of postoperative soundness of up to 74%.[1,4]

Once the cyst has been identified, a rongeur can be used to remove the articular cartilage overlying the SCL. A motorized cartilage resector also may be used to remove the cartilage, but it is often cumbersome and time-consuming, because the cartilage is often too thick and pliable to be easily removed this way. Once all the cartilage not supported by underlying bone is removed, the contents of the cyst are evacuated with the help of a curet (Figure 89-7). Osteostixis of the adjacent bone is not recommended, because it can lead to expansion of the cyst.[1,37]

While the contents of the cyst are evacuated, care is taken not to traumatize the articular cartilage adjacent to the rim of the cyst. Although this is easy to accomplish when using the articular approach, it must be verified using fluoroscopy or radiography during a transosseous approach.

Varied success rates for return to previous function after surgical débridement of SCLs of the MFC have been reported, ranging from 56% to 74%.[1,29,38] One study reported on a series of 85 horses with SCL managed with surgical débridement under arthroscopic supervision. In that study, 64% of horses younger than 3 years returned to soundness, compared to 35% of horses older than 3 years.[38]

Following surgical débridement, 64% of Thoroughbreds raced, compared to 77% of siblings.[29] Of this population, 28% of the horses raced as 2-year-olds, 61% as 3-year-olds, and 51% as 4-year-olds. There was a difference in racing percentage based on the width of the surface defect: 60.6% of horses with surface débridement of 15 mm or less, and 39.3% of horses with surface débridement of at least 15 mm started a race. The authors concluded that the amount of cartilage surface involved seemed to be a better predictor of success than depth of the lesion. Recently, a report tried to correlate the amount of débrided cartilage surface with kissing lesions on the opposing cranial meniscal horn and meniscal ligamentous injury.[39] No direct conclusions could be drawn, but these observations led to a change in technique, whereby more emphasis is being placed on sparing the cartilage covering the SCL.

Because direct supervision of curettage of cystic lesions using a transosseous approach is impossible, the curet was always directed away from the articular cartilage, concentrating on the deeper aspects of the cyst. The cysts approached transosseously were subsequently filled with different materials (see later) with good results, although in small populations.[34,40]

Intralesional Corticosteriod Injection

The technique of injecting corticosteroids into the lining of the SCL under arthroscopic or ultrasonic guidance is based on earlier work, where inflammatory mediators were detected in the cystic contents leading to bone resorption.[7,41] The initial use of methylprednisolone has given way to triamcinolone according to one study.[41,42] Although the injection can be performed with ultrasonographic or arthroscopic guidance, with the later technique, identification and débridement of any significant cartilage lesions can be achieved. The corticosteroid is deposited throughout the cyst lining, with the most effective technique involving multiple redirections of the needle to distribute the medication. Lameness resolved in 35 of 52 (67%) horses receiving injections. A total of 73 SCLs were treated and 77% were classified as successful based on followup lameness examination. A success rate of 90% for unilateral and 67% for bilateral SCLs was reported.[42]

Grafting Procedures

CANCELLOUS BONE GRAFTS

Multiple grafting techniques have been developed and evaluated for their ability to accelerate and improve healing of the débrided SCL. Packing the lesion with autogenous grafts also has been recommended (Figure 89-8).[43,44] However, a study comparing healing of surgically created subchondral defects filled with compacted cancellous bone grafts compared to empty defects revealed no difference in the healing patterns after 6 months.[45] This type of management is no longer used.

MOSAIC ARTHROPLASTY

Mosaic arthroplasty (autologous osteochondral grafting) has been studied as a potential treatment for SCLs.[46-48] One study evaluated the bone and cartilage quality of donor sites and concluded that material properties of the grafts from the trochlear groove and axial aspect of the lateral trochlear

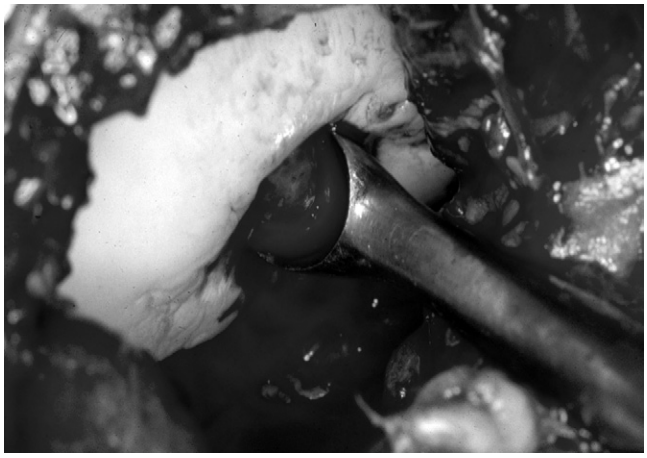

Figure 89-7. Intraoperative arthroscopic view under CO_2 insufflation of a distal femoral SCL being enucleated using a curet. The cyst has a wide cloaca into the MFC.

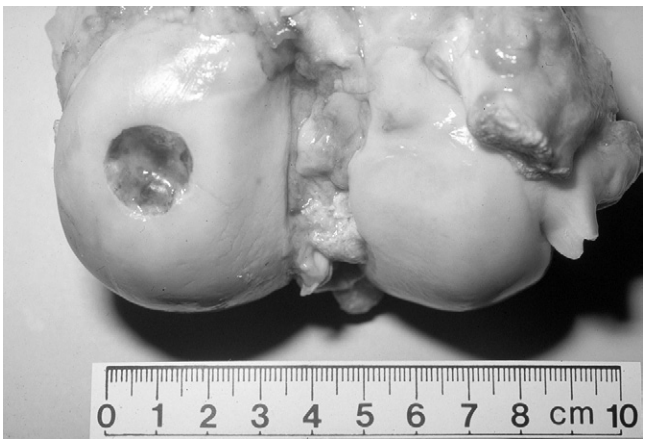

Figure 89-8. Postmortem view of a SCL of the MFC 1 month after grafting the curetted cyst with a cancellous bone graft. The graft had collapsed to some extent but had a smooth fibrous tissue cover over it. (The horse was euthanized for other reasons.)

ridge were the closest match for those found in the medial condyle, whereas properties of the lateral condyle were most similar to those found in the trochlear groove and axial aspect of the medial trochlear ridge.[47] Differences of graft performance were found between species.[49] Cartilage surface integrity was best for equine grafts, which contained the highest percentage of bone and lowest fibrous tissue, when compared to cystic lesions treated similarly in bovine, ovine, and human patients.

Osteochondral autograft transplantation (mosaic arthroplasty) was performed in a clinical case series, where grafts were harvested from the abaxial border of the medial femoral trochlea of the unaffected limb (Figure 89-9).[46] Graft implantation was achieved through a small arthrotomy or by arthroscopy depending on SBC location. All horses improved postoperatively; 10 horses had successful outcomes with radiographic evidence of successful graft incorporation, and 7 returned to a previous or higher activity level. On follow-up arthroscopy (5 horses), there was successful reconstitution of a functional gliding surface. One horse had delayed incorporation of a graft because of a technical error but became sound. One horse had recurrence after 4 years of work and soundness. One stallion

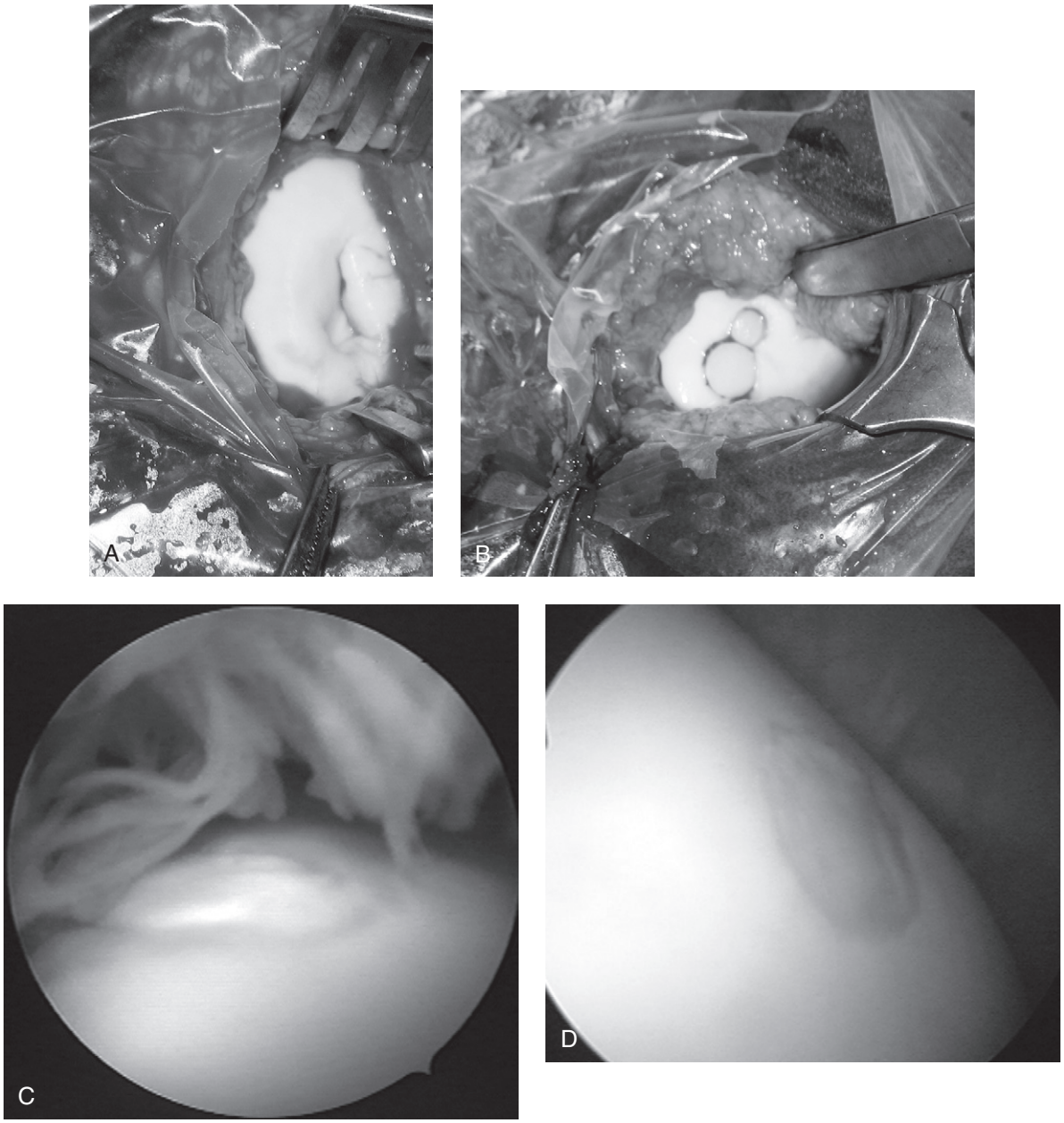

Figure 89-9. Mosaicplasty. **A,** SCL located in the MFC of a 5-year-old Hungarian half-bred mare, viewed through an arthrotomy. **B,** Postoperative view after insertion of two differently sized osteochondral grafts taken from the medial femoral trochlea of the unaffected limb of the same horse. **C,** Typical appearance of a 9-month follow-up arthroscopic view of 6.5-mm defect created on the MFC and treated with an osteochondral graft in a 2-year-old research horse. **D,** Typical appearance of an 8-month follow-up arthroscopic view of a donor site at the medial femoral trochlea. The defect is covered with uniform fibroid tissue. (Courtesy G. Bodo, Budapest, Hungary.)

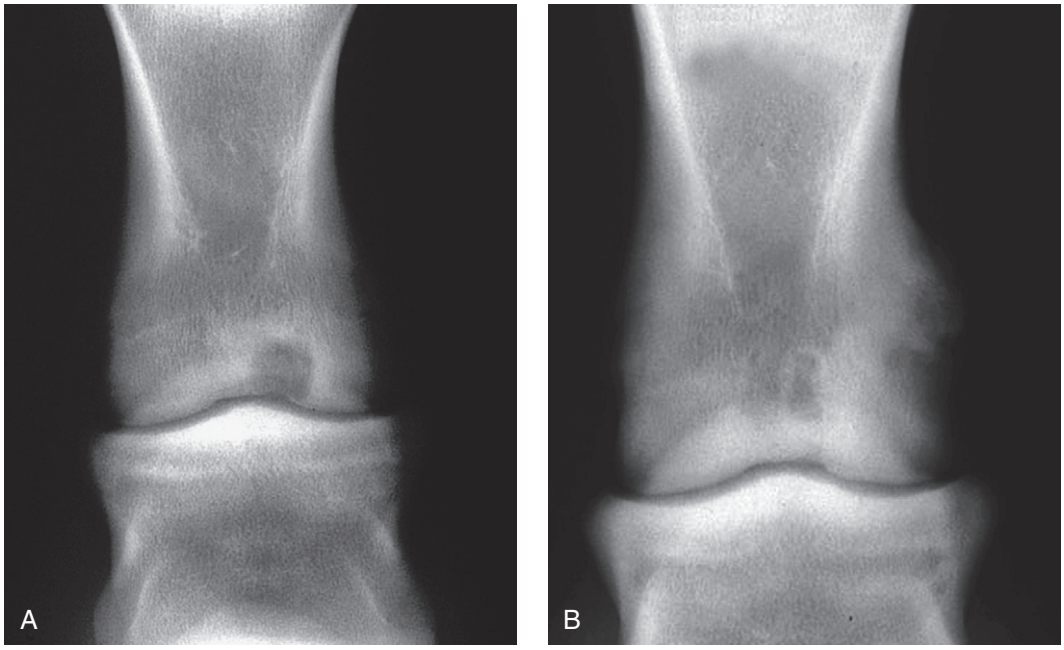

Figure 89-10. A, Preoperative dorsopalmar radiographic view of an SCL in the distal articular aspect of the proximal phalanx. The cyst was curetted and filled with tricalcium phosphate granules through a transosseous approach. **B,** Dorsopalmar radiographic view 2 years later. Complete healing has occurred.

was used for breeding and light riding because of medial meniscal injuries on the same limb.[46]

TRICALCIUM PHOSPHATE GRANULES

Tricalcium phosphate (TCP) granules can be implanted in SCLs following transosseous curettage.[41,42] Before implantation, granules are placed in a syringe, autologous whole blood is added, and a vacuum is applied for several minutes. This procedure evacuates air from the granules, and the blood fills the spaces between granules. Both the drill hole into the cyst and the cyst cavity are filled with this mixture (Figure 89-10). Good results were reported with this technique; however, the population was small.[41,42] TCP also was used as the base for other techniques (see later).

HYDROGELS AND PARATHORMONE

In a clinical case series of five horses, parathyroid hormone peptide (PTH_{1-34})-enriched fibrin hydrogel was used in transosseously curetted SCLs. All horses responded with osseous healing of cysts and return to soundness.[34] A prospective study is currently being conducted on the application the same product. Data are available in the first 15 cases, involving 16 joints, after a 1-year follow-up (Figure 89-11).[50] The cysts were curetted under arthroscopic supervision or through a transosseous approach followed by injection of the activated hydrogel. In cases where *arthroscopic supervision* was used, the fluid irrigation was stopped and joint expansion was maintained by switching to gas (CO_2). The cystic cavity was dried, and blood and fluid were removed. Finally, PTH_{1-34} in fibrin was injected through a needle into the surgically prepared cystic cavity. Gelation of the hydrogel took place within 1 to 3 minutes. When the transosseous approach was used, after curettage of the cysts, the lesions were flushed and as much liquid as possible was removed from the cyst through suction, followed by injection of the PTH_{1-34} in fibrin through the drill hole into the cystic cavity, using a blunt arthroscopic probe placed through the most proximal part of the drill hole. Of the 15 horses in the recent study, 11 (73%) were sound at 2 to 6 months postoperatively; at 12 months, soundness reached 91% to 100% in 5 cases, 61% to 90% in 5 cases, and 31% to 60% in 1 case. With these encouraging results, it is hoped that this currently experimental composite will soon be available to equine surgeons across the world.

MISCELLANEOUS TECHNIQUES

Autogenous fibrin plugs containing allogenic chondrocytes and insulin-like growth factor-1 placed over cancellous bone grafts or TCP granules and packed into débrided cysts resulted in success rates of approximately 75%.[51,52] Autologous patient-side grafting, using bone marrow aspirate concentrate and platelet-rich plasma with TCP, showed positive results with regard to the rapidity of bone formation and thickness of the resultant subchondral plate.[53] The injection of a hydraulic biodegradable cement led to a remarkable result in restoring a severely undermined distal metacarpals condyle (Figure 89-12).[54]

Aftercare and Prognosis

Postoperative management of the treatment techniques presented is the same as for any arthroscopic surgical intervention. The healing of treated SCL normally is slow and can take several months to years if just surgical débridement is used, but using bone replacements and growth factors seems to enhance bone healing of the curetted lesion and therefore shortens healing time considerably. Younger horses have a better prognosis for complete recovery compared to older horses. This is especially true if SCLs are associated with osteoarthritis in older patients, where a cautious prognosis must be given.

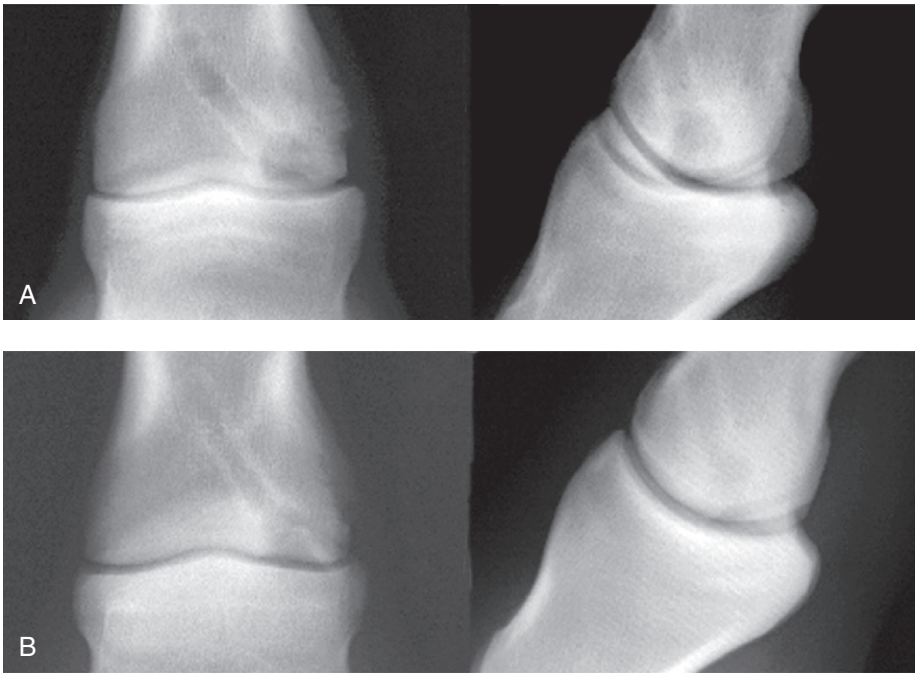

Figure 89-11. A, Immediate postoperative dorsopalmar (*left*) and lateromedial (*right*) radiographic views of a young horse with a subchondral cystic lesion (SCL) in the distal proximal phalanx. The cyst was enucleated through a transcortical approach and was treated with fibrin-based hydrogel and $PTH_{1\text{-}34}$. **B,** Four-month follow-up radiographs of the horse seen in **A.** Note that bone density has increased considerably, and the SCL is hardly visible on radiographs.

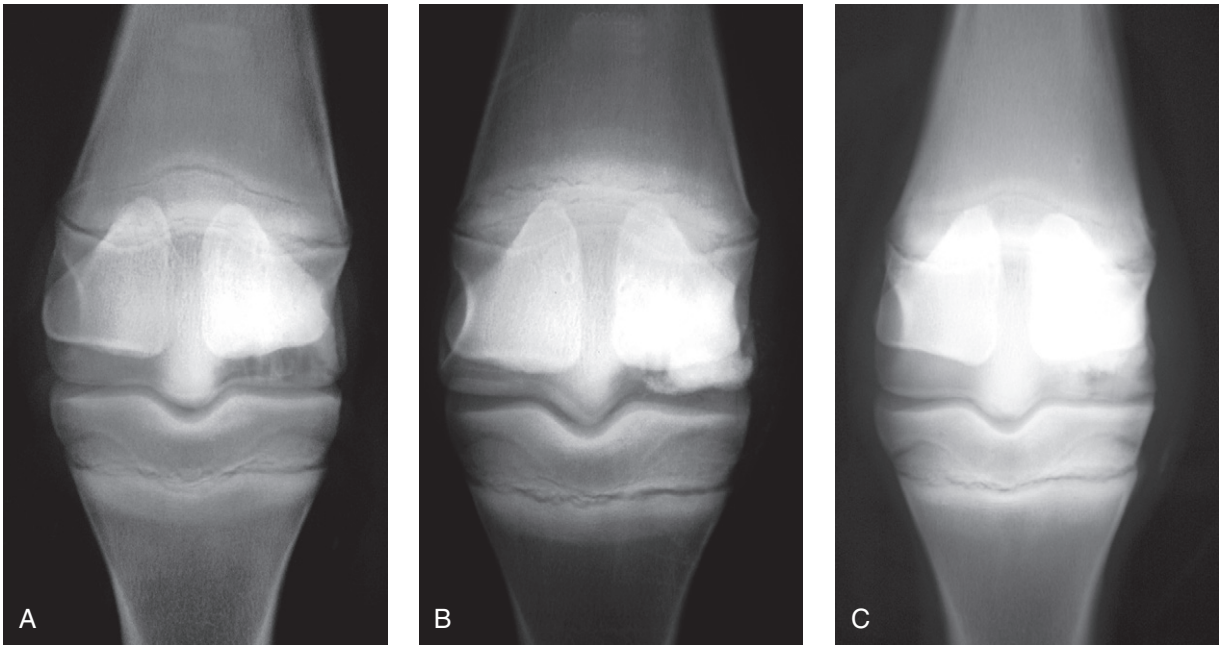

Figure 89-12. A, Dorsopalmar radiographic view of the distal MCIII depicting a cystic lesion that involved the entire medial condyle. **B,** The defect was approached using an 8-mm diameter drill bit and filled with injectable biodegradable bone cement (chronOS Inject). **C,** The 12-month follow-up radiograph shows satisfactory healing of the defect. The animal was pain free at a walk and trot, despite the loss of medial joint space.

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CHAPTER
90 Foot

Anton E. Fürst and Christoph J. Lischer

A multitude of disorders affect the equine foot. When the problem is diagnosed, a decision has to be made whether to treat it conservatively or surgically. In some conditions, surgery becomes necessary after conservative management has failed to bring about improvement. For other problems, surgical intervention is the best or even the only treatment option. In this chapter, frequently encountered disorders of the foot managed by surgery are discussed.

DISORDERS OF THE HOOF CAPSULE

Often, changes in the hoof horn quality are noticed that jeopardize the use of the horse for competition or pleasure riding.[1,2] The old saying "no hoof, no horse" holds true even today with all the sophisticated treatment possibilities available in veterinary medicine, which clearly affirms the importance healthy hooves have for the everyday use of horses. Therefore, special attention should be given to the feet not only during lameness evaluations but also during prepurchase examinations, where the clinician is asked to provide the potential new owner of the horse with a statement regarding the future usefulness of the horse for its intended purpose.

Many hoof problems have a number of predisposing causes, such as faulty feeding regimens, including vitamin and mineral deficiencies, as well as excessive and uncontrolled work load, especially on hard and dry ground. Poor hygiene and inadequate hoof care propagate hoof problems because urine and manure weaken or in some cases destroy horn integrity. Trimming and shoeing practices can exacerbate problems with hooves that initially had minimal changes in horn quality. The hoof capsule can be damaged by different external insults. Last but not least, some horses are born with hooves of inferior quality, possibly triggered by genetic factors. These animals may be predisposed to hoof problems despite good stall hygiene, excellent feeding practices, and adequate exercise. Supplementation with biotin improves horn quality over time in some horses.[1,2]

Thrush

Etiology and Diagnosis

Thrush refers to infection that leads to necrotic processes in the frog area, especially in the central and lateral sulci.[3] The sulci become soft and slimy and emit a characteristic foul-smelling odor commonly recognized with this disorder. Horn damage can progress to involve the sensitive lamina; then it is referred to as *pododermatitis*. As a result of the soft nature of the altered horn, foreign body penetration is more likely.

Manure and urine accumulation in the foot dissolve horn in the frog area in association with bacteria and fungi. Therefore, this condition is seen in conjunction with poor stall hygiene and neglected hoof care. However, a contracted hoof and lack of exercise can also predispose a hoof to thrush. Different bacteria and fungi have been shown to colonize the horn of the frog and eventually destroy it.[4-6]

Treatment and Aftercare

Meticulous hoof care and good stall hygiene are important considerations in the management of this condition, along with local treatment of the altered horn in the frog area. Box stalls should be thoroughly cleaned at regular intervals, and all urine-soaked bedding material should be removed. Intially, the sulci of the frog need to be cleaned twice daily to remove any manure packed into the hoof sole.

All damaged horn is removed, which effectively reduces bacteria and fungi colonization and provides better contact of locally applied medications to the affected tissues (Figure 90-1). After removing the diseased horn, the sulci are cleaned with a disinfecting solution.[7] In cases where the sensitive lamina is involved, a protective bandage containing sponges soaked in povidone-iodine (Betadine) should be applied for a few days. Subsequently, the sulci are treated with solutions possessing disinfectant, drying, and hardening properties.[7] In most cases, solutions containing formalin in one form or another are used. These solutions are applied to gauze sponges or small cotton balls and pushed into the affected sulci.

The majority of thrush medication is toxic to epithelium and therefore must be applied with utmost care, especially in the presence of severe horn damage and sensitive lamina. It is better to initiate treatment with povidone-iodine–soaked sponges and then proceed to stronger medications when a thin horn layer has grown over the defect.

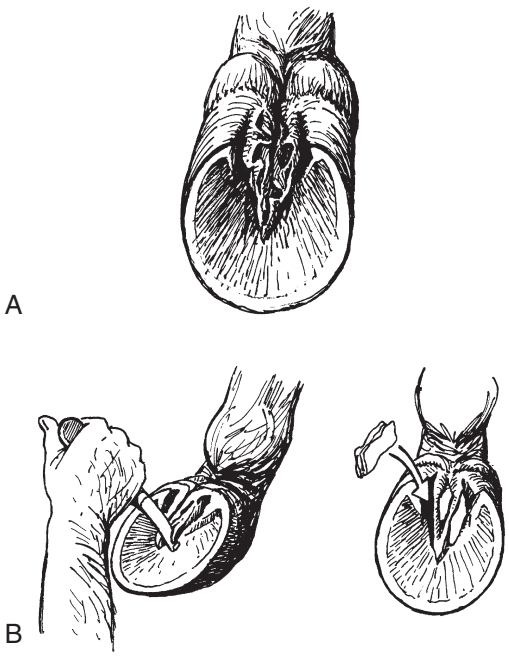

Figure 90-1. A, Graphic illustration of a hoof affected with thrush. **B,** Initially the lateral and middle sulci of the frog are thoroughly cleaned and subsequently medicated.

Figure 90-2. The sole of a hoof of a mule with severe white line disease. Necrotic and dessicated horn has been removed from the white line area.

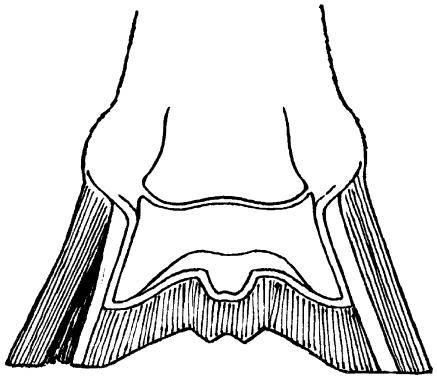

Figure 90-3. Graphic illustration of a hoof with a hollow wall shown on the left *(dark area).*

Prognosis

Horses suffering from thrush are predisposed to acquiring the problem again, mainly when the inciting causes of inadequate stall hygiene and neglect in hoof care and exercise have not been resolved. Therefore, complete resolution of the problem is possible only if good hygiene is maintained for the rest of the horse's life.

White Line Disease

Etiology and Diagnosis

White line disease refers to deterioration of the white line of the hoof capsule, resulting in the loss of the bond between the hoof wall and the sole (Figure 90-2). White line disease is incited through the presence of poor-quality horn, which allows the colonization of different bacteria and fungi.[8] These microorganisms are not able to grow in normal horn of the white line. They are more commonly encountered in geographic regions with a warm and humid climate.

When the hoof wall begins to separate from the sole, the hoof wall is exposed to increased tensional forces, which leads to the development of inflammatory processes in the sensitive lamina and results in lameness. Additionally, the selective placement of hoof nails into the white line is jeopardized, preventing solid attachment of horseshoes to the foot. If the necrotic processes extend farther proximad, entire parts of the hoof wall may become undermined and eventually form a hollow wall.

Treatment and Aftercare

Treatment consists of meticulous stall hygiene and local management of the affected hooves. All the altered and necrotic horn must be removed and the defects treated with solutions to destroy the remaining bacteria and fungi. The solutions used are the same ones used to manage thrush (see earlier). The débridement might need to include hoof wall resection (see later). The horseshoe nails should be placed higher than normal to prolong the duration the shoe is firmly attached to the sole. Additionally, the nail holes and the underside of the cleats should be sealed with bactericidal and fungicidal substances.

Hollow or Loose Hoof Wall

Etiology and Diagnosis

A hollow or loose hoof wall develops when the the most axial layers of the hoof wall, near the sensitive lamina, become separated (Figure 90-3).[9] A loose, hollow wall can develop from an extension of white line disease. In other cases, bleeding into the hoof wall seems to be the inciting cause. Chronic tearing as a result of continuous excessive use can lead to a separation within the hoof wall. As an example, many years ago, hollow walls were commonly diagnosed in pulling horses. Soil, bacteria, and fungi can get access to the dead space between the hoof wall layers and induce a local infection.

As long as no infection is present and the sensitive lamina is not involved, it is possible to apply horseshoes to the foot. However, there is always a risk that the hollow wall will continue to extend proximad, which makes treatment more difficult. Therefore, effective treatment of a hollow wall should be initiated early in the course of events, especially in the presence of an infection.

Treatment and Aftercare

Successful therapy can be achieved only if all diseased, necrotic horn is removed. The remaining hoof defect must be treated with solutions effective against bacteria and fungi, using moist hoof bandages. Depending on the extent of the hollow wall, a considerable amount of horn may have to be removed (Figure 90-4, *A*), which can result in a prolonged convalescent period. Treatment can last several months and the problem must be completely resolved before the horse can be ridden again.

Several artificial hoof products are on the market that allow the lost hoof wall to be effectively replaced. Such products as Keralit, Sigafoos, or Vettec have properties similar to normal hoof wall in respect to stability and flexibility, and they are

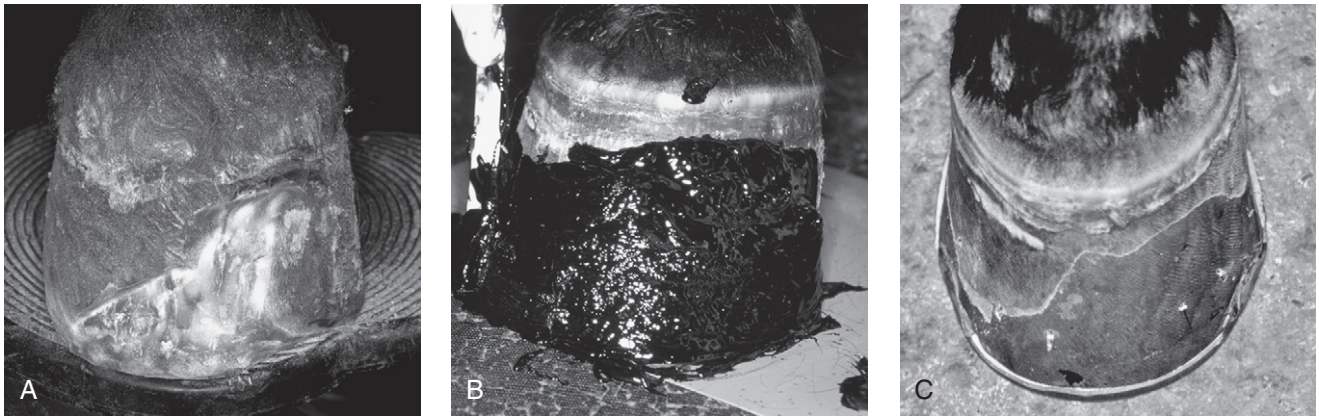

Figure 90-4. A, A hollow hoof wall is removed; **B,** The defect is filled with artificial horn; **C,** A shoe is nailed to the foot, with nails placed into the artificial horn.

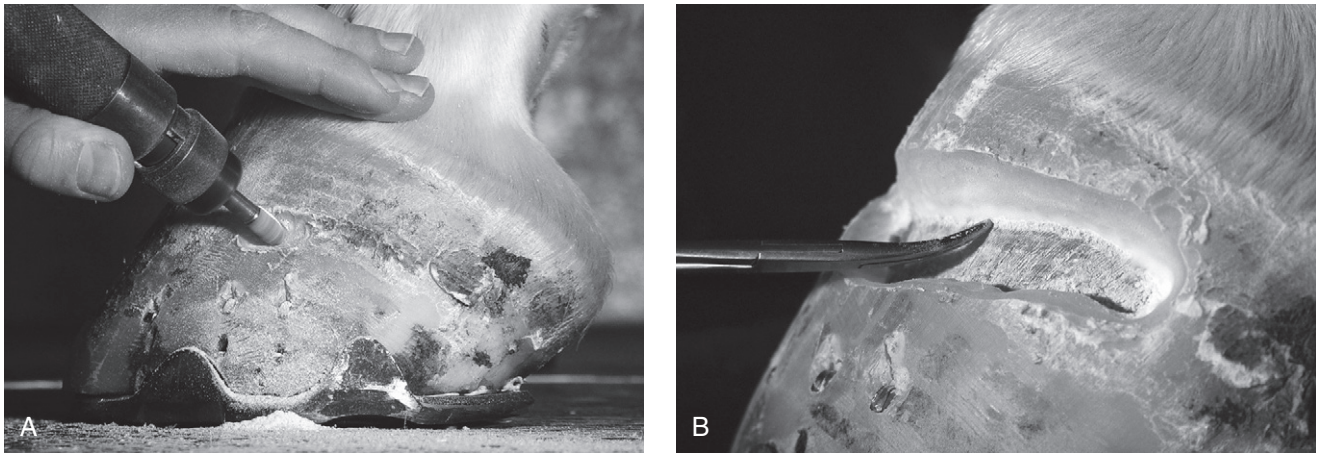

Figure 90-5. A, A loose portion localized only in the proximal part of the hoof wall is removed with a Dremel tool; **B,** The exposed dried laminae are shown.

ideally suited to replace horn defects.[10,11] The artificial horn products contain two components that are mixed together and, when polymerized, develop into a sticky, easily moldable mass. After it is molded into the clean and dry hoof wall defect, artificial horn is shaped to the contours of the hoof wall (see Figure 90-4, *B*). Within a few minutes, the material hardens into horn-like material. The hoof replacement material can be reinforced with fiberglass webbing for additional strength and resistance to wear.

The advantages of these products are that the susceptible laminae are covered with a durable protective layer, which increases the stability of the entire hoof wall. The artificial hoof wall also allows the placement of horseshoe nails (see Figure 90-4, *C*).

There is a potential danger that an infection can develop under the artificial horn, necessitating its immediate removal. Therefore, careful monitoring of the healing process is required until the defect is completely replaced by new hoof wall, which will grow down from the coronary band. In less severely involved hooves, instead of removing the entire wall, just the affected portion of the wall should be eliminated (Figure 90-5).

Hoof Wall Cracks

Etiology

A hoof wall crack occurs as a longitudinal disruption of the hoof wall parallel to the horn tubules and lamellae. It can involve the entire length of the hoof wall, only the proximal hoof wall near the coronary band, or only the distal hoof wall (Figure 90-6). The crack can penetrate the superficial layers only or can extend into the sensitive lamina. The latter cracks are called deep or perforating hoof cracks, which result in inflammation and lameness (Figure 90-7). Horizontally oriented hoof defects parallel to the coronary band are called hoof crevices, but they have the same etiology and treatment as hoof cracks.

The causes of the hoof cracks are diverse. Poor horn quality or a horn wall that is too thin are predisposing factors. Abnormal hoof angles can produce significant tension gradients within the hoof wall, which develop into cracks.[12]

DISTAL HOOF WALL CRACKS

Poor horn quality associated with excessive workload and poor hoof hygiene can lead to development of hoof cracks at the distal hoof wall (see Figure 90-6). Usually, these cracks are

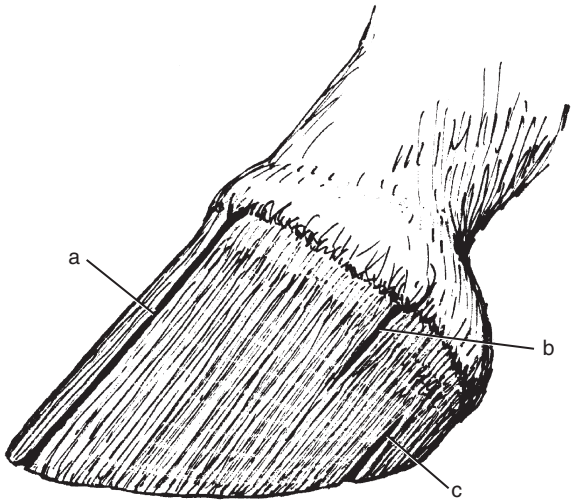

Figure 90-6. Different locations of hoof wall cracks. *a,* Dorsal crack extending over the entire length of the dorsal hoof wall; *b,* proximal lateral hoof wall crack; *c,* distal lateral hoof wall crack.

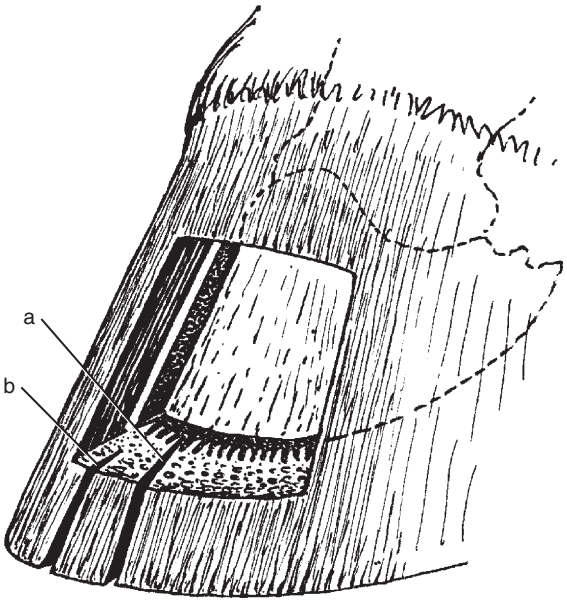

Figure 90-7. Hoof wall with a window, showing cracks penetrating to different depths. *a,* A deep or perforating hoof wall crack involving the sensitive lamina; *b,* superficial hoof wall crack.

superficial initially and do not cause lameness unless they extend and develop into a perforating crack. A horizontal groove may be cut at the most proximal aspect of the crack, and the foot should be trimmed very short to prevent proximal and deep extension of the crack. If these management practices do not stop the progression of the crack, more aggressive surgical techniques will be required (see later).

PROXIMAL HOOF WALL CRACKS

Proximal hoof wall cracks develop as a result of local trauma (Figure 90-8), inflammation, or scar tissue formation near the coronary band. Because of such damage, horn of poor quality is formed, promoting the development of a hoof wall crack.

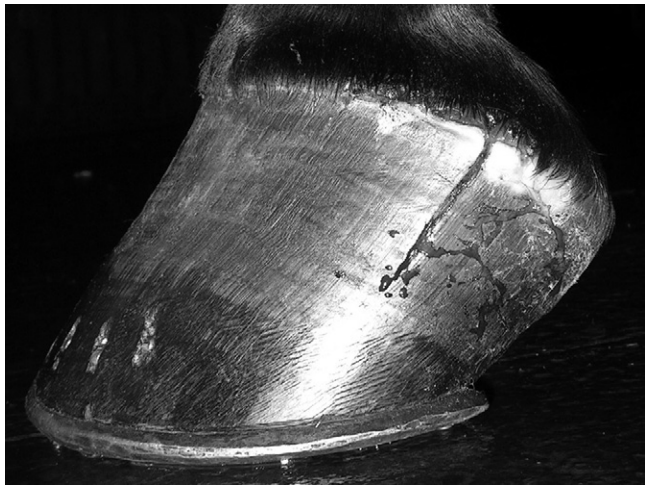

Figure 90-8. Lateral view of a proximal hoof wall crack after self injury by the contralateral horseshoe.

These cracks slowly extend distad with concomitant horn growth.

HOOF CRACKS INVOLVING THE ENTIRE LENGTH OF THE HOOF WALL

There are many etiologies of hoof cracks along the entire hoof wall, aside from local trauma to the coronary band. Most likely, tension gradients inside the hoof wall combined with extensive use and poor horn quality are major predisposing factors in their development. Often such hoof cracks are found in hooves with excessively long sidewalls (Figure 90-9). Hoof cracks are more commonly seen on the medial hoof wall than on the lateral wall.

Uneven heels and displaced bulbs of the heel are predisposing factors. Hoof wall cracks can also be caused by faulty shoeing practices, such as shoes that are too short, branches that are too narrow or side clips applied too far back, uneven hoof soles, and hoof nails inserted too far caudally. When unphysiologic conditions such as these are present for several months, poor-quality horn can grow, predisposing the hoof wall to form cracks. The combination of long shoeing intervals and excessive work foster hoof crack development.[13]

Diagnosis

Most of the time the diagnosis is made on close visual inspection of the hoof. The deeper layers of the crack often are impacted with dirt and manure. Lameness is a prominent feature of deep or perforating hoof wall cracks. Conversely, superficial horn cracks are not associated with lameness and many do not require treatment. Nevertheless, there is always a risk that a superficial crack will extend into one that eventually initiates an inflammatory process.

Treatment and Aftercare

Because hoof cracks are encountered often and have been a common problem for hundreds of years, many treatment methods are described. However, although many methods and techniques exist, successful management of hoof cracks

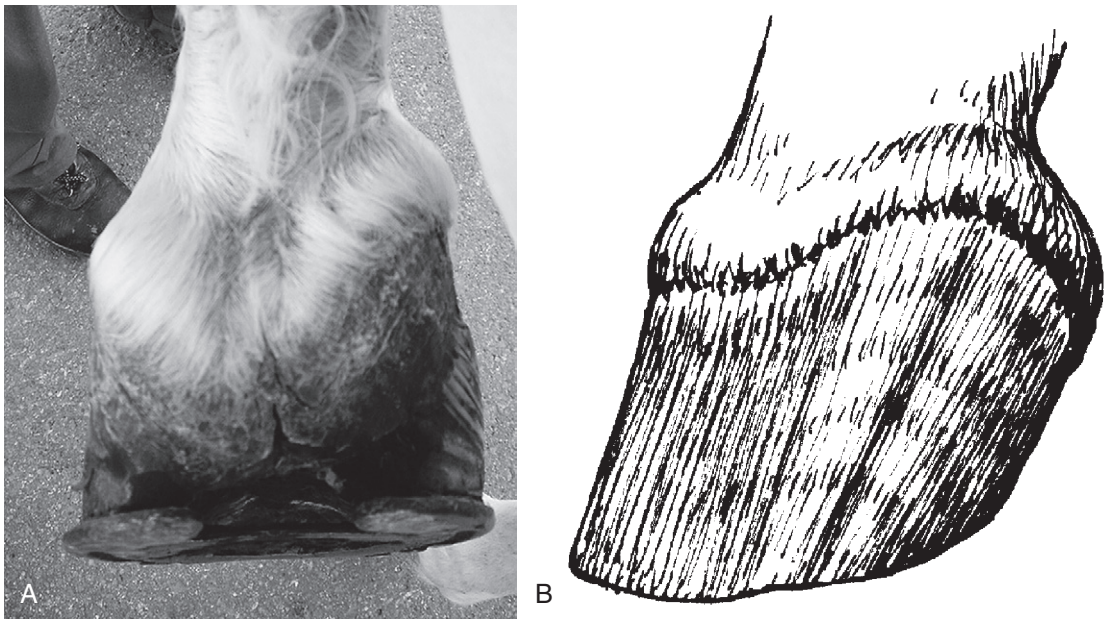

Figure 90-9. A, A hoof with a long and steep inside hoof wall and uneven heels that promoted a hoof crack. **B,** Graphic illustration of a contracted hoof with a distorted coronary band and a hoof crack.

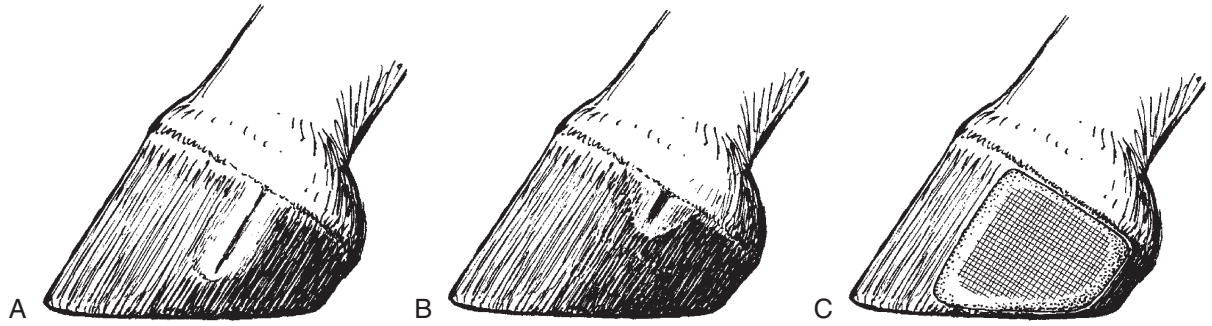

Figure 90-10. A and **B,** The hoof surrounding the two different-sized cracks is carefully removed, taking care to establish smooth transitions before each crack is covered with artificial horn **(C).**

demands close cooperation among the clinician, the farrier, and the owner.

The type of treatment selected depends on the location and depth of the crack, and the length of treatment can be from a few days to several weeks or even months. The first line of attack involves improvement of hoof care and shoeing.[14] The repair of the hoof crack without initial débridement can reduce the movement of the two sides relative to each other, effectively protecting the underlying sensitive laminae. In these instances, lameness will be attenuated. In rare cases, the crack can heal completely, allowing regrowth of normal horn from the coronary band distad.[15] In most cases, however, more-involved techniques are necessary to bring about healing of the defect, especially if the crack is accompanied by infection and lameness.

First, the condition of the shoes has to be assessed, correcting present abnormalities, such as uneven heels and abnormal hoof angles. The old shoe is removed and the hoof is properly trimmed. By cutting the heels on the affected side short, local pressure in the hoof wall can be partially reduced.

Subsequently, the horn adjacent to the crack is carefully removed with a Dremel tool (Figure 90-10).[16] All altered horn down to the sensitive lamina is removed, taking special care to establish smooth transitions toward the sides. When completely cleaned, the defect is covered with gauze sponges, and a hoof bandage is applied.

The foot is shod with a bar shoe, and the sole is filled with silicone and covered with a metal plate. This reduces pressure in the heel region, which is especially beneficial if the crack is located in this area. In some cases, the coronary band immediately proximal to the crack is displaced dorsad. Trimming the hoof wall short just below this crack reduces the continuous movement at the coronary band and leads to the production of better-quality horn.

Last, a fixation device should be applied over the crack. If the crack was débrided down to the sensitive lamina, fixation has to be delayed until a subtle layer of horn covers the defect.

An effective repair unites the two separated hoof parts and reduces individual movements at the transition to the normal horn proximally at the level of the coronary band. There are a

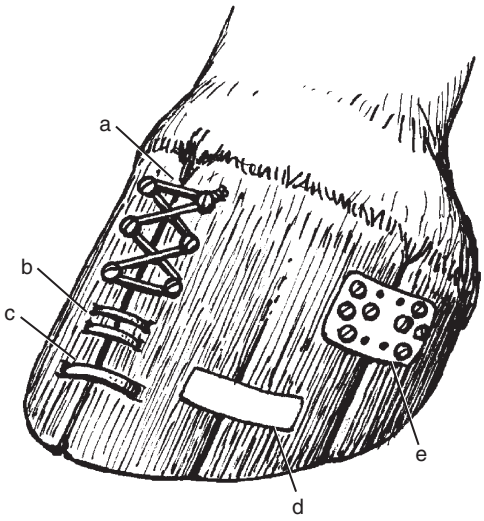

Figure 90-11. Various devices applied to repair hoof cracks. *a,* Umbilical tape laced around small protruding screws in the hoof wall; *b* and *c,* different clamps whose sharp points were tapped into the hoof wall; *d,* a metal plate glued over the crack; *e,* a metal plate spanning the crack fixed to the hoof wall with small screws.

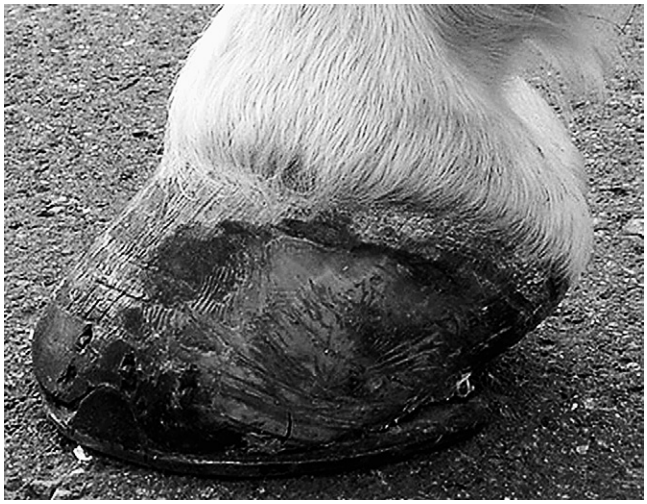

Figure 90-12. Lateral view of a hoof crack covered with artificial horn.

variety of methods that can be used to repair a hoof crack (Figure 90-11). The introduction of the two-component hoof replacement materials mentioned earlier have significantly improved the effectiveness of hoof crack repair.[17] These materials provide a natural covering of the crack with a material having properties similar to the normal hoof wall. These materials cure with an exothermic reaction. Therefore, it is important that at least a thin layer of horn be present to protect the sensitive lamina. It is even possible to install a drainage system underneath the artificial horn. Even so, hoof abscesses can develop after application of the two-component hoof replacement materials.

The hoof wall must be covered with artificial horn material over almost its entire length to prevent a recurrence of a crack (Figure 90-12). Poor horn quality might dictate that a covering

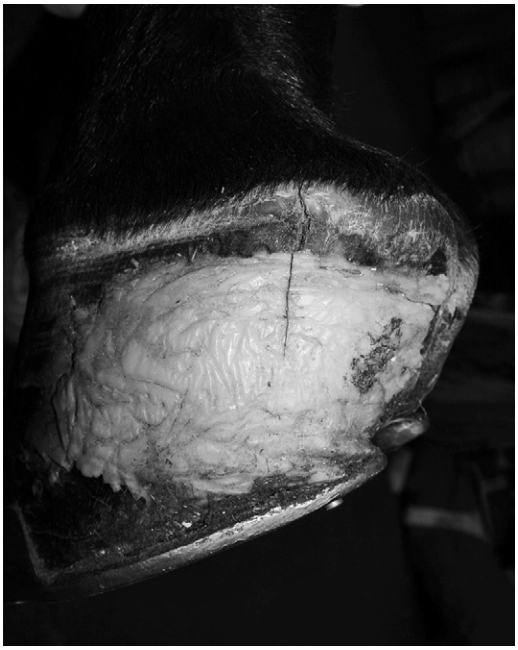

Figure 90-13. An inadequate repair with artificial horn led to recurrence, with the crack extending partially across the artificial horn.

of artificial horn be maintained for several months. New horn growing from the coronary band eventually completely replaces the repaired hoof wall.

Immediately after the repair, the patient should be kept in a stall for a few days before gradually being returned to exercise. Depending on the quantity and quality of new horn production, the patient may go back to light exercise after 4 to 8 weeks.

Prognosis

The prognosis for a successful treatment of a hoof crack is guarded to good. Often, the crack recurs, especially if the predisposing factors are not abolished (Figure 90-13). Because the properties of the horn vary with the location on the hoof, the prognosis varies as well.[18-20]

Keratoma

Etiology

A keratoma represents a columnar thickening of the hoof horn that extends toward the inside of the hoof (Figure 90-14). Because it is rare, the disorder is often overlooked. In addition to the classic keratoma, with its cylindrical appearance and protrusion parallel to the horn tubules, a spherical form of keratoma has been described that can develop anywhere in the hoof capsule.[21]

Most keratomas are found in the dorsal or dorsolateral aspect of the wall. A local inflammation reaction or trauma at the transition of the new horn produced at the coronary band and hoof wall is the most likely inciting cause. This results in the formation of scar tissue in that location, which gradually grows distad as new horn is produced.

A hoof abscess also can lead to the development of a keratoma. Because of the expansiveness of the keratoma,

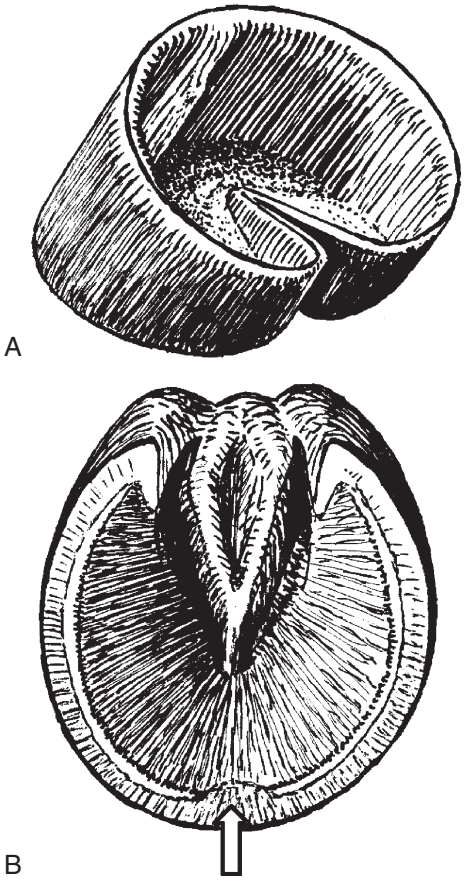

Figure 90-14. Graphic illustration of a keratoma in the hoof shell. **A,** Representation from the inside. Note the horn tissue that has formed at the toe of the hoof. **B,** First manifestation at the median of the white line *(arrow).*

unphysiologic pressures are exerted on the sensitive laminae and the distal phalanx, leading to inflammation and lysis of the underlying distal phalanx. The keratoma consists of poor quality horn (Figure 90-15), which decays early, allowing bacteria and fungi access to the inside of the hoof wall. Therefore, keratomas are often associated with local hoof wall infections and are the reason horses are presented to veterinarians. If a patient is presented with a chronic recurrent hoof abscess, it is prudent to assume a keratoma is the actual cause of the problem.

Diagnosis

Careful inspection of the sole is important for the diagnosis. Trimming and cleaning of the sole surface is necessary to recognize the abnormal configuration of the white line. Typically, the lamellar horn of the white line is replaced by tubular horn and scar tissue (Figure 90-16, *A*). The pathologic tissue displaces the white line toward the sole. In advanced cases, a circular lytic area of the distal phalanx can be recognized radiographically (see Figure 90-16, *B*). This area should not be mistaken for the naturally occurring crena at the dorsal aspect of the distal margin of the distal phalanx. Often, a sclerotic border delineates the lytic area (see Figure 90-16, *B*). If the diagnosis is unclear, a computed tomographic (CT) examination will show the defect in the distal phalanx very clearly (Figure 90-17).

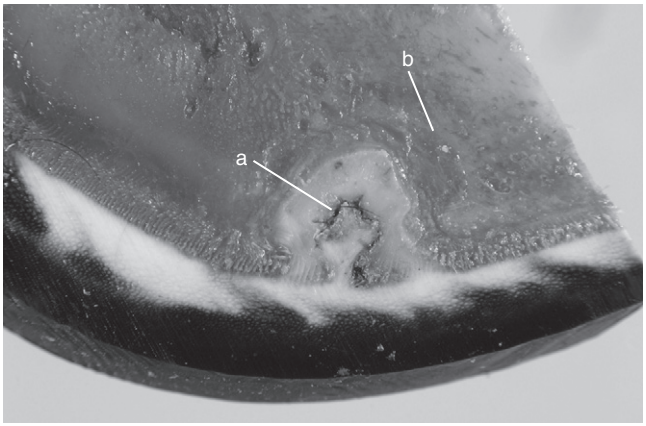

Figure 90-15. Photograph of a sagittal section of the hoof that has keratoma formation. *a,* Keratoma; *b,* the bone surrounding it.

Treatment and Aftercare

In selected cases, it is possible to temporarily abolish the signs of a keratoma with the help of conservative management. However, even with meticulous hoof care and good hygiene it is not always possible to prevent the formation of a hoof abscess. Therefore, an early surgical approach toward solving the problem is advised. For successful resolution, two main principles must be observed[21,22]: the keratoma must be completely removed up to its origin, and support has to be given to the hoof wall.

The timing of the surgery depends to a certain extent on the condition of the patient and the intentions of the owner. The main dilemma is whether to first manage the inflammatory processes medically and remove the keratoma after it has resolved, or to immediately enucleate the structure. The choice should be based on the amount of pain shown by the patient; early intervention may be indicated in very lame patients to avoid the development of laminitis in the contralateral foot. Aside from the altered horn, the altered sensitive laminae also must be removed. The hoof wall defect may be filled with artificial horn as soon as the sensitive lamina is healed. Additionally, a special shoe with large clips placed on either side of the defect should be applied to the foot.

The surgery is performed in two steps, with a tourniquet applied above the foot. First, as much horn as possible is removed with the horse standing until the Dremel tool has to be exchanged for the scalpel and curettes. The second stage of the procedure is carried out under aseptic conditions, and the altered lamina and the entire keratoma are removed *in toto* (Figure 90-18). This procedure also may be carried out on the standing horse if a ring block at the level of the metacarpophalangeal joint is used. The advantages of performing the removal under general anesthesia include the more comfortable position for the surgeon and immobility of the patient. It is of utmost importance to remove all pathologically altered tissue in addition to the altered hoof horn. The keratoma usually has a characteristically round appearance at its origin.

At the end of the procedure, an antiseptic pressure bandage is applied to the phalangeal region, and the tourniquet is removed. The bandage is changed at 3- to 4-day intervals under aseptic conditions. As soon as the bone is covered by granulation tissue, a medication plate shoe is applied. The horse remains confined to a box stall for 4 to 6 weeks, during which

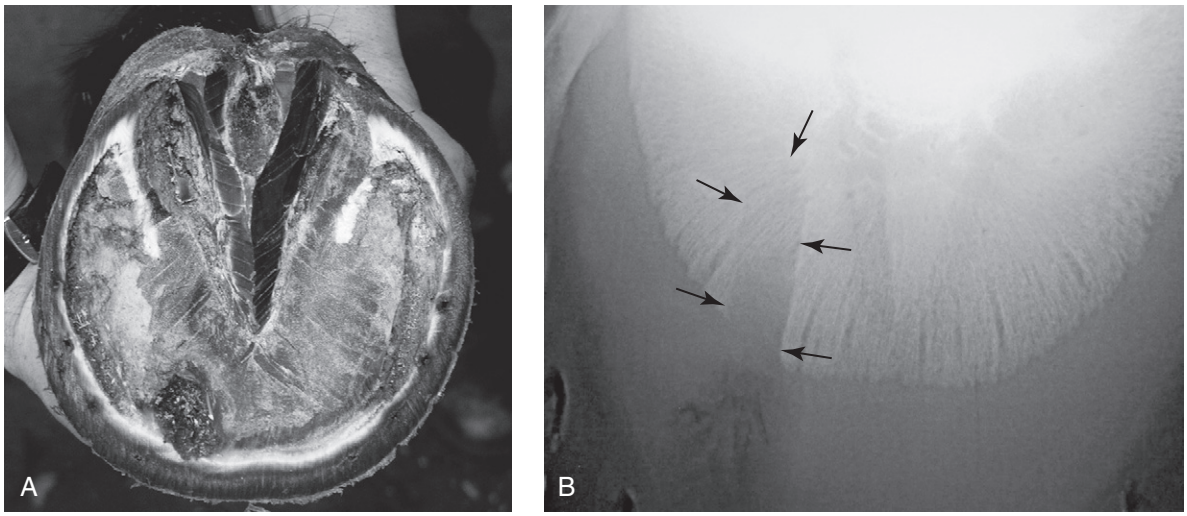

Figure 90-16. A, Solar view of a foot with a keratoma demonstrating a local chronic infection at the tip of the sole. **B,** Dorsopalmar radiographic view of the same patient showing a lytic area in the distal phalanx extending up along the hoof wall *(surrounding arrows),* which is typical of a keratoma.

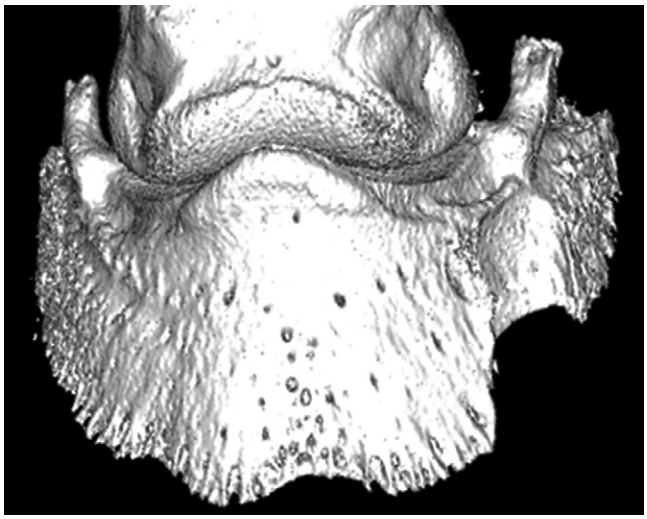

Figure 90-17. Three-dimensional reconstruction of a computed tomographic image of a third phalanx affected by a keratoma. Note the large defect in the pedal bone where the keratoma formed.

Figure 90-18. Partial hoof wall resection for keratoma removal. The portion of hoof wall left near the ground surface provides stability.

the wound is treated routinely with dressings. After this time, a new shoe is applied. Depending on the healing process, different avenues may be taken. If healing progresses normally, the defect may be filled with artificial horn and the patient subjected to light walking exercise and, after approximately 2 to 4 months, light riding work. If signs of infection recur, all affected tissues have to be removed and the process of postoperative management started again.

Prognosis

With surgical treatment, a significantly better prognosis can be given, with an 83% success rate, compared to conservative management, where an approximate success rate of 43% has been published.[23] The complication rates vary with surgical techniques. Complete hoof wall resection from the coronary band to the sole has a greater complication rate than partial wall resection directly over the mass.[24] Therefore, the hoof wall should be preserved to provide stability to the hoof capsule. Overall prognosis for return to soundness and the previous level of performance is very good for surgical treatment of keratomas.[24]

SEPTIC AND ASEPTIC DISORDERS OF THE SOFT TISSUES

Acute Hoof Abscesses

Etiology

"Hoof abscesses have 1000 faces." Hoof abscesses are among the most frequently diagnosed causes of lameness seen in equine practice. It is interesting to realize how many symptoms

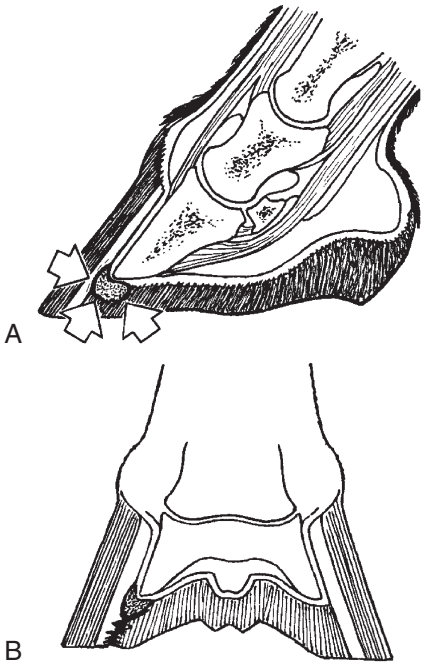

Figure 90-19. Location of a typical hoof abscess at the tip of the foot (**A,** *arrows*) and at the side **(B).**

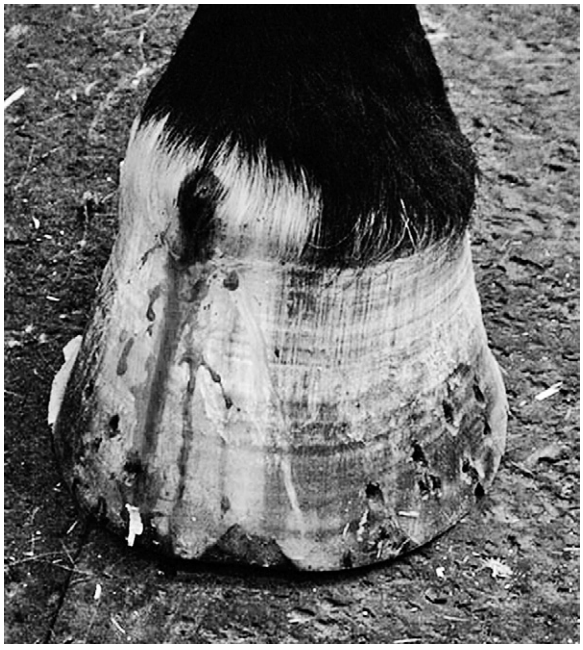

Figure 90-20. Delayed diagnosis or delayed treatment can lead to the abscess breaking out at the coronary band.

Box 90-1. Possible Etiologies for a Hoof Abscess

- Placement of hoof nail too close to the sensitive lamina, allowing bacteria along the nail to induce an infection in close proximity to the sensitive lamina
- Insertion of a hoof nail into the sensitive lamina, resulting in an infection
- Small pointed rocks penetrating the solar horn
- Any penetrating foreign object, such as a street nail[22]
- Any sole bruise, which creates a blood culture medium for the growth of bacteria that enter the tubules in the horn normally

a hoof abscess may generate. It is therefore not surprising that this problem is occasionally misdiagnosed.

Hoof abscesses develop in the unshod horse—often foals—as well as in shod horses. No breed or age predispositions are known. After penetration of bacteria across the hoof wall, infection develops in the region of the sensitive lamina, which includes the accumulation of purulent material inside the hoof capsule (Figure 90-19). Because the hoof capsule provides a solid external shield, increased pressure builds up inside, leading to severe, non–weight-bearing pain. Box 90-1 lists some possible etiologies for the development of a hoof abscess. A sole bruise is the most likely inciting factor.

Diagnosis

The cardinal symptom is a sudden severe lameness, which might be described as "fracture lameness," sometimes resulting in the horse being admitted to a clinic as a fracture patient. The horse might be febrile and have increased warmth of the foot, with strongly pulsating distal palmar and plantar arteries. A generalized swelling of the distal limb might be noticed, which can lead to the erroneous diagnosis of septic tenosynovitis or other similar conditions. Local application of pressure through the hoof tester generally elicits a marked localized positive response. Local perineural anesthetic blocks (deep palmar anesthesia, middle palmar/plantar anesthesia) aid in localizing the problem, allowing subsequent pain-free treatment. Complete blood count (CBC) and blood chemistry analyses are usually unrewarding, but in some cases radiography supports the tentative diagnosis of hoof abscess.

Treatment and Aftercare

If a hoof abscess is suspected, the horseshoe should be removed and each nail closely examined for wetness and odor. Subsequently, the exact location of the abscess is established with the help of the hoof testers. Location with the hoof tester can be difficult in the presence of thick and hard sole horn. In such a case, it might be necessary to apply a povidone-iodine or creosote bandage for overnight, which results in softening of the hoof horn, permitting localization of the abscess and subsequent drainage.

All the undermined and necrotic horn is carefully removed, piece by piece, until a smooth transition is achieved toward the surrounding normal sole. Subsequently, the infected area is cleaned with iodine solution or H_2O_2 and covered with a povidone-iodine–soaked gauze sponge and a hoof bandage. The bandage is changed after 2 to 3 days under aseptic conditions. At that time, the defect is closely examined and any additional undermined sole is carefully removed. As soon as the site is dry, the horse can be reshod. It is prudent to cover the site with a leather or synthetic pad.

If the abscess is not managed properly or in a timely fashion, it can break through at the coronary band (Figure 90-20) or extend deeper into the foot and involve the distal phalanx. In

Figure 90-21. The abscess is flushed daily from the coronary band toward the sole with diluted povidone-iodine (Betadine) solution.

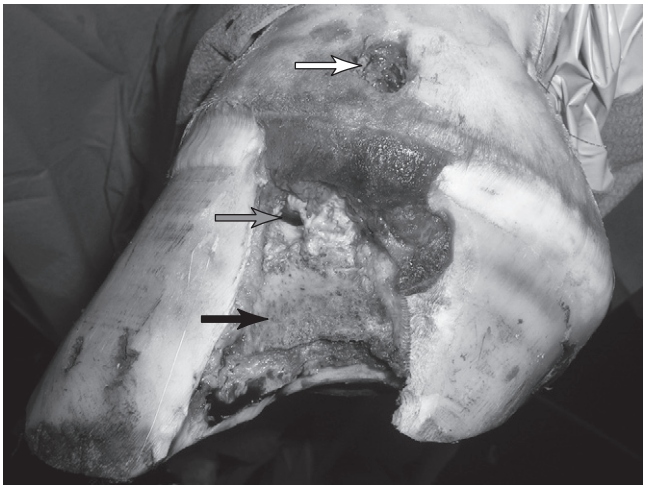

Figure 90-22. Delayed diagnosis or delayed treatment can lead to a septic arthritis of the DIP joint. The opening into the DIP joint is clearly visible *(gray arrow).* The diseased bone was curetted *(black arrow).* The abscess had to be opened proximal to the coronary band to allow effective flushing *(white arrow).*

these cases, the draining tract has to be flushed daily with diluted povidone-iodine solution from the coronary band distad (Figure 90-21). It is important that the flushing solution exits at the sole of the foot to establish ventral drainage. The foot must be kept in a bandage until drainage has stopped. Under extraordinary circumstances, extension of the abscess into the distal interphalangeal (DIP or coffin) joint can occur (Figure 90-22). The management of these cases is described later.

Administration of nonsteroidal anti-inflammatory drugs (NSAIDs; e.g., phenylbutazone) is indicated. Antimicrobial therapy is also indicated, when involvement of other deeper structures is suspected. Generally a good prognosis can be given, even though advanced abscesses can take weeks to heal completely.[25]

Chronic Hoof Abscesses and Septic Pedal Osteitis

Etiology

On rare occasions, a hoof abscess might not be diagnosed right away, especially if the horse has been treated with pain-relieving medications for a period of time. The clinical symptoms are more or less effectively masked, preventing recognition of the problem and its treatment. Also, administration of NSAIDs prevents the abscess from maturing and can permit the microorganisms to invade other tissues within the foot. The abscess can extend into the foot along three avenues: along the dorsal and lateral hoof wall in a proximal direction, eventually breaking out at the coronary band (see Figure 90-20) or invading the coffin joint (see Figure 90-22); or along the sole, potentially undermining it completely or along the distal phalanx, resulting in septic pedal osteitis.[26,27]

Diagnosis

Physical examination of the foot might reveal a draining tract. A thorough radiographic evaluation, with or without a probe positioned in the draining tract, often helps make the diagnosis, because at this point radiographic changes usually are visible on the distal phalanx. Lytic changes can develop as a result of a chronic abscess as well as after aseptic local inflammation.

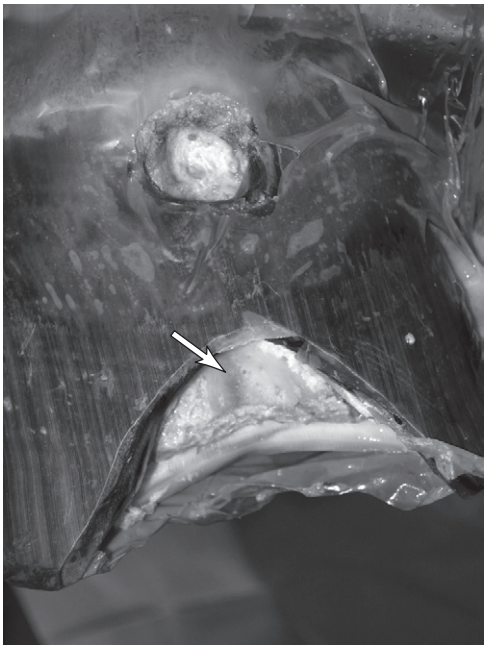

Figure 90-23. A chronic abscess had to be opened up near the coronary band and the distal phalanx had to be curetted as well *(arrow).*

Treatment and Aftercare

As described earlier, the abscess needs to be opened and all the undermined sole removed until normal horn is encountered.[26,27] If the infection spreads out along the side wall, all the undermined wall must be carefully removed, again until normal wall is encountered. If the distal phalanx is involved, all the affected bone must be curetted (Figure 90-23). The

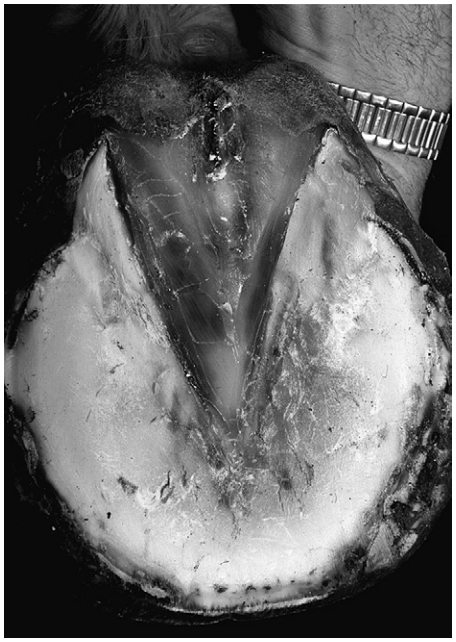

Figure 90-24. Marked reddening of the sole near the frog in a case of aseptic pododermatitis (severe bruising).

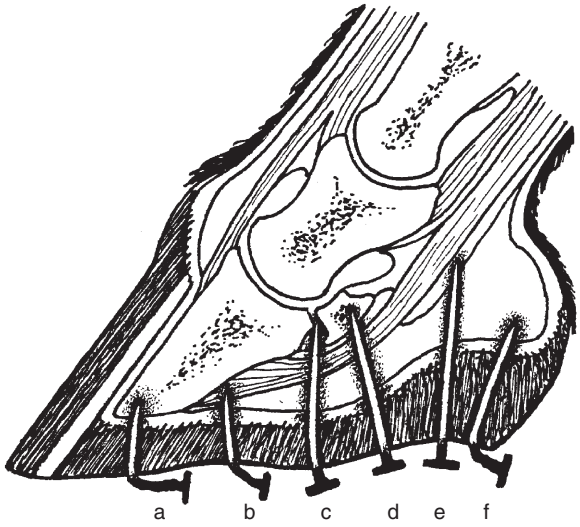

Figure 90-25. Potential puncture wound sites, resulting in the penetration of different anatomic structures. *a,* Tip of the sole, involving the distal phalanx; *b,* dorsal third of the frog, involving the insertion of the deep digital flexor tendon; *c,* middle of the frog, involving the deep digital flexor tendon, the impar ligament, and possibly the distal interphalangeal joint; *d,* palmar third of the frog, involving the deep digital flexor tendon, distal phalanx, and navicular bursa; *e,* palmar aspect of the frog, involving the hoof cushion, deep digital flexor tendon, and tendon sheath; *f,* most palmar frog, involving the hoof cushion.

treatment of a chronic abscess can take several weeks to months, especially if a substantial amount of hoof wall needs to be resected.

Aseptic Pododermatitis

Etiology

Aseptic pododermatitis refers to a severe bruising of the laminae and horn tissues of the foot and is a common cause of lameness in horses. Often this problem is not recognized immediately, and the concomitant lameness is attributed to navicular disease or osteoarthritis of the DIP joint. There are many possible causes for this problem: poor shoeing techniques, excessive use of the horse under difficult conditions, such as hard, uneven ground, and frequent kicking against the stall wall.

Diagnosis

The symptoms are typical but not very specific, leading occasionally to a misdiagnosis (Figure 90-24). The hoof is slightly warmer than normal and a positive pain reaction can be elicited with the hoof testers. Pulsation of the palmar arteries is usually increased. Palmar digital nerve anesthesia usually relieves the lameness. Radiography helps to differentiate the problem from a fracture in this region. Keratoma is part of the differential diagnosis in cases of chronic persistent pododermatitis.

Treatment and Aftercare

Management of this disorder is directed at removing the actual cause. In most cases, the shoe must be removed. Application of creosote bandages for three or more days is very effective. These bandages are followed by dry bandages for several weeks. Some patients require NSAIDs because of persistent pain. Some handwalking on soft ground is optional, and after 7 to 10 days the horse is reshod.

TRAUMATIC INJURIES

Puncture Wounds

Etiology

Nail prick refers to an injury caused by inadvertently driving a nail through the sole and sensitive laminae during shoeing. *Street nail* refers to puncture of the solar surface of the hoof by nails or other sharp objects, such as screws. A hoof is *nail bound* when a horseshoe nail is driven too deep into the horn, causing excessive pressure on the corium. All three types of injuries usually result in pain, inflammation, and infection.

Although puncture wounds of the sole can appear small, they are often deep and can have disastrous effects when such structures as the distal phalanx, distal sesamoid bone, DIP joint, navicular bursa, deep digital flexor (DDF) tendon, or tendon sheath are penetrated (Figure 90-25). The penetrating object is usually contaminated with soil, rust, or manure, which can lead to serious infection. The superficial wound in the sole usually seals quickly, leaving no area for drainage. The anaerobic environment created favors the growth of *Clostridium tetani*, the microorganism causing tetanus.

Depending on the location and depth of the puncture wound, various structures can be affected (see Figure 90-25).[28,29] Deep puncture wounds are extremely serious and difficult to treat. Affected horses are often referred to specialized clinics for surgical therapy. For these reasons, deep puncture wounds must be treated as an emergency to prevent the infection of bones, joints, and tendon sheaths.

Diagnosis

Horses usually have a moderate to severe supporting-leg lameness, often pointing the affected toe. The hoof is warmer than normal, and there is increased pulsation of the digital arteries.

The horse might have a fever. Examination with hoof testers usually elicits severe pain. In horses with severe acute lameness, the hoof must be thoroughly cleaned and examined for a foreign body or puncture wound (see Figure 90-25).

Treatment and Aftercare

The nail or foreign body should be promptly removed. However, it is imperative that the depth and direction of the tract be noted and the point of entry marked on the sole or recorded on paper, because it will rapidly become inapparent. The point of entry is cleaned, and the entire hoof is bandaged.

Based on the location, direction, and depth of the injury, the horse may be treated on site or referred to a clinic. When there is suspicion of injury to deeper structures, such as the navicular bursa, DIP joint, or the DDF tendon sheath, the horse must be referred immediately for surgical treatment. Broad-spectrum antibiotics are started, and tetanus antitoxin and toxoid are administered.

Treatment in a Clinic

A thorough clinical examination is carried out, after which local anesthesia is administered. The shoe is removed and the entire hoof is trimmed. The decision to pursue further treatment is based on the results of the clinical examination and radiography. The puncture tract is carefully cleaned and disinfected. A sterile metal probe is inserted into the puncture tract, and the hoof is radiographed in two planes (Figure 90-26, *A*). Placing a needle into the navicular bursa using aseptic technique and injecting a contrast medium can enhance the diagnosis (see Figure 90-26, *B*). Alternatively, a 20-gauge needle is placed in the DIP joint and synovial fluid is collected into an EDTA tube. Approximately 10 mL of contrast medium is injected into the joint. After a few minutes, the hoof is radiographed again to determine whether the contrast medium is exiting through the puncture tract.[28] The same procedure is repeated for the DDF tendon sheath.

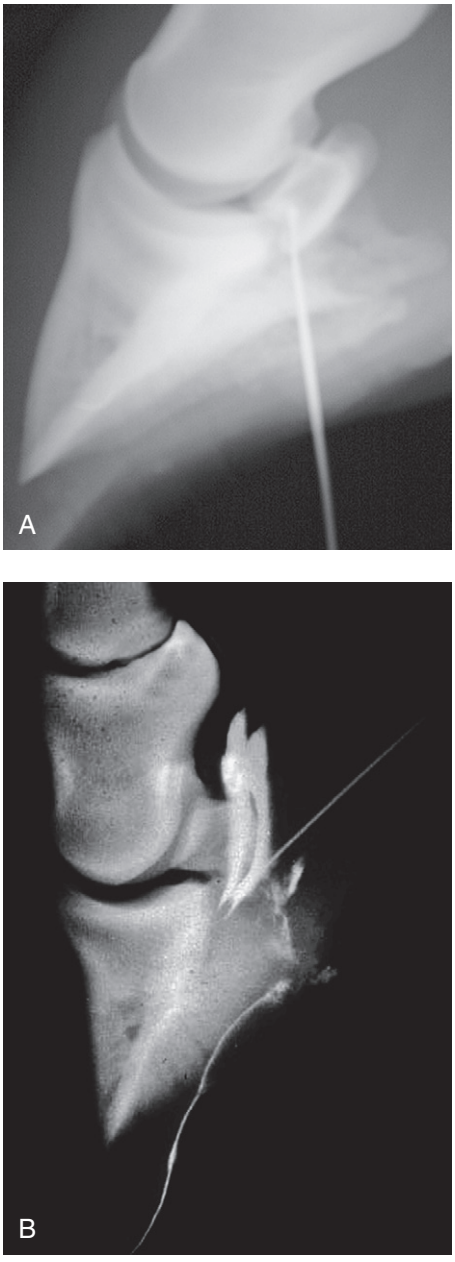

Figure 90-26. A, Lateromedial radiographic view showing a probe inserted through a puncture wound, which penetrated the navicular bursa and distal sesamoid bone. **B,** Lateromedial radiographic contrast study of the navicular bursa, showing contrast media exiting through the puncture wound.

SURGICAL DÉBRIDEMENT AND TREATMENT OF PUNCTURE WOUNDS

Surgical débridement of puncture wounds is usually performed with the horse under general anesthesia. This procedure entails two parts: initial débridement of the sole in the standing and sedated horse followed by aseptic treatment of the puncture wound and the involved deeper structures (Figure 90-27).[30] With the horse sedated and standing, the hoof is cleaned and the horn around the puncture tract is carefully removed down to a thin layer of horn that can be cut with a scalpel blade. The hair from the hoof to the fetlock joint is clipped. The prepared area is cleaned with chlorhexidine scrub (Hibiscrub) and covered with a bandage.

The horse is positioned in lateral recumbency under general anesthesia and a tourniquet is applied. Occasionally, it is necessary to remove additional horn at this time. All affected tissues around the puncture tract are excised. The horn around the tract is removed in an area measuring approximately 3 × 3 cm. The corium and subcutis are then removed and the underlying structures are exposed. If the foreign body has penetrated the DDF tendon, a 1.5 × 1.5 cm area of the tendon is resected. Curettage is necessary if the foreign body penetrates the distal phalanx or the distal sesamoid bone (Figure 90-28). With perforation of the impar ligament and penetration of the DIP joint, the ligament must be resected and the joint as well as the navicular bursa must be lavaged. Involvement of the digital tendon sheath requires thorough lavage.

The affected synovial structures are lavaged with copious amounts (several liters) of lactated Ringer's solution to which antibiotics have been added. A pressure bandage is applied, followed by a hoof bandage and a wedge under the heel. Repeated regional intravenous perfusion with an antibiotic is recommended.[31]

An arthroscopic technique has been developed to lavage and treat a deep puncture wound that penetrates the navicular bursa or DIP joint (Figure 90-29). This technique has a good outcome because it permits a less-invasive approach to the penetrated

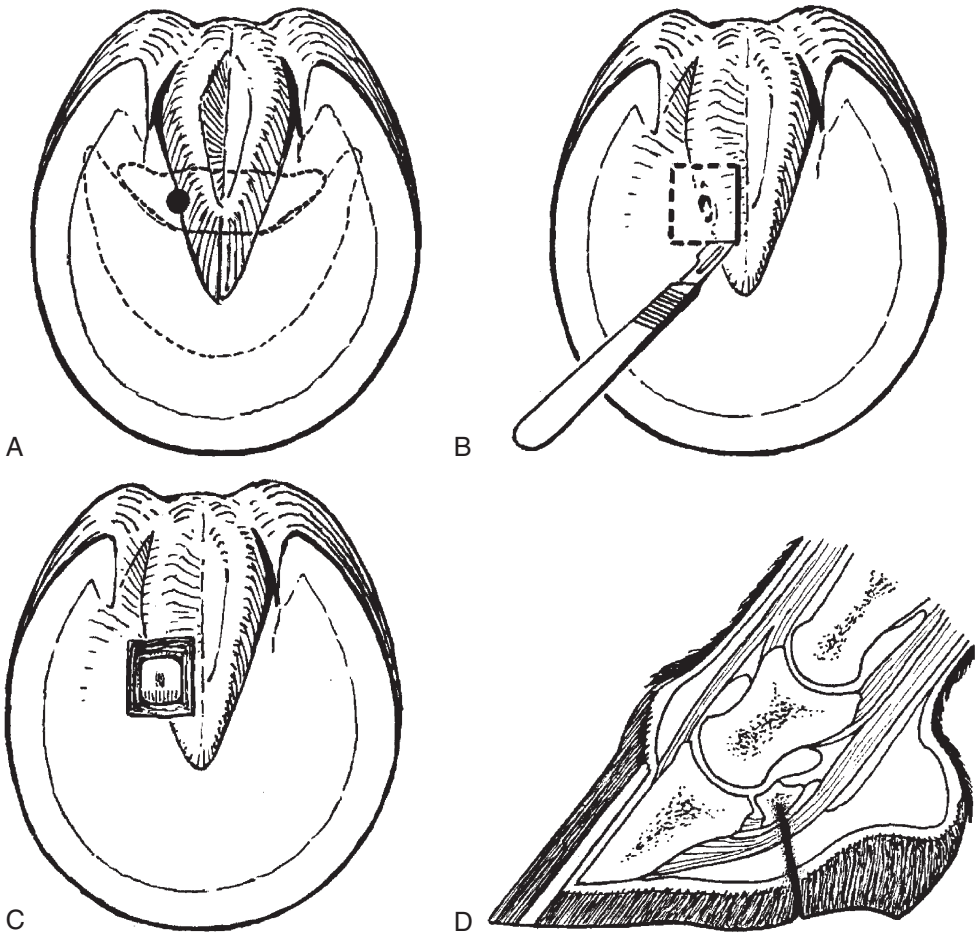

Figure 90-27. Street nail procedure. **A,** Localization of the entry point. **B,** Subcutis and sensitive lamina is removed. **C,** The deep digital flexor tendon is fenestrated. **D,** Representation of the puncture wound in a parasagittal plane.

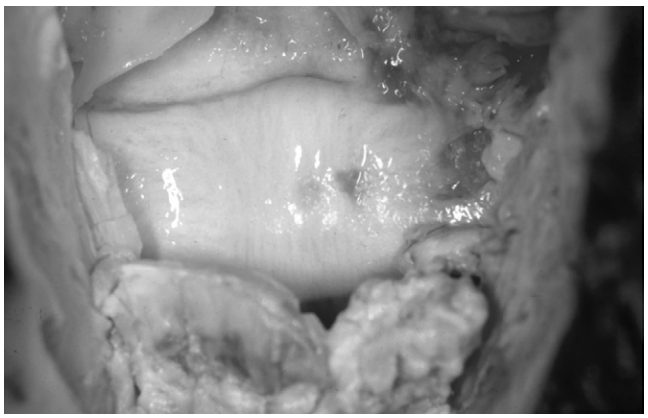

Figure 90-28. Ten-day postoperative appearance after a street nail procedure for a foreign body penetrating the distal sesamoid bone.

structures, allowing débridement to be carried out under endoscopic guidance.[32,33] Also, the area is under constant lavage during treatment, decreasing surgery time.

Depending on the severity of the injury, lavage may be repeated once or twice with the horse under general anesthesia. Soon thereafter, the bandage is changed under aseptic conditions on the standing and sedated horse. Systemic antibiotics and anti-inflammatory drugs are administered for at least 2 weeks and regional intravenous perfusion should be repeated several times. After horn tissue begins to cover the soft tissues, a medication-plate shoe can be applied (Figure 90-30).

Prognosis

The prognosis for puncture wounds of the sole is generally guarded. Horses that receive prompt surgical treatment and have few deep structures affected have the best prognosis.[30] Injury to the distal sesamoid bone can eventually result in adhesions between the bone and the DDF tendon, necessitating neurectomy. Bursoscopic treatment can lessen the development of morbidity.

Scalping Injuries

Etiology

Coronary band and heel injuries occur at the transition between the skin and hoof capsule. They are usually caused by overreaching, where one foot treads on the coronary band or heel of another foot. Heel calks on shoes can result in very deep injuries as well.

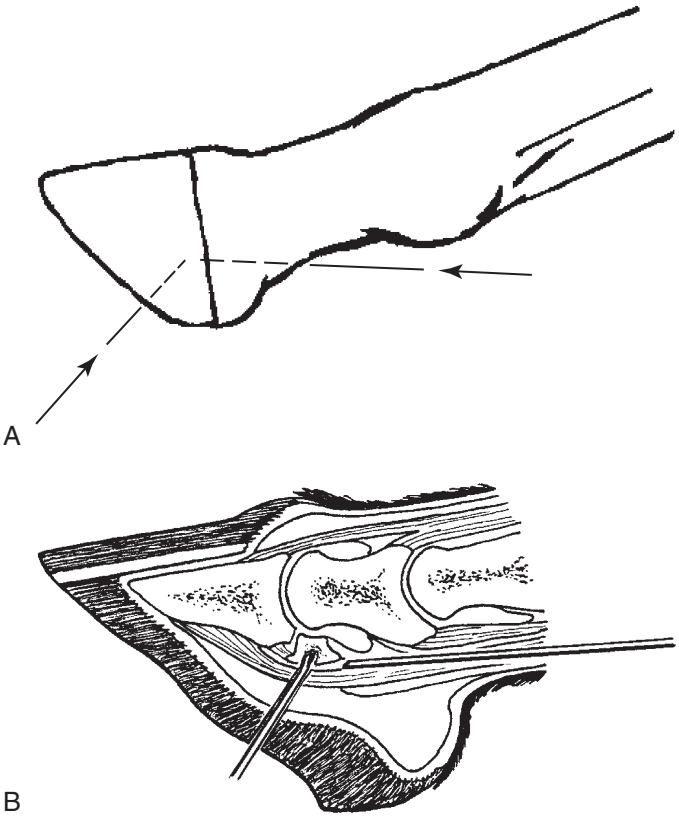

Figure 90-29. Bursoscopy in lateral recumbency. **A,** Overview of the surgical portals. **B,** Close-up view showing the anatomic structures involved.

Figure 90-30. A medication-plate shoe used for postoperative protection of a foot after surgery. The wound may be dressed daily by removing the plate.

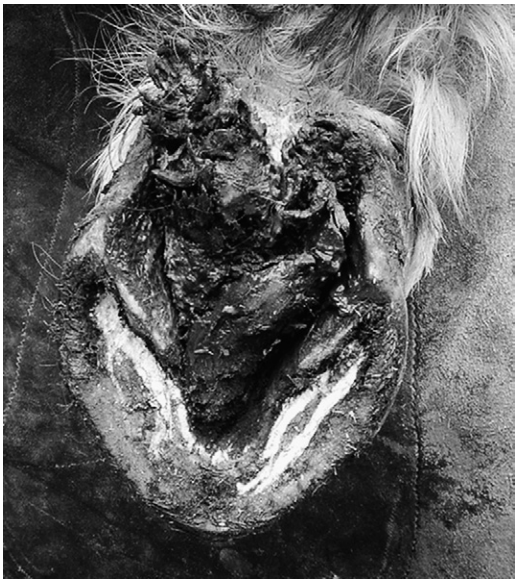

Figure 90-31. Canker in a horse showing a flaky, hypertrophic frog.

Diagnosis

These injuries are usually quite painful in the acute stages and can cause severe lameness. Inadequate treatment can lead to infection within the hoof capsule. There is also a risk of permanent damage to the horn-producing cells, resulting in abnormalities of hoof growth, such as cracks and ridges.

Treatment and Aftercare

The damaged horn should be removed to allow drainage of wound secretions. Heavy scissors, ronguers or a hoof knife are ideal for this. The wound is subsequently cleaned with a mild disinfectant solution and bandaged.

Other Hoof Lacerations

Injuries to the hoof capsule are relatively common. Trauma to the corium usually results in marked hemorrhage and severe pain. This type of injury is particularly problematic when the coronary corium is involved; damage to the horn-producing cells results in the production of poor-quality horn. A pressure bandage is applied to injuries that are bleeding severely. The bandage is changed daily until a stable and healthy layer of horn is formed.

DEGENERATIVE AND NEOPLASTIC DISEASES OF THE FOOT

Canker

Etiology

Canker used to be a common disease in working draft horses. Therefore, detailed descriptions can be found in older textbooks. Canker of the frog represents abnormal horn proliferation in the frog region and is rarely seen in other areas of the hoof (Figure 90-31). It is not a true neoplasm, but rather a chronic inflammatory reaction characterized by massive parakeratosis.[34] The sensitive lamina hypertrophies and the

superficial horn and corium degenerates. Canker can spread from the frog to the adjacent sole and even involve the hoof wall.

This disease is slowly progressive and spreads to adjacent structures over the course of months. In some horses, the skin adjacent to the hoof capsule is affected. The skin near the coronary band is reddened, the hair grows in all directions, and the area is painful when palpated. In rare cases, the horn of the chestnuts is affected.

The cause of canker is not known, although unsanitary stall conditions appear to be a predisposing factor. Long-standing thrush is thought to result in canker formation, because it causes the original degeneration of the horn cells. Horses that are kept in moist and warm stalls with urine-soaked bedding often suffer from canker. Also, there seems to be a breed predilection; draft horses are most commonly affected, but the disease is also seen in Thoroughbreds, Standardbreds, ponies, and even donkeys. Young horses rarely have canker. Spirochetes have been identified histologically in canker lesions in several horses but not in healthy hoof tissues.[35,36]

The hind hooves are more often affected than front hooves, and one or more hooves may be involved. Bacteriologic examination of affected tissue usually reveals gram-negative anaerobic bacteria. Although many microorganisms can be isolated, anaerobic bacteria seem to be most important in this disease.

A differential diagnosis must include advanced thrush and fungal infection of the coronary band.

Diagnosis

Horn of rubber-like consistency that breaks easily on the surface is characteristic of canker. The horn produced by the epidermal cells affected by canker is soft, greasy, and friable. Cauliflower-like growths that are not cornified at the surface but covered with a greasy, grayish-white material are typical. These hypertrophic horn growths have a foul odor somewhat reminiscent of an abscessed tooth. The area is painful to pressure and horses are usually lame. When traumatized, the area bleeds profusely. The poor horn quality can result in injury and infection of the underlying structures.

Treatment and Aftercare

Treatment of canker is very difficult and time consuming, and it does not always result in a cure. All damaged cells must be removed to allow the growth of healthy horn tissue. Treatment must be as hygienic as possible and involves a number of steps.[37,38]

SURGERY

The first step is surgical débridement under local anesthesia. All necrotic and abnormal horn is removed with the horse standing or, in severe cases, with the horse under general anesthesia. A tourniquet is applied to the limb in the middle metacarpal or metatarsal region. All abnormal horn is removed so that the area can be thoroughly cleaned and cut with a scalpel blade (Figure 90-32).

The transition between abnormal and healthy horn is determined during paring with a hoof knife. A cut is made around the frog to define the surgical field. Then, beginning from the palmar or plantar surface, the entire frog is removed (Figure 90-33). It is important not to cut too deeply in or near the apex of the frog. Alternatively, cryotherapy can be used.[39] Normal epithelium is then able to grow from the remaining healthy tissue.

In most cases, removing the abnormal horn while sparing the stratum germinativum is adequate. A new frog grows within a few months (Figure 90-34). The foot is bandaged during this time, so this treatment is labor intensive and time consuming for the owner.

LOCAL TREATMENT

The second step involves local treatment consisting of disinfection, drying, and hardening of the horn. The hoof is cleaned and disinfected with povidone-iodine scrub and treated with

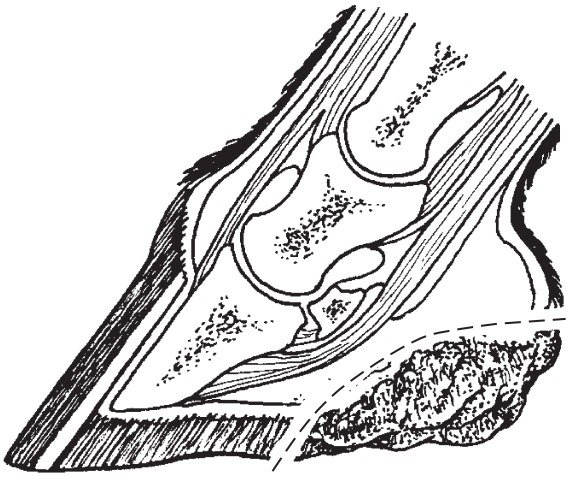

Figure 90-32. Sagittal view of a graphic illustration of a hoof with canker. The *dotted line* represents the level of the surgical cut needed for removing the frog.

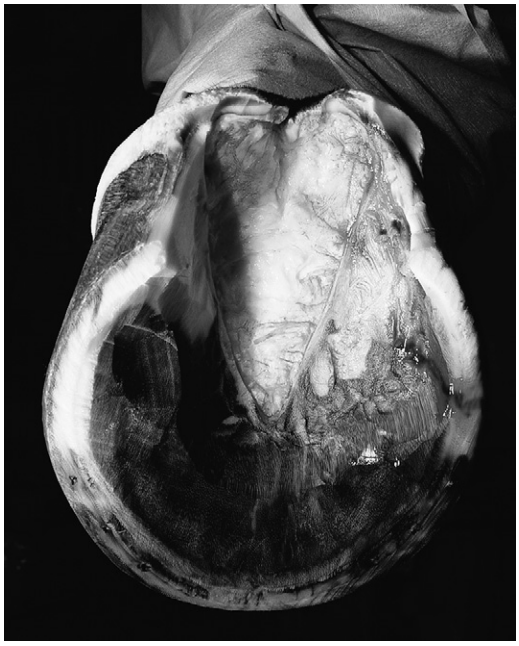

Figure 90-33. Postoperative picture following removal of the entire frog in a horse affected with canker.

Figure 90-34. Four-month follow-up picture showing progressive healing after removal of the entire frog.

povidone-iodine–soaked bandages for 2 to 3 days. Daily bandage changes are required thereafter.

When healing is observed, a shoe with a pad is applied. The pad must be removable so that the area can be cleaned daily with povidone-iodine scrub and rinsed with povidone-iodine solution. The defect is covered daily with a mixture of 20 g iodoform iodine, 20 g zinc oxide, 20 g tannic acid, and 40 g metronidazole; this mixture acts as a disinfectant, astringent, and drying agent. The wound is subsequently bandaged or covered with gauze and protected with the pad.

Administration of local or systemic antibiotics might be necessary; chloramphenicol and metronidazole are particularly effective.[40] Systemic antibiotics should be administered when more than one hoof is affected. Recently the local and systemic treatment with doxycycline and oxytetracycline has been recommended.[41] Biotin and zinc supplements should be added to the nutritional regimen.

The horn defect becomes smaller as it heals (see Figure 90-34). The persistence of soft greasy horn usually indicates recurrence of the disease. Repeated surgical débridement is required in some cases. Treatment entails many months and requires good owner compliance. Sanitary stall conditions also are important.

DISORDERS OF THE COLLATERAL CARTILAGES

Mineralization

Etiology

Mineralization of the collateral cartilages of the distal phalanx (side bone) is a common radiographic finding in the front feet of heavy horses.[42,43] Ossification of the collateral cartilages has been considered as a part of the normal aging process, and such factors as heavy body weight, working on hard surfaces, repetitive concussion, poor conformation, improper shoeing or trimming, and other foot problems, such as collateral ligament or distal phalangeal injuries,[44] are often mentioned as possible causes.

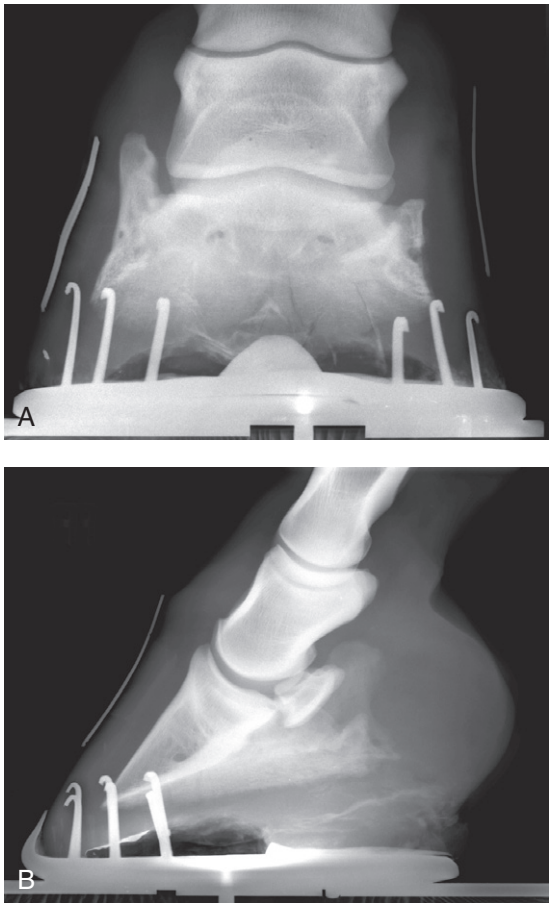

Figure 90-35. Dorsopalmar **(A)** and lateromedial **(B)** radiographic views of a hoof with mild ossification of the lateral collateral cartilage at the palmar process of the distal phalanx. A wire was taped to the hoof wall to identify its exact orientation.

However, large side bones have also been found in young horses that have not yet started working; therefore an inherited tendency to develop side bones has been suspected. Side bones are more common in mares than in stallions and geldings.[45,46] Extensive ossification is more commonly found in the lateral than the medial cartilage.

Diagnosis

Ossification of the collateral cartilages is most often an incidental finding. Lameness associated with side bone is difficult to confirm. It is important to correlate radiographic findings with signs of pain. Radiographs reveal ectopic ossification emanating from the proximal aspects of the palmar or plantar eminences of the distal phalanx in the area of the collateral cartilages (Figure 90-35). Because separate centers of ossification exist in these cartilages, these findings should not be confused with fractured side bones (Figure 90-36), although in rare cases the side bone can fracture (Figure 90-37).

Digital manipulation of the collateral cartilages can reveal loss of pliability and elicit a pain response. Lameness is most marked when the horse is turned at a trot on a hard surface. A palmar

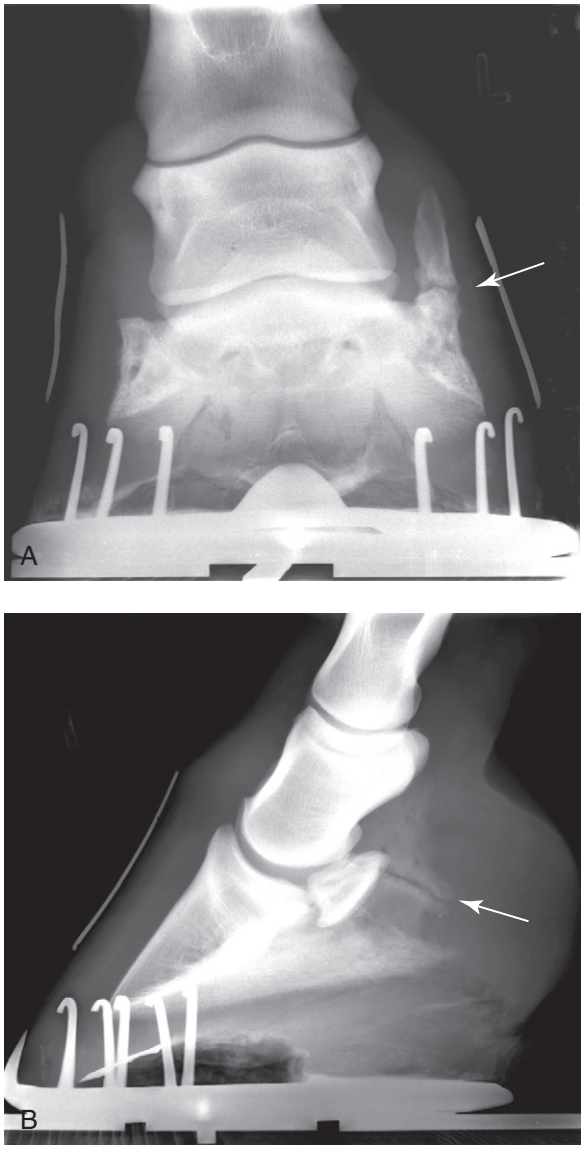

Figure 90-36. Dorsopalmar **(A)** and lateromedial **(B)** radiographic projections of a horse suffering from ossification of the lateral collateral cartilage in a left forelimb. Note the radiolucent line *(arrow).* This separate ossification center should not be confused with a fractured side bone. Dorsally and on each side of the foot, small wires were taped along the hoof wall to identify its surface.

digital nerve block on the suspected side of the hoof should improve the painful gait. Scintigraphy may provide information on the potential clinical significance of ossification of the cartilages of the foot.[47] Increased radiopharmaceutical uptake associated with a different radiographic appearance from that of other ossifications of the front feet was a conclusive sign of clinical significance in a report of 21 Finnish horses.[48] Although ossification of the cartilages is commonly associated with obscure lameness problems, most often ossification is not the cause.

Treatment

When ossification of the collateral cartilage has started, it cannot be stopped, and specific treatment is not available. However, if the side bones are suspected as the cause of lameness, rest and administration of NSAIDs would be the logical initial treatment. Any foot imbalances should be corrected, and breakover should be moved caudad on the foot by rolling or rockering of the toe. The heels should have as much opportunity for expansion as possible, which is especially important for unbalanced or sheared heels.

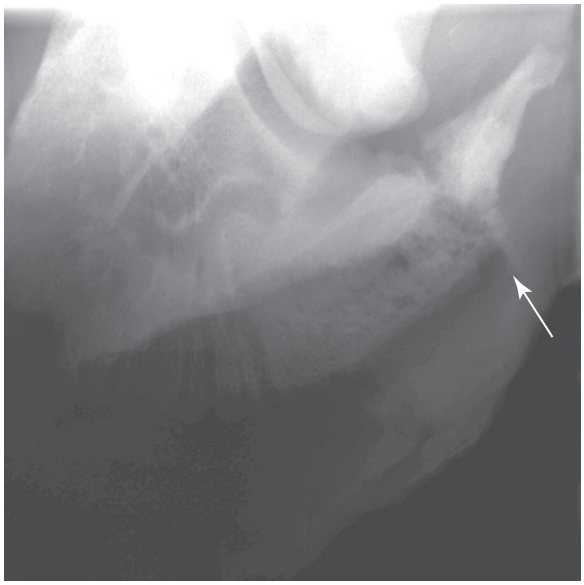

Figure 90-37. Oblique radiographic projection of the distal phalanx of an Arabian horse with a 6-month-old fracture of the medial side bone. Healing is in progress, but the fracture line *(arrow)* is still visible.

A fracture of an ossified cartilage is treated conservatively with NSAIDs and an extended period of stall rest or small paddock turnout (8 to 12 weeks) followed by controlled exercise on level surfaces for 6 to 8 months. It is most likely that after this period of time, the fracture line will still be visible on radiographs but surrounded by proliferative exostosis (see Figure 90-37). In refractory cases, unilateral palmar digital neurectomy can be performed.

Small fragments can be removed eventually if any signs of sequestrum formation are observed. An incision 1 to 2 cm parallel to the palmar or plantar aspect of the coronary band provides access to the proximal part of the collateral cartilage. No attempts should be made to remove large proximal fragments.[49]

Necrosis

Necrosis of the collateral cartilage (colloquially termed *quittor*) is characterized by an intermittent purulent discharge and sinus tract formation at or proximal to the coronary band in proximity to the collateral cartilage. These lesions are chronic and do not heal. Lameness occurs in the acute stages but might show remission when the lesion appears to be healing. Usually a mixed bacterial infection is present, associated with subsequent necrosis of the cartilage, as the result of injury to the cartilage itself or to the adjacent soft tissue.[50] The quittor condition can develop secondary to a penetrating wound through the sole where infection has gained access to the collateral cartilage.

Diagnosis

Clinical signs of quittor include enlargement over the affected collateral cartilage with one or more chronic sinus tracts that continue to drain. The most important differential diagnosis is a chronic ascending infection of the white line that breaks and drains at or slightly proximal to the coronary band. With quittor, the swelling is usually more diffuse and is located more proximally over the collateral cartilage.

Radiography can be helpful to detect foreign bodies or to rule out involvement of the middle or distal phalanx. The depth and direction of the draining tracts are determined radiographically after injection of a contrast medium or insertion of a flexible metal probe into the tract.[51]

Treatment

The treatment of choice is surgical débridement. Prior to surgery the region is clipped and the hoof is trimmed and rasped, scrubbed, and placed in a povidone-iodine–soaked bandage for 24 hours. After the horse is anesthetized, a tourniquet is applied. The foot is held in maximal extension during dissection to tighten the joint capsule and retract it from the area of dissection, minimizing the risk of accidental penetration into the DIP joint.[50] A slightly curved incision beginning just dorsal to the coronary band over the diseased collateral cartilage is made (Figure 90-38). The flap is dissected distad to expose the collateral cartilage, and a probe is used to identify the draining tract. Alternatively, diluted methylene blue can be injected into the tract to identify it during dissection.

Necrotic cartilage is recognized by its dark blue or reddish blue appearance. All of the necrotic tissue and cartilage is removed until healthy margins remain. Subsequently, arthrocentesis of the DIP joint is performed and the joint is distended with polyionic fluid to assess the integrity of the joint capsule axial to the removed portions of the collateral cartilages. In case of accidental laceration of the joint capsule, the joint must be flushed with large amounts of fluid through a needle placed in the dorsal aspect of the joint capsule.

After débridement, the defect is closed with 2-0 monofilament absorbable suture material in a simple interrupted pattern. An injection of 2 mL of amikacin into the joint protects it from developing an infection. If necrotic cartilage extends distal to or below the coronary band, a hole is drilled in the hoof wall over the ventral-most limits of the excised cartilage to provide drainage (see Figure 90-38). After extensive flushing, the proximal skin incision is closed with nonabsorbable tension sutures, and the wound is packed through the hole in the hoof wall using antiseptic-soaked gauze sponges. The surgical site is protected with a sterile bandage.

The following day the bandage is removed and the wound is flushed. Bandages are changed every other day until all evidence of infection has subsided and a healthy bed of granulation tissue is present. When this occurs, a short limb cast can be applied to the limb for 8 to 10 days to minimize movement at the suture line and to encourage primary healing. After removal of the sutures, bandaging is continued until granulation tissue fills the defect and epithelium begins to cornify.

FRACTURES OF THE HOOF REGION

Fractures occurring in the hoof region are separated into distal phalanx fractures and fractures of the distal sesamoid bone.

Fracture of the Distal Phalanx

Fractures of the distal phalanx are diagnosed in horses of all ages, even very young foals.[52,53]

Etiology and Classification

Fractures of the distal phalanx are caused by acute trauma, such as a kick toward a hard, nonmovable object. Most often fast or excessive work induces fractures of the distal phalanx.[54]

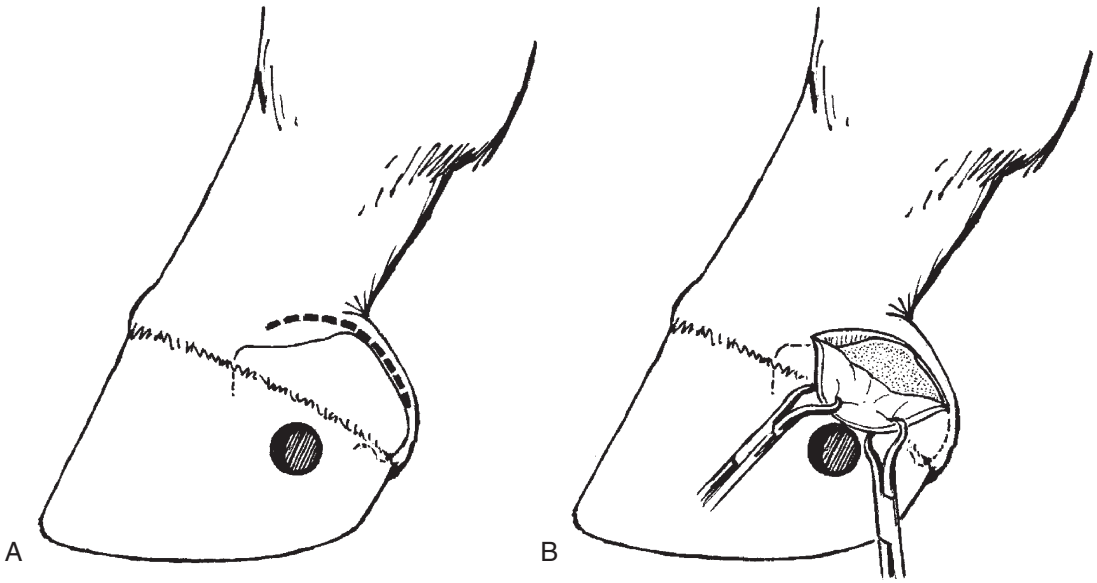

Figure 90-38. Graphic illustration of the surgical treatment for quittor, or necrosis of the collateral cartilage. A hole drilled in the hoof wall is required for adequate drainage if infection and necrosis extend distal to the coronary band. **A,** The *dotted line* represents the surgical incision line. **B,** A slightly curved incision beginning just dorsal to the coronary band over the diseased collateral cartilage produces a skin flap, which allows removal of the proximal part of the necrotic cartilage.

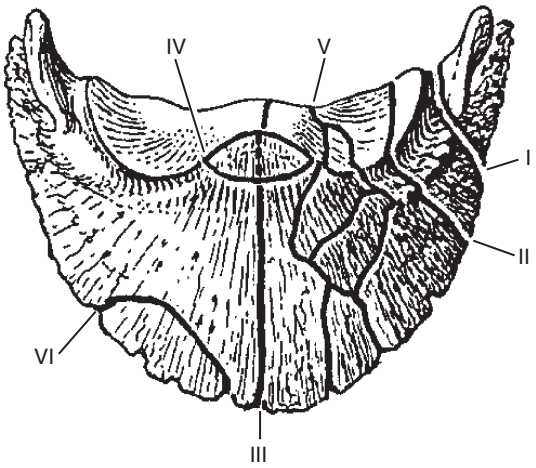

Figure 90-39. Classification of distal phalangeal fractures: *I*, Abaxial nonarticular fracture; *II*, abaxial articular fracture; *III*, axial and periaxial articular fracture; *IV*, extensor process fracture; *V*, multifragment articular fracture; *VI*, solar margin fracture.

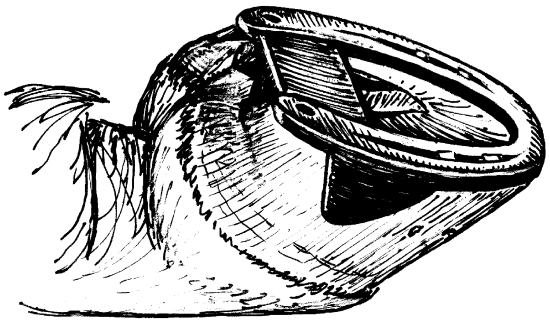

Figure 90-40. Bar shoe with large side clips to limit hoof expansion laterally during weight bearing. This shoe is often used after hoof wall resection is performed to access the interior of the hoof capsule, as well as for pedal bone fractures.

Laceration of the hoof capsule may result in fractures as well. The forelimb is more commonly involved than the hindlimb.[55]

Fractures of the distal phalanx are classified into the following types (Figure 90-39)[55]:

I. Abaxial/paramedian fractures without joint involvement
II. Abaxial/paramedian fractures with joint involvement
III. Axial/sagittal and perisagittal fractures with joint involvement
IV. Fractures of the extensor process
V. Multifragment (comminuted) fractures with joint involvement
VI. Solar margin fractures

Some complicated fractures involve several fracture types and therefore they cannot be assigned to one of these classifications alone.[54,56]

Clinical Symptoms and Diagnosis

The patient usually shows an acute, moderate to severe lameness accentuated during turns. The hoof and distal phalangeal region are warm to the touch, and an increased pulse can be palpated over the palmar or plantar arteries.[52] Pressure exerted with the hoof testers might elicit a positive response. Arthrocentesis of the DIP joint results in a blood-tinged synovial sample when there is articular involvement. Signs are relieved by regional anesthesia of the distal phalangeal region. In the differential diagnosis, a hoof abscess should be considered.

Radiography confirms the diagnosis in most cases. However, occasionally it is difficult to detect a fracture line because of minimal displacement. Additionally, the irregular border of the distal phalanx and debris on the hoof capsule can make recognition of the fracture difficult. It is important to take several radiographs from different angles.[55] Abaxial nonarticular fractures are usually difficult to recognize because they are normally only minimally displaced.

It is important to distinguish vascular channels from potential fractures. The presence of thin lines crossing vascular channels at different angles indicates a fracture. If no fractures can be recognized, the animal should be placed in a box stall and the radiographs repeated after 7 to 10 days.[57] Because of displacement of the fracture or osteolysis occurring at the fracture margins, the radiographic fracture gap enlarges with time, aiding in recognition of the fracture. Scintigraphy, CT, and magnetic resonance imaging (MRI) are often successful in delineating an obscure fracture (see Chapters 69 to 71).[57,58] CT and MRI are especially useful to assess articular fractures and to identify displacement of the fragments, which, if they result in a step in the articular surface, have a poor prognosis.

Treatment

Fractures of the distal phalanx can be managed with fragment removal, cast application and special shoeing, compression screw fixation, and neurectomy.[59-61] Foals are best treated with stall rest.[53,60]

ABAXIAL FRACTURES (TYPES I AND II)

These fractures are supported for 2 months by application of a fiberglass cast around the hoof capsule. The sole must be filled with silicone or polyurethane to impede any movement of the hoof capsule. Alternatively, a bar shoe with large side clips is applied to the hoof, providing support to the heels to limit hoof expansion during loading (Figure 90-40). NSAIDs are administered to reduce the pain level and allow the horse to bear weight on the foot. Stall rest for 2 to 4 months is required. Follow-up radiographs are taken to evaluate fracture healing.

Usually after 4 months, the horse can be ridden at a walk on even terrain. Depending on the reaction, the workload is slowly increased.

A reasonable prognosis can be given for future use.[59,62] It usually takes 4 to 6 months for the fracture to heal. However, radiographically the fracture line is visible much longer. Initially a fibrous union develops, which ossifies at 6 to 12 months. In some cases, a nonunion develops (Figure 90-41).

Abaxial articular fractures with substantial displacement may also be treated surgically using lag screw technique as described later (Figure 90-42). There is room for one screw, but insertion in this location is technically difficult and good intraoperative imaging is mandatory for this procedure. Computer-assisted surgery is recommended (see Chapter 13).[63]

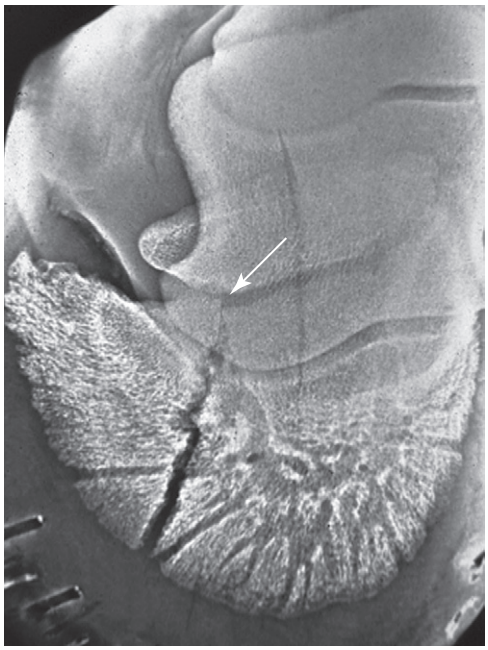

Figure 90-41. Dorsolateral-palmaromedial oblique radiographic view of a slightly displaced abaxial fracture of the distal phalanx with joint involvement, 6 weeks after the injury. The displacement of the fracture is easily recognized at the palmar articular edge *(arrow)*.

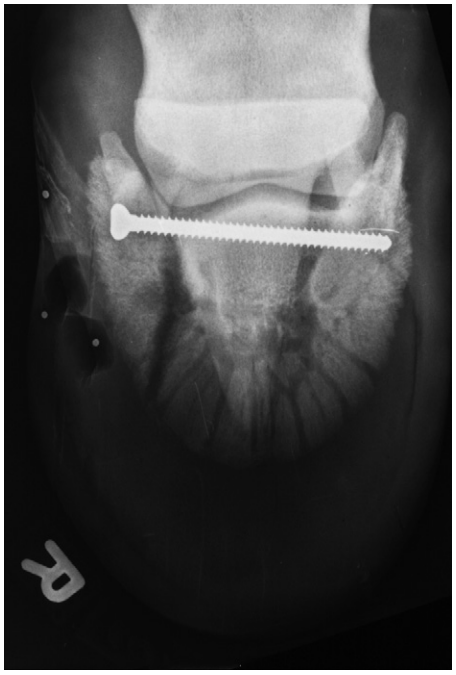

Figure 90-42. Dorsopalmar radiographic view of a displaced articular abaxial fracture repaired with a 4.5-mm cortex screw inserted in lag fashion from plantarolateral to dorsomedial.

AXIAL AND PERIAXIAL FRACTURES (TYPE III)

Axial fractures may be managed conservatively or surgically.

Nonsurgical management

The same type of treatment is used as that for abaxial fractures (see earlier). However, it is important to prolong the rest period: 4 months of stall rest followed by 4 months of hand-walking exercise. Additionally, the horse should be shod for 6 to 8 months with a bar shoe. A guarded prognosis is given for horses older than 3 years that are to be used as future riding animals, whereas horses younger than 2 years have a good prognosis.[61,64]

Surgical management

On the day prior to surgery, the entire hoof is thoroughly cleaned, the sole is trimmed, and all crevices are removed.[64] The entire hoof wall surface is rasped and the foot is placed overnight in a bandage soaked with an antiseptic (Betadine). A preoperative CT technique to determine landmarks for screw insertion has been established in the standing horse.[65]

For surgery, the animal is anesthetized and positioned in lateral recumbency on the surgery table. The use of a fluoroscope during the surgery aids in achieving correct positioning of the screw. With the aid of an arthroscope in the dorsal pouch of the DIP joint, anatomical reduction of the fragments can be observed and if necessary adjusted (Figure 90-43). The entry point through the hoof capsule is determined by taping a radiodense object to the lateral hoof wall and taking a lateromedial radiograph. By comparing the radiographic image with the objects on the hoof wall, the entry point is determined and marked on the hoof wall with an awl. The final preparation of the hoof and phalangeal region for aseptic surgery is then completed.

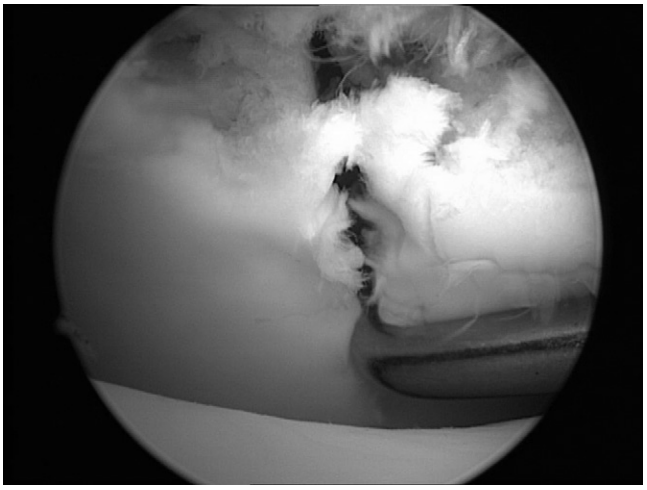

Figure 90-43. Arthroscopic view of a displaced axial fracture of the distal phalanx using a dorsal approach.

Using an 8- to 10-mm diameter drill bit, a hole is prepared through the hoof capsule parallel to the sole surface and aligned with the bulb of the heel. The hole is continued until the drill bit reaches the distal phalanx. A pilot hole is drilled into the distal phalanx with a small drill bit until it crosses the fracture. This depth cannot usually be determined without the help of fluoroscopy or intraoperative radiography. When the desired location and direction of the drill bit is determined, the glide hole is prepared (Figure 90-44). Frequent fluoroscopic views will help identify the moment when the fracture plane is crossed with the drill bit. At this point, the drill bit is removed, the drill sleeve is inserted into the glide hole, and the thread hole is

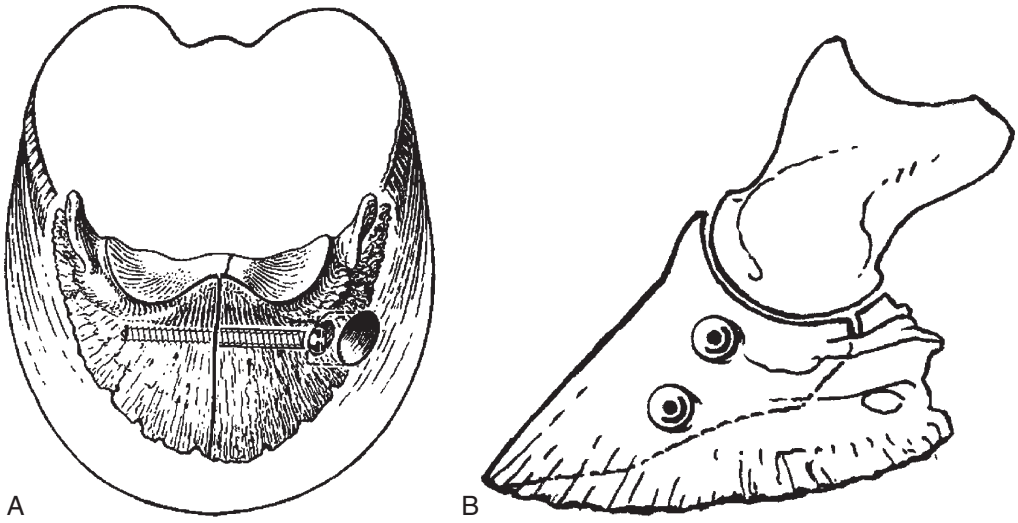

Figure 90-44. A, Graphic illustration of a dorsopalmar view of a fixation of a type III fracture with one cortex screw inserted in lag fashion. **B,** Graphic lateromedial illustration of a fixation of a type III fracture with two cortex screws inserted in lag fashion.

prepared concentrically across the bone. Care is taken to identify the exit point through the opposite fragment to prevent inadvertent trauma to the sensitive lamina. After countersinking and tapping, a 4.5-mm or a 5.5-mm cortex screw of predetermined length is inserted and solidly tightened.

Tangential radiographs are taken directed at the exit point of the screw tip to ensure correct length. Screws that are too long are exchanged for shorter ones. Conventionally, one screw is inserted without taking too big a risk of entering vital structures. With the help of computer-assisted navigation (see Chapter 13), two screws can safely be implanted.[66,67] The hole in the hoof capsule may be filled with an antibiotic-soaked sponge and placed under an aseptic pressure bandage or closed with antibiotic-impregnated polymethyl methacrylate (PMMA). When the horn defect is dry (if it is not immediately filled with PMMA), it is filled with artificial horn.

Postoperatively the horse is kept in a box stall for 4 months followed by hand-walking exercise for an additional 4 months. Barring any complications, the screw may be left in place; otherwise it is removed if problems develop. The prognosis for a pleasure horse to return to athletic use is guarded to good. Complications include postoperative infection and abscess formation.

EXTENSOR PROCESS FRAGMENTS (TYPE IV)

Etiology

These fragments have three known etiologies: hyperextension injury, avulsion by the extensor tendon, and a separate center of ossification (osteochondrosis, which may be bilateral).

Clinical symptoms and diagnosis

Some horses show no clinical signs, and the presence of a fragment of the extensor process is found incidentally. Often the clinical signs are similar to those exhibited with other articular disorders of the DIP joint. A routine lameness examination including perineural and intra-articular anesthesia and a radiographic examination permit a definitive diagnosis (Figure 90-45).

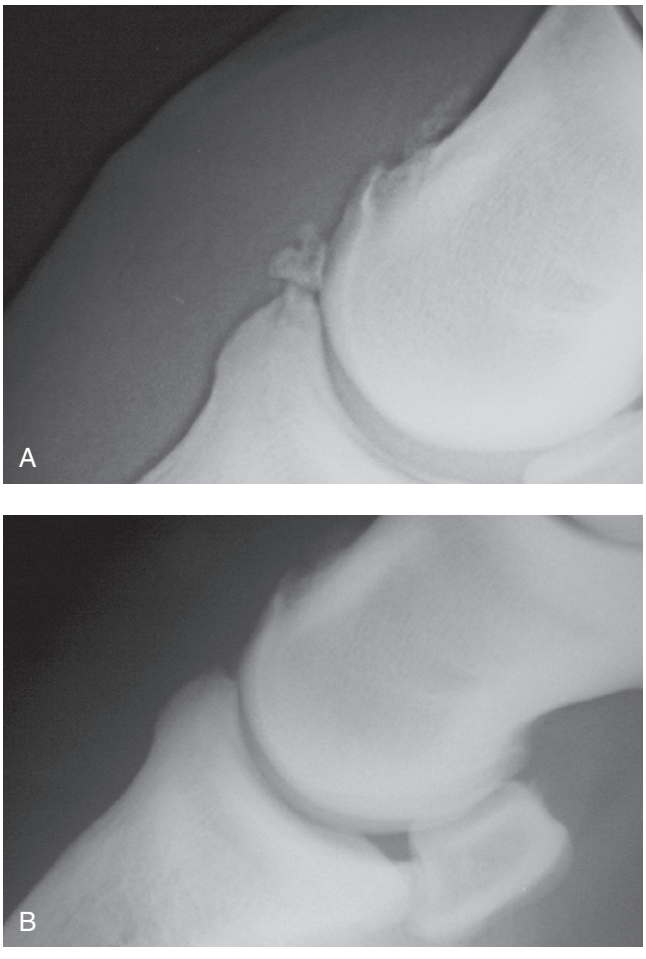

Figure 90-45. A, Preoperative lateromedial radiographic view of an extensor process fracture. **B,** Postoperative lateromedial radiographic view of the same horse after removing the fragment.

Treatment

Small fragments (see Figure 90-45) should always be removed because they are mobile and have contact with the articular surfaces.[68] Fragment removal is accomplished by routine arthroscopy (Figure 90-46). Adjunctive therapy with intra-articular hyaluronan is encouraged. Postoperatively, the phalangeal region is kept under a bandage for 3 weeks and the horse is rested for 6 to 8 weeks.

There is no ideal treatment for large fragments (Figure 90-47). Therefore, conservative therapy is usually tried first. Insertion of one or two screws in lag fashion has been described, but only rarely is a rigid fixation achieved.[69-71] Therefore, if lameness persists after conservative management, surgical removal of the fragment is recommended.[71] The fragment is divided into smaller pieces and these smaller pieces are removed under arthroscopic supervision. Generally, a good prognosis can be given.[72] Postoperatively, a fiberglass cast or a compression bandage should be applied over the surgery site.

MULTIFRAGMENT FRACTURES (TYPE V)

Multifragment fractures are always associated with severe lameness. Radiographically, several fracture lines can be appreciated (Figure 90-48). Application of a fiberglass cast may be tried; however, these fractures have a poor prognosis for future soundness. Therefore, euthanasia has to be considered. Alternatively, a neurectomy can be performed if euthanasia is not an option for the owner.

SOLAR MARGIN FRACTURES (TYPE VI)

Solar margin fractures do occur often and are underdiagnosed.[55,73] Direct or blunt trauma, possibly caused when the horse kicks a hard, immobile object, are common causes of solar margin fractures. In the spring of the year in some climates, when freezing and thawing produces hard uneven ground in the mornings, these fractures are prone to occur. These fractures can also develop as a result of chronic laminitis.[55] Most solar margin fractures heal by bony union; rarely,

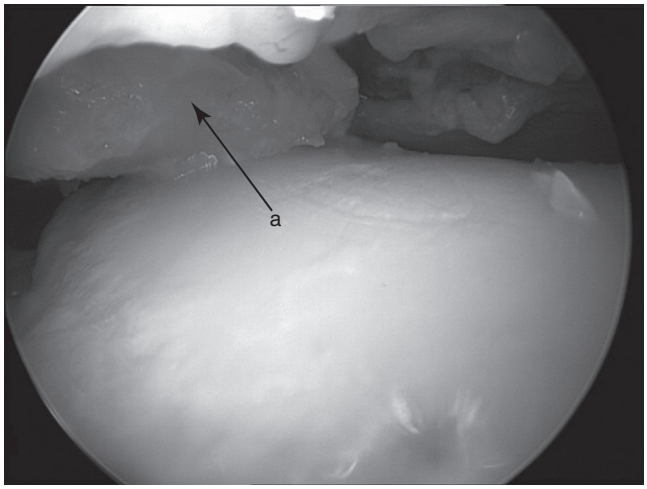

Figure 90-46. Arthroscopic view of the fractured extensor process *(a)*. The *arrow* points toward the fragment.

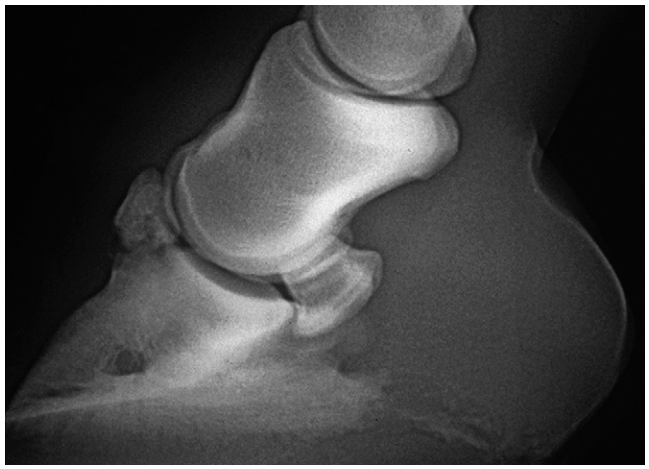

Figure 90-47. Lateromedial radiographic projection of a 3-year-old Friesian horse with a large fragment of the extensor process.

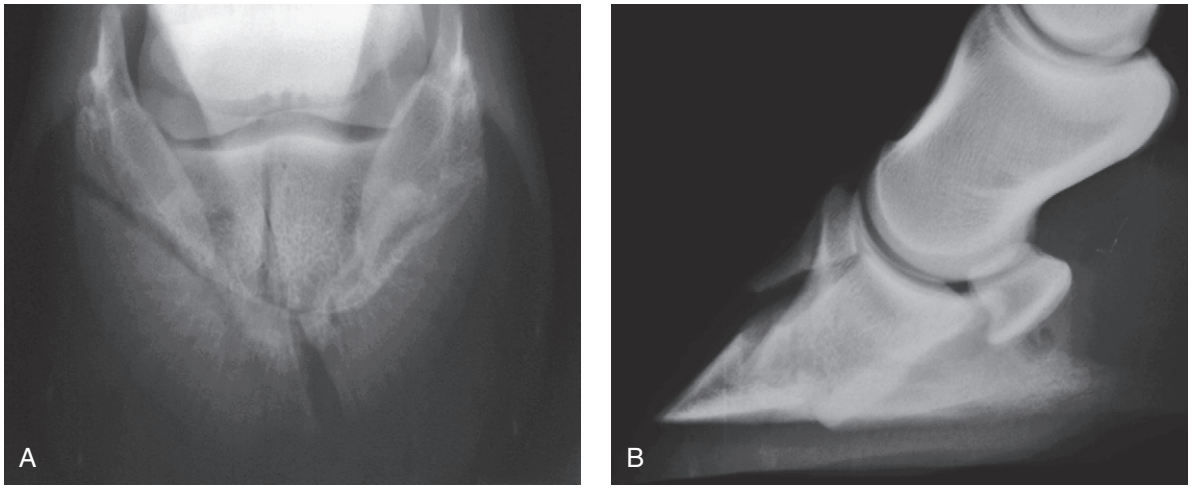

Figure 90-48. Dorsopalmar **(A)** and lateromedial **(B)** radiographic views of a Thoroughbred racehorse with a multifragment (comminuted) fracture of the distal phalanx.

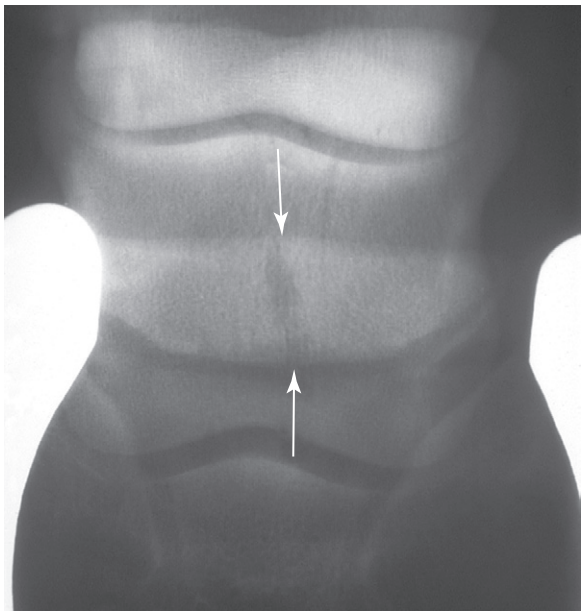

Figure 90-49. Dorsopalmar radiographic projection of an axial fracture of the distal sesamoid bone *(between the arrows)* showing some displacement.

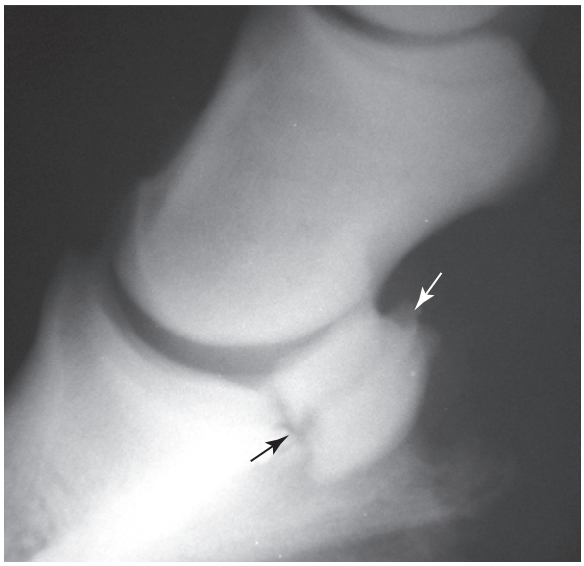

Figure 90-50. Lateromedial radiographic view of a frontal plane fracture *(between the arrows)* of the distal sesamoid bone.

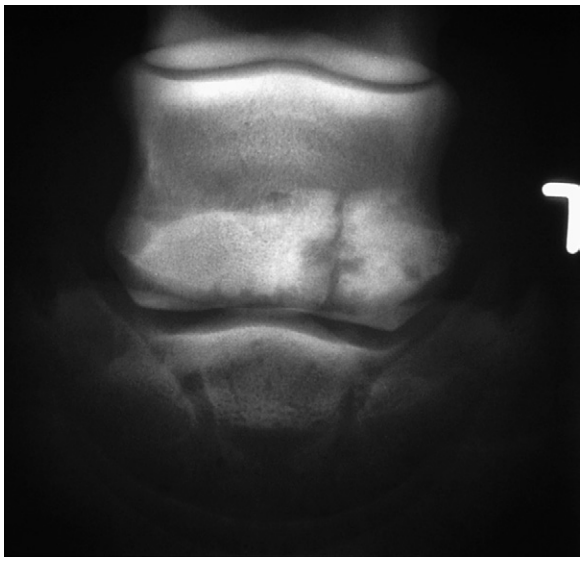

Figure 90-51. Dorsopalmar radiographic view of a pathologic fracture of the distal sesamoid bone. Note the large radiolucent area in the center of the fracture and the proliferative new bone formation on the proximal rim.

resorption of small fragments can occur.[55] Surgical removal of fragments has been reported in individual cases, especially if sepsis develops.

Fractures of the Distal Sesamoid Bone

Fractures of the distal sesamoid bone (DSB) occur rarely. They are diagnosed more often in the forelimbs. Four different types are distinguished: avulsion fractures, simple fractures of the body, comminuted fractures, and frontal fractures.

Avulsion fractures of the distal margin are often found in conjunction with navicular disease. Therefore, these fractures are considered part of an osteoarthritic syndrome (see later in this chapter).

Most of the simple fractures occur abaxially in a vertical or slightly oblique direction (Figure 90-49). Some displacement is possible.

Multifragment fractures are rare and in most cases carry a poor prognosis. Fractures in the frontal plane are highly unusual (Figure 90-50).

Etiology

Most fractures of the DSB have a traumatic origin and are the result of excessive or repetitive loading through the middle and distal phalanges and the DDF tendon.[74] Occasionally, a preexisting pathologic condition in the DSB predisposes it to fracture (Figure 90-51). In these cases, chronic navicular disease is usually implicated.[75]

Clinical Signs and Diagnosis

Clinical signs include an acute moderate to severe weight-bearing lameness associated with an intense pain reaction to turning, increased pulsation of the palmar or plantar arteries, and positive reaction to the application of hoof testers. The low palmar or plantar nerve block is usually diagnostic. All standard radiographic views of the DSB should be taken (lateromedial, tangential, and oblique) to rule out the potential presence of a bipartite or tripartate DSB (two or three ossification centers) or other disorders. Air shadows of the sulci of the frog will project over the DSB and can be mistaken for fracture lines. Therefore, it is important to fill them with a suitable modeling compound before taking the radiographs.

Treatment

Management options for fractures of the DSB include conservative and surgical techniques. Callus formation is rare, and often syndesmoses are formed (fibrous tissue union).[74]

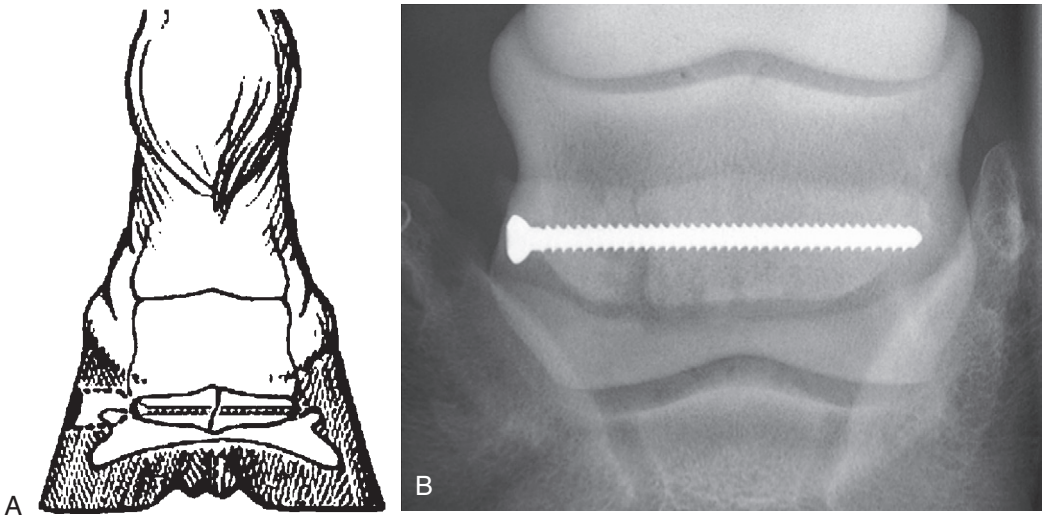

Figure 90-52. A, Graphic illustration of a screw inserted through the hoof wall into the distal sesamoid bone, stabilizing the fracture. Note the large hole drilled through the hoof wall to get access to the distal sesamoid bone. **B,** Dorsopalmar radiographic view showing a 3.5-mm cortex screw inserted across the fracture.

NONSURGICAL MANAGEMENT

Corrective shoes are recommended to protect the DSB. Therefore, nonsurgical management involves stall rest and an elevated heel and bar shoe for 4 months, followed by small paddock exercise and a shoe with thickened branches.[76] Even if the patient improves clinically during the first few months, an osseous union cannot be detected radiographically. Healing requires 10 to 12 months. The prognosis for future athletic activity is unfavorable.[77]

One option involves fixation of the distal limb in an extremely flexed position using a fiberglass cast, which relieves excessive stresses on the DSB. Acceptance of the cast takes a special patient, and healing requires about 6 months. Despite these measures, the prognosis for a future pleasure horse is guarded.

SURGICAL MANAGEMENT

The surgical options include screw fixation and neurectomy. Insertion of a cortex screw in lag fashion provides stability and promotes bone union.[78] The difficulty lies in correct placement of the screw, avoiding penetration of the DIP joint and the palmar tendon sheath (Figure 90-52). Close intraoperative monitoring through fluoroscopy or multiple radiographic images is of utmost importance. The application of a specially developed radiolucent aiming device (Figure 90-53) is mandatory for a correct screw placement. Computer-assisted navigation is an alternative technique (see Chapter 13).

Effective preoperative preparation of the hoof is initiated the day before surgery, as described earlier. The entry point through the hoof wall is determined and marked with the help of fluoroscopy. The horse is placed under general anesthesia and positioned in lateral recumbency on the surgery table. The lateral hoof wall and the pastern region is prepared for aseptic surgery.

After preparing an 8-mm drill hole across the hoof wall at the previously marked location, the aiming device is applied to the hoof and adjusted with the help of fluoroscopy. There is considerable variation among horses in relation to the structures that have to be navigated to get access to the lateral aspect of the DSB. In some horses, only the collateral cartilage has to be traversed, whereas in others part of the distal phalanx and/or the ossified collateral cartilage has to be traversed. It is important to prepare a large enough opening to allow the screw head to pass easily down to the DSB. The 3.5-mm glide hole is prepared, just crossing the fracture plane. The insert drill guide is inserted into the glide hole, and a 2.5-mm thread hole is drilled into the opposite fragment. After tapping, a 3.5-mm cortex screw of estimated length is inserted and tightened. With the help of a dorsopalmar radiograph, correct positioning and length are verified.

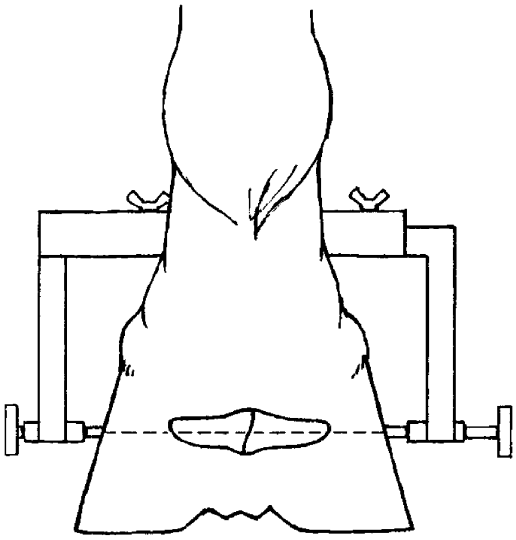

Figure 90-53. Graphic illustration of the aiming device used to ensure intraosseous placement of a screw into the distal sesamoid bone.

Computer-assisted surgery has significant advantages over screw insertion via an aiming device.[79] Using a computer-assisted surgery system, 3.5-mm and 4.5-mm screws were implanted into normal DSBs.[80] The glide hole was drilled from the surface of the hoof wall down to the lateral aspect of the

DSB. Subsequently, the drill bit was exchanged with a new one of the same size, with which the glide hole was created across the DSB just beyond the imaginary fracture plane. Finally, the thread hole was drilled using routine technique. This was followed by expanding the hole in the hoof wall across the distal phalanx/collateral cartilage to an 8 mm diameter. The lateral aspect of the DSB was countersunk and the thread hole was tapped. The screw length was determined with computer-assisted navigation. Using this technique, it was possible to insert all 3.5-mm and 4.5-mm cortex screws without penetrating the DIP joint or flexor surface of the DSB. The problem with the 4.5-mm cortex screw was their relatively large screw head relative to the depth of the DSB.[80] In 8 out of 10 cases, the screw head modified either the articular rim or the rim of the flexure surface.[80] The use of the 4.5-mm cortex screws would be preferred but the screw head is too large. The screw head was machined to a 5-mm diameter, which solved the problem. The use of the 4-mm cortex screws could be considered to improve the stability of the fixation and reduce the risk of screw breakage through cyclic loading.

Healing of the fracture takes 6 to 8 months, during which stall rest and hand-walking exercise are implemented. Barring any complications, the screw is left in place.[81] The prognosis for athletic competition is guarded, but for pleasure riding it is favorable.[77] To allow the horse to return to competitive work, it is often necessary to perform a neurectomy.[78]

ARTICULAR SURGERY

Arthroscopy

Arthroscopy of the DIP joint (Figure 90-54) has replaced arthrotomy, as it has in other joints. The advantages include the small approach portals, the small risk of postoperative infection, and the shortened convalescent period.[68] Indications for arthroscopic interventions include fragment removal from the extensor process, lavage in cases of infection, and curettage of subchondral cystic lesions.[82]

Preparation

The hoof should be thoroughly cleaned the day before surgery, and the coronary band and the phalangeal region should be clipped. The entire area is scrubbed and placed overnight under an antiseptic bandage. The shoe may be left in place.

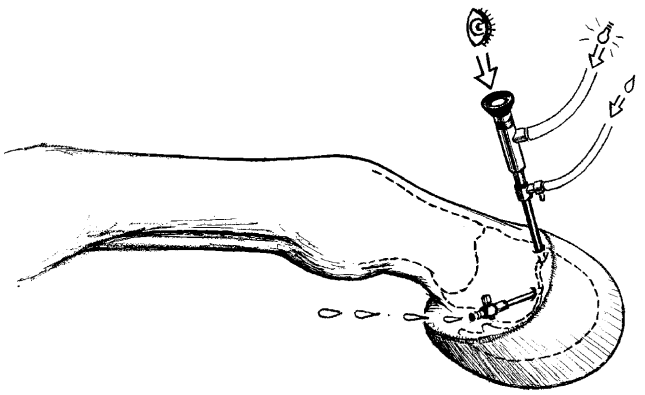

Figure 90-54. Typical location of the arthroscopic and instrument portals (or egress needle shown here) used for arthroscopy of the distal interphalangeal joint.

For arthroscopic interventions in the forelimb, the animal is positioned in dorsal recumbency. Lateral recumbency is usually selected in the hind limb because the reciprocal apparatus makes manipulation easier in this position. An Esmarch bandage is applied to prevent intraoperative bleeding. Because of the location of the surgery site, meticulous attention is paid to the draping procedure. The use of a sterile incise drape is advisable.

Surgical Technique

DORSAL APPROACH

With the DIP joint in an extended position, a needle is advanced in a dorsolateral to distomedial direction from 2 cm proximal to the coronary band and 1.5 cm abaxial. Withdrawal of synovial fluid ensures the correct placement of the needle. The joint is distended with gas or with 20 mL Ringer's lactate solution.

The arthroscope portal is prepared with a No. 11 scalpel blade introduced into the joint along the needle, and the needle is withdrawn. Immediately after the incision is prepared, the sleeve and the blunt obturator are carefully advanced together into the joint with a rotating movement. The obturator is subsequently replaced by the arthroscope.

Working with a dorsolateral arthroscope portal allows inspection of a medial fragment and its removal through an instrument portal positioned directly over the fragment (see Figure 90-54). The joint is very small, providing little room for recognition of the anatomic landmarks. The middle phalanx is recognized by its articular cartilage surface and the proximal prominent ridge, which provides access to the dorsal outpouching of the joint. The synovial membrane, located at the dorsal aspect of the joint cavity, is covered with synovial villi, which partially obstruct the view. Often the osteochondral fragment of the extensor process is hidden behind the villi. The surface of the fragment is usually rough and irregular.

The key to identifying the fragment is to recognize the separation between the fragment and the parent portion of the bone. The abaxial aspect of the joint should be inspected initially to provide an overview of the condition of the joint. It is possible that displaced small fragments can be found in these locations.

Most fragments are well attached to the soft tissues. Insertion of an elevator between the fragment and the distal phalanx and careful manipulation loosens most fragments. It is important to disrupt a significant portion of the attachments of the extensor tendon and surrounding tissues from the fragment before attempting to remove the fragment with rongeurs. When good purchase of the fragment is achieved, rotating movements are performed to disrupt the remaining attachments. Because of the softness of the fragment, it is usually removed in pieces.

The fracture bed is inspected and curetted if deemed necessary. Introduction of the arthroscope through the instrument portal allows inspection of the fragment bed from a different view and occasionally the detection of other small fragments. The joint is then flushed and the portals closed with two simple-interrupted sutures of an absorbable monofilament material.

A postoperative pressure bandage is maintained for 2 weeks.[83] The portals for the surgery heal slower than similar portals in other joint regions. Therefore, there is an increased risk of infection. Additionally, intra-articular injections (such as hyaluronan) are delayed for about a week. The horse is kept in a box stall for 4 weeks and can then slowly return to work.

The prognosis depends on the nature of the problem, the age of the patient, and the arthroscopic findings during surgery. In young horses, a good prognosis can usually be given, whereas in older horses with concomitant osteoarthritis, the prognosis for successful return to work decreases.

PALMAR APPROACH

An arthroscopic approach to the palmar aspect of the DIP joint has been described.[82] The horse is positioned in lateral recumbency and the joint is distended through a dorsally placed needle. Observation of the process of joint distention allows identification of the best palmar location to access the joint. A needle is placed into the site to facilitate correct scope positioning. A 5-mm skin incision is made over the lateral/medial aspect of the palmaro/plantaroproximal pouch, axial to the collateral cartilage and the neurovascular bundle and abaxial to the deep digital flexor tendon, and the blunt obturator is directed toward the apex of the frog. The midsagittal ridge of the dorsal articular border of the navicular bone, the entire proximal border of the navicular bone, the medial and lateral aspect of the joint, parts of the collateral sesamoidean ligaments can be evaluated. Osteochondral fragments and navicular cysts can be found.

PALMAR OR PLANTAR DIGITAL NEURECTOMY

General Considerations

Neurectomy is one of the oldest surgical procedures described in horses.[84] Palmar or plantar digital neurectomy is a viable option for alleviating pain in horses with certain kinds of chronic lameness, particularly navicular disease. A thorough clinical examination and proper case selection are critical. For example, digital neurectomy is contraindicated in horses with laminitis or infection of the DIP joint.

Various techniques for digital neurectomy have been described. The operation may be performed with the horse standing or in lateral or dorsal recumbency under general anesthesia.[85,86] With the horse standing, the procedure is carried out quickly, avoiding the risks of general anesthesia. With the horse in dorsal recumbency, repositioning the horse to access both sides of the limb is not required, and a tourniquet is not necessary because minimal bleeding occurs.[86] A nonsteroidal anti-inflammatory drug should be administered postoperatively and the horse should be kept in a box stall for 4 weeks. The horse is then slowly reconditioned by walking for another 4 weeks.

Re-innervation can occur when the severed nerve endings re-establish contact with each other (Figure 90-55). This can happen as early as 6 months postoperatively, depending on the technique used. However, ideally this does not happen for many years.[82,87] Neuritis and neuroma formation are common complications, particularly in the proximal nerve stump; depending on the study, up to 20% of patients are affected. These lesions are very painful, and in selected cases they must be treated with local injections of triamcinolone acetate (5 to 10 mg for each neuroma), sarrapin, or alcohol blocks. Rupture of the DDF tendon and subluxation of the coffin joint can occur after neurectomy, especially if it was compromised before surgery (Figure 90-56). Another sequela is laminitis, which can develop after a second operation or with injury of the blood vessels.

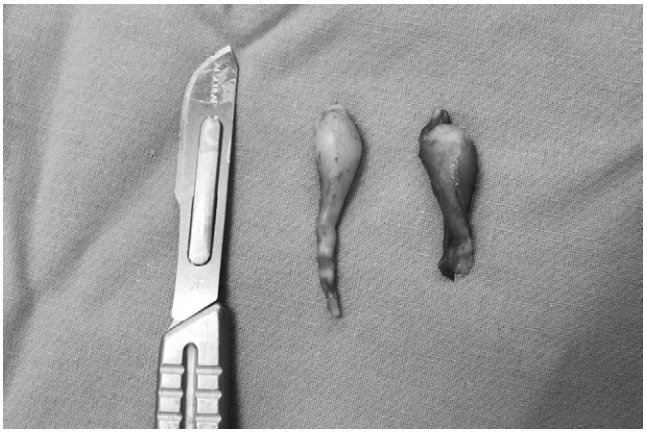

Figure 90-55. Specimens of neuromas excised following re-innervation of a neurectomized digit.

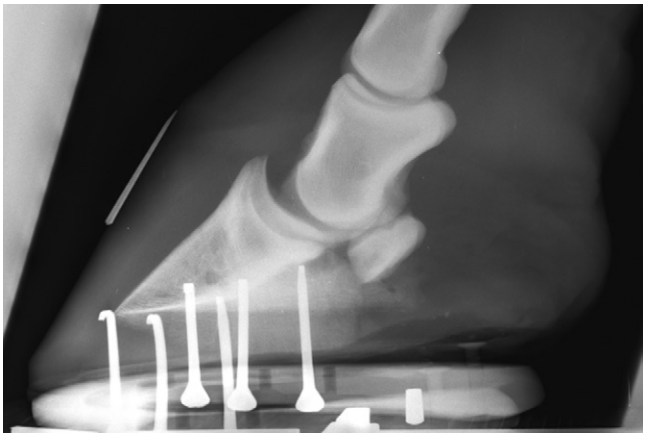

Figure 90-56. Lateromedial radiographic view of a subluxation of the distal interphalangeal joint, which occurred after palmar digital neurectomy.

Special techniques are used to prevent or at least delay regrowth of the severed nerve ends as well as to reduce the risk of neuroma formation. Various methods have been described for cutting the nerve and treating the severed nerve ends. However, the "guillotine technique," meaning simple scalpel transection of the nerve, does not use any form of nerve end treatment and is the most commonly used method.[82,87] This technique can be applied through one large or two small incisions, as described later.

For the sake of completeness, discussion of the methods that have been used to treat severed nerve ends is presented here. Electrocoagulation is a rarely used method. Other methods include epineural capping, inserting the nerve in a hole drilled into the proximal phalanx, or ligating the nerve.[88,89] A valid alternative to digital neurectomy is cryoneurectomy, in which

the nerve is frozen to as low as −30° C transcutaneously.[90] Other alternatives include topical application of chemicals and injection of neurotoxins, such as cobra venom.[91] Carbon dioxide laser treatment of the proximal nerve stump has reduced the development of neuromas.[92]

In certain countries, animal welfare legislation and equine sports associations (e.g., the International Equestrian Federation [FEI]) prohibit horses that have had a digital neurectomy from competing in official events. As a consequence, various methods of detecting a neurectomy have been developed.[93,94]

Palmar Digital Neurectomy in the Pastern Region Using One Skin Incision

The horse is positioned in dorsal recumbency, and the limbs are extended and tied to restraints attached to the ceiling.[86] The surgical field is clipped, prepared for aseptic surgery, and draped. The nerve is palpated, and an approximately 4-cm incision is made in the pastern skin directly over the nerve (Figure 90-57, *A* and *B*).[82] Usually the skin incision is made along the dorsal edge of the DDF tendon, which directly exposes the nerve.

The ligament of the ergot is identified and split longitudinally (see Figure 90-57, *C* and *D*), allowing access to the neurovascular bundle located beneath it. Splitting the ligament provides an extra layer to be closed over the severed nerve and potentially reduces postoperative irritation and subsequent neuroma formation. Additionally, mistakenly transecting the ligament instead of the nerve can be prevented with this technique. The nerve is isolated within the neurovascular bundle and isolated from the artery (see Figure 90-57, *E* and *F*). The neurotomy is initially performed proximally and then distally, followed by removal of the loose piece of nerve (see Figure 90-57, *G*).

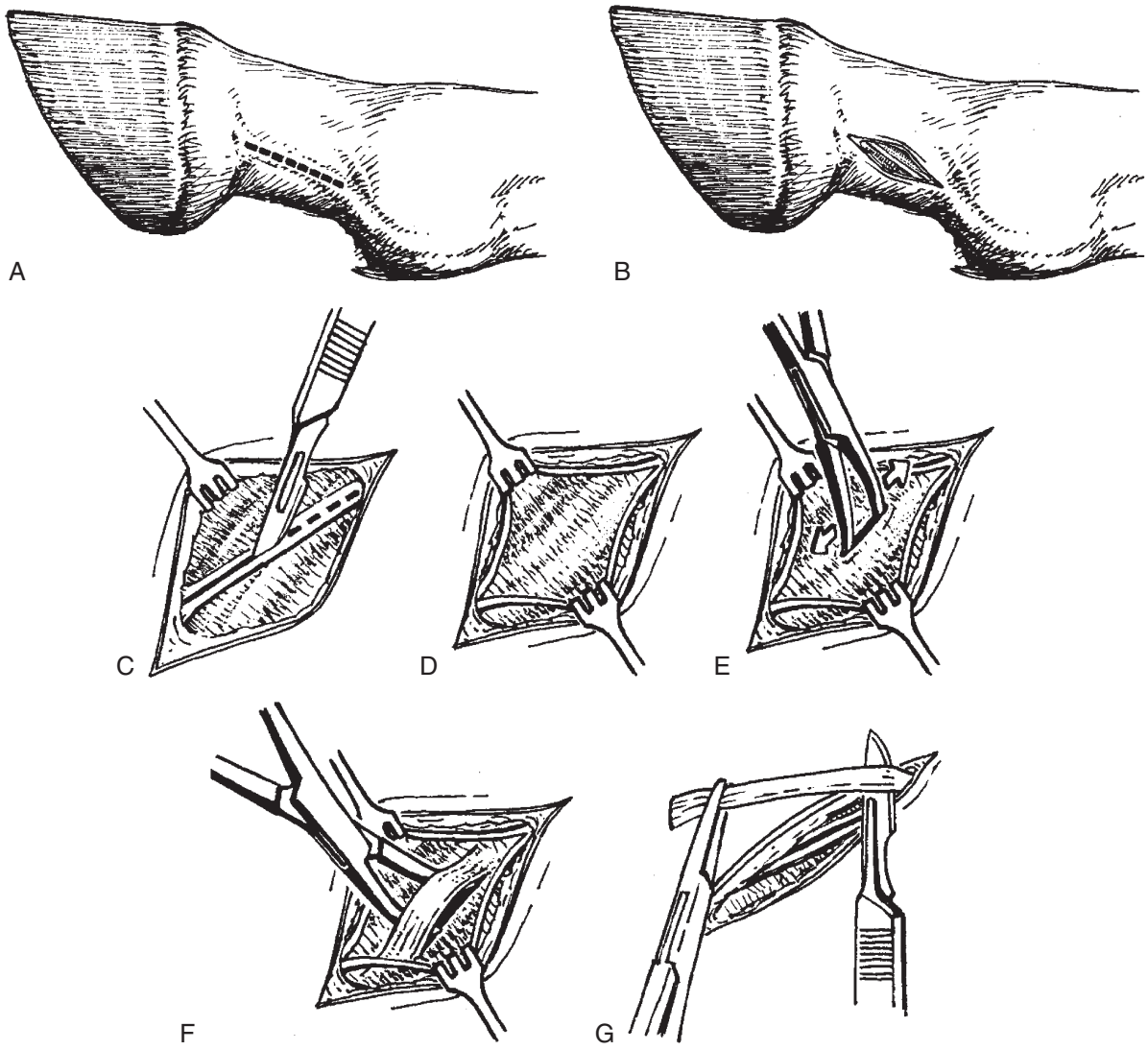

Figure 90-57. Graphic illustration of the palmar digital neurectomy procedure performed in the phalangeal region through one incision. **A,** Location of the skin incision. **B,** The skin incision has been made. The ligament of the ergot is split longitudinally **(C)** and separated **(D),** giving access to the underlying tissues and the neurovascular bundle. The structures of the neurovascular bundle are separated **(E),** and the nerve is isolated **(F). G,** A 2-cm piece of nerve is excised.

The ligament of the ergot is closed in a continuous pattern, and the skin is closed with simple interrupted sutures. Aftercare is the same as that for the method using a small incision, described under "General Considerations."

An alternative and simpler technique involves a small stab incision about 1 to 1.5 cm in length at the mid pastern region. The nerve is isolated palmar to the artery and elevated up to the skin, applying some tension. Subsequently the nerve is transected at the proximal aspect of the incision and, applying considerable tension, the distal transection is performed as far distal as possible. Skin closure is routine in two layers. This technique allows the removal of 2 to 3 cm of nerve.

Palmar Digital Neurectomy in the Pastern Region Using Two Small Incisions—Pull-Through Technique

The location of the nerve is palpated and two approximately 1.5-cm skin incisions are made directly over the nerve (Figure 90-58, *A* and *B*). The distal skin incision is located at the distal end of the pastern region at the transition to the bulb of the heel. The proximal skin incision is selected at the proximal end of the pastern region at the transition to the metacarpophalangeal joint.

The tissue is bluntly dissected longitudinally with a mosquito forceps so that the dorsal and palmar aspects of the nerve are isolated (see Figure 90-58, *C*). It is important that the nerve and artery are well separated. Proximally the nerve might be obscured by the ligament of the ergot, which runs superficial to the nerve at a slightly different angle. The nerve is always covered by fascia, and it can be surprising how deep a dissection is required for its isolation.

When the nerve has been freed proximally and distally, it is alternately pulled at either end to ensure that the correct structure has been exposed. The nerve is pulled up with a mosquito forceps, held with tissue forceps, and cut sharply as far distally and proximally as possible with a No. 15 scalpel blade (see Figure 90-58, *D* and *E*). One clamp is applied to the proximal end and another one to the distal end of the severed nerve segment, and tension is applied to ensure that all attachments are eliminated and the nerve segment can be completely removed (see Figure 90-58, *F* and *G*). The skin incisions are closed with 2-0 nonabsorbable suture material.

The advantages of this technique are the small size of the skin incisions, short time of surgery, and low incidence of neuroma formation.[95]

Repeated Palmar Digital Neurectomy

A second neurectomy might be necessary when re-innervation occurs and the horse becomes lame again. For this procedure, a long incision is usually made so that the nerve can be adequately isolated. This procedure is often more difficult than the initial operation because the nerve is generally very closely attached to the artery.

Small neuromas are always seen in the proximal aspect of the previous surgical field. These nodules can be easily palpated in horses that have had a neurectomy. Sometimes the regrown nerve is surrounded by a large amount of scar tissue, which renders its isolation more difficult. Only the neuroma and part of the distal nerve should be removed. Skin closure and aftercare are the same as for the other neurectomy procedures.

High Lateral and Medial Palmar Neurectomy

The goal of high lateral and medial palmar neurectomy is to disrupt afferent and efferent nerve tracts proximal to the division into palmar and dorsal branches to prevent pain sensation from chronic disease processes innervated by the dorsal branches of the lateral and medial palmar nerve. This is usually performed proximal to the flexor tendon sheath, because locating and removing the nerve is easy in this location (Figure 90-59). Also, there is little relative movement in this area, so that the nerve endings are less traumatized. The operation is carried out with the horse in dorsal recumbency. The skin is incised immediately proximal to the flexor tendon sheath on the lateral and medial aspects of the metacarpus. The nerve is located, isolated, and removed. Aftercare is the same as for the other techniques.

SURGICAL MANAGEMENT OF NAVICULAR SYNDROME

Navicular disease is a degenerative disorder that involves the DSB and its surrounding structures. The etiopathogenesis of the condition is multifactorial, and many theories have been proposed.[96-99] Vascular and mechanical theories continue to be debated as to the primary cause of the disease.

Treatment

There is no actual cure for navicular disease. Its management concentrates on abolishing the symptoms and clinical signs.

Nonsurgical Management

Management strategies include corrective shoeing, NSAIDs, and vasodilators (such as isoxsuprine or metrenperone).[100] Intra-articular administration of hyaluronan with or without corticosteroids into the DIP joint or in the navicular bursa can provide transient relief of clinical signs. The bisphosphonate tiludronate (Sanofi-Aventis, Paris), a drug designed to inactivate osteoclast activity, has demonstrated positive results in the treatment of navicular after disease in horses with lameness of less than 6 months duration.[101,102]

Another novel medical treatment is based on the pathophysiologic mechanism that podotrochlosis is associated with an increase of the intraosseous pressure in the navicular bone. The increase of pressure might be caused by an accumulation of osmotically active substances in the interstitium, as has been described in cases of compartment syndrome. Therapy with benzopyrone is proposed.[103] This drug is used successfully in humans to reduce high-protein edema.[104]

Surgical Management

Surgery is usually reserved for cases of navicular syndrome that have not responded to such conservative treatments as corrective shoeing and medical management. Three surgical procedures are currently available: navicular suspensory desmotomy, palmar digital neurectomy, and periarterial sympathectomy. These surgical procedures should be combined with balancing the feet and applying corrective shoes.

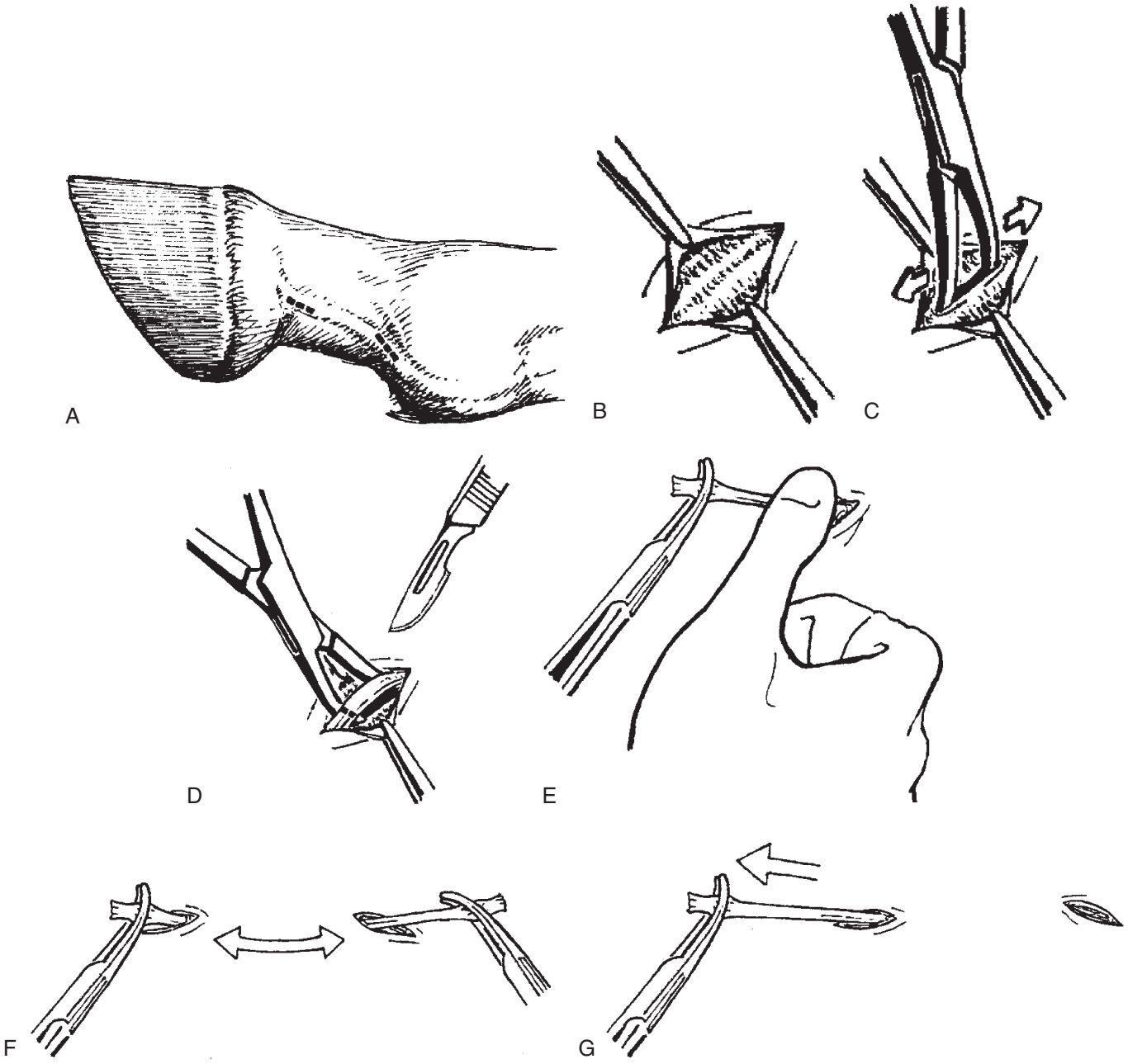

Figure 90-58. Graphic illustration of a palmar digital neurectomy performed through two small incisions. **A,** Location of the two incisions. **B,** The neurovascular bundle is visible through an incision. The nerve is isolated **(C)** and elevated above the incision **(D).** The proximal end is transected **(E),** followed by the distal end **(F),** and the piece of nerve is pulled out of the distal incision **(G).** The incisions are subsequently sutured.

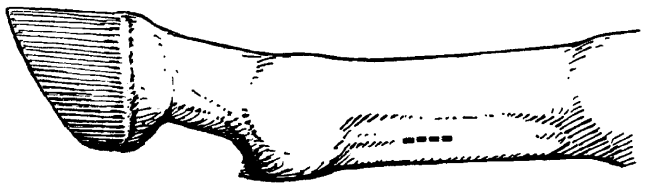

Figure 90-59. Graphic illustration of the location of the surgical site for the high palmar neurectomy.

DESMOTOMY OF THE SUSPENSORY LIGAMENTS

Desmotomy of the suspensory ligaments (DSL) of the DSB has been proposed on the assumption that the syndrome has a mechanical basis. The proposed mechanism of action of the surgery reduces the forces on the navicular bone and perinavicular structures associated with the caudal weight-bearing phase of the stride (the portion of the stride where the suspensory ligaments are loaded).[105-107]

The DSB has its own supporting ligaments: proximally, the collateral sesamoidean ligaments (CSLs) and distally, the impar ligament and the chondrosesamoidean ligaments (Figure 90-60). The suspensory ligaments of the DSB are broad, elastic

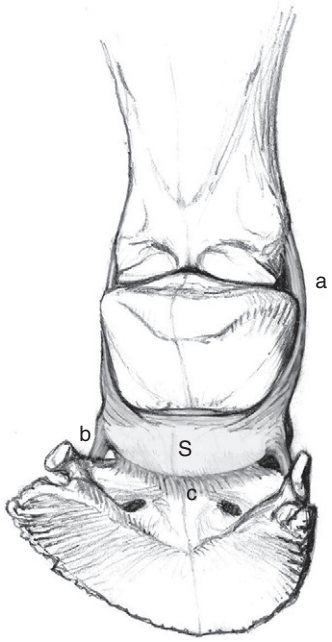

Figure 90-60. Graphic illustration of the supporting ligaments of the distal sesamoid bone (palmaromedial view). *a,* Collateral distal sesamoidean ligament; *b,* chondrosesamoidean ligament; *c,* impar ligament; *S,* distal sesamoid bone.

structures containing an abundance of nerve fibers. They originate at the dorsodistal aspect of the proximal phalanx and insert primarily on the proximal border of the DSB (Figure 90-61, *A*). A branch of each ligament, the chondrosesamoidean ligament, also inserts on the axial surface of the adjacent collateral cartilage and the palmar process of the distal phalanx.

The biomechanics of the DSB and its suspensory apparatus are not completely understood and are explained mainly with the help of theoretical models. It is generally accepted that the CSLs anchor the DSB and prevent its descent during weight bearing.[106] However, the exact biomechanical implications of the CSL during loading are difficult to explain because the ligament passes over two joints, and time and maximum amount of tension depend on the foot or pastern axis and hoof conformation, as well as on the phase of the stride. Nevertheless, it is argued that under maximal extension of the CSL, third-order acceleration (vibratory) forces between the DSB and the deep flexor tendon can develop.[107] Desmotomy of the CSL would remove these vibratory forces, which could potentially damage the DSB and impar ligament with all the associated vasculature.

Surgical transection of the CSL is performed as described in the literature.[97,105] The horse is anesthetized and positioned in dorsal recumbency. An Esmarch bandage is applied as a tourniquet proximal to the metacarpophalangeal joint. A 4-cm vertical incision is made midway between the common digital extensor tendon and the distal eminence of the proximal phalanx, from the level of this eminence to the coronary band (see Figure 90-61, *B*). The incision continues deeper through the subcutaneous fascia to permit identification and isolation of the coronary plexus (see Figure 90-61, *C*) on the proximal margin of the collateral cartilage. Dissection axial to the cartilage allows identification of the dorsal margin of the CSL. A mosquito forceps is passed in the dorsopalmar direction under the CSL to elevate the ligament, and a No. 11 scalpel blade is used to transect it (see Figure 90-61, *D*). The subcutaneous tissues are closed with a simple-continuous suture pattern using 2-0 absorbable suture material. Skin closure is routine. The opposite CSL in the same limb and the ligaments of the opposite limb are transected applying identical technique.

Postoperative care includes protective bandages for 10 days until the sutures or staples are removed. Hand-walking exercise should be initiated soon after surgery and is continued for 3 weeks. Thereafter, a gradually increasing exercise program is implemented with the goal of achieving full work levels by 3 months after surgery.[97] Postoperative complications are rare but include wound dehiscence, infection, local swelling, and postoperative scarring. Recently, an arthroscopic approach for transecting the CSL has been evaluated in an experimental study.[108]

Preliminary results from the United Kingdom were encouraging: 13 of 16 horses treated with CSL desmotomy were able to work without lameness.[105] In the same year, another study revealed clinical improvement in 50% of 57 horses.[109] Two additional studies reported success rates of 15 of 21 horses and 12 of 17 horses, with the horses in the second group being sound at least 6 months after surgery.[110,111] In a review of 118 horses that had navicular syndrome and that were treated with CSL desmotomy, 76% were sound at 6 months, and 43% were sound after 36 months.[112]

All of the following conditions were associated with a diminished response: flexor cortex defects, proximal border enthesiophytes, mineralization of the DDF tendon, and medullary sclerosis of the DSB. Horses with more than 1 year's duration of clinical signs had a poor prognosis. The procedure is not recommended in horses with inflammatory disease of the DIP joint and the navicular bursa, but horses with new bone growth at the site of insertion of the CSLs (enthesiophytes) are considered excellent candidates for this surgery. The procedure should be attempted early in the course of the syndrome.[113]

It was originally stated that desmotomy of the CSL should modify the biomechanical forces acting on the DSB, but clinical improvement could also be the result of transection of the sensory fibers that course within the suspensory ligaments. It seems that the procedure is more effective in improving clinical signs on a short-term basis (6 to 12 months), and the lameness can recur later on because of re-innervation of the sensory fibers in the CSL.

PALMAR DIGITAL NEURECTOMY

The most commonly performed surgical technique to abolish lameness associated with the navicular syndrome is palmar digital neurectomy (see earlier).

PERIVASCULAR SYMPATHECTOMY AND FASCIOLYSIS

Perivascular sympathectomy and fasciolysis around the lateral and medial digital artery and vein have been described for the treatment of navicular syndrome.[114] A 10-cm skin incision is centered over the lateral and medial proximal sesamoid bones. The subcutaneous tissues and fascia are carefully transected as well and the neurovascular bundle is identified. The artery and vein are isolated from their perivascular tissues and adventitia over the entire length of the surgical site. The nerve is isolated from its surroundings as well. Inadvertent trauma to the vessels

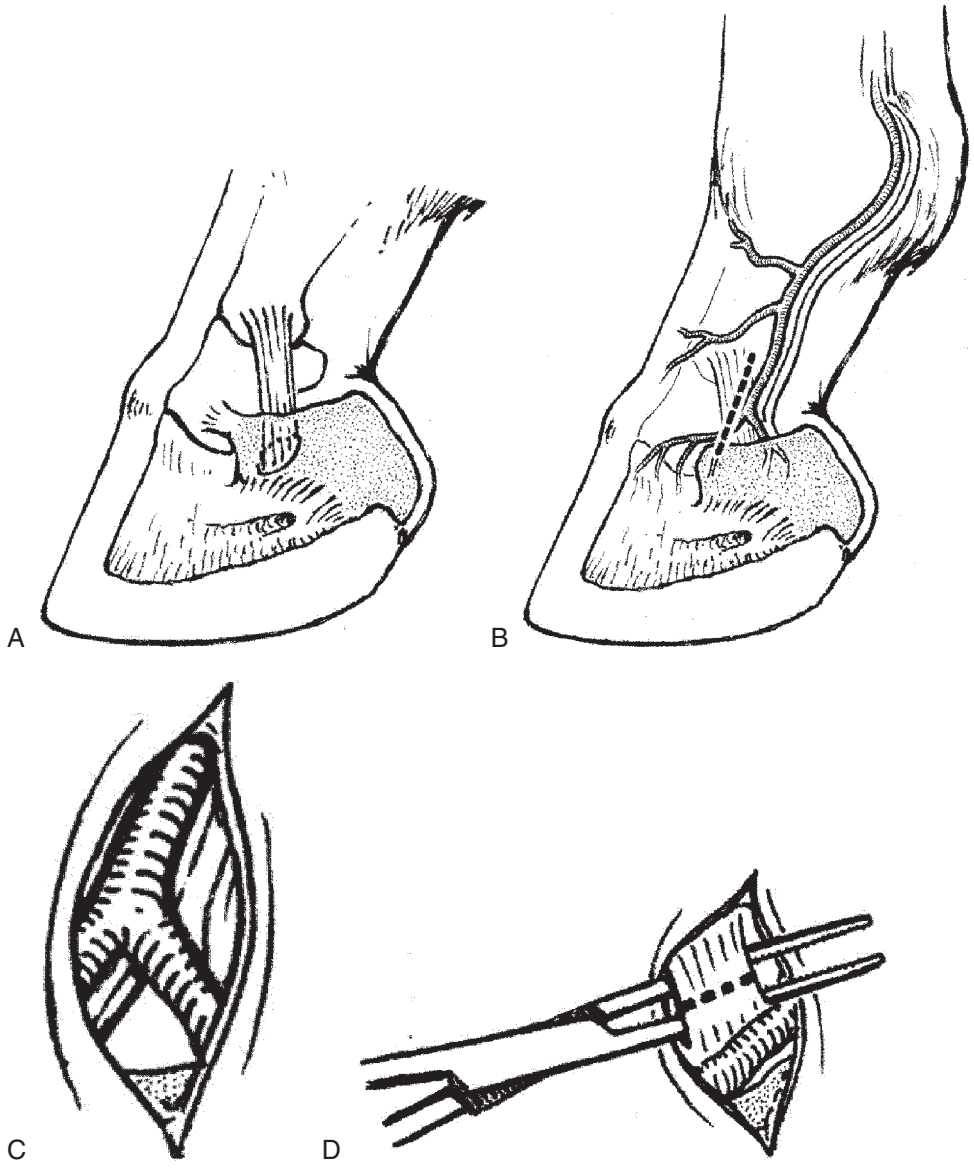

Figure 90-61. Graphic illustration of the desmotomy of the collateral distal sesamoidean ligament. **A,** Overview of the anatomic structures. **B,** Location of the skin incision over the ligament. **C,** After the skin is incised, the ligament can be seen underneath the superficial vessel. **D,** The ligament is isolated, elevated above the skin incision, and transected.

and the nerve should be avoided at all costs. Through this dissection the sympathetic nerve supply of the vessels is stripped, which results in a prolonged vasodilation. The subcutaneous tissues and the skin are closed in routine fashion.[115]

A significant increase in skin temperature caused by increased blood circulation in the region could be demonstrated at 8 and 20 weeks postoperatively with the help of thermography in 30 horses.[114] In each horse, one forelimb was operated on, whereas the other served as a control. A significant increase in hoof horn growth could be seen in the experimental limb compared to the control limb. In a review of 79 horses that had navicular syndrome and that were treated with perivascular sympathectomy and fasciolysis, 73% were sound and returned to the intended use. The mean duration of follow-up was 23.6 months (range, 3 to 72 months).[114]

SURGICAL MANAGEMENT OF LAMINITIS

Laminitis is characterized by a breakdown of the connective tissue suspensory apparatus of the distal phalanx inside the hoof wall. As a result of lamellar pathology, the distal phalanx is no longer supported by the laminar attachment and starts to displace. As soon as the first signs of displacement are recognized, the disease becomes chronic.

There is a direct relationship between the degree and speed of movement of the distal phalanx away from the hoof capsule and the severity of damage to the laminae at the initial insult. Therefore, the most useful diagnostic and monitoring aids in the course of the disease are high-quality radiographs in both lateromedial and dorsopalmar (horizontal) projections.

Clinical Signs

Clinical signs, severity of damage, and response to therapy vary among individual horses, but it is widely accepted that the single most important factor influencing the final outcome of equine laminitis is the severity and extent of the initial damage to the internal anatomy of the foot.

Diagnosis

Clinical signs are very typical, and the radiographic examination further helps to diagnose this condition.

Treatment

Treatment of laminitis consists of dietary management, medical treatment, soft bedding, and hoof care. In all phases of the hoof care, corrective shoeing and trimming should aim at reducing stress to the damaged lamellae by minimizing the distracting forces affecting the displacement of the distal phalanx (rotation or sinking). DDF tenotomy is a salvage procedure for horses with chronic refractory laminitis accompanied by rotation of the distal phalanx and persistent pain. However, it is reported that even foundered horses with persistent draining tracts caused by osteomyelitis and excessive heel growth respond favorably to the procedure.[116]

Tenotomy of the Deep Digital Flexor Tendon

The rationale for tenotomy of the DDF tendon is based on the biomechanical forces in the foot. The procedure is performed to reduce the palmarly directed pulling forces of the DDF tendon on the distal phalanx and subsequently decrease the shearing stresses on the lamellae of the dorsal aspect of the hoof capsule. It also serves to reduce the pressure of the apex of the distal phalanx on the corium of the sole. Tenotomy of the DDF tendon permits lowering the heels to allow a more normal alignment (derotation) of the distal phalanx using orthopedic shoeing.[115]

DDF tenotomy can be performed at the midmetacarpal or the pastern area of the limb. Both procedures are equally effective, but tenotomy at the level of the midmetacarpus is preferred for various reasons. It is easier to perform and can be performed in a standing horse, the digital tendon sheath is not invaded, it bears less risk of postsurgical infection, and it leaves some support to the DIP joint through fascial attachments. Pastern region DDF tenotomy must be performed under general anesthesia and should be reserved for cases that require a second tenotomy.

Before either surgery, horses should be shod with a heel extension to stabilize the foot and help prevent postoperative hyperextension or subluxation of the DIP joint.

MIDMETACARPAL APPROACH

The hair is clipped circumferentially from the level of the metacarpophalangeal joint to the carpus. The horse is sedated, and local anesthesia is achieved through a high palmar ring block.

After aseptic preparation of the limb and appropriate draping, a vertical incision through the skin, subcutaneous tissue, and paratenon is made directly over the lateral aspect of the DDF tendon, centered at the junction of the proximal and middle third of the MCIII (Figure 90-62, *A*). With the help of curved Kelly forceps, the DDF tendon is separated from the neurovascular bundle (see Figure 90-62, *B*), the accessory ligament, and the superficial digital flexor (SDF) tendon. The DDF tendon is elevated out of the incision (see Figure 90-62, *B*). During this part of the procedure, an assistant should lift the limb off the ground to relieve the tension on the DDF tendon. Care must be taken to avoid elevating the neurovascular bundle located medially to prevent its inadvertent transection together with the tendon. The elevated tendon is subsequently transected with the scalpel blade. An immediate separation of the ends of 1 to 3 cm is usually noted after complete transection of the tendon.

An alternative tenotomy technique involves the blind transection of the DDF tendon with the help of a blunt bistoury while the animal is weight bearing.[117] Concomitant transection of the medially located neurovascular bundle can occur with this technique.

The subcutaneous tissue is closed with an absorbable monofilament suture material in a simple-continuous pattern. The skin is closed with stainless steel staples.

PASTERN APPROACH

DDF tenotomy is performed with the horse in lateral recumbency under general anesthesia.[118] A 3-cm vertical skin incision is made along the palmar midline of the pastern region, 1 cm proximal to the bulb of the heel (Figure 90-63, *A*). The skin, subcutaneous tissue, and sheath of the DDF tendon are incised, and the DDF tendon is exposed, elevated, and transected (see Figure 90-63, *B* and *C*). The amount of separation of the tendon ends is greater after tenotomy at this level (6 to 10 cm) because there are no attachments to the distal tendon other than the insertion site to the distal phalanx.[117] Closure of the tendon sheath, subcutaneous tissue, and skin is routine.

Postoperative Management

Bandages should be maintained for a minimum of 6 weeks to minimize swelling and prevent contamination of the surgery site. The horse is administered phenylbutazone for pain as needed and confined to the stall, wearing a heel extension shoe for 6 to 8 weeks. For midmetacarpal tenotomy, this type of shoe seems to be less necessary.[116]

A mild degree of hyperextension of the DIP joint occurs, but it is usually self-limiting.[113] Most horses show an initial improvement within 2 to 3 days of surgery. After tenotomy in either location, flexor support for the distal phalanx should develop through attachments of the distal tendon end by 6 to 8 weeks after surgery, and maintenance of a normal hoof–pastern axis without extended heels should be possible.[118] Tension relief after tenotomy appears to last for several months.[117]

Complications directly related to the surgery are rare but can include incision infection and postoperative pain. Chronic pain can result from overloading the SDF tendon before healing, osteoarthritis of the DIP joint, or chronic infection of the digit.[117] A flexural deformity of the metacarpophalangeal joint (dorsiflexion) can develop from chronic pain, resulting in an inability to bear weight on the limb and contracture of scar tissue at the tenotomy site.

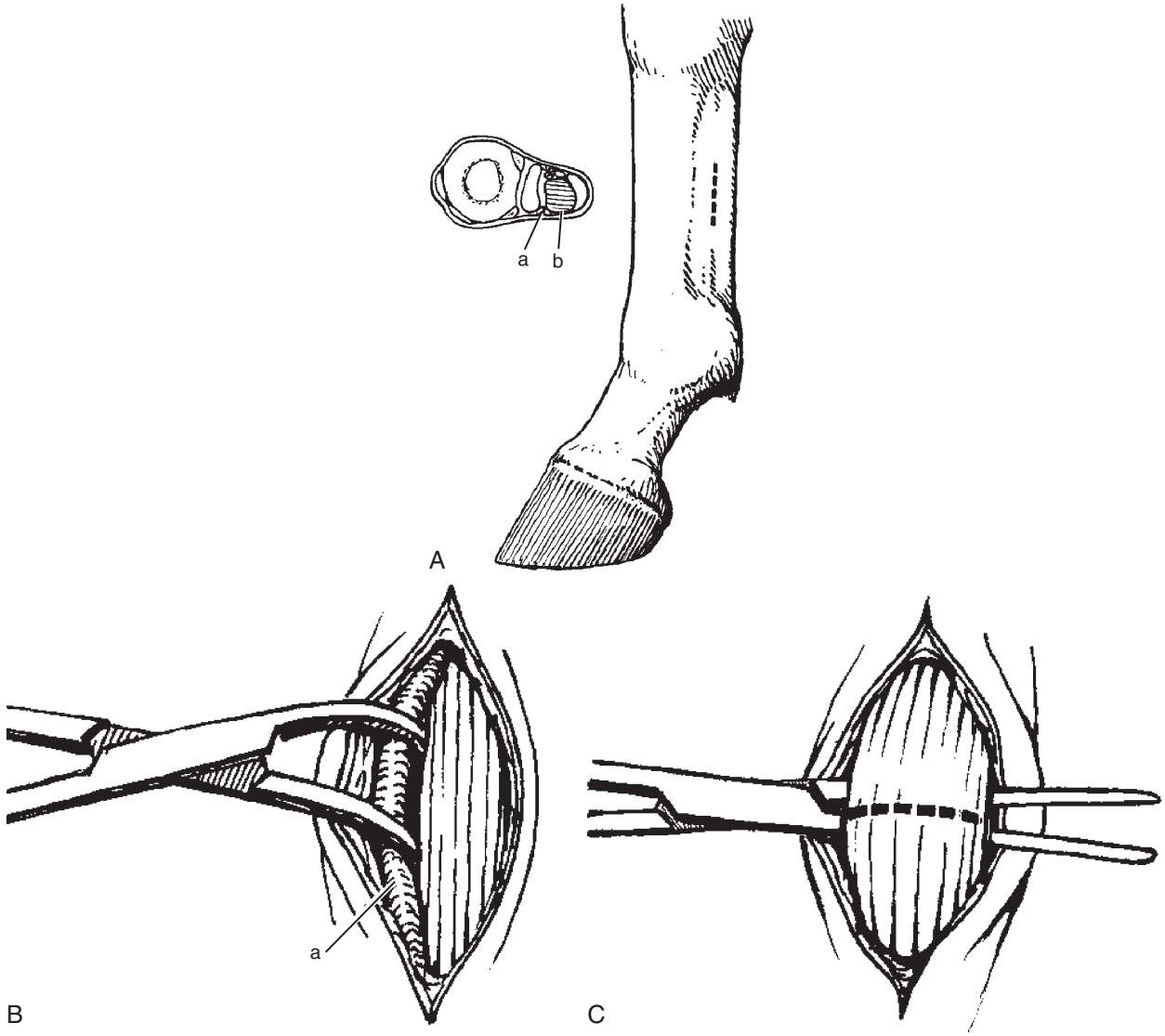

Figure 90-62. Graphic illustration of the deep digital flexor tenotomy in the mid-metacarpal region. **A,** Location of the surgical site on the lateral aspect of the limb. **B,** The deep digital flexor tendon is separated from the neurovascular bundle. **C,** The isolated deep digital flexor tendon is elevated above the incision and transected. *a,* Neurovascular bundle; *b,* deep digital flexor tendon.

Prognosis

In a study of 13 horses with chronic laminitis and 12 to 36 degrees of rotation of the distal phalanx from the dorsal hoof wall, the first clinical improvement was noted by the third or fourth day after midpastern tenotomy of the DDF tendon.[118] At the end of 2 weeks after surgery, the horses continued to improve and were willing to walk without the benefit of nerve blocks. Eventually, five horses could be lightly ridden, seven horses were pasture sound, and one horse was euthanized.[118] In another report of DDF tendon transection at the midmetacarpal level in 20 horses with severe acute or chronic laminitis unresponsive to conventional treatment, 11 patients survived less than 1 month after surgery, and six horses survived longer than 6 months. Three horses remained lame, and no horse returned to athletic performance.[119]

One study evaluating the effect of DDF tenotomy as a treatment for chronic laminitis in 35 horses (midmetacarpal, 30 horses; midpastern, 5 horses) found that 27 horses (77%) were alive 6 months after surgery, and 19 of 32 (59%) horses were alive for at least 2 years.[120] Body weight at the time of surgery and the degree of distal phalangeal rotation had no effect on the 2-year survival. Of the horses in this study, 10 became sound enough for light riding, and there was no correlation between the Obel grade of lameness, the degree of rotation, and the ability to be ridden.[119]

Another study evaluating the effect of DDF tenotomy in the midcarpal region of nine horses with severe laminitis associated with complications, such as intense pain, rotation more than 15 degrees, perforation of the sole, or evidence of infection of the sole or distal phalanx found an initial survival rate of 100%.[121] Six of the nine horses survived more than 21 months, and the other three were convalescing at the time the report was published. Four of the nine horses could be used for pleasure riding.

Although the recent studies report surprisingly good results, it should not be forgotten that these horses require prolonged

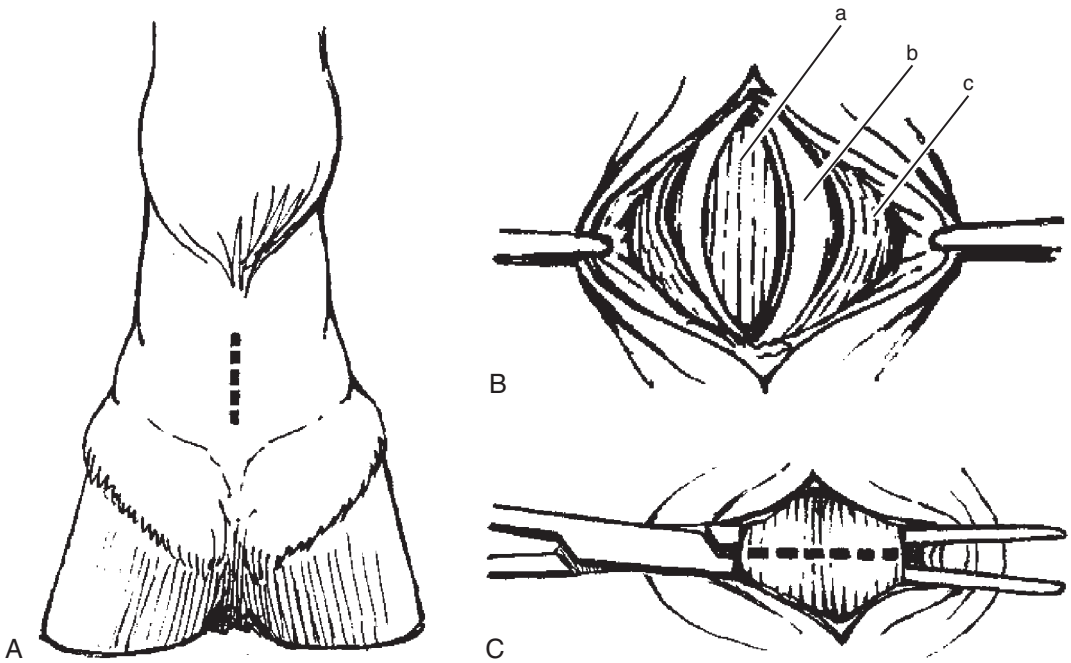

Figure 90-63. Graphic illustration of the deep digital flexor tenotomy in the mid-pastern region. **A,** Location of the surgical site. **B,** The skin, subcutaneous tissues *(c),* and tendon sheath *(b)* are sharply transected. **C,** The deep digital flexor tendon *(a)* is elevated above the incision and sharply transected.

and expensive supportive care and often additional surgical interventions. They suffer months of crippling foot pain and recumbency, and everyone involved must be dedicated to the patient.

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CHAPTER
91

Phalanges and the Metacarpophalangeal and Metatarsophalangeal Joints

Alan J. Nixon

MIDDLE PHALANX

Anatomy

The middle phalanx is a relatively compact bone that is loaded predominantly in axial and torsional planes during athletic activity. It articulates with the distal phalanx through a high-motion joint and proximally with the proximal phalanx through a relatively low-motion joint. The middle phalanx is enclosed distally within the confines of the hoof and coronary band, but it is more exposed proximally, particularly over the palmar and plantar eminences.

The middle phalanx is bounded by multiple ligaments and tendons. The common (or long) digital extensor tendons dorsally and the digital flexor tendons palmarly (or plantarly) contribute to motion of the proximal and distal interphalangeal (PIP and DIP) joints and provide resistance to overextension of the distal limb. In addition, the middle phalanx is stabilized by strong lateral and medial collateral ligaments of the proximal and distal interphalangeal joints and the suspensory ligament of the distal sesamoid bone. Additional perimeter support to the distal aspect of the middle phalanx is derived from the surrounding hoof capsule.

Etiology of Fractures

Fractures of the middle phalanx are most common in Quarter Horses and Arabians but occur in all breeds.[1-3] Horses that turn predominantly on their hindlimbs, such as cutting and reining horses, incur comminuted fractures of the middle phalanx almost exclusively in the hindlimbs. More than 70% of middle phalanx fractures in one report involved the hindlimb.[1] Similarly, distraction fractures of the plantar eminences occur primarily in the hindlimbs because of the forces incurred during sudden stops and turns, most likely as an avulsion of the bony insertion of the superficial digital flexor (SDF) tendons.

Clinical Signs and Diagnosis

Lameness is acute and severe. Comminuted fractures generally result in non–weight-bearing-lameness and attempts to bear weight often result in hyperextension of the digits and sinking of the metacarpophalangeal (MCP) joint. More inherently stable fractures, such as palmar/plantar process fractures, and axial fractures improve within 24 hours and allow weight bearing. Diagnosis is confirmed by radiographic examination. Computed tomography (CT) is often used to better define fracture planes in comminuted fractures.

Fracture Configurations

Middle phalangeal fractures are classified as simple or comminuted and fall into one of four categories: 1) dorsal or palmar/plantar intra-articular osteochondral chip fractures, 2) palmar or plantar eminence fractures, 3) axial fractures, or 4) comminuted fractures.[1]

Osteochondral Fractures

Dorsal or palmar/plantar osteochondral fractures are rare.[2-5] Most osteochondral fractures occur on the palmar or plantar aspect of the middle phalanx, either immediately lateral or medial to the axial midline (Figure 91-1). These fractures do not involve the insertion of the SDF tendon or distal sesamoidean ligaments and therefore do not distract. The fragments do tend to grow *in situ*, which increases the likelihood of lameness. Despite the size and intra-articular location of these fragments, development of osteoarthritis is relatively slow. Nevertheless, they have been reported as a cause of lameness, and surgical removal is recommended if a lameness examination confirms the mid-phalangeal region as the site of pain. They can be incidental findings.

Surgical removal of palmar or plantar fragments using arthroscopy is recommended,[6] although open approaches through the digital sheath are described. The arthroscopic portal is made into the proximal aspect of the voluminous palmar pouch of the PIP joint (Figure 91-2). Access for instruments is provided by a second portal 2 cm distad to the arthroscope, and often an additional portal on the contralateral side of the palmar region of the pastern, especially when removing large

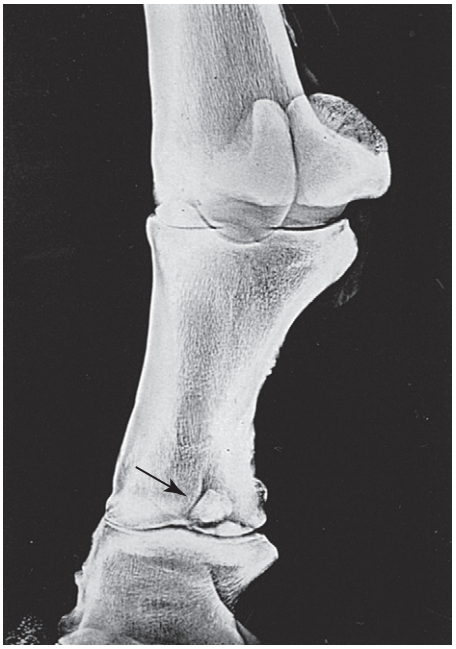


Figure 91-1. Oblique radiographic view of a plantar osteochondral fracture of the proximal margin of the middle phalanx *(arrow)*. Arthroscopic access can be used to remove the fragments that cause lameness.

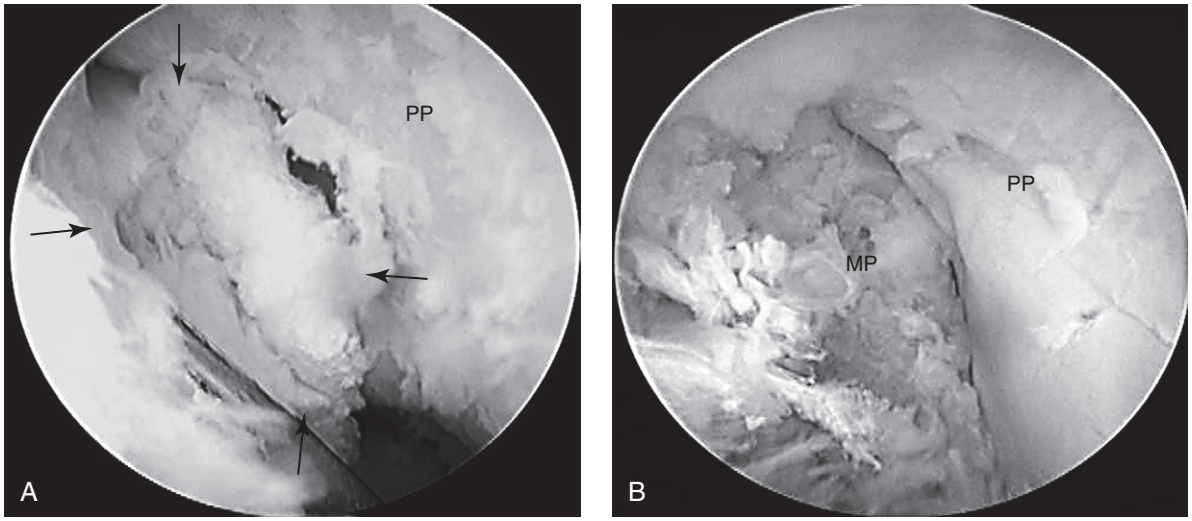


Figure 91-2. A, Arthroscopic appearance of a middle phalangeal palmar fragment *(arrows).* Rongeurs have been introduced through an ipsilateral portal adjacent to the palmar condylar surface of the proximal phalanx (PP). **B,** After removal of the fracture fragment, the fracture bed on the proximal aspect of the middle phalanx (MP) is evident.

palmar fragments. Fragment dissection and removal can be difficult. Fragments located more abaxial to the midline are obscured by the middle scutum and may need removal by open approaches.

Dorsal fragments also can be removed arthroscopically with an approach over the dorsal joint pouch.[5,7] Most fragments develop lateral or medial to the midline and tend to be small and in close proximity to the dorsal rim of the middle phalanx (Figure 91-3). Limited maneuverability results from the attachment of the extensor tendon immediately distal to the joint. Arthroscope entry is usually abaxial to the extensor tendon. Instrument access is adjacent to the fragment, and careful dissection is required in the tight dorsal recess of the PIP joint.

The prognosis after surgical removal of dorsal and palmar fragments is favorable, unless the fragmentation is associated with early joint degeneration, which can develop relatively quickly after the initial chip fracture.

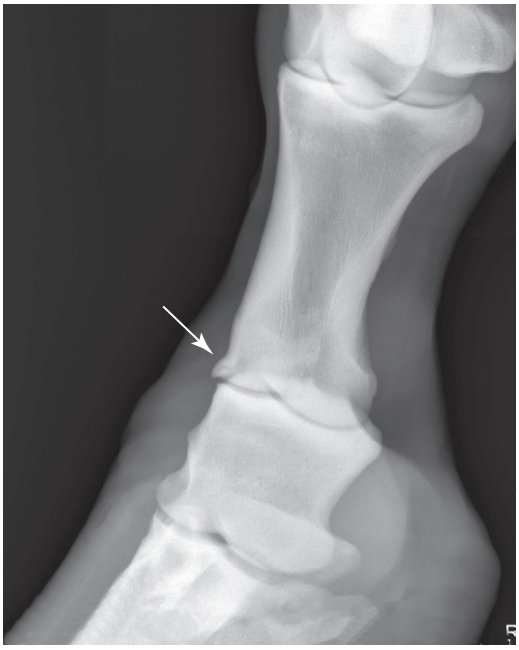


Figure 91-3. Oblique radiographic view of a dorsal osteochondral fragment in the PIP joint *(arrow).*

Palmar or Plantar Eminence Fractures

Fractures of the palmar or plantar eminence of the middle phalanx can be uniaxial or biaxial.[3] The treatment and prognosis vary considerably depending on this configuration. These fractures result from hyperextension of the pastern joint, with tension on the palmar or plantar attachments of the SDF tendon, or the palmar/plantar middle scutum and distal sesamoidean ligaments. Uniaxial fracture does not result in pastern subluxation or the considerable lameness associated with biaxial fracture. Occasionally, fracture fragments do not involve the entire eminence. Rarely, the palmar soft-tissue structures are disrupted without fracture of the bony insertion (Figure 91-4).[8] Radiographs verify the type and extent of the fracture.

UNIAXIAL FRACTURES OF THE PALMER OR PLANTAR EMINENCES

Uniaxial palmar or plantar eminence fractures (Figure 91-5, *A*) can be treated with selective screw fixation or pastern arthrodesis, depending on the duration and configuration of the fracture.[9] Screw fixation in lag fashion can be performed through stab incisions over the proximal extremity of the fractured eminence, with the screws inserted under radiographic control (see Figure 91-5, *B* and *C*).

Fracture reduction can be difficult depending on the duration of the fracture. The interfragmentary screw is inserted in a distal axial direction, with the primary access to the eminence gained by splitting the insertion of the SDF tendon longitudinally. A delay in presentation or the presence of biaxial palmar eminence fractures necessitates pastern arthrodesis. Even with stable articulations and uniaxial eminence fracture, arthrodesis can be preferable and is best performed using a combination of plates and screws (see Chapter 81).[10-13] Cortex screws that are 5.5 mm in diameter provide more rigid fixation than 4.5-mm screws. A

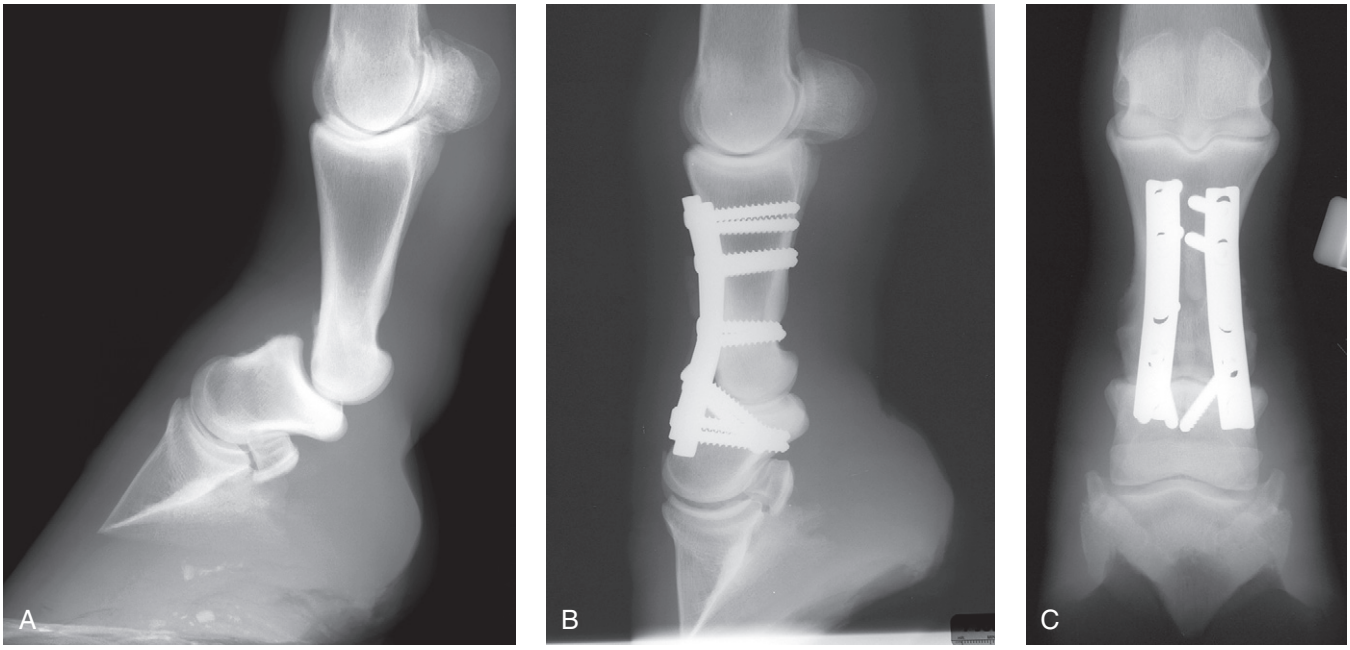


Figure 91-4. Palmar luxation associated with distruption of the palmar insertion of the middle scutum and SDF tendon. **A,** Preoperative lateral radiograph. **B,** Postoperative lateral projection showing reduction and application of two narrow 4.5 mm DCPs with four screws in the middle phalanx. **C,** Dorsoplantar projection showing plate position and distal screws angled to engage the middle phalanx.

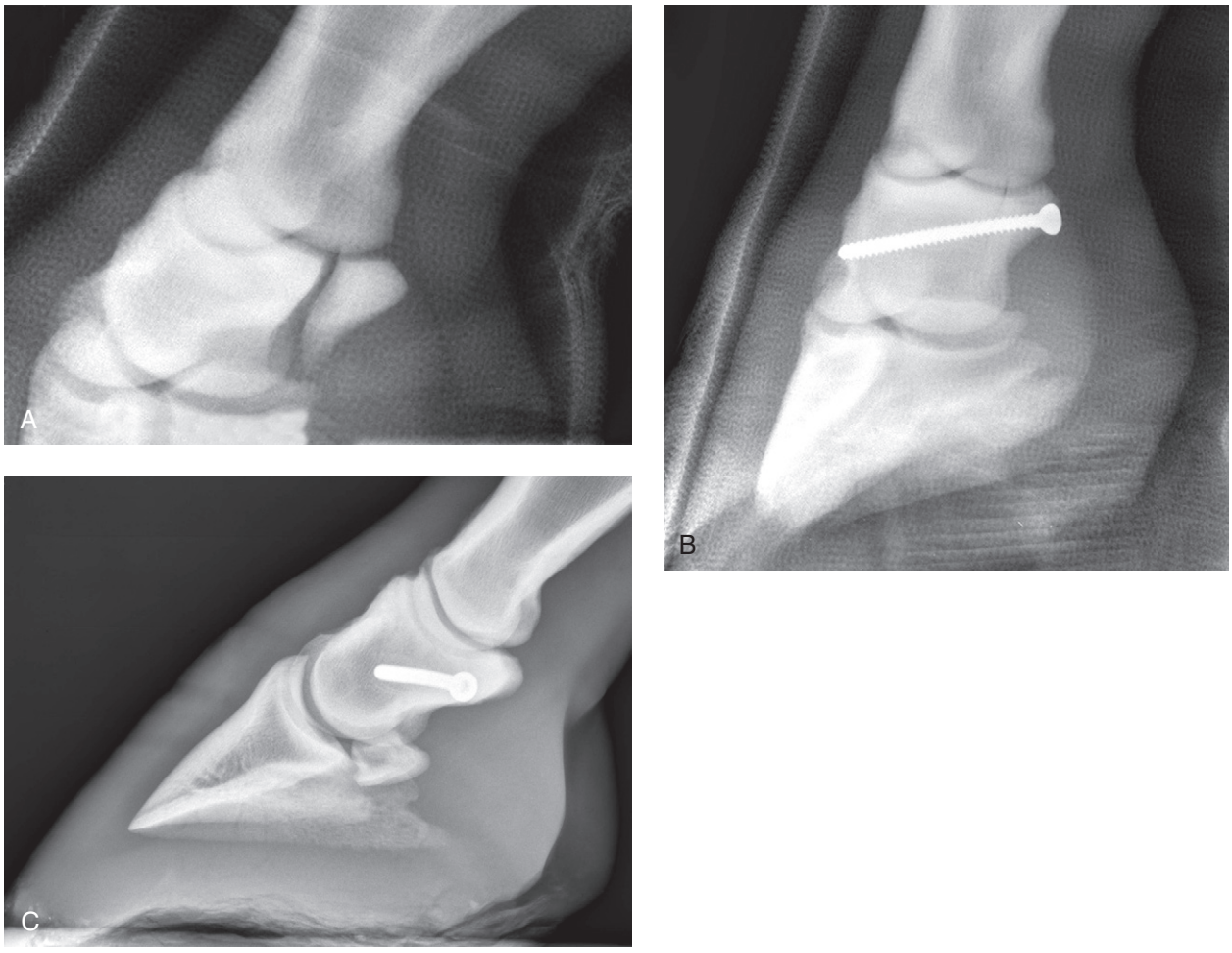


Figure 91-5. A, Oblique radiographic projection of a unilateral palmar eminence fracture of the middle phalanx. The limb is supported in a fiberglass cast for the transport to the clinic. **B,** Postoperative oblique radiographic projection following insertion of a 4.5-mm cortex screw across the fracture, providing interfragmentary compression. The limb is supported in a fiberglass cast. **C,** Lateromedial radiographic projection of the healed fracture at 5½-month follow-up. No arthritic changes are visible and the animal was sound at a walk and trot. (Courtesy J. Auer, Zurich, Switzerland.)

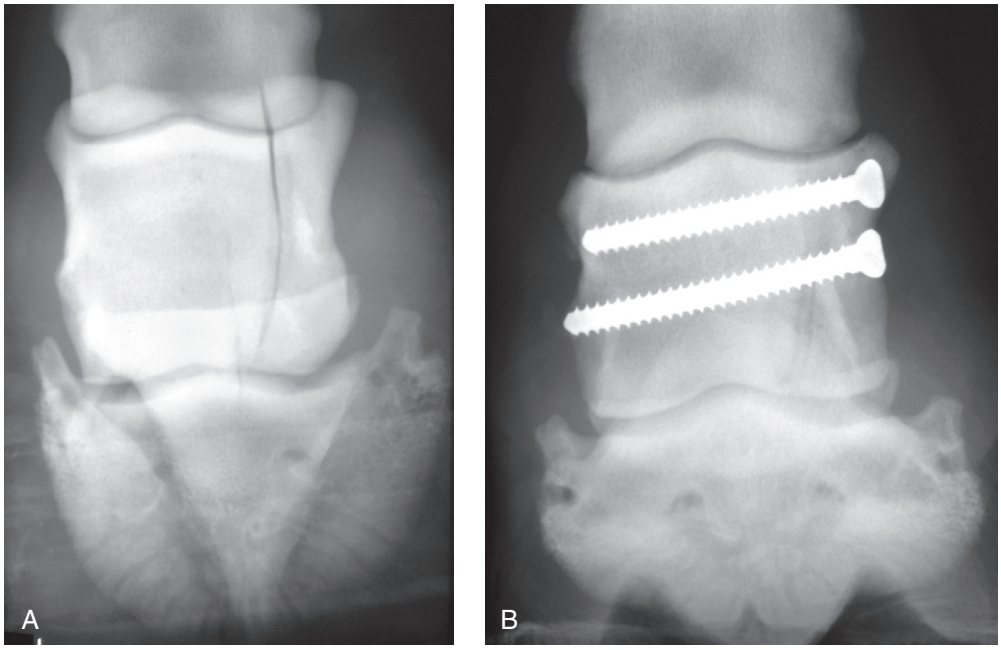


Figure 91-6. A, Dorsopalmar projection of a rarely occurring axial middle phalanx fracture. **B,** Dorsopalmar postoperative radiographic projection after implantation of two 5.5-mm cortex screws using lag technique. (Courtesy C. Lischer, Free University of Berlin.)

bone graft is not usually necessary, although forage of the subchondral bone of the proximal and the middle phalanx is used to promote vascular invasion and access to bone-forming cells.

A postoperative cast is usually maintained for 10 days to 2 weeks. The prognosis after arthrodesis for uniaxial fractures is fair to favorable.[1,11,14] The outlook for return to active work is improved with fractures of hindlimb versus forelimb eminences. Recuperative periods of up to 12 months are required, which can be shortened by the use of plate fixation for pastern arthrodesis.

BIAXIAL FRACTURES OF THE PALMER OR PLANTAR EMINENCES

Biaxial fractures of the palmar or plantar eminences result in considerable lameness and instability of the PIP joint. Pastern arthrodesis is preferred by dorsal application of a locking compression plate (LCP) or, if one is not available, a dynamic compression plate (DCP) application.[15,16] Plate configurations include a single broad LCP or two narrow LCPs placed on the dorsal aspect of the proximal and middle phalanges.[14,17] The palmar or plantar eminence fractures are stabilized by screw fixation placed in lag fashion through the distal plate screw holes. This will require additionally bending an LCP if it is used in this repair. If space is available (in larger fragments), individual interfragmentary screws unassociated with the plate should be added to strengthen the repair. Pastern arthrodesis by plate fixation without attempted perfect reduction of the individual eminence fractures is satisfactory; however, callus production and induced secondary lameness are minimized if the eminence fragments can be incorporated into the repair.

Prognosis after biaxial eminence fracture repair by arthrodesis is fair, particularly for fractures involving the hindlimbs.[1,11] For additional information on pastern arthrodesis, see Chapter 81.

Axial Fractures

Simple axial fractures of the middle phalanx occur rarely (Figure 91-6). These fractures can be repaired with cortex screws applied in lag fashion. The fracture configuration dictates the approach. Adequate reduction is confirmed radiographically before placement of the first screw. Cast fixation during the initial 4 postoperative weeks is important. The prognosis for soundness is guarded because some proliferative new bone formation, especially at the articular margins, is expected as a result of the initial trauma.

Comminuted Middle Phalanx Fractures

Comminuted fractures are the most common configuration encountered in the middle phalanx.[1,3] Clinical appearance includes severe non–weight-bearing lameness with palpable instability and, occasionally, crepitus of the distal limb. The configuration of the fracture is confirmed with radiography, and the treatment is selected based on the extent of the fracture and possibility of adequate repair. The initial and most important decision to be made is whether to pursue treatment or elect euthanasia. Treatment options include screw fixation in lag fashion of minimally comminuted fractures, application of a single broad LCP or DCP with additional screws, use of two narrow LCPs or DCPs for fracture fixation and pastern arthrodesis, or application of strategic cortex screws applied in lag fashion in combination with a transfixation cast.

An extensive series of preoperative radiographs is required to establish the extent of comminution. Most comminuted fractures involve proximal portions of the phalanx, and many extend into the DIP joint, reducing the prognosis. A CT scan is particularly helpful in defining multiple fracture planes entering

the DIP joint. Most are more complex than suggested by plain radiographs.

The aim of surgical repair is reconstruction of the distal, and to some extent the proximal, articular surfaces of the middle phalanx. If reconstruction of the DIP joint is inadequate or the bone is severely comminuted and unlikely to be adequately repaired with a plate and screws, humane destruction should be considered.

The most frequent configuration of comminuted fractures involves fractures in both a sagittal and frontal (transverse) plane, often with added comminution on the palmar or plantar proximal aspect. Marked instability because of connection of the attachments of the palmar or plantar ligaments and tendinous structures is common. Rarely, the distal sesamoid bone is also fractured. Treatment for most comminuted middle phalanx fractures involves application of two narrow LCPs or DCPs, although use of a single broad DCP and a T-plate have been described.[1,3,15,16] The use of a T-plate is discouraged because of the biomechanical weakness of this thin plate combined with the fact that the plates are applied on the compression side of the phalanges. Use of LCPs for comminuted fractures tends to limit screw insertion angle when using a locking screw in the distal plate hole, and DCPs are often preferred. However, the LCP can be used with cortex screws only, where it will act as a limited-contact DCP (LC-DCP).

Plate fixation in combination with independent screws placed in lag fashion to achieve pastern arthrodesis optimizes the chances for adequate reconstruction of the distal articular surface of the middle phalanx. This region is poorly visualized at surgery and accurate reconstruction, to avoid later osteoarthritis of the DIP joint, relies on precise alignment of the proximal portion and fixation as far distally as practically possible. An extensive dorsal approach to the proximal and middle phalanx is used. The application of two narrow LCPs or DCPs allows greater versatility in screw placement than a single plate.

The proximal articular surface of the middle phalanx is exposed to allow initial reduction and stabilization of the palmar or plantar portions of the fracture to the dorsal struts of bone.[3] The sagittal fractures are reduced later with individual screws inserted in lag fashion. Intraoperative radiographs or fluoroscopy ensure reconstruction of the articular surface within the DIP joint. Three-dimensional fluoroscopy and portable CT units provide a more precise method to assess alignment, and they may be used during reduction and fixation to assess screw trajectory and fracture reduction.

The cartilage of the PIP joint is removed, and the plate screws in the proximal phalanx are inserted. If possible, one or two plate screws should be placed transarticularly. Postoperative cast fixation for 4 to 8 weeks is used, particularly where fracture stability is tenuous. Follow-up radiographs at the time of cast removal dictate the period of stall confinement, which is generally an additional 6 to 12 weeks. Postfixation callus development can be extensive, depending on the extent of fracture line reduction and rigidity of the stabilization. The union of the proximal and middle phalanges usually progresses quickly. The most common cause of persisting lameness is osteoarthritis of the DIP joint.

Repair of severely comminuted fractures with cast or transfixation cast techniques is reserved for fractures that cannot be adequately reduced and stabilized by implants and when humane destruction is not an option for the owner. Fractures entering the DIP joint usually result in some osteoarthritis, and the degree of residual lameness varies accordingly. The prognosis is partly determined by the development of weight-bearing laminitis on the opposite limb. The selection of fixation should provide the earliest return of comfortable weight bearing on the fracture to minimize this complication.

Complete resolution of lameness with implant fixation can occur; however, residual lameness is anticipated if there is inadequate reduction of the fracture fragments entering the DIP joint.[1,3,15-17] Concurrent fractures of the distal sesamoid bone reduce the prognosis further. The vascular supply to the distal limb and the soft tissue integrity can occasionally be compromised through the initial fracture or during attempts at repair. Preoperative immobilization in a supporting splint is important for the long-term outcome of these cases (see Chapter 73). Infection can also be a serious complication, and local and systemic antibiotics are used to minimize bacterial growth.

PROXIMAL INTERPHALANGEAL JOINT

Osteoarthritis

Osteoarthritis of the PIP joint (high ringbone) is a relatively common condition affecting multiple breeds, particularly those with a tendency for short upright pastern conformation. In addition, horses that make quick stops and hard turns with rapid twisting, such as Western performance horses and show jumpers, have a high incidence of pastern osteoarthritis. In young horses, there is also the possibility of arthritic change induced by osteochondrosis (OC).[18]

Osteoarthritis can occur in the PIP joints of the forelimbs and hindlimbs and often results in grade II to III/IV lameness. Palpable enlargements of the PIP joints are a feature after the development of periarticular new bone. Confirmation of the PIP joint as a site of lameness usually requires perineural anesthesia and radiographs to confirm the involvement of the joint. Nonarticular new bone production can be misdiagnosed clinically as true intra-articular osteoarthritic change and should be assessed with radiography before any discussion of surgical therapy. Pain on manipulation of the PIP region is occasionally elicited, and the horse can have a positive response to flexion tests of the distal limb. Heat and swelling over the PIP joint are indicators of pastern arthritis. The lameness is usually chronic and exacerbated by increased work. Radiographic evidence of osteoarthritis can be confined to one limb but occasionally occurs in both, if the problem is conformational in origin.

Treatment of true intra-articular PIP osteoarthritis is usually surgical fusion of the involved joint.[11,14] The response to intra-articular medication with corticosteroid preparations is usually short term. Horses improve considerably with phenylbutazone therapy. Recalcitrant lameness is treated with surgical fusion of the PIP joint. For simple arthritic conditions, this can be accomplished by dorsal exposure of the affected joint, removal of articular cartilage, and application of two 5.5-mm cortex screws in lag fashion to provide transarticular compression, as previously described (see also Chapter 81). Additionally, a three-hole LCP or DCP is applied to the dorsal midline of the two phalanges involved. The use of the LCP in combination with two abaxial 5.5-mm lag screws has been shown to be as strong as the 4.5-mm DCP applied with two adjacent 5.5-mm screws.[19] However, better *in vivo* performance of the LCP could be anticipated based on other studies of the cycles to failure of the locked screw design. Further, the single 4.5-mm DCP and two abaxial 5.5-mm screws have been shown to be biomechanically

superior to the three 5.5-mm lag screw arthrodesis technique.[20] For horses more than 500 kg, application of two LCPs or DCPs is preferred for additional fixation, using the distal screw in each to engage the palmar/plantar process of the middle phalanx and the second most distal plate screw to lag obliquely across the PIP joint and engage the more axial regions of the middle phalanx (see Figure 91-4). Postoperative cast fixation is needed for up to 2 weeks after arthrodesis. Cast bandage combinations may be substituted for the cast in the second week of the 2-week coaptation period and may be continued for an additional 4 weeks.

Minimally invasive techniques for arthrodesis of the PIP joint have been described, using percutaneous insertion of two LCPs and independent stab wounds for plate screw insertion.[12] Additionally in cases with advanced osteoarthritis, laser facilitated cartilage débridement of the PIP joint, and application of three 5.5-mm parallel lag screws through stab incisions, reduces surgical exposure and time, minimizes the need for postoperative casts, and provides early pain relief.[21] Two 5.5-mm screws may be adequate depending on the horse's size, given *in vitro* studies that show similar biomechanical performance of two and three 5.5-mm lag screw configurations for PIP arthrodesis.[22] Laser-facilitated arthrodesis is not suitable for horses with extensive residual cartilage in the arthritic PIP joint, which develop instability and extensive callus as the cartilage undergoes necrosis and reduces the initial compression provided by the screws. Use of ethyl alcohol for eliminating cartilage before applying compression with the screws as an alternative to laser-facilitated arthrodesis has also been described.[23]

Application of LCPs by percutaneous insertion, in combination with multiple passes of a 4.5-mm or 5.5-mm drill to remove residual cartilage, is a better choice for less-advanced osteoarthritis. Physical removal of cartilage may provide improved bone-to-bone contact, but more importantly, two LCPs enhace the rigidity of the repair and minimize postoperative callus formation. Additionally, contouring of the LCPs is lessened because direct cortical contact of the plate is not a prerequisite, allowing flexibility in plate positioning. For large horses, insertion of an independent 5.5-mm screw across the PIP joint in lag fashion, or angling the second plate screw across the PIP joint by using a 4.5-mm or 5.5-mm cortex screw inserted using lag technique, provides three or four stabilizing screws in the middle phalanx. Minimally invasive collateral ligament sparing techniques also allow considerable PIP cartilage to be removed by multiple passes of a 4.5-mm or 5.5-mm drill bit and provide better acute resistance to lateral bending in cadaver specimens.[24]

The rate at which bone union is achieved depends on the degree of stability afforded by internal stabilization. Most horses continue to form periarticular callus for 6 months after surgical repair. Unstable repairs result in exuberant callus, which can interfere with the action of the flexor tendons on the palmar or plantar surface of the pastern and occasionally with the extensor tendons over the dorsal middle phalanx region. Digital extensor tendons can adhere to exostoses on the dorsal aspect of the middle phalanx,pulling on the insertion to the extensor process of the distal phalanx, resulting in residual lameness.[11,25] Excessively long screws that penetrate the palmar or plantar surface of the middle phalanx and interfere with the action of the distal sesamoid bone are also causes of residual lameness.

The prognosis for riding soundness is fair to favorable, particularly for conditions involving the hind limb. The outcome in one report indicated 16 of 22 horses returned to full function.[10] A second study indicated 16 of 21 clinical cases became sound after arthrodesis, with a better response in the hind limbs.[11]

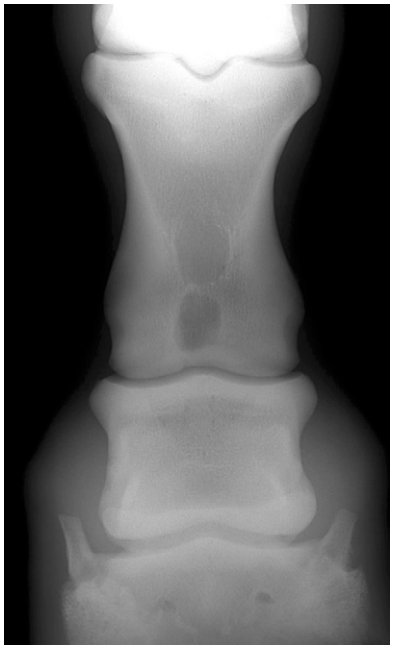


Figure 91-7. Subchondral cystic lesion of the distal aspect of the proximal phalanx. If conservative therapy fails, transcortical approaches either laterally or through the palmar cortex allow access to the cyst.

Osteochondritis Dissecans and Subchondral Cystic Lesions

Osteochondritis dissecans (OCD) can occasionally develop in the PIP joint. Subchondral cystic lesions (SCLs) are diagnosed more often than dissecting cartilage flap lesions and involve the distal articular surface of the proximal phalanx (Figure 91-7), or rarely the proximal articular surface of the middle phalanx. Late stages of OCD with secondary osteoarthritis have been diagnosed in young horses unilaterally and bilaterally. Solitary SCLs are readily apparent on radiographs, whereas multiple SCLs are associated with a persistent lameness leading eventually to osteoarthritis.

Intra-articular hyaluronan injections provide temporary pain relief in solitary SCLs. Surgical curettage of solitary SCLs of the proximal phalanx has been performed in five horses (three foals and two adults).[26] Because the lesion is often not approachable arthroscopically, transosseous drilling is used. Initially, a small drill bit is advanced under fluoroscopic control into the cyst, which is confirmed by the drainage of saline previously injected into the PIP joint. Proper alignment of the drill is verified radiographically, and the drill hole is enlarged with a 5.5-mm drill bit, allowing access with a curette to evacuate the cyst. After copious flushing from the joint, the cyst and drill hole are filled with tricalcium phosphate granules.[26] Long-term follow-up revealed that four of the five horses treated were serviceable and could engage in their intended athletic function. More recently, a similar cyst has been successfully packed with fibrous gel containing $PTH_{1\text{-}34}$.[27] Additional information about these treatments is found in Chapter 89.

Multiple cystic lesions show a poor response to medical treatment, leaving elective arthrodesis of the joint as the only

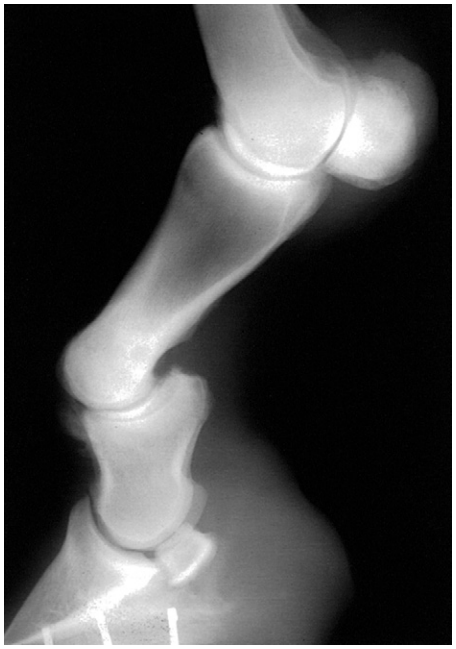


Figure 91-8. Lateromedial radiographic view of a dorsal subluxation of the proximal interphalangeal joint caused by loss of support from the extensor branches of the suspensory ligament. Note the distal location of the proximal sesamoid bones as a result of failure of the suspensory ligament.

treatment (see Chapter 81). Simultaneous bilateral arthrodesis can be performed in foals, whereas staged fusion is elected in adult horses.

Luxation and Subluxation

Luxation and subluxation of the PIP joint are usually treated with arthrodesis. Subluxation is more common and occurs in either a dorsal or a palmar direction. Dorsal subluxation is the result of damage to the suspensory ligament and its terminal extensor branches (Figure 91-8); it is occasionally caused by contracture of the distal sesamoidean ligaments. Palmar subluxation results from failure of the palmar supporting connective tissues such as the distal sesamoidean ligaments, the middle scutum, and the SDF tendon insertions (see Figure 91-4). This condition is manifest by marked hyperextension of the PIP joint and considerable swelling associated with the soft tissue disruption.

The clinical signs associated with dorsal and palmar subluxation include acute lameness initially, with the dorsal subluxations resulting in an obvious dorsal swelling over the PIP joint. Palmar subluxation, because of disruption of the support structures, usually results in hyperextension of the pastern and sinking of the MCP joint. Radiographic examination is used to confirm the diagnosis of subluxation and to differentiate lesions associated with osteoarthritis or bony disruption of the middle phalanx. The lameness associated with PIP joint subluxation is less severe than that associated with fracture disruption.

Dorsal subluxation occurs bilaterally in the hind limbs of some horses with upright conformation. When this occurs, it can be managed with anti-inflammatory medication and a controlled exercise program. As the horse gains condition, the subluxations often resolve.

Pastern arthrodesis is used to treat recalcitrant dorsal and palmar subluxations. Surgical arthrodesis is performed using either the slightly diverging three lag screw technique or, preferably, the application of a single bone plate or a pair of bone plates (see Figure 91-4), as previously described for larger horses. Some acquired dorsal subluxations are caused by excessive tension in the deep digital flexor (DDF) tendon, and successful resolution in three hindlimb cases resulted from release of the medial head of the DDF tendon.[28]

The prognosis after repair of subluxation is fair to favorable. With adequate surgical reduction and stabilization of the subluxation, the pastern can be fused without excessive callus formation. Few cases are described in the equine literature that provide information on long-term follow-up, but horses can return to athletic activity, particularly if a hindlimb is involved. Rarely, bilateral subluxations develop, and a staged pastern arthrodesis can provide a return to soundness.

PROXIMAL PHALANX

Clinical Signs and Diagnosis

Comminuted fractures of the proximal phalanx result in severe lameness, whereas stable sagittal or intraarticular dorsal and palmar/plantar fractures improve rapidly to mild or moderate weight-bearing lameness. The clinical diagnosis is confirmed by radiography. Additional information on complex fracture configuration can often be derived from CT evaluation.

Fracture Configurations

Fractures of the proximal phalanx can be divided into two categories: 1) proximal intra-articular osteochondral fractures and 2) fractures involving the shaft or diaphyseal region of the proximal phalanx. The cause of most of these fractures is trauma, with hyperextension being particularly important for osteochondral fractures, and torsion with axial weight bearing being more important for fractures of the shaft of the proximal phalanx.

Proximal Osteochondral Fractures

PROXIMODORSAL OSTEOCHONDRAL FRACTURES

Osteochondral fractures of the proximal dorsal margin of the proximal phalanx within the MCP joint are common in racehorses.[29-31] They also frequently occur in non-racing breeds.[32] They arise from hyperextension of the MCP joint with impact of the proximal and dorsal aspect of the proximal phalanx onto the dorsal region of the third metacarpal bone (MCIII). Proximodorsal fragments in immature horses have been described as OC, but it is uncommon to find histologic evidence to support these claims.[33] Initial clinical symptoms include several days of moderate lameness associated with obvious synovial effusion and pain on flexion of the affected joint. Depending on the size of the fragment, lameness dissipates quickly. Radiography with four views, including horizontal beam oblique views, demonstrates the lesion adequately. The most prevalent site for proximodorsal fragments is the dorsomedial eminence of the proximal phalanx. Occasionally, both lateral and medial eminences of the phalanx are involved.

The treatment of choice is surgical removal using arthroscopy.[7,30,31] However, small chip fractures (less than 2 mm in diameter) can be rapidly covered by synovial tissues, and many heal without causing clinical symptoms. Moderate to large dorsal fragments should be removed to prevent synovitis, cartilage degeneration, and chronic proliferative (villonodular) synovitis mass development. Large chip fractures result in erosion of the opposing metacarpal condyle and often result in persistent lameness.

The arthroscope is placed into the joint just proximal to the middle of the visibly distended dorsal joint pouch, adjacent to the extensor tendon, because this facilitates examination of both lateral and medial portions of the joint without damage to the dorsal aspect of the sagittal ridge. A second entry portal is then made for instrument entry and fragment retrieval.

Postoperative convalescence includes 6 to 12 weeks' rest from training, depending on the damage to the third metacarpal cartilage. Results of arthroscopic surgery for the treatment of osteochondral fractures of the proximal phalanx in 336 horses revealed a return-to-use rate of 86% for racehorses.[29] Additionally, 68% of horses returned at the same or higher level of racing performance. Removal of osteochondral fractures also may be useful even when moderate levels of osteoarthritis are present. Seventy-five percent of horses with other MCP lesions, including arthritis, were able to return to previous use. Other studies indicate similar excellent results after chip fracture removal, with 89% of 461 Thoroughbreds returning to racing, and 82% racing at a similar or better class.[31]

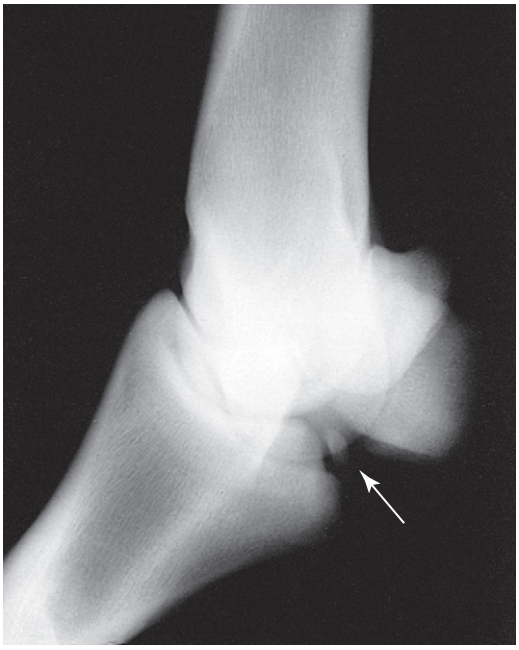


Figure 91-9. Elevated oblique radiographic projection (D15Pr20L-PlDiMO) of the rear MTP region of a Standardbred pacer with a type I (axial) osteochondral fracture *(arrow)* in the plantar pouch. These fragments need to be differentiated from type II fractures on the abaxial corner of the proximal phalanx, many of which do not need surgery.

PROXIMOPALMAR AND PROXIMOPLANTAR OSTEOCHONDRAL FRACTURES

Bone fragments associated with the plantar or palmar portions of the proximal phalanx within the metatarsophalangeal (MTP) or MCP joint have been recognized for many years.[34,35] The cause of fragmentation is controversial; one suggestion being that they are OCD lesions. Recent data support a fracture etiology.[33,36]

The osteochondral fragments have been categorized as *type I fractures* when they are avulsed from the axial, proximal, plantar or palmar rim of the proximal phalanx and are mostly articular. Most have extensive regions of short sesamoidean ligament insertion still attached to the avulsed fragment. Larger, abaxially located, partly articular osteochondral fragments have been categorized as *type II fractures*. Type II fractures extend distad 2 to 3 cm and contain minimal articular cartilage. Lameness associated with type I fractures is minimal and usually only evident at higher speeds.[37,38] Type II fractures do not appear to produce persistent lameness.

Affected MTP or MCP joints often exhibit synovial effusion, moderate pain on flexion, and a mild to moderate response to flexion tests. Intra-articular anesthesia is often required to confirm the diagnosis. The fragments are less common in the forelimb, possibly as a result of a more angled shape to the equine palmar aspect of the proximal phalanx compared with the plantar counterpart. Fragments have occurred in weanlings and yearlings, presumably the result of hyperextension of the MCP or MTP region with avulsion of one or both short sesamoidean ligament insertions, but at this age it has been common to consider them a form of OCD.[37,39] Because most occur in younger animals, they are often covered by synovial tissue at the time of surgical removal. They are most prevalent in Standardbreds; in surveys, 11.8% of all Standardbred yearlings in Norway and 28.8% of yearlings in Sweden were affected.[40,41] In Warmblood horses, these lesions are often overlooked until they are diagnosed in 3- to 6-year-olds, when the training level for show jumping is increased.

Radiography is used to confirm the diagnosis. Routine oblique views are not particularly satisfactory for delineating type I fractures. A better projection results from raising the radiographic beam 20 degrees above horizontal and taking the oblique projections only 15 to 20 degrees dorsal to a standard lateromedial projection (Figure 91-9).[42] These projections, the proximo(20)dorso(15)lateral—distal palmaromedial or the opposite view, the proximo(20)dorso(15)medial—distal palmarolateral projection, highlight the plantar rim of the phalanx and the associated base of the proximal sesamoid bone. These projections allow differentiation of type I axial fractures from the type II abaxial fractures (nonunited proximal plantar tuberosity of the proximal phalanx).[43,44] Type II fractures are quite evident on standard oblique radiographs. Both types of fragments are common in the MTP joints.

Type I plantar fracture fragments are often removed in yearlings to prevent the development of lameness during training and racing. Lameness in yearlings is rare, and the prediction of later lameness induced by these plantar fragments is difficult. An arthroscopic approach through the plantar pouch of the MTP joint provides access to the intra-articular type I fractures.[7,38] The instrument portal is prepared at the base of the proximal sesamoid bone; the exact location of the portal is identified with a hypodermic needle. The fragments are dissected from the covering of synovial membrane and remnants of the attached short sesamoidean ligaments and removed with sturdy rongeurs. The use of motorized resectors, radio-frequency cutting loupes, or diode or CO_2 laser for removing excessive soft

tissue proliferations and resection of the fragment reduces intraoperative bleeding and improves visualization.[38,45]

Surgery is rarely indicated for type II abaxial osteochondral fractures. Additionally the incidence of these fragments is lower (2.4% of 753 Standardbred yearlings), and they might not be fractures but rather constitute a form of delayed ossification.[44] Lesions in 11 of 18 horses had radiographically united to the parent proximal phalanx after 12 months. Occasionally nonhealing abaxial "fractures" and true traumatic fractures with fresh margins and associated lameness require screw fixation in lag fashion to achieve radiographic union. This type of fixation is only warranted when lameness is present, and simple absence of progressive bony fusion is not a reason for surgical fixation.

Convalescent care for type I fracture patients includes 6 to 12 weeks' rest from training, depending on the damage to the distal sesamoidean ligaments at the time of surgery. In a review of 119 surgery cases, follow-up race performance information was available on 87.[38] Fifty-five horses (63%) returned to racing at or above their preoperative level. Abnormal arthroscopic findings, including articular cartilage loss over the palmar condyles and extensive synovial proliferation, had a negative impact on successful outcome.

DORSAL FRONTAL FRACTURES OF THE PROXIMAL PHALANX

Dorsal frontal fractures entering the MCP or MTP joints are relatively rare; in a case series, they represented only 9 of 123 (7%) fractures involving the shaft of the proximal phalanx.[46] These fractures occur predominantly in Thoroughbreds and are prevalent in the hindlimbs. Dorsal frontal fractures tend to be short, extending from the articular surface 2 to 5 cm distad in the dorsolateral cortex of the proximal phalanx. Fractures are generally complete and minimally displaced. Fracture configuration and degree of displacement dictate the need for surgical repair.

Complete fractures are better repaired using screw fixation in lag fashion, although nondisplaced fractures heal with conservative therapy.[46] Complete displaced fractures should be repaired with interfragmentary screws. The surgical repair can be supplemented by arthroscopic examination of the dorsal intra-articular region of the affected joint and débridement of associated cartilage damage. Insertion of one or two 3.5-mm cortex screws using lag technique under arthroscopic guidance is recommended (Figure 91-10). Other screws such as the 4.0-mm cancellous screw also suffice, although the shaft on these screws is fragile.

Most horses recover in a soft bandage. Two weeks of complete stall rest is followed by 6 weeks of limited daily handwalking. The implants are removed only where lysis or reaction has developed beneath the screw heads. If conservative therapy is selected, adequate radiographic healing of the fracture occurs in 4 to 6 months.[46] The prognosis for a return to racing is favorable, with 6 to 9 horses reported to have returned to satisfactory performance with nonsurgical therapy.

Diaphyseal Fractures

Most axial fractures of the proximal phalanx occur in the sagittal plane and propagate distad from the articular surface of the MCP or MTP joint. The mid-sagittal groove is mechanically predisposed to the initiation of the fracture, possibly as a result of torsion applied to the sagittal groove from the opposing

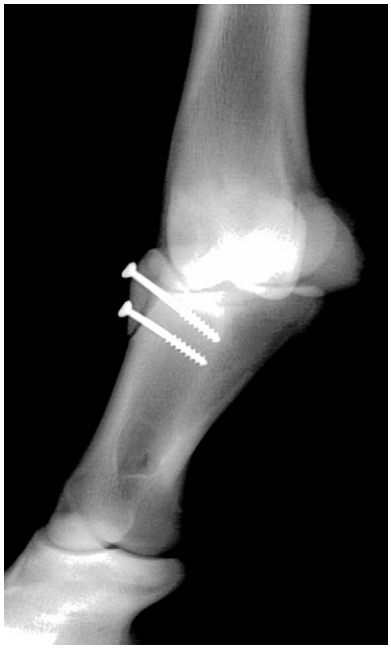


Figure 91-10. Oblique radiographic view of a dorsal frontal fracture of the dorsolateral cortex of the proximal phalanx in the rear limb. Repair using two 4-mm cancellous screws has resulted in improved joint congruity.

articular surface of the sagittal ridge of MCIII or MTIII. Fractures of the proximal phalanx are more common in the forelimbs and are particularly prevalent in racehorses, although proximal phalanx fractures are recorded in most breeds of horses.

The clinical signs associated with an axial fracture of the proximal phalanx depend on the extent of fracture propagation. Affected horses with incomplete sagittal fractures show moderate pain of relatively short duration. Fractures extending the full length of the proximal phalanx and those that tend to comminute result in non–weight-bearing lameness and moderate swelling of the pastern. Short, incomplete proximal fractures of the phalanx are sometimes difficult to isolate through a routine lameness examination, and perineural anesthesia is necessary to localize the site of lameness. The use of desensitizing nerve blocks in this situation increases the risk of propagation of the fracture lines during the lameness examination.

Radiographs define the extent of the fracture; most fractures commence in the sagittal groove and extend distad. Complete fractures exit on either the lateral or, rarely, medial cortex of the proximal phalanx or enter the proximal interphalangeal joint (Figure 91-11, *A*). Short sagittal fractures can be difficult to localize on routine dorsopalmar radiographs. Several different dorsopalmar projections, using proximodistal and distoproximal dorsopalmar projections, better demonstrate the fracture. Short sagittal fractures are often detected using nuclear scintigraphy, with radiographs performed to verify the extent of the fracture. Computed tomography or MRI scans are also exquisitely sensitive at detecting these fractures, but many are identified using scintigraphy in the standing horse without the need for more elaborate equipment.

Most short sagittal fractures are initially treated conservatively because the fracture is incomplete and stable. Screw fixation applying lag technique is reserved for fractures that do not heal after 3 months of conservative therapy. Screw stabilization

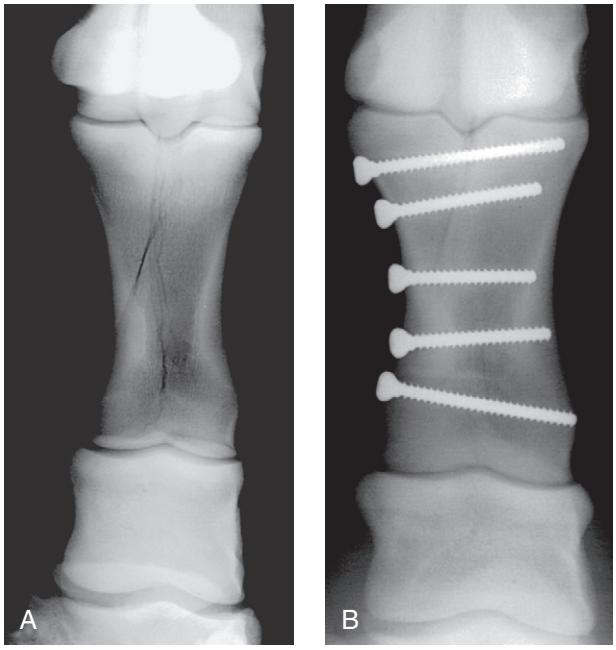


Figure 91-11. A, Dorsopalmar radiographic view of a complete nondisplaced fracture of the proximal phalanx commencing at the sagittal groove within the MCP joint and spiraling distally to enter the PIP joint. **B,** Repair using 4.5-mm cortex screws placed in lag fashion through stab incisions.

induces primary bone union and reduces the chance for further propagation of the fracture in the convalescent period.[47,48] Because of this, interfragmentary screw fixation may be considered as the initial treatment, especially in Warmblood horses.[49] Complete nondisplaced and displaced fractures require surgical repair.[48,50-52] The extent of comminution varies considerably, and as a result, the inherent stability of the fracture configuration varies accordingly. Use of CT imaging can increase the success of surgical reconstruction by improved definition of the fracture planes for screw insertion. The prerequisite for selecting screw reconstruction of proximal phalanx fractures over transfixation casts or external fixator devices is the presence of an intact strut of bone spanning from the MCP to the PIP joint. Disruption of weight-bearing support without an intact bony column is generally a contraindication to screw repair alone. Occasionally, the articular surface of the MCP and PIP joints can be reconstructed with interfragmentary screws, but without an intact strut axial stabilization must be provided with a transfixation device.

SURGICAL TECHNIQUES

Nondisplaced fractures

For routine nondisplaced fractures, the proximal phalanx can be stabilized by inserting screws in lag fashion through stab incisions (see Figure 91-11, *B*). The most proximal screw in the series preferably should be a 5.5-mm cortex screw for additional compression of the articular surface, although 4.5-mm screws usually suffice. Intraoperative radiographic and arthroscopic monitoring is recommended to ensure that the proximal screw does not penetrate the MCP joint and the fracture planes are being adequately compressed.

Ideally, the proximal screw should pass within 5 mm of the most distal point of the sagittal groove of the proximal phalanx to provide maximum compression to the articular surface. This screw should penetrate the transcortex, and the screw head should be adequately countersunk to avoid torque on the screw shaft from the oblique angle of the cortex beneath the head. Additional screws are placed at intervals of 20 to 22 mm until the entire fracture line has been stabilized.

A less common configuration involves fractures in a frontal plane. These fractures are often uniplanar and can be repaired similarly, placing screws in a dorsal-to-palmar direction (Figure 91-12). More comminuted frontal plane fracture configurations can result in trauma to the distal sesamoidean ligaments either by sharp fragments or instruments during repair. This can result in subluxation of the proximal interphalangeal joint after fracture healing. This potential complication should be discussed with the owner of the horse prior to fracture repair.

Postoperative support is provided using a fiberglass cast if the fracture is extensive. Simple fractures that are repaired with several screws can be safely recovered in a firm bandage or a splint. Follow-up radiography is used to assess bone union, and most horses can return to light training 4 months after repair. Proximal phalanx fractures of the hindlimb are more likely to require cast support for the recovery phase because of increased torsional loads on the hindlimbs during recovery.[50] Implant removal is not necessary for fractures of the proximal phalanx. Rarely, lysis beneath the screw head, drainage from an incision, or excessively long screws are identified, and all are indications for screw removal.

Mildly to moderately displaced fractures

Complete fractures that involve sagittal and oblique fracture planes with mild to moderate displacement can generally be better reduced with open approaches to the proximal phalanx followed by screw stabilization using lag technique.[50,51] Access for fracture débridement and reduction is provided by an incision over the dorsal aspect of the sagittal fracture. The incision usually enters the MCP or MTP joint for direct inspection of the articular surface, particularly to ensure accurate realignment of the subchondral bone plate and overlying cartilage. More complicated fractures require a more extensive longitudinal incision and occasionally use of an "I" incision or curvilinear flap for extensive fracture plane manipulation. Separate stab incisions can be used for placing the screws in lag fashion in a lateral plane to stabilize the sagittal fracture and reduce the extent of soft tissue dissection.

Extensive fractures that involve multiple sagittal and frontal planes require an extensive open approach either using an I-shaped incision that exposes the entire dorsal and abaxial cortices of the proximal phalanx or an S-shaped incision.[51] The latter approach involves severing a collateral ligament of the MCP or MTP joint to expose the proximal articular surface of the proximal phalanx. The exposure of the fracture planes and the proximal articular surface allows placement of interfragmentary screws in lag fashion in multiple directions to anatomically reconstruct the bone around the residual intact strut. Most screws are placed in a lateromedial or dorsopalmar direction to provide stabilization of sagittal and frontal fracture planes, respectively. Alternatively, using the dorsal I-shaped incision, the dorsal articular margin of the proximal phalanx can be evaluated during screw insertion, and intraoperative radiographic control is used to ensure the remaining fracture planes are adequately aligned during reduction and stabilization (Figure 91-13). This preserves the collateral ligaments and the

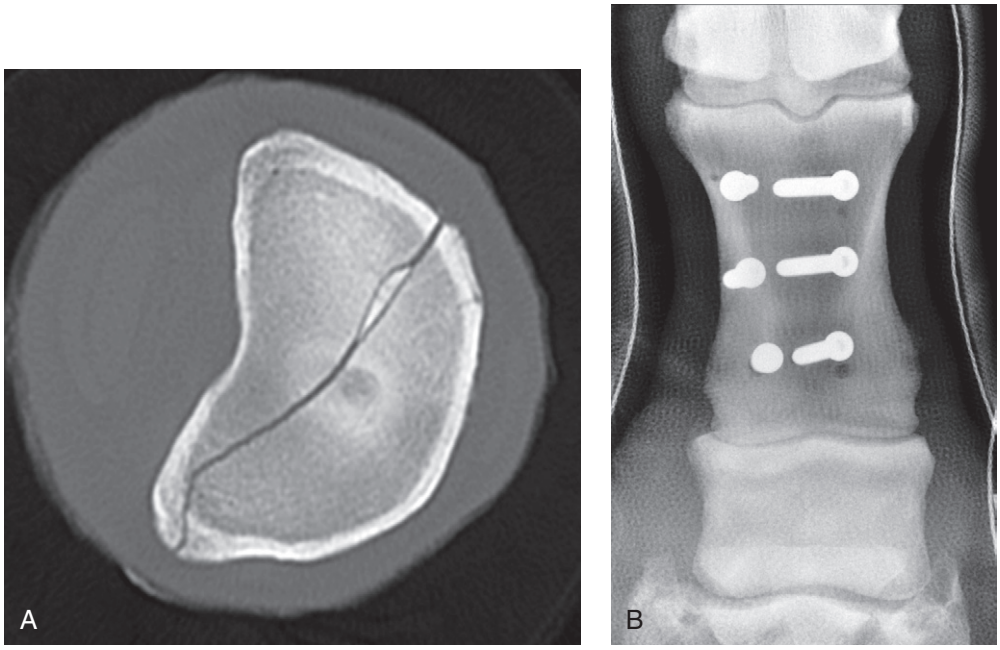


Figure 91-12. A, Transverse computed tomographic view of an oblique frontal plane fracture of the proximal phalanx. **B,** Postoperative dorsopalmar radiographic view showing the direction the cortex screws were inserted in lag fashion. The CT image allowed the surgeon to orient the screws perpendicular to the fracture plane. (Courtesy Lisa Fortier, Ithaca, NY.)

metacarposesamoidean ligaments, and it provides some opportunity for a return to competition.

The limb is cast to the proximal aspect of MCIII or MTIII for recovery from anesthesia and for the next 3 to 6 weeks. Radiographic evaluation is used to determine the appropriate cast time required. Preoperative antibiotics are continued for 3 to 5 days after surgery to minimize the risk of sepsis. Intraoperative culture during extensive and prolonged procedures might also be useful before closing the soft-tissue structures.

Severely fragmented (comminuted) fractures

Fractures without an intact strut of bone are poor candidates for internal fixation. They are generally high-energy fractures and the bone is extensively fragmented (Figure 91-14). Lameness is marked, and considerable swelling can develop if the limb is left unbandaged for any length of time. Extensively fragmented fractures need to be stabilized within a cast before transfer of the horse for evaluation and surgery (see Chapter 73). Radiographs can often be taken through the cast to define the fracture configuration and determine if surgical repair is possible or humane destruction is indicated. For cases where repair is desired, transfixation casts usually provide a reasonable possibility of salvaging the horse. Return to athletic activity is not possible.

Simple cast fixation does not provide resistance to axial collapse of the fracture within the cast. The complications of collapse include continued lameness, a high risk for development of pressure necrosis of the skin leading to an open fracture, and laminitis of the unaffected weight-bearing limb. Transfixation techniques reduce the collapse of the fracture within the cast by using transcortical pins in the distal and mid portion of the MCIII or MTIII.[53] Pins with positive threads located in the center are preferable, and slow sequential drilling followed by preparation of the threads in the hole with a suitably sized tap are generally required before the pin is inserted (see Chapter 76).

An alternative to a transfixation cast is the external skeletal fxaton device, which uses a series of transfixation pins through the MCIII or MTIII joined to side-connecting tapered bars attached to a foot plate (see Chapter 76).[54,55] The most significant complications of transfixation casts and the external skeletal fixator is ring sequestrum formation around the pin tracts, especially in heavy horses, and subsequent fracture through a pin hole (see Figure 76-9).[56] Despite these complications, this fixator is a good choice for salvaging horses with extremely comminuted fractures.

THE METACARPOPHALANGEAL JOINT

Palmar Metacarpal Fragmentation

Palmar fragmentation of the metacarpal condyles is a trauma-induced disorder that results from the accumulated stress and sclerosis that develops during racing. This syndrome has also been referred to as *palmar osteochondral disease* during more detailed studies of the pathogenesis.[57] Previously, this disease was thought to belong to the osteochondrosis complex, in this joint located at the palmar or plantar surface of the MCIII condyles.[58,59] However, there are no histologic data to verify this etiology, and classification as an OCD lesion has been abandoned. Most patients are Thoroughbred racehorses that are 3 years old or older and have moderate to severe MCP lameness.

The palmar lesions are identified on standard lateromedial and flexed lateromedial projections (Figure 91-15). Additional information can occasionally be derived from 125-degree dorsopalmar projections.[60] The radiographic lesions appear on the lateral projection as differently shaped palmar defects, including crescent, flattened, concave, oval, or circular lesions.

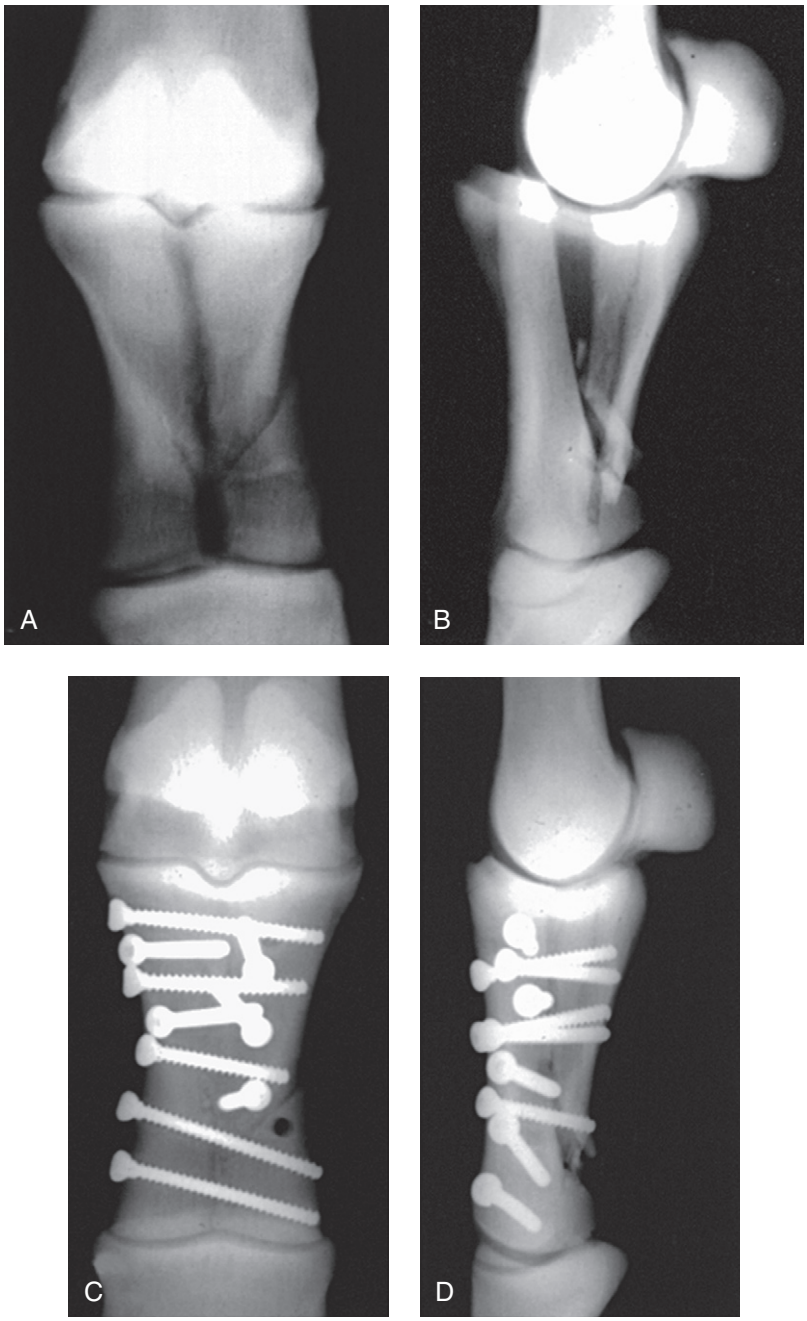


Figure 91-13. Dorsopalmar **(A)** and lateromedial **(B)** preoperative radiographic projections of a multifragment fracture of the proximal phalanx showing sagittal and frontal plane components. There is an intact medial strut of bone that is to be used in reconstructing the fragmented portions. Dorsopalmar **(C)** and lateromedial **(D)** postoperative radiographic projections show the repaired fracture.

Secondary arthritic changes are common, with periarticular osteophytes or enthesiophytes, narrowing of the joint space, and supracondylar lysis. A prominent secondary feature is intense sclerosis of the palmar region of the condyle, deep to the lytic region. Morphologic assessment of these degenerate metacarpal condyles indicates an area of acellular and apparently necrotic bone over the entire distal palmar region of the affected metacarpus.[61] Deep to the necrotic area is a zone of new bone formation producing a sclerotic barrier that compensates for the biomechanical dysfunction of the palmar fracture. These remodeling and fracture changes are the result of accumulated stress fractures in the palmar region of the condyle, largely as a result of hyperextension of the MCP joint. Early lesions may have little overlying cartilage damage; however, eventual fracture and displacement of the subchondral bone result in complete bone and cartilage loss (Figure 91-16). Lameness, although initially mild or moderate, then becomes severe.

Scintigraphy is sensitive in detecting the early stress fracture and bony deposition forming the sclerotic zone within the palmar region of the condyle. Early recognition is particularly helpful because the sclerosis can be reduced or allowed to remodel rather than progress to palmar fracture with prominent symptoms. CT imaging and MRI also are both important diagnostic modalities to define early lesions and track progress. This condition is difficult to treat, and efforts at prevention are important. Access to the palmar region of the distal metacarpus is very limited and is made more difficult because most of these lesions are close to the sagittal ridge.

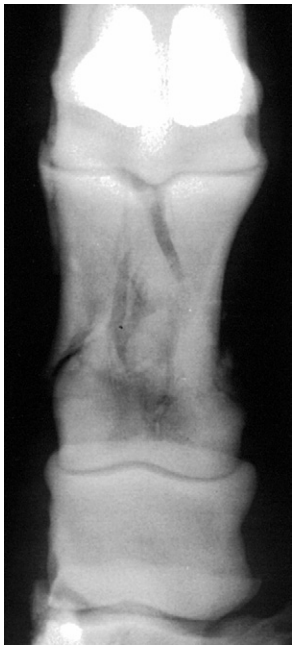


Figure 91-14. Dorsopalmar radiographic projection of a severely fragmented fracture of the proximal phalanx, without an intact strut and lacking inherent stability. Screw repair is generally inadequate, and transfixation casting or external fixators are required for salvage.

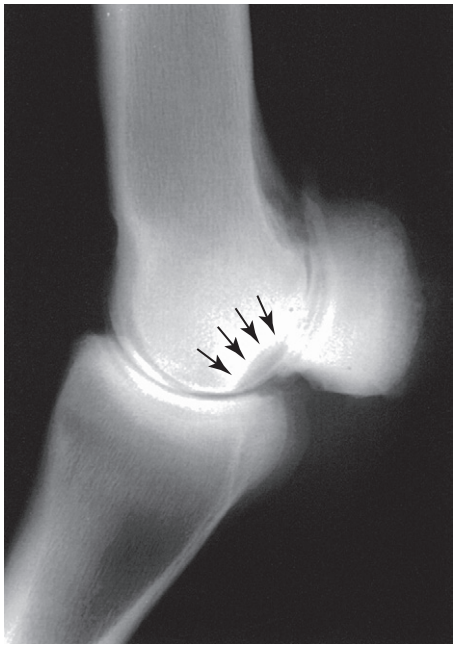


Figure 91-15. Lateromedial radiographic projection of a 5-year-old Thoroughbred with marked palmar fragmentation, bone loss, and extensive subchondral sclerosis of the metacarpal condyles *(arrows).* Enthesiophyte formation is noted at the proximal and distal ends of the proximal sesamoid bones, indicating chronic arthritis.

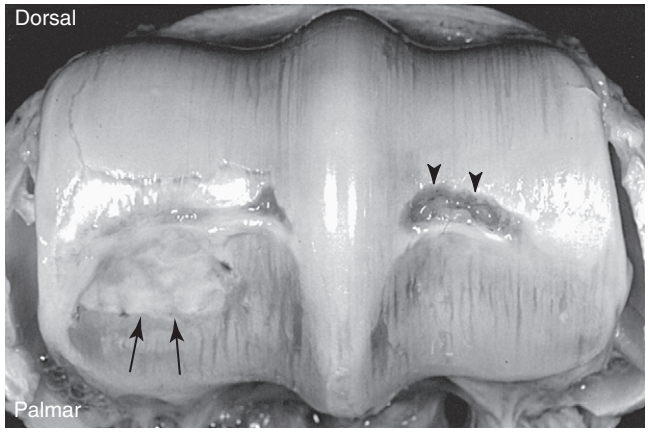


Figure 91-16. A cadaveric specimen of palmar metacarpal fragmentation of the condyles in a 4-year-old Thoroughbred racehorse. Extensive score lines have developed over the entire cartilage surface, with fragmentation and granulation laterally (*arrowheads*) and fibrous healing tissue in the defect medially *(arrows).*

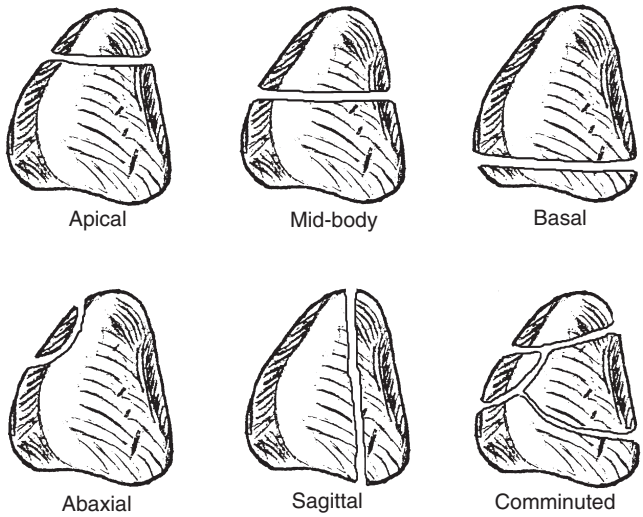


Figure 91-17. Proximal sesamoid fracture configurations.

Proximal Sesamoid Bone Fractures

Fractures of the proximal sesamoid bones are relatively common, particularly those involving the apical portion.[62-75] Other configurations include basal, midbody, abaxial, sagittal, and comminuted fractures (Figure 91-17). Most proximal sesamoid fractures occur because of excessive tension from the suspensory apparatus.

Apical Fractures

Apical fractures are particularly common in the hindlimbs of Standardbreds, and the resultant prognosis often is dictated by the extent of loss of suspensory ligament insertion and preexisting suspensory desmitis.[62] All fracture configurations, except abaxial, nonarticular fractures, result in MCP effusion and moderate to severe lameness of short duration.

Apical fractures involving the proximal one fourth to one third of the proximal sesamoid bone always contain an articular

component. However, they do not result in extensive suspensory derangement, and lameness diminishes rapidly. Removal of apical fragments up to one third of the proximodistal dimension of the proximal sesamoid bone is recommended. Better results are evident with smaller apical fractures, where there is less disruption of suspensory ligament insertion[62,64] because removal of the proximal third of the proximal sesamoid bone results in delay of return to racing and a considerably reduced likelihood of a successful postinjury career. Large apical fractures with obliquity to the fracture plane may be better candidates for internal fixation.[75]

Arthroscopic removal of apical fractures results in less-extensive dissection and secondary fibrosis. The arthroscope is inserted in the proximal aspect of the palmar or plantar pouch, and the instrument portal is made at the level of the fracture, allowing dissection of the fragment from the suspensory attachment. Use of the contralateral palmar pouch for arthroscope entry often provides better visualization of the apical fracture and an unfettered region for instrument access.[75]

Midbody Fractures

Midbody and basal fractures present special problems for surgical fixation. Both of these injuries are slightly more prevalent in Thoroughbreds, and internal fixation is required for adequate fracture union. Midbody proximal sesamoid fractures should be repaired with either circumferential cerclage wire or screw fixation using lag technique.[67,68] Recent publications suggest screw fixation may be the only option for Thoroughbreds to return to athletic capacity.[76] Previous therapy, including cerclage wire fixation accompanied by insertion of a bone graft into the separated fracture line, combined with extended periods of cast fixation, are unreliable in returning horses to work.

Screw fixation in lag fashion is performed from the base or the apex of the bone, depending on the orientation of the fracture plane. An oblique fracture with the fracture plane declining from medial to lateral is best treated with interfragmentary screws placed from the apex, whereas a fracture declining from lateral to medial is best approached from the base or from a contralateral approach allowing insertion of oblique proximal to distal oriented screws.[75] Interfragmentary compression and reduction during screw insertion is maintained with large pointed reduction forceps. Frequent intraoperative radiographs and/or arthroscopic supervision are essential for a high-quality repair. For screw insertion from the base, the screw head should come to lie between the oblique and straight distal sesamoidean ligaments in a natural fossa in the base of the proximal sesamoid bone. Proximal to distal oriented screws are inserted proximolaterally at the apex of the bone, embedded in the insertion of the lateral suspensory branch. Insertion technique for the screws is routine after the correct screw position has been verified radiographically (see Chapter 76). Interfragmentary screw fixation in lag fashion returns up to 60% of Thoroughbred horses to active racing,[67,68] whereas circumferential wiring has largely been reserved for Standardbreds.[68,76]

Basal Fractures

Basal slab fractures involve the origin of all the distal sesamoidean ligaments, and the prognosis is certainly unfavorable. Lag screw fixation is more challenging because of the thin fragment profile (Figure 91-18). There is an inverse relationship between dorsopalmar fragment length and the likelihood of return to racing.[72] Basal osteochondral fragments that do not extend to the palmar surface have a much better prognosis, with 59% returning to racing in a study of 57 horses.[66] Many of these fractures can be removed arthroscopically. Similarly, in a second study, 57% of horses with fragments involving less than 25% of the base successfully returned to racing after fragment removal, compared to 40% with fragments involving more than 25% but not the entire base.[72]

Abaxial Fractures

Abaxial fractures must be assessed by a 60-degree skyline projection of the abaxial surface of the proximal sesamoid, which demonstrates whether the abaxial fracture enters the joint or is located palmarly/plantarly and is extraarticular (Figure 91-19).[69] Intra-articular fractures can be removed under arthroscopic visualization, whereas the nonarticular fractures are best treated conservatively.[70,71] Return to function is fair to good (61%), depending on the length of the intra-articular fracture.[71]

Sagittal Fractures

Sagittal fractures are rare and tend to occur on the axial margin of the sesamoid bone in conjunction with other MCP injuries such as condylar fractures (see Figure 92-7).[73] Very rarely, they can develop as isolated fractures, but they occasionally can be repaired using several 3.5-mm cortex screws placed in lag fashion in a lateral-to-medial orientation. Postoperatively, application of a cast is recommended for the initial 2 to 3 weeks. At that time, if everything looks satisfactory, the cast is exchanged for a heavy support bandage. The remainder of the postoperative management is routine. Because the fractures usually occur in conjunction with displaced lateral condylar fractures, return to athletic activities is unlikely and retirement from racing is inevitable. Rarely, MCP arthrodesis is necessary, including fractures compounded by injury to the intersesamoidean ligaments or fractures of the proximal phalanx. For additional information see Chapter 92.

Biaxial Fractures

Multifragment and biaxial fractures are severe breakdown injuries involving the MCP joint. In most cases, these horses require MCP arthrodesis for a return to comfortable weight bearing (see Chapter 81) (Figure 91-20).[77,78]

Prognosis

Follow-up on 109 horses with apical sesamoid fractures indicated that 64% of the horses treated surgically raced afterward, whereas only 37% of those treated conservatively raced again.[62] Another outcome study of 84 Thoroughbreds indicated 77% of horses returned to racing after removal of apical fractures, with a slightly better outcome when hindlimbs were involved.[74] For midbody fractures repaired with cortex screws inserted in lag fashion, 9 of 12 horses were able to race after surgery.[62,67] Similarly, in Standardbred horses with midbody fractures repaired by wiring, 11 of 15 were able to resume an athletic career; none of the Thoroughbreds, however, returned to racing.[68,76]

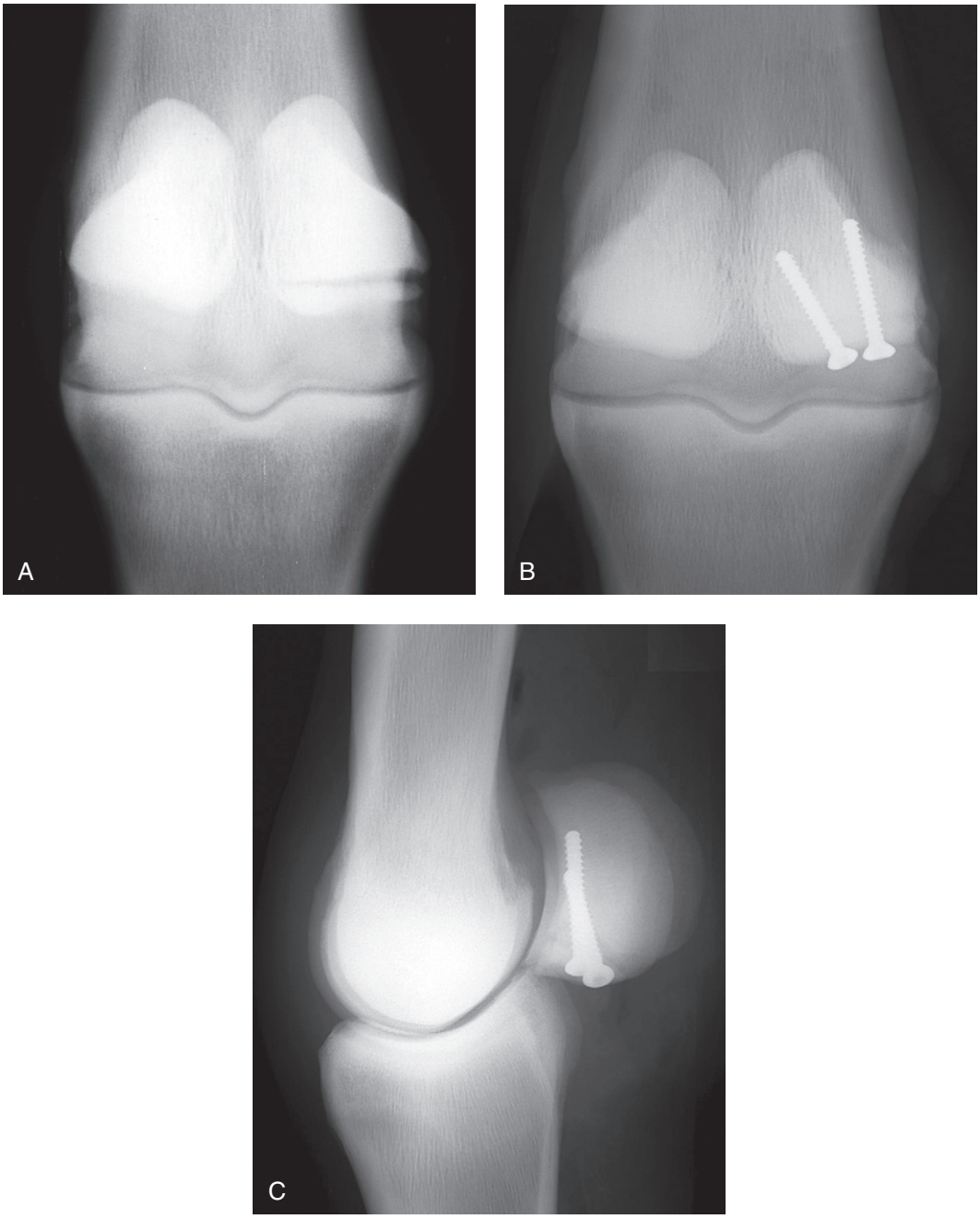


Figure 91-18. A, Dorsopalmar preoperative radiographic projection of a basal fracture of the lateral proximal sesamoid bone. Dorsopalmar **(B)** and lateromedial **(C)** postoperative radiographic projections following repair of the fracture by means of two 3.5-mm cortex screws inserted in lag fashion. Because of the small size of the basal fracture fragment, 3.5-mm screws were selected.

Chronic Proliferative (Villonodular) Synovitis

Development of soft-tissue masses in the dorsal aspect of the MCP joint is common secondary to chronic fibrosing synovitis.[79-83] The most frequent cause is proximodorsal osteochondral fractures of the proximal phalanx that are not immediately treated by fragment removal. In addition, chronic proliferative masses commonly develop with advancing osteoarthritic changes within the joint. Horses with long pasterns are predisposed to this problem. A visible and palpable mass is evident at the proximal dorsal aspect of the affected MCP region. Lameness is usually grade II to III/V depending on the underlying disease. Radiographs occasionally show cortical lysis on the dorsal region of MCIII, under the enlarging mass. Involvement of the hindlimbs in chronic proliferative synovitis is rare. Definitive diagnosis requires either arthrography or ultrasonographic examination.[30] Ultrasonography is more convenient.

Most masses are 7 to 10 mm in diameter. The minimum thickness that warrants surgery is 4 mm; smaller masses can be treated with intra-articular atropine and steroids. The enlarged medial portion of the chronic proliferative plica is usually thicker than the lateral portion.

A critical factor in the decision for surgery is the extent of osteoarthritis present in the joint. Osteochondral fragments and

other fracture diseases in the MCP joint result in secondary fibrous mass development, and these fibrous masses can be removed without difficulty and with reasonably favorable prognoses.[30,82] Masses secondary to osteoarthritis are generally larger and more chronic, and the associated arthritis dictates the likelihood of successful return to work after the mass is removed. A critical evaluation of the joint space on preoperative radiographs and the use of ultrasonography to measure the dorsal articular cartilage thickness prevent inadvertent operation in these cases. Increasing use of MRI has helped define the extent of concurrent cartilage loss and the value of surgical débridement.

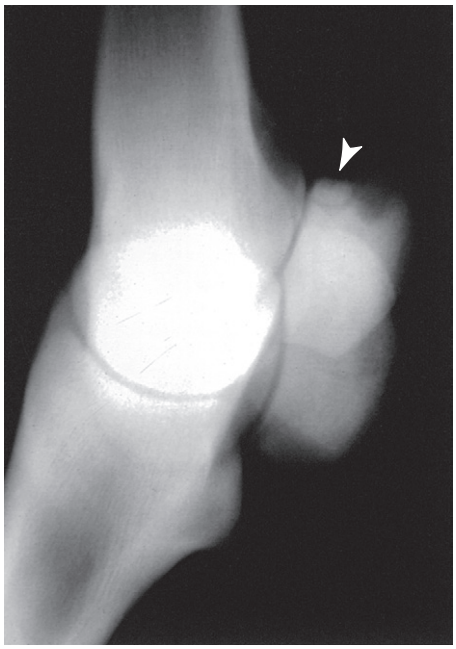


Figure 91-19. Elevated 60-degree lateromedial radiographic projection (Pr60L-DiMO) of the proximal sesamoid bones to evaluate the intra-articular component of an abaxial sesamoid fracture *(arrow)*. This fragment enters the joint.

Primary chronic proliferative masses can develop without an obvious initiating factor, and these most likely result from hyperextension of the MCP joint and impact trauma on the normal synovial plica. which primarily cushions and lessens the impact of the dorsal margin of the proximal phalanx onto the metacarpal cortex.[79,82] Hemorrhage and fibrosis in this plica eventually result in significant enlargement, which is then more easily and repeatably injured during exercise. This is quite painful, but the horses do very well after surgery to remove the mass.

Arthroscopic approaches are suitable for removing chronic proliferative masses (Figure 91-21), although some masses are so large that they may be easier to remove by arthrotomy.[30,82] Arthroscopic visualization allows mass removal using synovectomy instrumentation, guarded scalpels, or the biopsy suction punch rongeur for smaller masses (Dyonics). Large masses can also be efficiently removed using larger biopsy punch rongeurs (Sontec Instruments), or CO_2 or diode laser where this equipment is available.[83] Radiofrequency probes are increasingly used to excise these masses, often using a second instrument portal to stabilize the mass during excision.

Postoperative therapy includes intra-articular injections of 20 to 40 mg of hyaluronan. Repeat doses in 3 to 4 weeks are recommended. The use of polysulfated glycosaminoglycan (Adequan) is another option depending on the state of the articular cartilage. Return to race training depends on concurrent arthritis.

Osteoarthritis

Deterioration of articular cartilage is common in mature racehorses and can lead to moderate to severe unrelenting lameness, with the development of periarticular osteophytes, enthesiophytes, and joint space collapse, particularly medially. These horses respond poorly to intra-articular medication and require

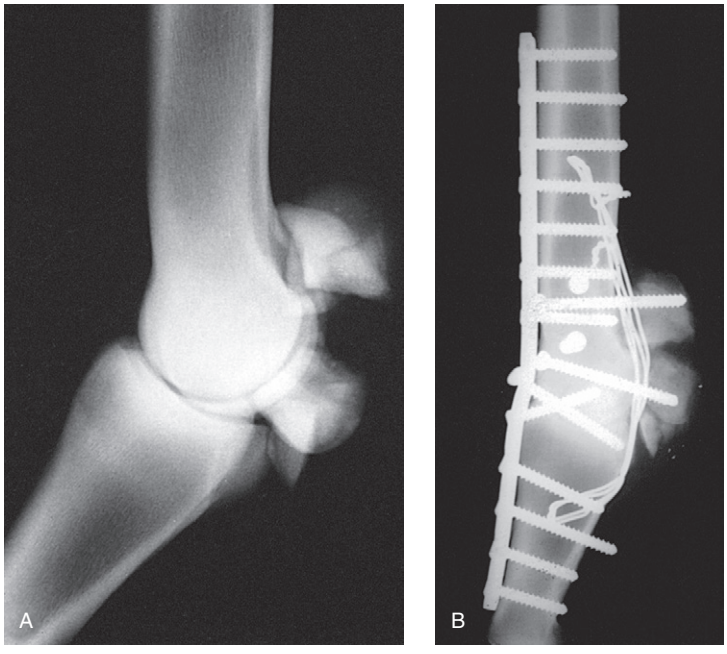


Figure 91-20. A, Lateromedial radiographic projection of the MCP region depicting bilateral transverse mid-body fractures of the proximal sesamoid bones resulting in a breakdown of the suspensory apparatus. **B,** Repair was achieved through an MCP arthrodesis using a dynamic compression plate (DCP) applied to the dorsal surface of the limb and a tension band wire for palmar support.

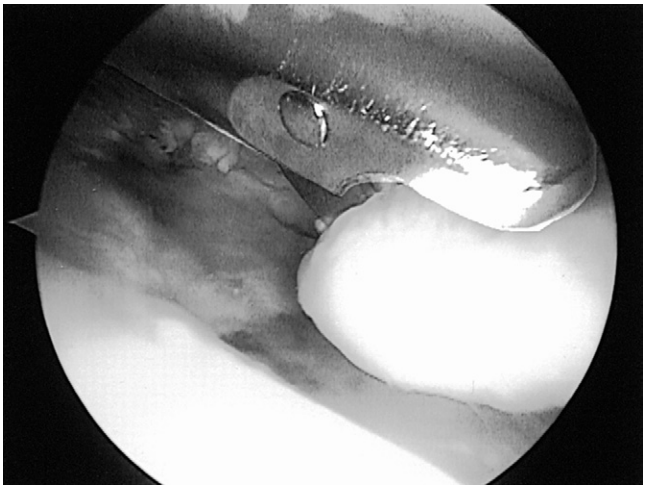


Figure 91-21. Arthroscopic removal of an 8-mm chronic proliferative (villonodular) mass from the MCP joint using a biopsy punch rongeur.

frequent injections to remain comfortable. Collapse of the medial compartment of the MCP joint is more prevalent than the lateral and often indicates the likelihood of a career-ending lameness.

Therapeutic options are limited. Pain relief can be effected by intra-articular steroid administration, but continued cartilage degradation is inevitable. Injection or surgical implantation of cultured bone marrow stromal stem cells (MSCs) may provide generalized improvement in joint function. Some cells may adhere to fibrillated cartilage, although the broader impact seems to be anti-inflammatory, antiapoptotic, and trophic effects of the stem cells at the synovial membrane boundary. In the final stages of joint deterioration, some improvement in the level of lameness has resulted from the administration of silicone oils to the joint. Ultimately, some horses need MCP arthrodesis to become comfortable (see Chapter 81).

Osteochondritis Dissecans and Subchondral Cystic Lesions

OCD can develop as palmar dissecting flaps on MCIII or MTIII, or more frequently in the form of dissecting cartilage flaps[84] that appear as mineralizing lesions on the dorsal sagittal ridge of the MCIII or MTIII, and these are common in young horses 8 to 24 months old.[85] Dorsal OCD lesions can affect all four MCP joints. Palmar metacarpal OCD lesions, which result in a debilitating lameness, often progress to osteoarthritis and represent the most serious form of MCP OCD. Palmar lesions occur in horses as young as 10 months but are more common in young racing-age horses. (Distopalmar metacarpal fragmentation in 3- to 4-year-old racehorses was originally described as OCD but is a different disease from that described here as OCD.) For more information on this disease complex see Chapter 88.

Subchondral cystic lesions (SCLs) of the distal MCIII or MTIII condyle occur most commonly in weanlings and yearlings but can be first diagnosed in 2-year-olds in training. The resulting lameness is variable, and occasionally severe, but osteoarthritis is a rare initial sequela. Most subchondral cysts open to the joint just dorsal to the transverse ridge by a narrow communicating channel. Occasionally, multiple cysts are present in the same bone, and the cysts can be bilateral. Cysts of the proximal articular surface of the proximal phalanx also can be seen, often opening in or near the sagittal groove.

The degree of lameness largely depends on the type of MCP OC. Dorsal sagittal ridge lesions result in mild to minimal lameness, although flexion tests of the involved joints are often positive. The other forms of OCD and SCL result in more significant lameness. Lameness and pain on flexion are most pronounced with palmar OCD lesions in yearlings. Regardless of the site, MCP effusion is a prominent feature. Most horses have sufficient lameness, synovial effusion, and exacerbation of the lameness with flexion tests that a tentative diagnosis can be reached without regional or intra-articular anesthesia. Radiographs are necessary to determine the exact cause of MCP disease and to provide prognostic guidelines. Dorsal sagittal ridge lesions often manifest as mineralized densities adjacent to the sagittal ridge (Figure 91-22, *A*). They often arise from the proximal condylar cartilage and are more correctly termed dorsal parasagittal or dorsal condylar OCD lesions.

Treatment options vary according to the site of involvement. Palmar MCIII and plantar MTIII lesions are relatively inaccessible, and surgical therapy is not possible unless the lesion is located unusually far caudad on the condyles or sagittal ridge. Occasionally, part of these lesions can be débrided under arthroscopic guidance with a palmar or plantar pouch approach. On most occasions, however, the lesions are inaccessible, and osteoarthritis with permanent lameness is the usual sequela. These young horses rarely enter and sustain an athletic career.

Dorsal sagittal ridge and parasagittal condylar lesions should be surgically removed under arthroscopic guidance (see Figure 91-22, *B*). The technique as described previously should be applied.[30] These lesions can achieve considerable size, and removal can leave a substantial subchondral defect. However, in this location, joint stability is unaffected, and the outlook remains favorable. Subchondral cystic lesions of MCIII or MTIII that have narrow openings to the joint can be treated successfully with several injections of hyaluronan (Figure 91-23, *A*). Most yearlings do well, and the lameness quickly resolves. The cystic cavity generally fills in but may take 1 to 2 years. Occasionally, lameness persists despite intra-articular medication and limited exercise, and surgery should then be considered.

Cystic lesions with wide channels to the articular surface do not respond to conservative therapy nearly as well as cysts with narrow openings, and débridement is often recommended at the initial diagnosis (see Figure 91-23, *B*). Surgical débridement of the cysts can be accomplished via dorsal arthroscopy with the MCP joint flexed. Most cysts can be curetted; bone grafts are not usually necessary. In mature horses, chondrocyte or stem cell grafts have been used to assist in repair of the subchondral architecture and overlying cartilage.[86,87] A recent study on the long-term effect of filling of the defect with a $PTH_{1\text{-}34}$–enriched hydrogel following débridement via an articular or transosseous approach revealed good results in cystic lesions in the proximal sesamoid bone (3 out of 3 became sound and radiographically filled), and distal MCIII/MTIII (2 out of 2 became sound and radiographically improved), whereas one lesion each in the proximal aspect of the middle and proximal phalanx remained lame and became larger.[88]

Arthroscopic techniques can be used only with cysts opening on or dorsal to the transverse ridge of MCIII. More palmar cysts are rare and best approached transosseously through drilling. Cysts of the proximal phalanx can be débrided with difficulty

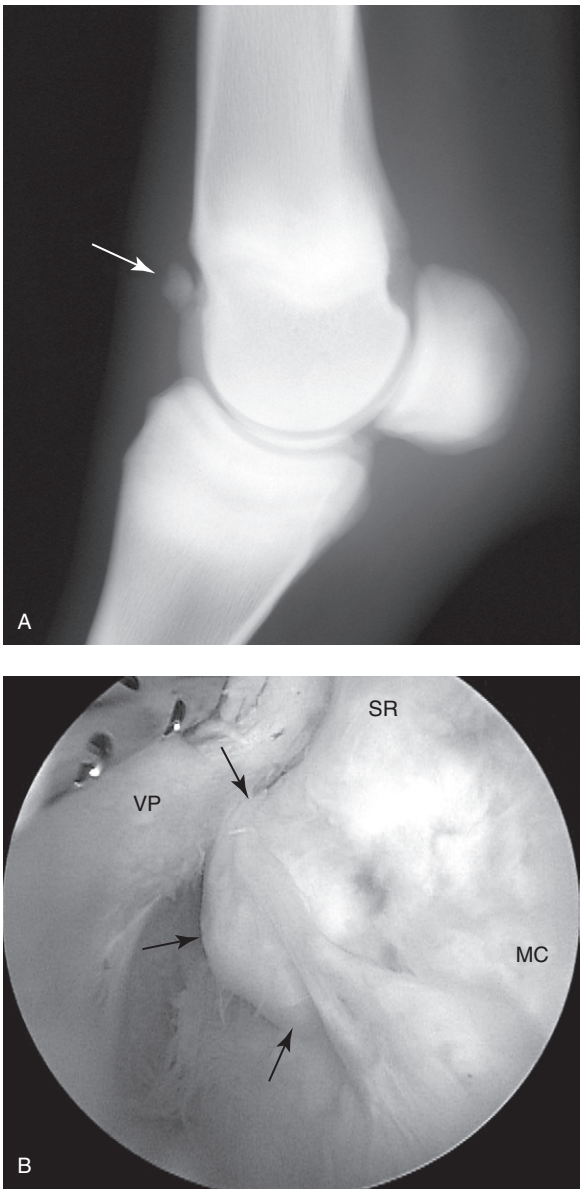


Figure 91-22. A, Lateromedial radiographic view showing dorsal sagittal ridge OCD *(arrow)* that involved the adjacent condyle. **B,** Arthroscopic examination shows the sagittal ridge *(SR)* distal to the fragment *(arrows)*, the metacarpal condyle *(MC)*, and villonodular pad *(VP)*.

from the articular surface, particularly in MTP joints where the joint separates with flexion.

The outlook for a sustained athletic career is favorable for subchondral cystic lesions of the distal MCIII or MTIII, reasonably favorable for dorsal sagittal ridge and parasagittal OCD, and poor for palmar metacarpal OCD flaps.[89]

Luxation

Lateral or medial complete luxation of the MCP and MTP joints occurs after rupture of either the medial or lateral collateral ligament.[90,91] Occasionally, there is an avulsion of the origin or insertion of one of these ligaments, resulting in marked instability. The condition is relatively rare and results primarily from entrapment of the distal limb in holes in the ground or cattle grates. The diagnosis is obvious when an angular deviation can be easily induced on manipulation (Figure 91-24). Radiography with a dorsopalmar projection with the limb under lateral or medial bending stress usually provides clear evidence of the extent of the luxation. Ultrasonographic examination may add additional information when radiographs are inconclusive, but stressed radiographs are a vital source of information regarding the extent of physical disruption.[91] Ultrasonographic studies have identified cases that are stable but have rupture of portions of the collateral ligament. Some injuries associated with trailer accidents result in an open luxation with disruption of the collateral ligament and some loss of bone structure because of pavement injury.

The treatment of closed luxations is simple and involves cast fixation for 6 weeks.[90] Imbrication or repair of the collateral ligament is not usually necessary. The cast is applied with the horse anesthetized, maintaining the lateral–medial stability as the cast cures. Surgery for open luxations is necessary for adequate wound débridement and soft-tissue closure. Extensive antibiotic lavage and removal of all foreign matter are essential. Treatment of the luxation initially in a splint is an option for the acute inflammatory phase, until the infection has been controlled. Later, a cast can be applied for increased stabilization, or arthrodesis by application of a DCP or LCP and screws can be used if the joint surface is extensively damaged (see Chapter 81).

Two case series of MCP luxation have been published.[90,91] One series, involving 10 horses, included five horses that had open luxation and five with closed luxation.[90] The open luxations were treated with joint lavage and cast immobilization, and the majority were sound at follow-up, including one horse that was being actively ridden. A more recent series of 17 horses, combined cases with stable and unstable rupture of the collateral ligaments based on ultrasonographic detail, also concluded the prognosis was favorable for return to function after conservative therapy.[91]

The development of obvious sepsis within the MCP joint can be minimized by aggressive early therapy and the placement of lavage drains and ingress antibiotic portals. Later application of antibiotic-laden bone cement assists in bringing the infection under control. Overall, the prognosis after the development of infection is guarded, and chronic lameness can be expected. Some of these cases can be salvaged with aggressive débridement and elective arthrodesis later.

ANGULAR LIMB DEFORMITY OF THE METACARPOPHALANGEAL REGION

Angular limb deformities originating at the distal physis of the MCIII or MTIII develop relatively often in foals; most are varus deformities. Concurrent valgus of the carpus or tarsus and a varus deformity of the MCP region are common. Many varus deformities are congenital and improve during the early postnatal period. A detailed discussion of angular limb deformity is found in Chapter 86.

FLEXURAL DEFORMITY OF THE METACARPOPHALANGEAL JOINT

Flexural deformities of the MCP joint can be classified as congenital or acquired.

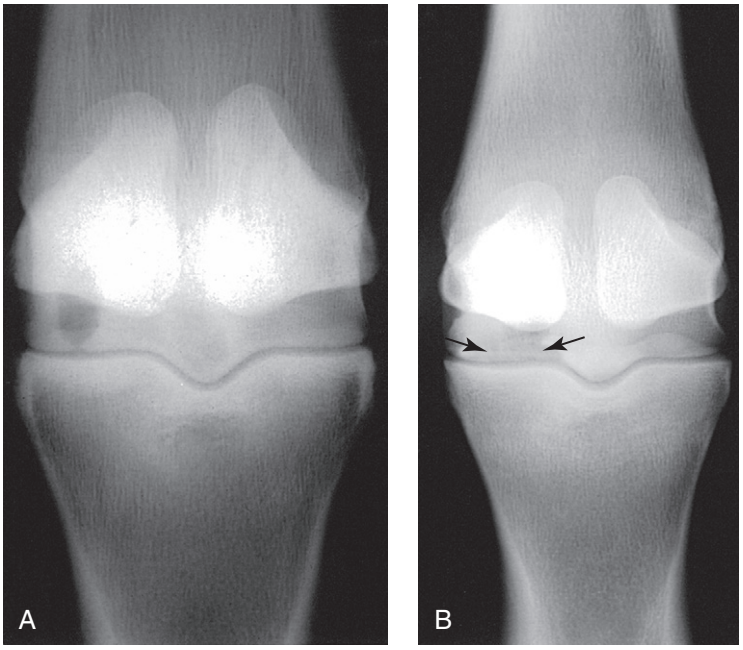


Figure 91-23. Dorsopalmar radiographic views of the MCP joint. **A,** Subchondral cystic lesion with small communication into the joint generally respond well to conservative therapy, such as intraarticular hyaluronan or intralesional corticosteroid injections. **B,** SCLs with wide canals *(arrows)* often need surgical débridement.

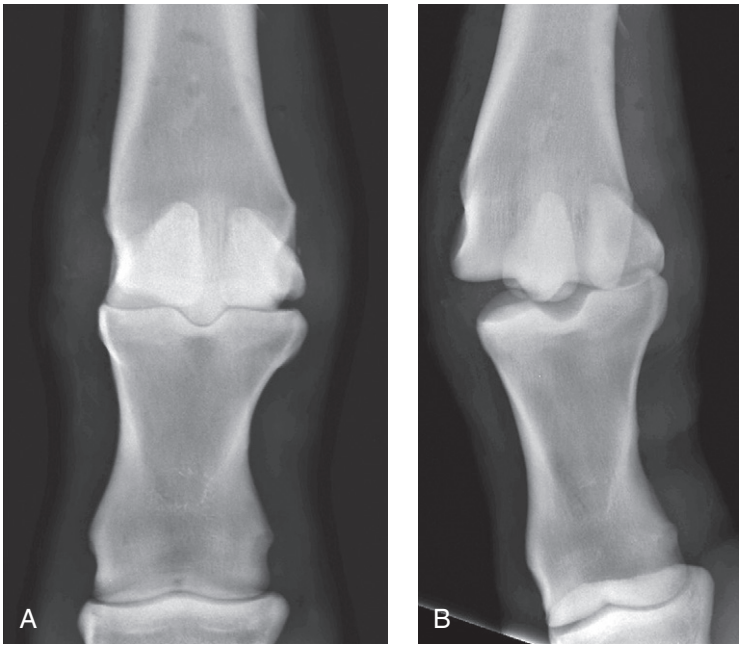


Figure 91-24. Luxation of the metacarpophalangeal joint. **A,** Standing dorsopalmar radiographic projection shows normal MCP alignment. **B,** Bending stress luxates the joint, confirming complete medial collateral ligament rupture.

Congenital Deformities

Congenital deformities are often relatively severe. If they are bilateral, the foal can have difficulty rising or, if it does stand, it tends to knuckle over at the MCP joint. In some congenital cases, tension is apparent in both the deep and superficial digital flexor tendons; in others, the common digital extensor tendon is ruptured. Occasionally, even the suspensory ligament is taut on palpation. The diagnosis and management of these deformities are discussed in detail in Chapter 87.

Acquired Deformities

Acquired flexural deformities can be unilateral or bilateral. Both forelimbs are commonly affected, one generally more severely than the other. The pathogenesis of the acquired form of flexural deformity is thought to involve pain-induced reflex loops in the ipsilateral limb, often precipitated by other skeletal disease. The diagnosis and management of these deformities are discussed in detail in Chapter 87.

ANNULAR LIGAMENT CONSTRICTION

Chronic tenosynovitis is the most common condition of the digital sheaths of horses, developing mostly in the hindlimbs as an insidious reaction to increased workloads. Most cases of tenosynovitis are successfully treated by changing exercise intensity with or without intrasynovial medication. Untreated tenosynovitis or tenosynovitis associated with more serious disruption of the tendon sheath, the various mesotenons and manica flexoria, the annular ligament, or the flexor tendons themselves can result in lameness and a self-perpetuating cycle of tendon sheath fibrosis, repeat tearing, and annular ligament thickening referred to as *complex tenosynovitis*.[92-94] Tenosynovial masses and adhesions can develop as a consequence of this chronic inflammatory action. These latter types of cases do not respond particularly well to medical therapy and not only require surgical intervention but also generally have a reduced prognosis for a cosmetic outcome and a return to complete soundness.

In animals in which the annular ligament causes constriction, chronic lameness (exacerbated by flexion of the fetlock), and tendon sheath effusion and thickening with a notching of the sheath over the width of the palmar/plantar annular ligament are common (Figure 91-25). Ultrasonographic evaluation of the tendons within the digital sheath and fetlock canal is useful to determine the extent of tendon lesions and the thickness of the synovial tendon sheath and annular ligament and to define the number and attachments of tenosynovial masses. Several reports suggest that annular ligament desmitis can be the sole initiator of annular ligament constriction, and ultrasonographic evaluation is critical in making the diagnosis.[95,96] Tears within the DDF tendon also are recognized as a cause of chronic severe tenosynovitis.[97,98]

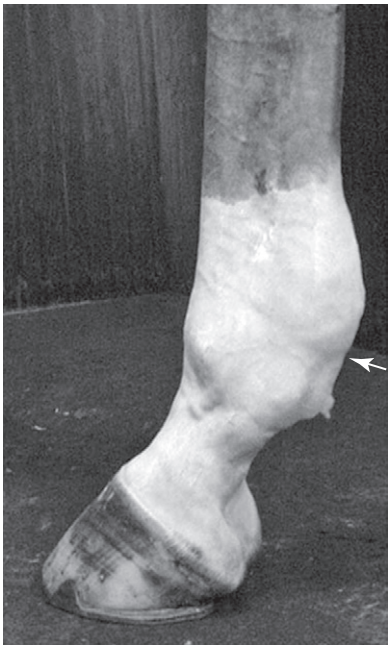


Figure 91-25. Lateral view of a horse suffering from constriction of the annular ligament, demonstrated by the synovial effusion in the tendon sheath and the characteristic notching of the tendons proximal to the annular ligament *(arrow)*.

Medical treatment is required in chronic cases when a noticeable lameness has developed in conjunction with persistent swelling. Where ultrasonographic examination reveals obvious tendon or digital sheath pathology such as tendon sheath masses and adhesions, tears in the digital flexors, rupture of the manica flexoria, or annular ligament thickening, surgical therapy is preferred for definitive resolution of symptoms.

Treatment

Mass Removal

Open tendon sheath approaches to mass and adhesion removal or resection have been successful. The disadvantage to extensive open approaches is the delay in the initiation of exercise postoperatively and adhesion formation or reformation. However, early postoperative walking increases the risk of wound dehiscence with the attendant risk of sepsis. For these reasons, endoscopic examination of the sheath (tenoscopy), using a standard rigid arthroscope, is preferred.[7,92,94,99]

The entry portal for routine tenoscopy of the digital sheath is on the palmarolateral or plantarolateral surface immediately distal to the annular ligament.[99] This entrance allows examination of most regions of the sheath, and instrument portals can be made proximal to the annular ligament under tenoscopic guidance. Tenosynovial masses can be resected either with motorized synovial resectors or by division at their attachments with biopsy-cutting forceps or retractable blades and subsequent removal with grasping forceps using an additional instrument portal (Figure 91-26). For extensive synovial resection, hemorrhage control is required, and a tourniquet is applied proximal to the digital sheath. If annular ligament transection is also needed, it can be accomplished initially to open further access for manipulation within the sheath, or it can be done at the end of the procedure, using the slotted cannula (Dyonics) available for carpal tunnel release in humans. Where space for surgical manipulation permits, the annular ligament can be transected free-hand with a variety of right-angled blades under arthroscopic visualization.

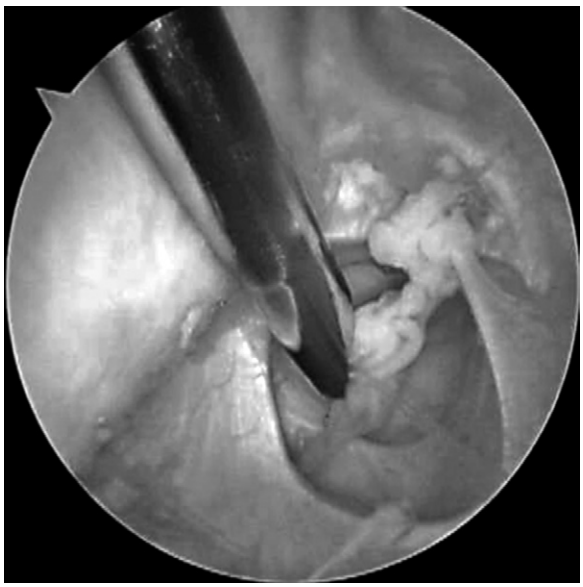


Figure 91-26. Tenoscopic removal of digital sheath masses from the dorsolateral recess of the sheath cavity using motorized equipment.

Annular Ligament Transection

The only effective treatment for the annular ligament constriction syndrome is division of the ligament. This can be done by limited open incision or, in more complex cases, by using tenoscopic access, which provides more complete assessment and access to other lesions. Preoperative planning is important for determining the degree of exposure of the tendons required to optimize treatment. Where only the annular ligament is involved, the ligament can be severed in the standing animal with a curved bistoury introduced through a 1-cm incision proximal to the ligament. In original descriptions of surgical division of the annular ligament, the horse was anesthetized and the skin was incised over the entire proximodistal length of the annular ligament. This incision provided access to allow sectioning of the annular ligament and subsequent division of adhesions under direct view. Where the annular ligament is the only involved structure, the exposure required for division of the ligament can be reduced to a 2-cm skin incision over the proximal border of the annular ligament.[100] Another surgical approach that can be performed open with less risk of dehiscence uses a paramedian skin incision over the entire length of the annular ligament, followed by palmar or plantar transection of the ligament over the mesotenon. This technique, if performed correctly, prevents opening of the tendon sheath. It can be applied if only the annular ligament is involved. Depending on the presence of tendon core lesions or peripheral adhesions, other surgical procedures such as tendon injection, mass removal, or adhesiolysis are performed as indicated. Currently, minimally invasive techniques are superior for these extensively affected cases and allow earlier initiation of exercise and physiotherpay. This translates into improved outcome.[92,94,101,102]

Tenoscopic examination of the digital sheath with an arthroscope entering distal to the annular ligament allows visual inspection of the entire sheath cavity and division of the annular ligament (Figure 91-27). Second entry portals allow adhesion resection and mass removal, as previously described, and endoscopic release of the annular ligament can be accomplished.[93] The obvious advantages of tenoscopic annular ligament release include the extensive dissection that can be performed through limited entry wounds, allowing better wound healing with less risk of dehiscence and earlier postoperative exercise.

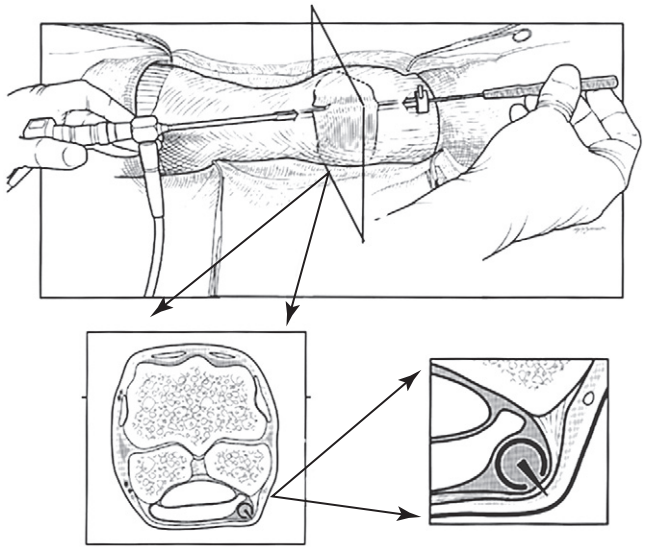


Figure 91-27. Illustration of tenoscopic annular ligament release, using a slotted cannula inserted from proximal, and an arthroscope inserted from distal to the base of the sesamoid to visualize a 90-degree–angled blade used to sever the fibers of the annular ligament. *Inset* shows the position of the blade adjacent to the sesamoid abaxial perimeter.

The entry and angle of the slotted cannula are critical to facilitate insertion of the arthroscope and 90-degree angled blade. The proximal entry portal should be dorsal in the digital sheath and the distal exit point should be plantar or palmar to allow the arthroscope or blades to clear the bulbs of the heel. The cannula with obturator in place is inserted under direct arthroscopic visualization as it traverses from proximal to distal. The insertion path must be external to the manica flexoria, or this ring of the SDF will be divided along with the annular ligament. As the obturator nears the distal portal, the arthroscope is removed, and the obturator and cannula are exteriorized through the vacant portal.

The obturator is removed, and the unsheathed arthroscope is inserted to view and verify the flexor tendons, sesamoid surface, and annular ligament. The slot is oriented to open directly toward the annular ligament, and the 90-degree-angle sharp blade is drawn across the fibers of the annular ligament to sever the full thickness of this structure. Arthroscopic guided free-hand division of the annular ligament using right-angled blades, radio-frequency probes, or bistoury can also be used (Figure 91-28). Hemorrhage is flushed from the cannula, which is then removed, and the tendon sheath is flushed before the skin incision is closed. When additional flexor tendon or sheath lesions need to be addressed, transecting the annular ligament before débriding other lesions improves maneuverability.

Tendon Débridement

Linear tears or clefts within the DDF tendon, and occasionally the SDF tendon, present special problems in repair, with most requiring débridement and some requiring suture.[97,98,102] Ultrasonographic examination is not sensitive in detecting linear clefts in the tendon structure. Any ultrasonographic suggestion of echolucencies within the palmar or plantar third of the surface of the flexor tendons is highly suspicious, and this region should be carefully assessed during tenoscopic exploration. The linear clefts can penetrate a variable distance into the substance of the tendon.

The treatment of choice is tenoscopic débridement (Figure 91-29). Débridement has proved superior to suture repair following open surgical approaches.[94] Tears in the DDF tendon can extend 4 to 10 cm in length. They often involve the DDF tendon from the level of the apex of the proximal sesamoid bones and extend distal to the mid-portion of the proximal phalanx. The depth of the linear cleft varies from penetration to the center of the DDF tendon to more superficial fiber erosion. Trimming of exposed tendon fibers can be accomplished using a combination of biopsy punch rongeurs and motorized resectors with both side and forward aperture, which are also effective in trimming epitenal and tendon fiber damage. The aim should be a relatively smooth tendon surface. Short linear tears in the DDF tendon generally have a good prognosis, however, long tears (more than 4 cm) are more difficult to return to function by débridement.

Tears of the manica flexoria of the SDF tendon can develop acutely and result in lameness and severe digital sheath

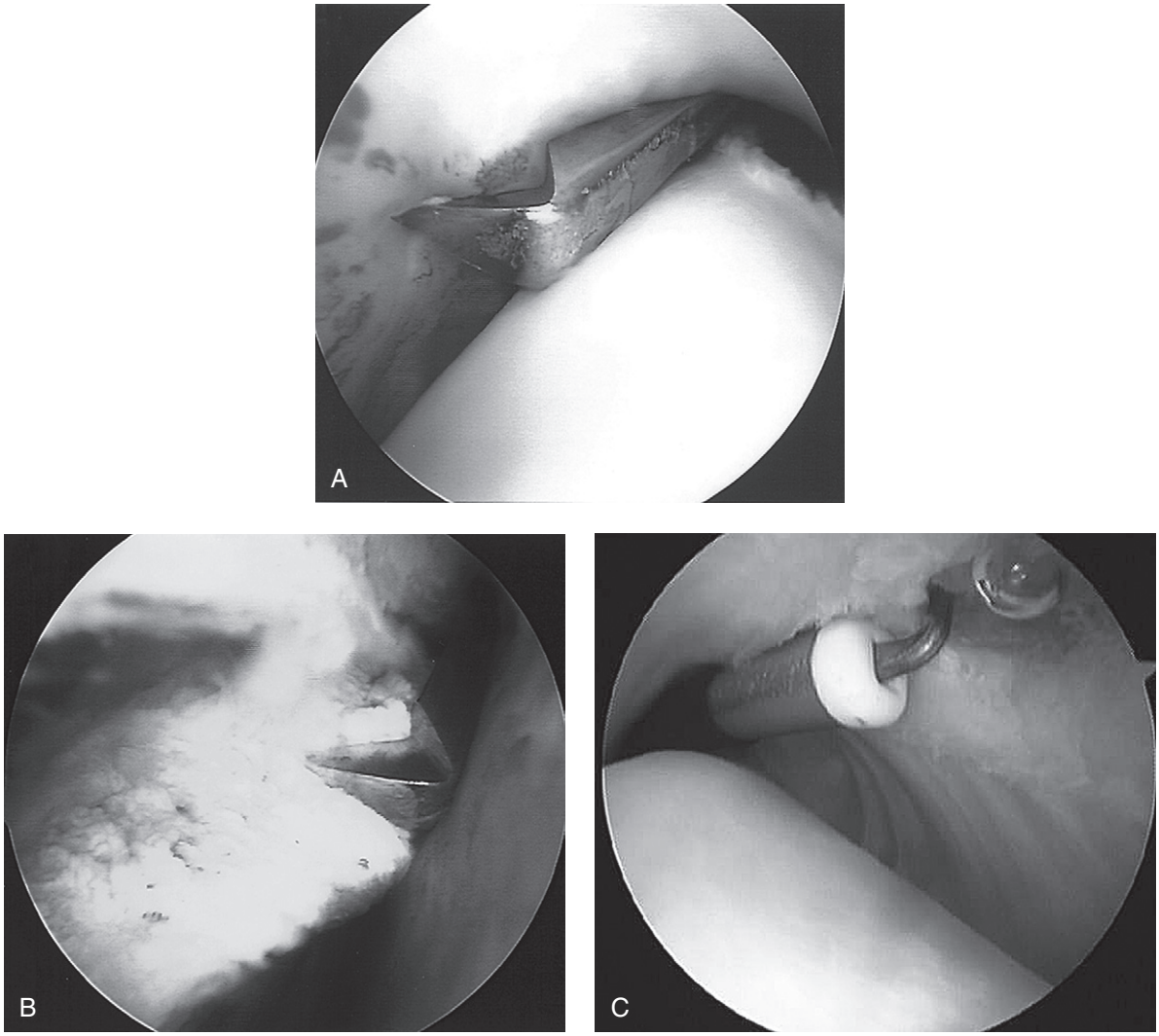


Figure 91-28. Tenoscopically guided free-hand transection of the annular ligament. **A** and **B,** The right-angled blade from the slotted cannula kit can be used free-hand if the MCP canal is not excessively tight. **C,** The 90-degree hook radiofrequency probe provides a clean and more precise cut.

swelling. Incidence varies among countries and sports activity; however, treatment by tenoscopic débridement is often curative. Most manica tears develop along the medial attachment of the SDF tendon and the manica recoils laterally (Figure 91-30). Resection of the torn portion is required. This can be accomplished by biopsy rongeur resection of the torn portion or by using an additional instrument portal to allow retraction of the mobile end of the torn portion of the manica and resection along the lateral attachment to the SDF tendon. Occasionally a thin band of the manica persists intact, and this should be converted to a complete tear and resected.

Postoperative Management

Hyaluronan (20 to 40 mg) can be injected into the tendon sheath at the time of wound closure or later at suture removal. Research in horses and experimental animals indicates hyaluronan can reduce the formation and re-formation of tendon adhesions in the sheath area and can enhance intrinsic tendon healing.[103] The incidence of synovial fistulas associated with the extensive skin incision in the original description of annular ligament transection makes the smaller exposure and the use of tenoscopy attractive alternatives. Nevertheless, the maintenance of a firm bandage with careful changes and sterile nonadhesive padding is important when walking exercise is initiated before suture removal. The limb is usually bandaged for 3 to 4 weeks after surgery. Although extensive tendon synovial mass and adhesion resection can be painful postoperatively, the majority of horses with simple annular ligament section do not require extensive analgesia.

Stall confinement without exercise is recommended initially, but hand-walking should be instituted after 5 days and the time period should be increased rapidly. Long periods of walking exercise are particularly helpful if tendon adhesions were present at surgery. If adhesiolysis was performed at surgery, follow-up injections of hyaluronan are recommended 2 and 5 weeks postoperatively.

Prognosis

Tenoscopic mass removal and annular ligament division in 25 horses followed for 6 to 68 months revealed cosmetically acceptable results in 22 of the horses. Lameness resolved in 18 of the 25 horses (72%).[92] The poorest response was evident in

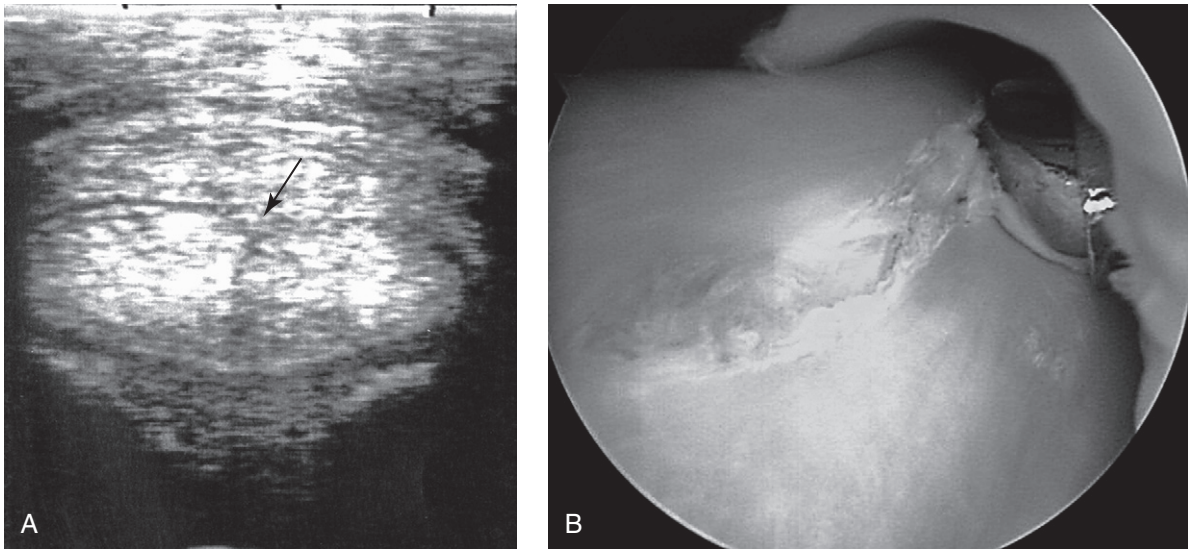
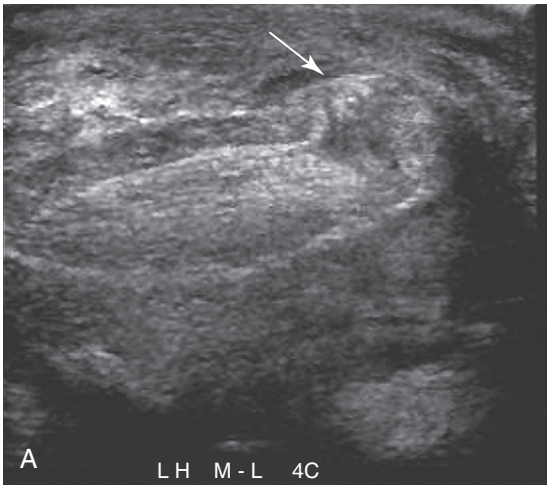


Figure 91-29. Deep digital flexor (DDF) tendon linear tears. **A,** Ultrasonographic image of the DDF tendon with a suspicious echolucency on the palmar surface of the tendon *(arrow)*. **B,** Tenoscopic trimming of the linear cleft in the deep digital flexor.



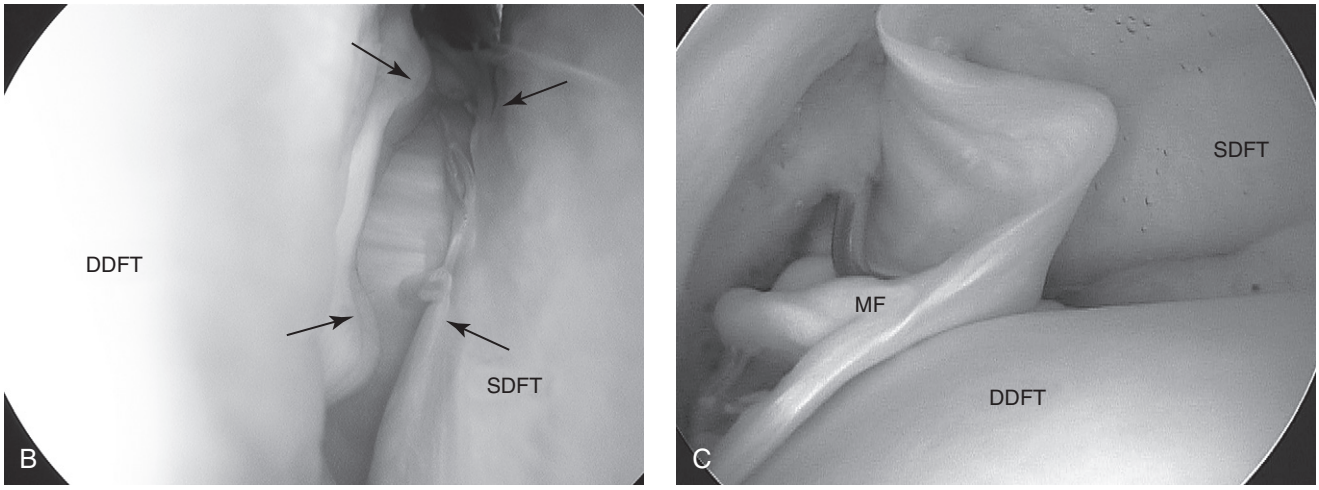


Figure 91-30. Torn manica flexoria of the SDF tendon *(SDFT)*. **A,** Ultrasonographic appearance showing the retracted mass *(arrow)* alongside the DDF tendon *(DDFT)*. **B,** Tear along the medial side of the manica *(arrows)*. **C,** Free portion of manica *(MF)* tear.

two horses with concurrent tendinitis in the region of the digital sheath. The cosmetic outcome was inversely related to the preoperative duration of signs and the thickness of the annular ligament. In addition, a longer history of symptoms led to a thicker annular ligament on preoperative ultrasonography. In a larger series of 76 cases having tenoscopy for nonseptic conditions of the digital sheath, tears of the manica flexoria were found in 23, and most had a favorable outcome.[102] Marginal tears of the DDF tendon, marked preoperative distention, and open surgical repair of deep digital flexor lesions were associated with reduced levels of postoperative performance. Marginal tears were associated with postoperative lameness, and long tears were associated with a reduced performance level compared with short tears.[102]

Simple constrictive syndromes as a result of a wound, desmitis of the annular ligament, or chronic fibrosing synovitis of the tendon sheath have a good prognosis for return to work after sectioning of the annular ligament. The outlook is guarded where extensive tendon adhesions are resected, because these cases often have residual obliteration of the tendon sheath cavity with tendon tie-down in the proximal and distal limits of the sheath. Additionally, long linear tears of the DDFT have a guarded prognosis for recovery of function.[98,102]

Constriction caused by tendinitis of the flexors in the MCP canal is readily relieved by annular ligament division; however, return to work is governed by the poorly healing bowed tendon. A guarded prognosis is common, and extended periods of measured increases in activity are recommended. Follow-up ultrasonograms are useful to evaluate the repair of tendon lesions, particularly where core lesions were injected at the time of annular ligament transection.

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CHAPTER 92

Third Metacarpal and Metatarsal Bones

Dean W. Richardson

ANATOMY

The third metacarpal (MTIII) and third metatarsal (MTIII) bones are important and vulnerable elements of the forelimbs and hindlimbs, respectively. The small metacarpal and metarsal bones (splint bones, MC/MT II and IV) are often termed *vestigial*, but both MCII and MTII have major weight-bearing functions, and MCIV and MTIV serve as sites for important carpal and tarsal ligamentous insertions (see Chapter 93). MTIII is slightly longer and typically more round in cross section than MCIII. The cross section of MCIII changes dramatically in response to increased loading as the horse ages and trains.

There is little soft tissue coverage of MCIII/MTIII except for their palmar/plantar aspects where the superficial and deep digital flexor tendons, accessory (check) ligament of the deep digital flexor and the suspensory (interosseous) ligament are located. The suspensory ligament originates in a broad attachment on the proximal palmar/plantar aspect of the bones. The soft tissues in the proximal palmar/plantar metacarpal and metatarsal regions also are invested in a retinacular sheath.

In the forelimb, the major neurovascular structures course along the length of the metacarpus both medially and laterally just dorsal to the flexor tendons. In the hindlimb, the great metatarsal (dorsal metatarsal III) artery's location between MTIII and MTIV make it particularly vulnerable to accidental or surgical injury.

FRACTURES OF THE THIRD METACARPAL AND METATARSAL BONE

Fractures of the MCIII/MTIII are common long bone injuries of athletic horses. These are the most intensely loaded bones of the appendicular skeleton and therefore vulnerable to single-event catastrophic failures, as well as repetitive cyclic fatigue injuries. The most common site of major fracture of MCIII/MTIII is in the distal articulation. Repetitive loading during intensive exercise leads to focal accumulated injury in the parasagittal groove region that eventually results in complete fracture.[1,2] MCIII/MTIII also are common sites of injury because of external trauma, such as kicks.

Lateral Condylar Fractures

Vertical fractures in the sagittal plane occur in the distal condyle of MCIII/MTIII, predominantly in young racehorses either during fast work or a race (Figure 92-1). In Thoroughbreds, MCIII fractures are at least twice as common as MTIII fractures, but in Standardbreds, the ratio of forelimb to hindlimb fractures is nearly equal.[3-5] There is not a large difference in prevalence between left and right forelimb fractures, but fractures

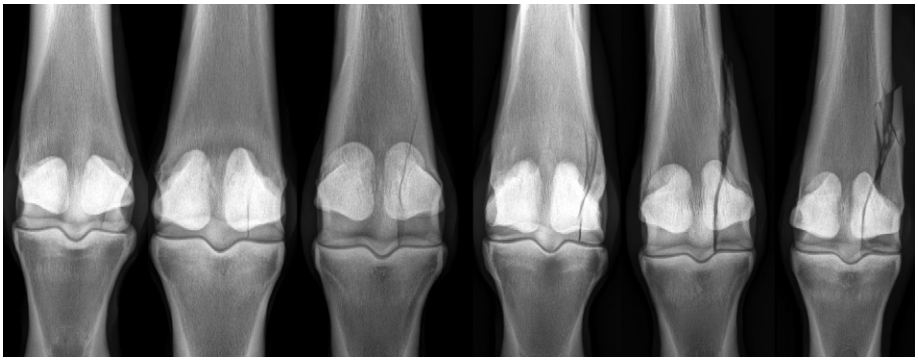

Figure 92-1. Lateral condylar fractures are vertical, occur in the sagittal plane, and range from faint, short linear cracks to complete displaced fractures with extensive comminution.

involving the right MCIII are more likely to be displaced than those in the left.

Clinical Signs

Clinical signs of lateral condylar fractures are typically straightforward, but incomplete fractures involving only the distal few centimeters of the condyle may present a diagnostic challenge. Horses may have a history of metacarpo/metatarsophalangeal (MCP/MTP) lameness preceding the onset of a more severe lameness; however, the most typical presentation is an acute onset of severe lameness after intense exercise or a race. The MCP/MTP joint almost always develops effusion, and there is overt pain on manipulation of this region. The degree of lameness is not well correlated with the amount of fracture displacement; many horses with nondisplaced fractures are extremely lame, whereas some horses with large, displaced fractures walk fairly well on the fractured limb.

Approximately 85% of all condylar fractures involve the lateral condyle.[3,4,6] In the forelimbs, lateral fractures are 7 times more common than medial fractures, and in the hindlimbs, lateral fractures are about 3 to 4 times more common.

Nearly all lateral condylar fractures propagate from the mid to midaxial portion of the lateral condyle for a few centimeters in a sagittal plane and then proximolaterally toward the lateral cortex. There is variation of their height, but the typical fracture breaks out 1 to 3 cm above the physeal scar. Unlike medial condylar fractures, lateral condylar fractures only rarely propagate in a spiral plane or course centrally through the diaphysis.

Diagnosis

Radiographic diagnosis is not difficult, provided good-quality radiographs are obtained. An underexposed dorsopalmar/dorsoplantar (DP) projection or a radiograph in which the distal margins of the proximal sesamoid bones are superimposed over the MCP/MTP joint surface may not be diagnostic. This is avoided by taking the routine DP view in a slightly proximal to distal direction. Many horses have pre-existing degenerative disease involving the distal palmar/plantar condyle adjacent to the base of the ipsilateral proximal sesamoid bone, so an additional radiographic projection further defining this region should be taken. A silhouetting of the distal palmar/plantar condyle can be obtained in slight flexion with the beam angled proximally[7] or by putting the joint in more flexion with the limb pulled forward and using a horizontal beam. Such a radiographic projection should be used in all displaced condylar fractures because comminution along the fracture line is commonly identified in this location (Figure 92-2). As newer imaging modalities are used, such as computed tomography (CT) and magnetic resonance (MRI), earlier recognition of the repetitive stress injury in this location that predisposes to condylar fractures should be feasible. Scintigraphy is probably the best means of early identification of condylar injuries.

First Aid

First aid for a lateral condylar fracture includes compression bandaging, nonsteroidal anti-inflammatories, and absolute rest until definitive therapy is undertaken. The expense of cast coaptation is not justified because very few lateral condylar fractures propagate if the horse is kept quiet in a heavy bandage. Manufactured splints and boots also are not indicated.

Surgical Management

Surgical management of lateral condylar fractures involves compression of the fracture with cortex screws applied in lag fashion. The screws prevent proximal propagation of the fracture and enhance articular healing, therefore minimizing the potential

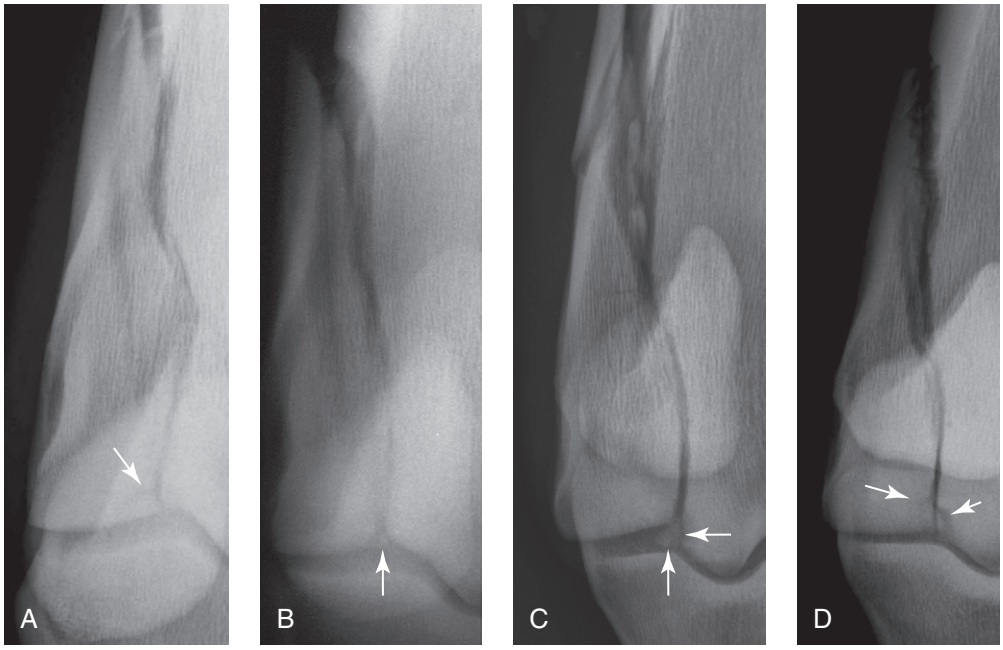

Figure 92-2. For preoperative evaluation of the distal palmar/plantar surface of the MCIII/MTIII, a 120-degree tangential view **(A, B)** or a flexed horizontal beam DP view **(C, D)** should be taken. *Arrows* indicate typical appearance of comminution along that margin that may be very difficult to see on a standard DP projection.

for developing degenerative joint disease. Surgical treatment is essential for displaced fractures because it provides anatomic reduction of the articular surface. Internal fixation renders the horse immediately more comfortable on the limb, thereby avoiding laminitis in the contralateral limb. The disadvantages of surgery are those attendant on all general anesthetic procedures as well as the risk of infection and surgical error. However, condylar fractures can heal successfully without internal fixation, and a conservative approach is a reasonable consideration in nondisplaced fractures where there are economic limitations.

NONDISPLACED FRACTURES

Nondisplaced fractures are repaired with screws applied in lag fashion using minimal soft tissue dissection. The site of screw insertion is determined by topographic landmarks and intraoperative radiography or fluoroscopy. Radiopaque skin markers, such as sterile skin staples, can be used preoperatively to select the sites for screw insertion. Alternatively, needles or other markers can be used intraoperatively. The distal screw is placed as centrally as possible in the lateral condylar fossa. This site can be accurately estimated by taking the midpoint of an imaginary line drawn between the proximal palmar prominence of P1 and the most dorsal aspect of the lateral condyle, both readily palpable landmarks. A stab incision is made down to the surface of the bone in this location and parallel with the collateral ligament fibers to minimize injury to them. The surgeon can feel the scalpel slide into the fossa, confirming accurate placement of the incision. Standard lag technique is used (see Chapter 76). As many additional screws as needed are placed at 15- to 20-mm intervals more proximad. Blindly placing a screw too close (less than 20 mm) to the proximal end of the fracture increases of the risk of splitting the narrow end or entering a fracture line. Correct positioning of the drill during the procedure is ensured through intraoperative radiography/fluoroscopy (Figure 92-3). Some surgeons prefer to place small-diameter (2 mm) marker bits prior to placing screws; the position and direction of the drill bit allows the surgeon to adjust drill/screw position. With either technique, two perpendicular views should be taken to check positioning in the dorsopalmar and lateromedial planes. Either 4.5- or 5.5-mm cortex screws can be used. It remains to be proved whether the larger screws yield improved results. Screw failure is rarely a problem in nondisplaced, noncomminuted fractures. The 4.5-mm screws have the advantage of being replaceable with a larger cortex screw if the bone threads are inadvertently stripped.

The majority of lateral condylar fractures can be treated with two screws. A few propagate proximad enough to justify more screws but usually the more proximal portion of the fragment is quite narrow in its dorsopalmar thickness, and accurate positioning of the screw in the center of that part of the fragment is difficult. There is little evidence to suggest that placing a third or fourth screw in the longer fractures results in a better outcome, particularly in nondisplaced fractures.

The most common technical errors in lag screw fixation of nondisplaced condylar fractures include inadequate compression and incorrect drill/screw placement. The former can be avoided by drilling the thread hole fully through the far cortex and being certain to completely tap the far cortex. The chances of incorrect proximal to distal aiming of the drill can be minimized by predraping positioning of the fractured bone parallel to the ground. Most horses have some degree of external rotation of the limb (especially hind limbs) when they are in lateral recumbency. The surgeon must account for this or the drill will be directed toward the palmar/plantar articular surfaces. Palpation of the medial and lateral wings of the proximal phalanx provides a good topographical key for the surgeon to direct the drill in a true lateral to medial direction.

Headless, variable-taper screws have been used successfully,[8] but their only advantage is the absence of the head and problems with a screw head in this location have never been documented. The headless screws provide less compression than standard cortex screws, have variable compression according to fragment thickness, and lack the versatility of standard lag technique, especially in displaced fractures.[9,10]

Unless there is a strong economic reason not to do so, arthroscopic examination of the dorsal and palmar/plantar

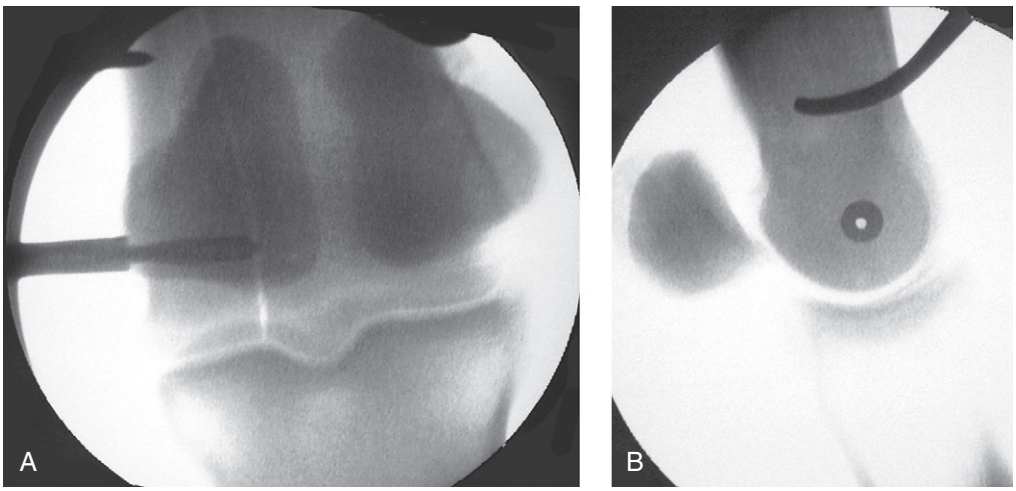

Figure 92-3. Intraoperative imaging is an essential element of consistently successful lag screw repair of condylar fractures. Imaging should *always* be done in two planes. A fluoroscopic DP view **(A)** showing the correct length of the glide hole and the lateral view **(B)** taken with the insert sleeve in place confirms that the hole's dorsopalmar orientation is correct. The distal screw is usually positioned in the condylar fossa to maximize compression at the articular surface.

joint should be done to confirm fracture reduction and characterize pre-existing or fracture-associated cartilage damage at the time of surgery.

DISPLACED FRACTURES

Similar lag technique and positioning of the screws is used for treatment of a displaced fracture. Unlike nondisplaced fractures, displaced fractures demand accurate anatomic reduction and often exhibit more severe injury to the joint surfaces. There is little room for error in the reduction of a displaced fracture in an athletic horse. Complete radiographic obliteration of the entire fracture line does not always occur, even with accurate reduction and screw placement and correct technique. This is caused by fibrin lodged within the fracture plane or bone loss along its margins. However, if the articular surfaces are aligned, the horse has a good chance to heal with minimal arthritic change. If there is a step on the major weight-bearing surface of the condyle after reduction, degenerative joint disease is likely. A significant malalignment of the condyle also puts very high bending and shear loads on the screws that may lead to bending or breaking of these implants.

The best technique for reduction of displaced lateral condylar fractures in most cases is by arthroscopic guidance.[11] To tentatively hold the reduction, the involved joint is dorsiflexed, and large pointed bone-reduction forceps are applied through the intact skin at about the level of the physeal scar.

The 4.5 mm glide hole is subsequently drilled through the fracture fragment using radiographic/fluoroscopic guidance. The glide hole should be drilled exactly to the fracture plane; this may be facilitated by temporarily removing the clamp so that it is possible to feel the drill enter the fracture plane. The insert drill sleeve and a 3-mm pin are placed in the fragment to use as a "handle." The arthroscope is then introduced into the proximal dorsolateral joint pouch. The fetlock can be flexed, extended, rotated and valgus/varus stresses can be applied until articular alignment is perfect. The pin inside the centering drill sleeve can be used for additional manipulation (Figure 92-4). When reduction is judged perfect, the reduction forceps are firmly reset and the remainder of the lag screw procedure is performed. The screw is inserted but not tightened. The arthroscope is repositioned and the articular compression and alignment are checked as the screw is firmly tightened. The palmar/plantar aspect of the joint is then examined arthroscopically through a second stab incision. Many comminuted fragments at the distal margin of the MCIII/MTIII are immobilized by the compression of the major fragment. If there are loose fragments, they can be removed under arthroscopic guidance. This is a difficult arthroscopic maneuver in some horses suffering from chronic arthritis and capsular fibrosis. The instrument portal must be carefully positioned at the base of the lateral proximal sesamoid bone and instruments (curettes/picks/probes) with angled ends are invaluable aids.

Occasionally, the fragment cannot be aligned properly because of interposed comminuted fragments. The pin and insert sleeve can be used to displace the main fragment laterally, allowing insertion of the arthroscope and cannula into the fracture plane. A dorsal instrument portal is positioned exactly over the fracture plane and the palmar fragments are retrieved. Obviously, this is done only in a displaced fracture.

If arthroscopic reduction is not feasible for some reason, displaced fractures should have enough of the fracture line exposed through an open approach to allow the surgeon to verify that reduction is accurate. In some cases this means a 2- to 3-cm incision over the proximal tip of the condylar fragment. In rare instances, it might mean a longitudinal arthrotomy along the dorsal fracture plane extending nearly to the proximal phalanx, but this should be a very unusual occurrence. It is worth noting that in many horses accurate reduction seems to get more difficult as the exposure is increased, presumably because more attachments are disrupted. It is always worthwhile trying to achieve accurate reduction using minimal exposure.

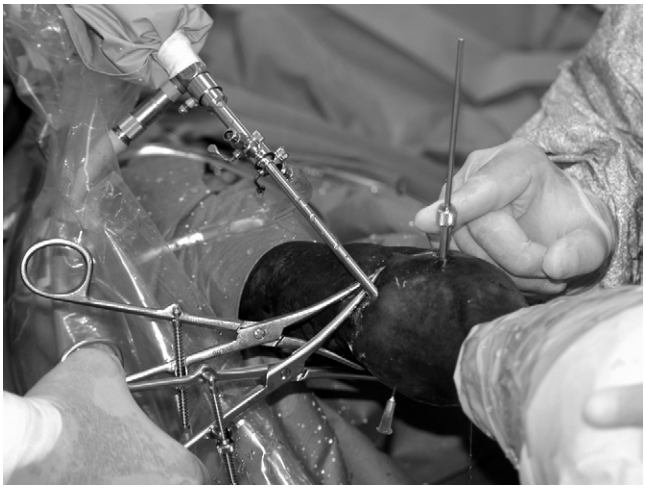

Figure 92-4. Arthroscopically assisted repair of a displaced lateral condylar fracture can be performed by drilling the glide hole and placing the insert sleeve before placing the scope. Fracture reduction is checked with the scope as the fragment is manipulated by flexion/extension/rotation of the hoof and manipulation of the fragment using the insert sleeve. Pointed reduction clamps are used to hold the fragment after reduction is achieved.

The majority of displaced lateral condylar fractures can be successfully treated with two distal screws, one in the epicondylar fossa and another 18 to 20 mm proximal to it (Figure 92-5). The fracture narrows quickly as it propagates proximad and comminution, sometimes occult, is common proximal to the physeal scar. More severe and displaced condylar fractures require more stability, so 5.5 mm screws should be considered. Placing three screws in a triangular pattern within the condyle also can increase the strength of the repair (Figure 92-6).

Since arthroscopic examination of the entire joint has become common, it has been recognized that injury of the proximal sesamoid bones is often associated with condylar fractures. Most proximal sesamoid bones damage involves varying degrees of erosions of articular cartilage, but more serious injuries also occur. Axial proximal sesamoid fractures are complications of displaced condylar fractures that should be identified preoperatively, because they are associated with a very unfavorable prognosis. If an axial sesamoid fracture is present, the condylar fracture should be repaired with the understanding that the horse has virtually no chance of becoming an athlete or resuming athletic activities. Axial proximal sesamoid fractures are crescent shaped and represent an avulsion of the axial margin of the lateral proximal sesamoid bone by the intersesamoidean ligament (Figure 92-7). With large displaced fractures, the fragment can be fixed with one or two 3.5-mm cortex screws, but primary fetlock arthrodesis is probably a better option. Most axial proximal sesamoid fractures have a slender rim of

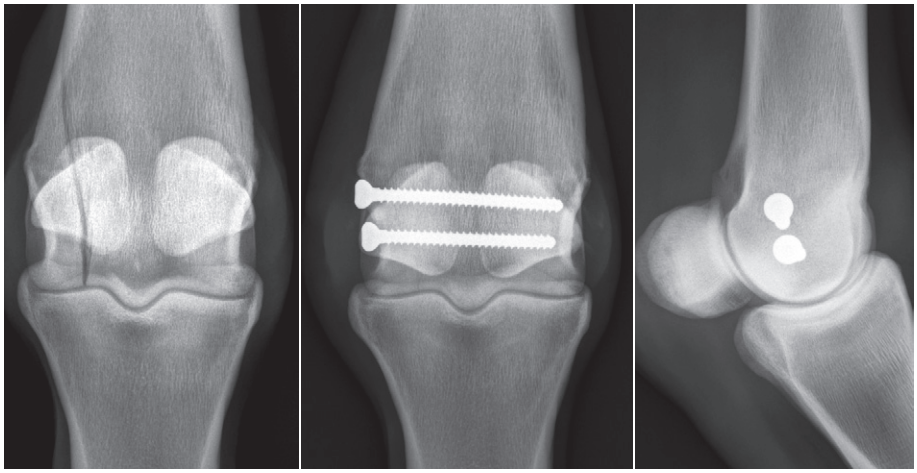

Figure 92-5. Nearly all lateral condylar fractures can be repaired with two cortex screws placed in lag fashion, one in the epicondylar fossa and the other near the physeal scar.

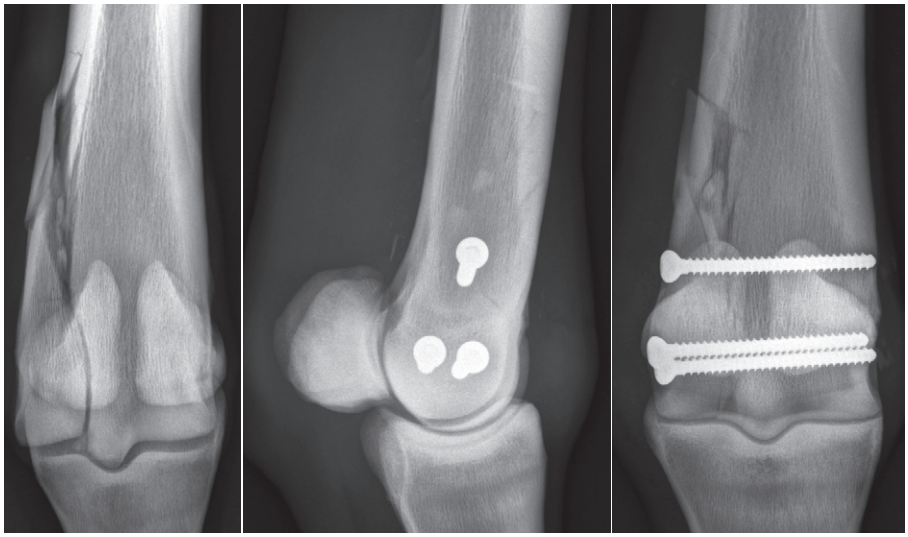

Figure 92-6. Badly damaged and displaced lateral fractures are inherently less stable and the screws are subjected to more bending loads. Both 5.5-mm screws and extra screws in the distal condyle should be considered to enhance stability. No effort is made to reconstruct the comminuted fragments at the proximal end of lateral condylar fractures.

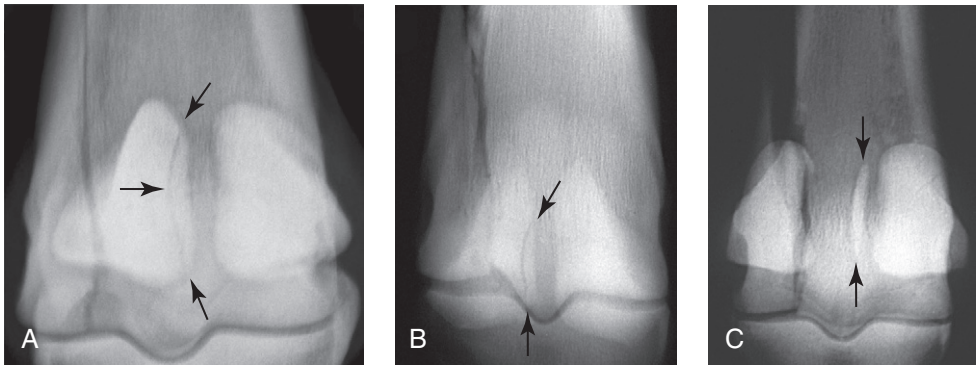

Figure 92-7. Axial sesamoid fractures associated with displaced lateral condylar fractures are a strong indication of major damage to the MCP joint. They can be seen on standard DP **(A)** and flexed DP **(B)** projections. **C,** If the fragment is completely separated *(arrowheads),* the best option is probably MCP arthrodesis.

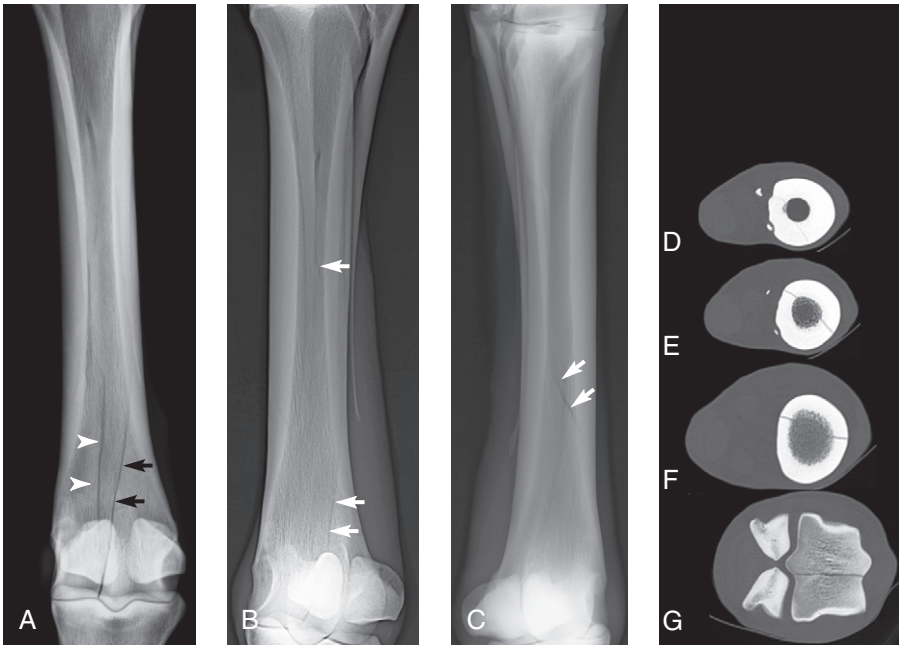

Figure 92-8. Dorsopalmar **(A),** DLPMO **(B),** and DMPLO **(C)** projections of a spiraling medial condylar fracture. Although the standard views can provide an adequate assessment of the fracture configuration, transverse CT images (proximal **[D]** to distal **[G]**) are even more helpful to the surgeon.

articular surface involved, but some fractures occur behind the articular surface and can be localized only with intraoperative radiographs or advanced imaging techniques, such as CT or MRI. Additional information on the management of proximal sesamoid fractures can be found in Chapter 91.

Nearly all lateral condylar fractures can be successfully treated with screws alone, but on occasion, lateral condylar fractures of MCIII also have diaphyseal spiral or oblique components that demand they be treated with more extensive fixation, as described later for medial condylar fractures.

Aftercare

Aftercare is discussed for all condylar fractures later.

Medial Condylar Fractures

Medial condylar fractures are seen in both the forelimb and hind limb of racehorses. Horses typically develop severe lameness immediately after exercise or are lame a few hours afterward. There is rarely significant soft-tissue swelling unless there is displacement of the fracture. In most cases, the site of the lameness is readily identified by manipulation of the MCP/MTP joint, and dorsopalmar/plantar radiographs clearly reveal the medial condylar fracture. Preoperative radiographs of any medial condylar fracture should include a full series of projections over the entire length of MCIII/MTIII. Medial fractures are quite different in terms of configuration, treatment, and complications. They are *not* the mirror image of a lateral fracture. Medial fractures nearly always propagate toward the axial aspect of the MCIII/MTIII, either spiraling up the diaphysis (Figure 92-8) or remaining nearly sagittal to the mid-diaphysis, where oblique fractures can later develop (Figure 92-9). Medial condylar fractures of MTIII are particularly prone to catastrophic failure with or without screw fixation, especially when the horse stands following general anesthesia. The risk extends for several weeks after repair.[12]

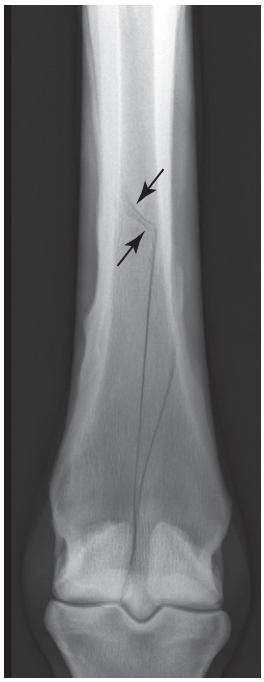

Figure 92-9. Medial condylar fractures of the third metatarsal that have an oblique diaphyseal component *(arrow)* are extremely dangerous fractures, prone to catastrophic dehiscence.

Treatment

Although internal fixation with multiple screws applied in lag fashion has been used successfully to treat many medial condylar fractures,[13] plating techniques are probably the most consistently successful technique. Screws alone are not optimal for any fracture with a known oblique diaphyseal component. If an oblique or transverse diaphyseal fracture is identified in the preoperative radiographs, a plating technique should definitely

Figure 92-10. Open exposure of a spiraling metatarsal fracture with the fracture line *(arrows)* "disappearing" in the mid diaphysis. The simplest method of defining the configuration of a medial condylar fracture in the diaphyseal region is to make an open approach with periosteal elevation. Implants can subsequently be placed accurately more easily.

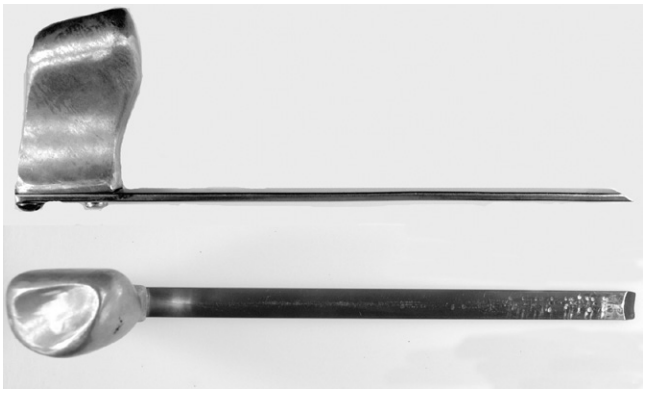

Figure 92-11. A custom-made plate passing device manufactured by sharpening the end of an old plate or by using a piece of steel stock of similar dimension. A solid handle is necessary to force it through the subcutaneous tissues.

be used. In hospitals (or horses) without options for controlled anesthetic recovery (e.g., pool or sling), *standing* screw fixation of the distal portion of the fracture has been shown to be surprisingly successful.[14] Preoperative CT imaging to evaluate fracture configuration and help optimize internal fixation may improve outcome, but a diaphyseal MTIII fracture of any configuration always poses a very serious risk.

OPEN APPROACH

The plate and screws can be placed from either the medial or lateral side, but the lateral approach has advanatages. The fragments are equal enough in size and the bone is so dense that adequate fixation can be obtained with lag screws even if the threads are in the smaller fragment. The advantages of a dorsolateral approach are that the operated limb is uppermost and a lateral plate is easier to remove under local anesthesia in the standing horse after the fracture has healed. The incision is made in one layer down to the level of the bone surface. The periosteum is elevated, and the skin and underlying subcutaneous tissues are retracted together. When the surface of the bone is exposed, the fracture line or lines are clearly identifed (Figure 92-10). The incision is continued distad to the level of the joint capsule, but the capsule is not incised. The distal two screws are placed through stab incisions using radiographic control similar to nondisplaced lateral condylar fractures. If the limb is positioned uppermost, the glide hole must traverse fracture plane and the thread holes engage the medial condylar fragment. This is ensured with intraoperative radiographs or fluoroscopy. A broad dynamic compression plate (DCP) or locking compression plate (LCP) is then positioned on the lateral aspect of the bone extending from just above the second screw to the most proximal portion of the bone. Typically 10- to 12-hole plates are used in MCIII and 12- to 14-hole in MTIII. It is possible to spirally contour the plate if the specific configuration of the fracture is evident, but a straight plate can usually be safely applied in these nondisplaced fractures. Exposure of the diaphyseal bone surface allows accurate placement of additional screws across the fracture plane and centrally within the fracture fragment. Either 4.5- or 5.5-mm cortex screws are used. Although anatomic placement of screws through the plate is straightforward, inadvertent injury to the suspensory ligament, splint bones, or both can occur. Obliquely directed screws or dorsopalmarly directed screws are drilled with care to avoid penetrating these structures, especially when using the 4-mm drill bit needed for the thread hole with 5.5-mm screws. Closure of the incision is routine if initially it was made in one plane. A simple-interrupted, cruciate, or simple-continuous pattern of synthetic absorbable suture material followed by routine skin closure is adequate. If special recovery systems (pool, sling) are not available, a full-length hindlimb cast should be considered for metatarsal fractures. Half-limb casts should not be used for metatarsal fractures. Forelimb medial condylar fractures are usually recovered in a full-limb Robert Jones bandage.

LESS-INVASIVE APPROACH

An alternative technique is to place the bone plate through a minimally invasive technique. This is particularly straightforward if there is no obvious proximal spiraling of the fracture. A 2-cm incision is made adjacent to the common digital extensor tendon at the level of the proximal MCIII/MTIII. A (previously used) broad DCP with a sharpened end attached to a handle (Figure 92-11) can be used to prepare a subcutaneous tunnel for the plate. After the subcutaneous (*not* subperiosteal) tunnel is made, a roughly contoured 10- or 12-hole broad 4.5-mm LCP is slid down along the bone (Figure 92-12). The "fit" of the plate is assessed palpably and with fluoroscopy. The plate is recontoured as needed and replaced. The holes in the plate can be easily palpated distally. Stab incisions are made over holes and screws are inserted routinely. After two screws are inserted, a plate of the same length is placed on the surface of the skin and incisions are made through that plate's holes. The remaining screws are inserted in routine fashion (Figure 92-13). The distal few screws in the plate may be placed in lag fashion if the fracture plane is radiographically visible at that level. Fluoroscopy or intraoperative radiography is used to check implant positioning. One or two skin sutures are used to close each stab incision.

Specific cautions with this technique include the need to avoid the contralateral MCIV/MTIV with the drill bits and screws, the difficulty in accurately measuring the depth of holes, and the need to check carefully that each screw is fully inserted into the plate. Although there is a possibility of inadvertently

placing a screw in a fracture line, the overall stability that the plate provides over the length of the bone appears to be adequate to prevent catastrophic dehiscence of the fracture. All possible precautions should be taken during recovery from anesthesia, especially in metatarsal fractures.

As mentioned earlier, another option for the treatment of high-risk condylar fractures is to place screws (typically two) to repair the distal portion of the fracture in the standing horse under sedation and local anesthesia. Although this still has a risk of catastrophic failure after fixation, the higher risk of fracture during recovery from general anesthesia can be avoided.

Aftercare

Convalescence of horses with condylar fractures involves stall rest with strictly controlled exercise for at least 2 months. Some turnout in a small paddock is allowed in the third month, and the patients usually returned to regular exercise in the fourth to fifth month. Displaced fractures or those with articular comminution are rested longer. The average time from surgery to first racing start is approximately 11 months. Although there is no uniform agreement on the need to remove screws from condylar fractures, many horses have returned to racing without screw removal, and most horses do not have screws removed after repair of typical fractures. When fractures extend into the mid-diaphyseal region, the screws are more likely to cause pain when horses return to intensive exercise. Therefore, diaphyseal screws are removed 3 to 4 months after surgery, usually with the horse under sedation and local anesthesia. The limb is clipped and scrubbed for routine aseptic surgery, and a number of sterile skin staples are placed over the heads of the screws to be removed. Radiographs are taken to facilitate exact placement of the stab incisions over the screws if they cannot be clearly palpated. Closure of the stab incisions is routine. After screw removal, the horse is continued in a walking and jogging program for 60 to 90 days before returning to galloping exercise.

Plates should be removed in horses expected to return to athletic pursuits. Removal of plates used to treat medial condylar fractures is performed with the horse standing under detomidine sedation and local anesthesia. These plates are typically removed about 70 to 80 days following surgery. Local anesthesia with direct infiltration over the implant or by means of a regional nerve block is applied and a short incision is made over the proximal 2 cm of the plate (Figure 92-14). The most proximal screw is partially removed. A matching bone plate is placed on the skin along the plate and stab incisions made through its holes. Each screw is backed out above the skin edge but not removed. After the screws are all counted, they are removed. This helps avoid groping for a screw that has already been removed as well as to avoid trying to remove a plate that still is secured with a screw. A battery-powered drill with the screwdriver attachment is quieter and easier to use in a standing horse. A 12-mm osteotome is used to pry the plate up at its proximal end. The osteotome is left under the end of the plate and then the plate is grasped with sterile vise grips and extracted proximally. A single skin suture per screw is placed and the

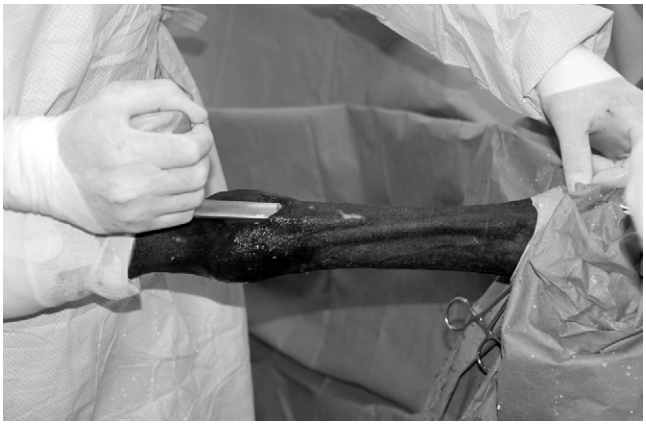

Figure 92-12. The plate passer creates a subcutaneous tunnel for the plate outside of the periosteum.

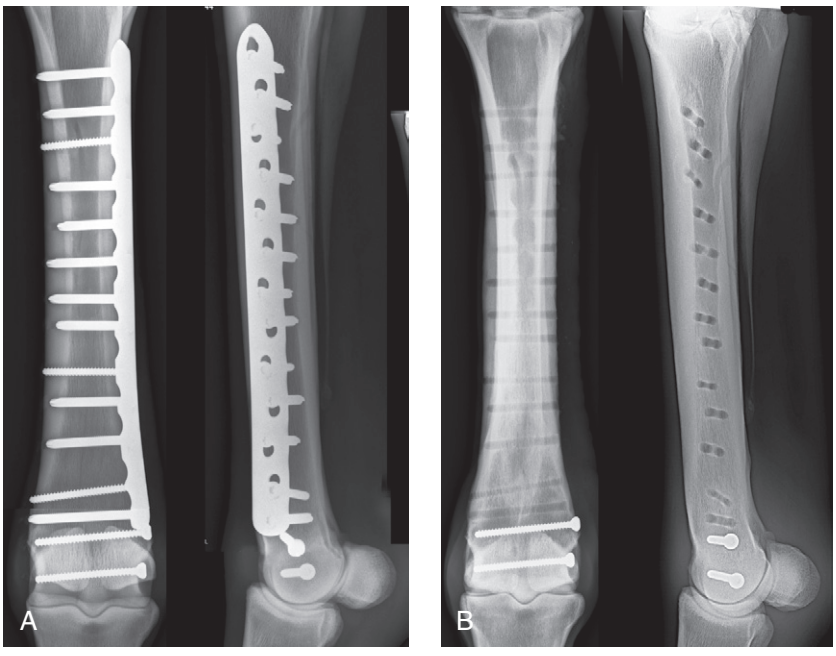

Figure 92-13. A, A medial condylar fracture repaired with distal lag screws and a subcutaneously positioned dorsolateral LCP. **B,** The plate was removed at 90 days. The lag screws are left in place.

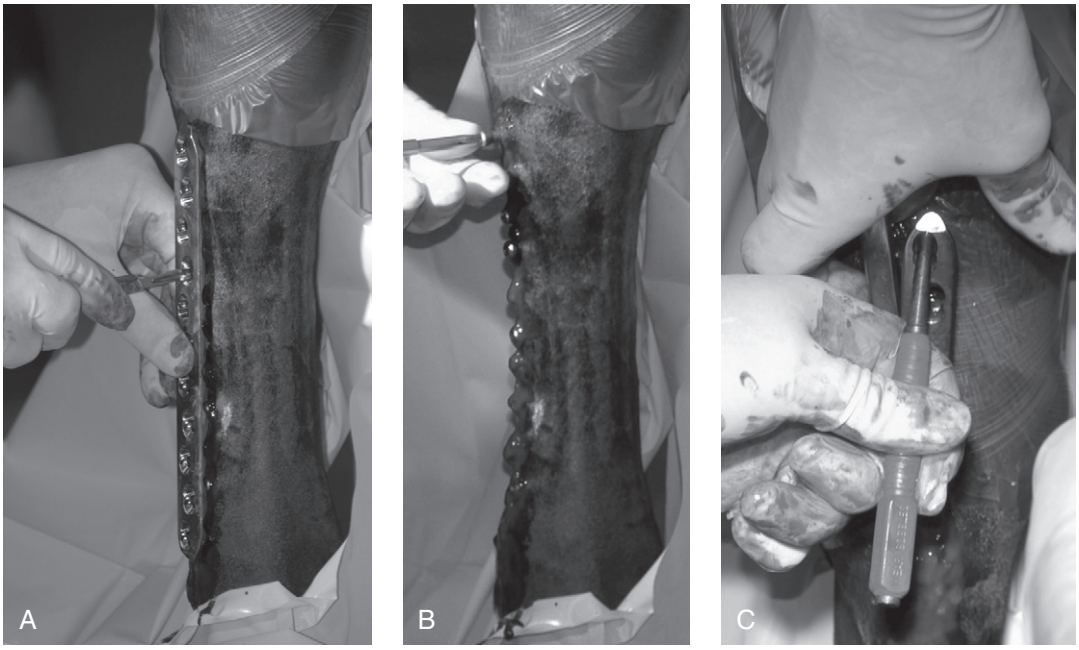

Figure 92-14. Plate removal is performed with the horse standing, using local anesthesia. **A,** A matching plate facilitates accurate stabs over the screw heads. **B,** All of the screws are partially removed. After all are loosened and counted, they are removed. **C,** A nail set and a mallet can be used to loosen the plate. If necessary, the proximal end is grasped with vise grips for removal.

larger proximal incision is closed in two layers. The horse is hand-walked for 60 days, receives paddock turnout for another month, then returns gradually to training.

Prognosis

Prognosis for condylar fractures is favorable (70% to 80% return to full function) if there is minimal pre-existing degenerative joint disease and the fracture is not displaced.[3-5] Displacement requiring open reduction of the fracture lessens the prognosis considerably, both because of the surgical trauma and because there typically is more injury to the joint surfaces (comminution, degenerative changes, proximal sesamoid bone injury, etc.). The prognosis for displaced lateral condylar fractures of MCIII is about 50% for return to racing, less if there is serious comminution or sesamoid injury. Lateral condylar fractures of MTIII have a better prognosis than those of MCIII. The prognosis for medial fractures is more variable and depends on the development of catastrophic complications. Because complications of medial condylar fractures can occur before surgery, during recovery, or even weeks afterward, by far the most important aspect of managing medial condylar fractures is to appropriately advise the owner of complications that can occur.

Diaphyseal Fractures

The most common major long bone fractured in horses is MCIII/MTIII, and diaphyseal fractures are encountered most frequently. They occur at any age and during any type of activity. Many occur as pasture accidents. There are favorable and unfavorable aspects to repairing fractures of MCIII/MTIII. Factors favoring successful repair are: (1) access and exposure of the entire diaphysis, making reduction and internal fixation feasible; (2) strong bone to which screws and plates can be affixed; and (3) the possibility of immobilization by external coaptation. Factors that do not favor repair are quite significant and include: (1) minimal soft tissue coverage, leading to many open fractures; (2) no muscles adjacent to the fracture, resulting in poor extraosseous blood supply to the fracture site; (3) sparse vascularity of the distal limb; and (4) frequent comminution, especially in older horses. Although the approach and treatment of MCIII/MTIII fractures is similar in all age horses, the chances of success are significantly greater in the younger, smaller patients.[15-17]

The diagnosis of diaphyseal MCIII/MTIII fractures is straightforward. In a frantic horse, radiography should be delayed until first aid has been administered and at least a preliminary decision has been made concerning treatment. It is particularly important not to risk a closed fracture becoming open by prolonged attempts to obtain radiographs. If the horse is unequivocally going to have definitive treatment, radiographs of an extremely unstable fracture can be taken through the external coaptation for preliminary assessment and can be repeated after the horse is anesthetized. Diaphyseal MCIII/MTIII fractures probably represent the single most important indication for appropriate external coaptation (see Chapter 17). Success or failure often hinges upon the immediate management.

Nonsurgical Management

Cast or splint coaptation as the sole treatment of displaced diaphyseal MCIII/MTIII fractures is rarely an acceptable approach. These fractures are unstable, horses almost never become comfortable with simple external coaptation, and problems in the contralateral limb develop quickly. Furthermore, the lack of soft tissue coverage over unstable cannon bone fractures may result in the fractures becoming open, even in a well-fitted cast. In young animals, even though fracture healing occurs

more quickly, cast coaptation is not the treatment of choice because permanent deformities develop rapidly in the contralateral limb and serious flexor/suspensory weakness occurs in the cast limb. Even if the fracture heals when treated in this manner, the problems associated with cast immobilization usually result in a less-than-optimal outcome.

Surgical Management

Internal fixation is the treatment of choice for diaphyseal MCIII/MTIII fractures. Although external skeletal fixation and intramedullary interlocking nails have been used, the optimal treatment remains double-plate fixation. Double plating is usually necessary in foals and always indicated in larger horses. A dorsolateral surgical approach is selected, splitting the common digital extensor tendon along its course down the bone. The skin incision is carried directly to the underlying bone down to the periosteum. The periosteum is elevated just enough along the fracture edges to allow visual control of reduction and reconstruction. Unlike in heavily muscled parts of the limb, reduction of MCIII/MTIII fractures is not usually difficult. Reduction of simple oblique or spiral fractures is maintained with large pointed reduction forceps or by insertion of strategically placed compression screws. The sites for screw insertion are selected after planning plate application. If the only practical sites for the screws will be under a plate, 3.5-mm screws that are deeply countersunk are placed to hold the reduction while the plates are applied. After reduction is secure with clamps, lag screws, or both, an aluminum template is used to help shape the two plates. Locking plates have major advantages in nearly all types of diaphyseal fractures. The plates are placed dorsolaterally and dorsomedially at 90 degrees to each other unless fracture configuration dictates otherwise. In a large adult horse, two broad plates with 5.5-mm cortex and 5-mm locking head screws are used, but in a foal, one broad and one narrow plate are adequate. The plates are staggered so screws placed perpendicularly through one plate will not interfere with those from the other. All screw holes in the plates should be used; even if a hole is directly over a fracture line. A screw can nearly always be inserted that will engage at least one cortex. Both plates should be as long as possible without involving the joints or physis. An autologous cancellous bone graft should always be used if there are any cortical defects. It is best to perform the surgery as soon as possible after the injury, because eburnation of the fragment ends occurs with delays and makes reduction difficult and the repaired fracture less stable. The tendon edges are apposed with interrupted synthetic absorbable sutures. Subcuticular and skin closure are routine.

A minimally invasive approach can also be used to repair selected diaphyseal metacarpal or metatarsal fractures.[18] For example, a severely comminuted fracture in a foal and a mildly comminuted fracture in an adult MCIII (Figure 92-15) may be spanned with two locking plates placed through stab incisions with screws inserted under fluoroscopic guidance.

The decision to place a cast versus use of a bandage on the limb depends on the horse's age, postoperative care, apparent stability of the repair, and available special recovery systems. A cast is used for protection of the repair during anesthetic recovery, but long-term protection of the repair with a cast is often undesirable because of the inevitable flexor weakness that develops. In general, foals younger than 1 year are not cast except for recovery. Casts are used in adult horses when the fixation is considered less than secure. The major complication of MCIII/MTIII fractures is infection because of the relatively poor vascularity of the region, poor soft-tissue coverage, a large metal-to-bone ratio, and the frequency of open injury. Implant failure (loosening or breakage) is possible but less of a concern if correctly sized and positioned implants are used.

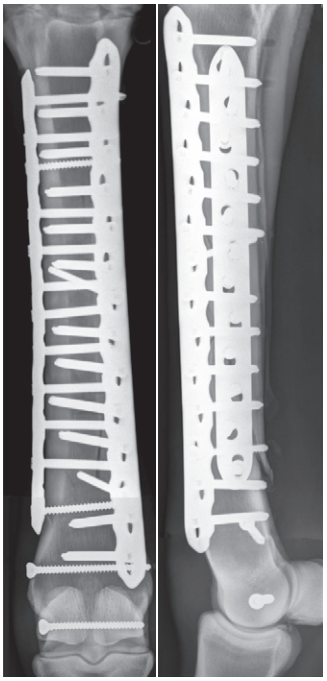

Figure 92-15. Postoperative radiographic views taken at right angles to one another of a multifragment MTIII fracture in an adult horse treated with two distal cortex screws applied in lag fashion, a lateral 12-hole LCP and a dorsal 14-hole LCP. In each plate, two 4.5-mm cortex screws were used with the remainder being of the locking head type. Note the proximal single screw was placed somewhat too far plantarad.

Aftercare

The same management as described earlier is applied. In any age horse, it is safest to stage the removal of the two plates. In foals, the first plate (usually the larger/longer of the two) can often be removed at around 3 months after implantation, and the second plate can be removed 45 to 60 days later. In neonates, these times can probably be safely shortened even more. An extraperiosteally positioned plate is usually still fairly easy to remove at 4 to 5 months, but overgrowth of bone can make the removal difficult. In most cases, the plate can be removed in a minimally invasive manner, but if bone overgrows the plate, a direct incision will be needed to remove the bone with an osteotome and mallet. In adult horses, staging also is recommended, although the earliest plate removal is likely to be around 4 months or so after implantation. As described earlier, standing removal should be strongly considered in adult horses. If general anesthesia is elected, great care should be taken during recovery from anesthesia.

Distal Physeal Fractures

Distal physeal fractures of MCIII/MTIII are common injuries in suckling and weanling foals. Nearly all are Salter-Harris type II fractures with a variable length of metaphyseal involvement

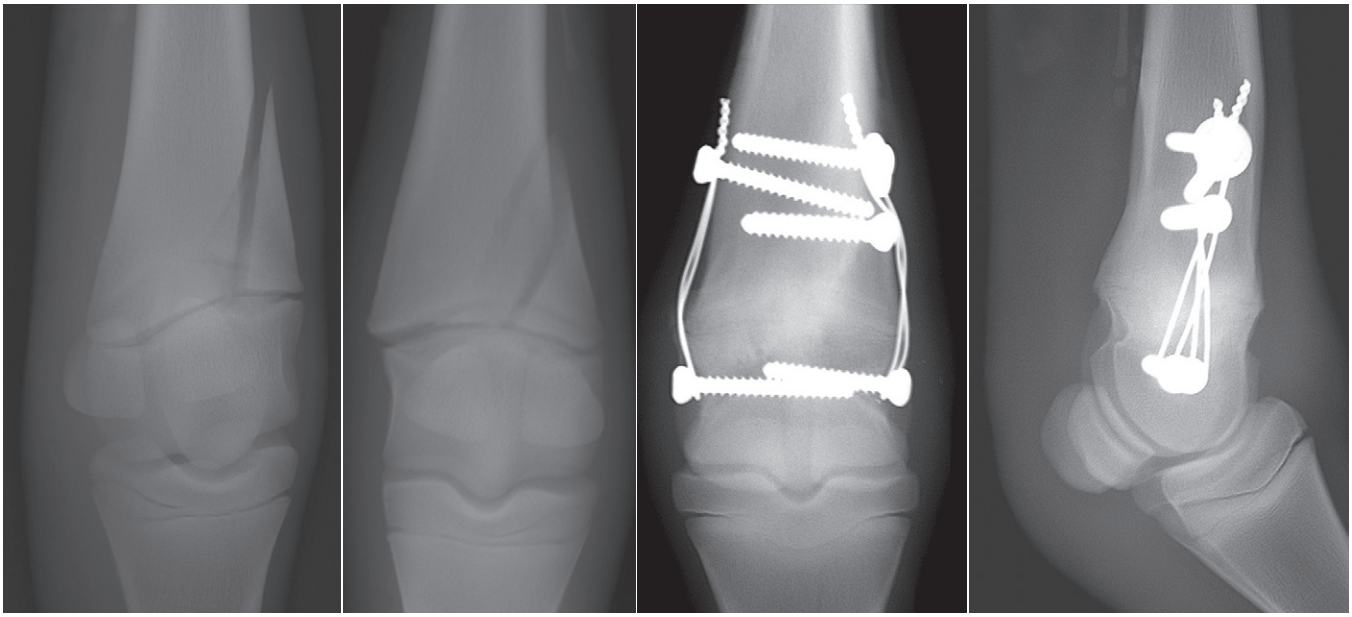

Figure 92-16. Although many methods can be successful for treatment of distal Salter-Harris II MC/MTIII fractures, a very reliable technique is to combine simple lag technique of the metaphyseal spike with a screw and wire transphyseal bridge. (Courtesy Dr. Alan J. Ruggles.)

(Figure 92-16). Such injuries usually heal relatively quickly with minimal surgical management. Young foals (less than 6 weeks old) with these fractures can be treated with cast coaptation alone for 2 to 3 weeks followed by 2 to 3 additional weeks in a splinted bandage. If the fracture appears reasonably stable and the foal is comfortable, even less coaptation may be adequate. In older, heavier foals or those with marked instability, the fracture can be repaired with compression screws through the metaphyseal component with or without a transphyseal bridge in addition to external coaptation. Although such minimal internal fixation is not inherently stable, it is adequate if combined with external coaptation in young foals because physeal injuries heal quite rapidly. It is preferable not to cross this growth plate with any implants in animals intended for athletic function. Regardless of the technique used to treat the fracture, the foal's return to exercise is carefully graduated over several weeks while the ligamentous and musculotendinous tissues regain strength.

The timing for implant removal depends on the age of the foal. If the foal is a neonate with significant remaining potential growth, the implants should be removed as early as possible in order to minimize the risk of a shortened bone. This could mean removal as early as 3 to 4 weeks in a neonate. In an older foal with negligible growth remaining, the implants can be removed at 2 to 3 months.

Proximal Articular Fractures

Proximal articular fractures of MCIII/MTIII occur sporadically in racehorses and can be confusing because the lameness associated with them is typically eliminated by local anesthetic injected into the middle carpal or tarsometatarsal joint. *Sagittal* fractures can be seen in any breed; most occur medial to the midline and are best diagnosed by nuclear scintigraphy and excellent-quality dorsopalmar/plantar radiographs. Occasionally, the fracture can be identified ultrasonographically as linear discontinuity in the proximal palmar/plantar cortex. *Frontal/dorsal* plane fractures occur in MCIII or MTIII. In Standardbreds, the fracture is most common in young pacers on the dorsomedial aspect of MCIII.[18] Proximal articular fractures have been treated conservatively with good success, but displaced frontal plane fractures are best repaired with internal fixation through compression screws (Figure 92-17). The sagittal plane fractures are rarely displaced and usually heal with stall rest alone (Figure 92-18). Implant removal is generally not necessary.

Dorsal Cortical Fractures

A large percentage of young Thoroughbred horses in race training develop pain and lameness associated with the dorsal cortex of MCIII; this is called *bucked shins.* In the majority of horses, no specific fracture line is identified, but some develop one or more oblique radiolucent lines in the dorsal cortex accompanied with focal pain and swelling. These so-called stress fractures occur most commonly in 3-year-old Thoroughbreds after they experience bucked shin problems in their 2-year-old season. The most common configuration of such fractures is a line coursing proximally at a 30- to 40-degree angle from the surface of the mid-distal dorsolateral cortex of the left MCIII (Figure 92-19). Typically, the fissure extends approximately 60% to 70% through the dorsal cortex and disappears, but in some cases, the crack curves proximad and courses back to the dorsal cortex, forming a true saucer fracture (Figure 92-20). Fractures may occur in the opposite configuration (proximodorsal to distopalmar, see Figure 92-19, *B*) as well as anywhere along the length of MCIII.

Nonsurgical Management

Treatment of dorsal cortical fractures can be conservative with anti-inflammatory agents and rest (stall and paddock) until the fracture is radiographically healed. Some trainers return the horse to a diminished exercise program as soon as the overt lameness abates and then simply follow the progress of healing

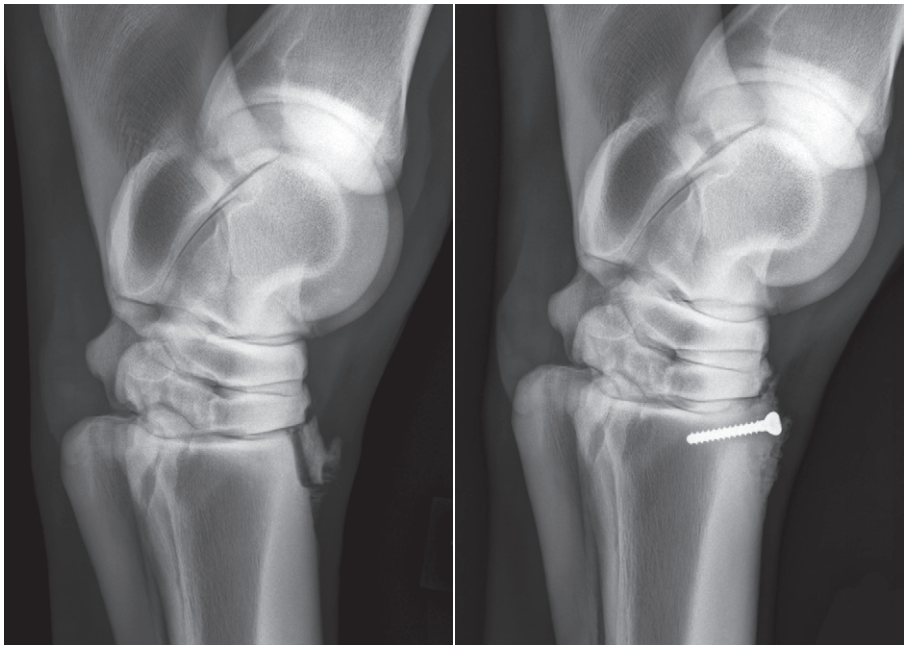

Figure 92-17. Displaced dorsal plane proximal metatarsal fracture treated with a cortex screw placed in lag fashion. This horse won two Eclipse awards after surgery.

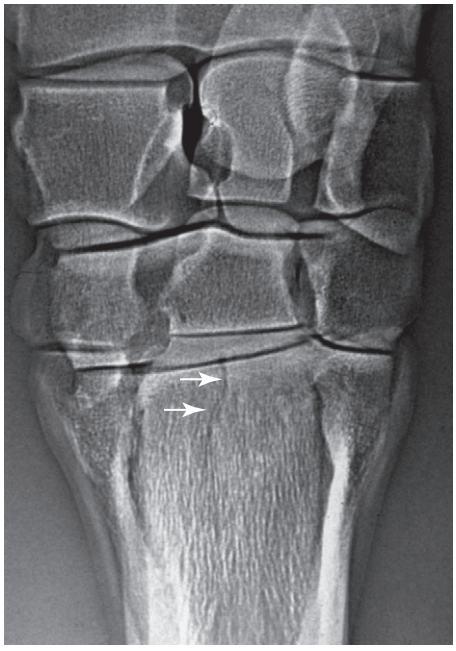

Figure 92-18. Proximal sagittal plane fracture of MCIII *(arrows),* which was treated conservatively.

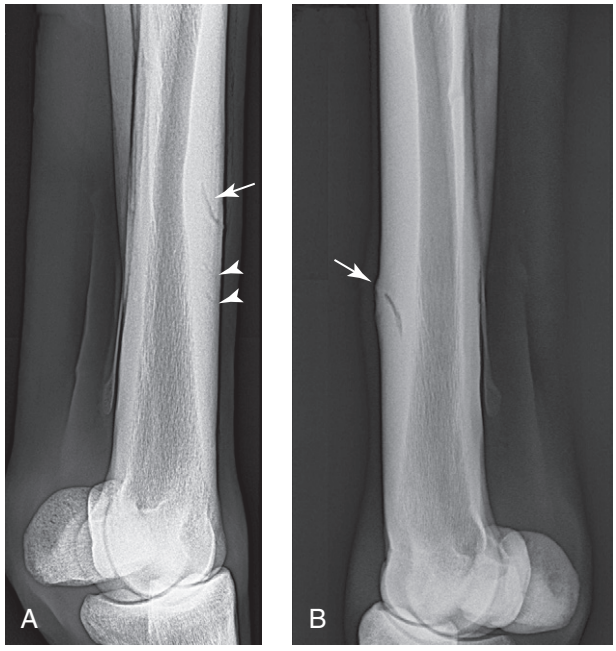

Figure 92-19. A, The most common configuration of dorsal cortical stress fractures of MCIII is shown with the *arrow*. Less distinct lucencies *(arrowheads)* can be seen separately or in combination with larger fractures. **B,** Occasionally, the opposite ("upside down") configuration is seen. The quality of digital imaging has made identification of these fractures more reliable.

with sequential radiographs. Many horses heal without surgical intervention, but a significant percentage has no signs of radiographic healing even after several months. The best candidates for conservative treatment are fractures in the distal or proximal metaphyseal regions of MCIII because they develop periosteal bridging callus much more rapidly than the typical intracortical fracture in the diaphysis.

Extracorporeal shock wave therapy has also been extensively used in the management of bucked shins and dorsal cortical fractures. There is little evidence that it has beneficial effects on healing and it is more likely that its primary effect is local analgesia.

Surgical Management

Surgical treatment of dorsal cortical fractures of MCIII is osteostixis alone or in combination with an intracortically placed screw.[19,20] The advantage of osteostixis alone is that it avoids a second operation to remove the screw. More consistent results have been achieved with the combination followed by routine screw removal at 60 days.

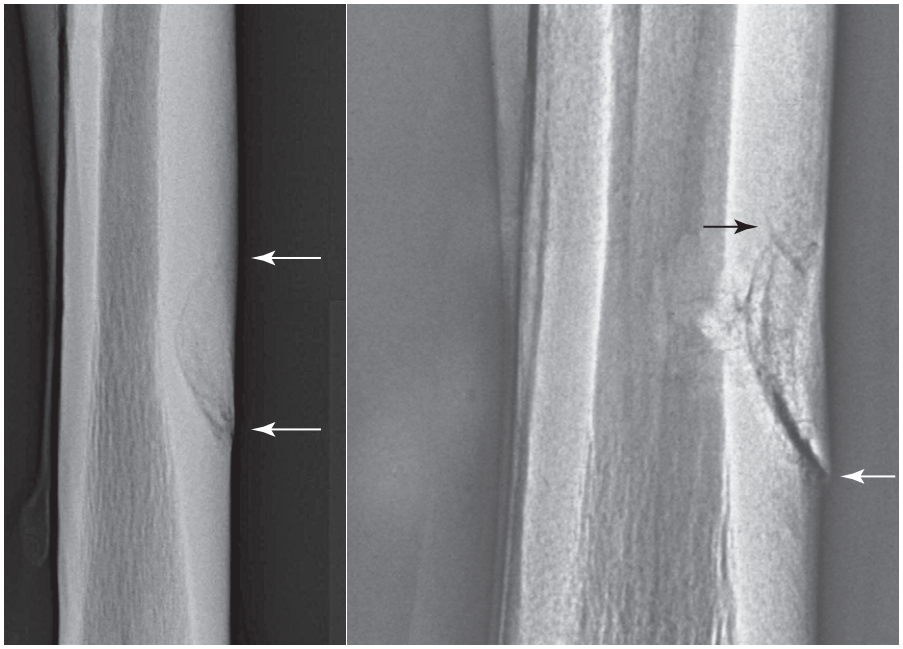

Figure 92-20. The term *saucer fracture,* originally derived from this uncommon configuration. This type of fracture is probably more prone to catastrophic dehiscence. *Arrows* depict the proximal and distal aspects of the fractures.

The surgery can be carried out either under general anesthesia or under sedation and local anesthesia. Local anesthesia has the obvious advantage of avoiding the risks of general anesthesia and recovery but the possible disadvantage of unexpected movement during drilling, leading to broken drill bits/taps. It is important to accurately locate the exact site of the fracture preoperatively because it can often be very difficult to identify it visually at surgery. A useful technique is to take a radiograph of the limb after surgically preparing the skin and place multiple skin staples in a grid over the affected region, varying the orientation of the staples so they can be easily identified. This allows precise positioning of the incision over the fracture.

A 4- to 6-cm incision is made over the fracture site. In the typical dorsolateral fracture, the incision is between the common digital and lateral digital extensor tendons. The incision is made boldly directly to the level of the periosteum to minimize dissection. The periosteum is elevated, and self-retaining retractors are placed to expose the bone surface. Although some surgeons use lag technique, typical dorsal cortical metacarpal stress fractures have no displacement and the effect of compression would be trivial. Because compression of the fracture is not necessary for successful treatment,[20] a position screw (without a glide hole) within the dorsal cortex is frequently placed. Because of the bending that occurs in the mid-diaphysis of MCIII, a screw across both cortices is not recommended. Drilling with a small bit in dorsal MCIII must be done with irrigation and frequent cleaning of the bit to avoid breaking it or causing thermal injury. The fractures can be difficult to identify even with open exposure of the dorsal cortex. An oblique drill hole is made into the medullary cavity and an intraoperative radiograph taken (Figure 92-21). If the hole is correctly placed, it can be used for the screw; if is too proximal or distal, an adjustment is made and the first hole serves as another osteostixis hole. Because the screw is inserted at an acute angle (perpendicular to the fracture line), countersinking is necessary to avoid bending at the head as it is tightened. The amount of countersinking should be minimal, however, because excessive depth of the head will make screw removal much more difficult. Most dorsal cortices accommodate a 22-mm screw. Six to eight 2.5-mm holes are made through the dorsal cortex around the screw. All holes must be drilled toward the medullary cavity (Figure 92-22) and separated at least 10 mm. Closure and bandaging of the area are routine.

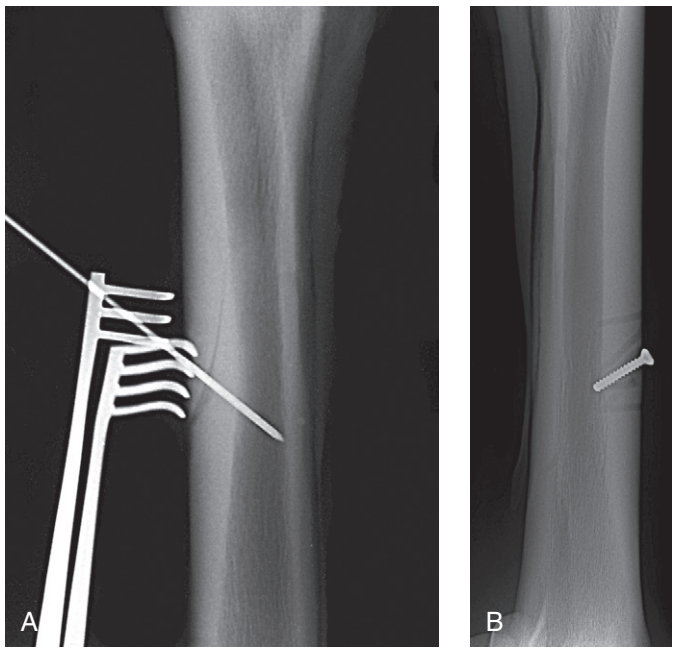

Figure 92-21. A, A single intraoperative image with a pin in the drill hole allows accurate repositioning if needed. **B,** Postoperative image showing the screw inserted and the surrounding osteostixis holes.

Postoperative care includes stall rest and hand-walking for 2 to 4 weeks followed by 4 to 6 weeks of stall rest and paddock exercise. At 60 days postoperatively, the horse is returned for screw removal, which is easily performed in the standing animal

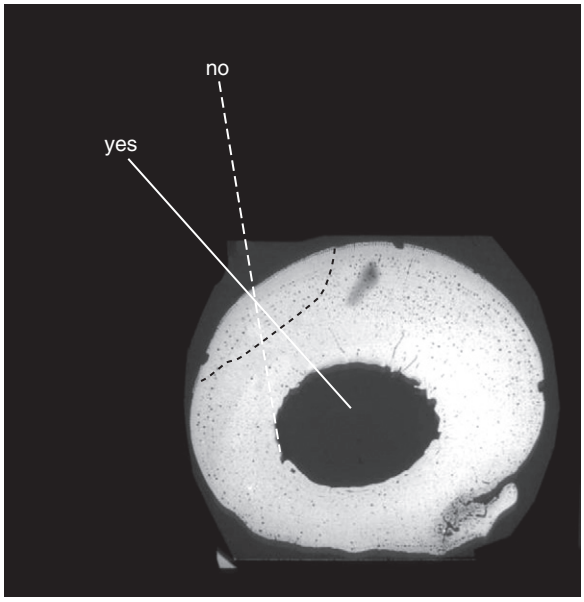

Figure 92-22. When drilling dorsal cortical stress fractures, it is important to aim toward the medullary cavity.

under sedation and local anesthesia. If the screw head cannot be palpated, a grid of sterile skin staples over the area can be placed and a radiograph taken to facilitate localization of the ideal site for a small incision. Local infiltration at the site of the screw head is adequate. Because the hexagonal recess of the 3.5-mm screw head is quite shallow, it is easily stripped; therefore, the screw head recess should be completely cleaned with a needle, sharp hook, or small pin before inserting the screwdriver tip to avoid this complication. If it does strip, the head is grasped with locking pliers and removed. After screw removal, the horse is hand walked for another month then begun in a jogging program for about 6 to 8 weeks before returning to regular race training.

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CHAPTER 93

Vestigial Metacarpal and Metatarsal Bones

Michelle A. Jackson and Jörg A. Auer

Although vestigial in nature, the small metacarpal and metatarsal bones (splint bones) are integral parts of the supporting and stabilizing structures of the equine limb and serve as a site for important carpal and tarsal ligament insertions. Traumatic injuries of these bones are predisposed by their relative anatomic exposure, the nature of the horse, and equine management practices.[1] The most common conditions affecting these bones are fractures and splint exostosis. Congenital defects like polydactyly occurs occasionally. Tumor-like lesions are very rare.[2]

ANATOMY

The proximal aspect of the small metacarpal or metatarsal bones articulates with the carpal and tarsal bones, respectively, and provides axial support to these structures. Unlike the other vestigial metacarpal/metatarsal bones, the fourth metatarsal bone (MTIV) has only a small articulation with the fourth tarsal bone, providing minimal weight transfer through this articulation.[3] The metacarpal/metatarsal interosseous ligament is very variable among horses, ranging from a pure ligamentous structure to osseous union (Figure 93-1, *A*). A firm fascia covers the tendons in the proximal region of the third metacarpus (MCIII) and metatarsus (MTIII) and runs from the medial to the lateral splint bone (see Figure 93-1, *B*). A bandlike structure extends distad from the distal end of the splint bone toward the proximal sesamoid bone (see Figure 93-1, *C*).[4] In the hindlimb, the lateral dorsal metatarsal artery's location between MTIII and MTIV make it particularly vulnerable to accidental or surgical injury.

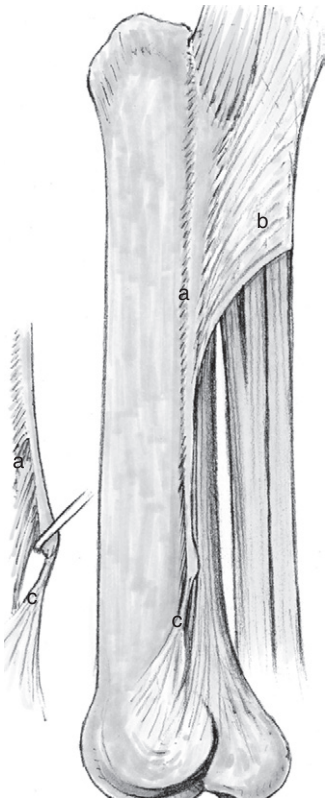

Figure 93-1. Important soft tissue structures surrounding the splint bone.[4] *a,* Metacarpal/metatarsal interosseous ligament; *b,* fascia metacarpi/metatarsi palmaris/plantaris; *c,* ligament-like structure originating from the distal end of the splint bone.

CONDITIONS

Fractures

Fractures of the small metacarpal or metatarsal bones are relatively common in horses of any age. Kicks from other horses are probably the most important cause of injuries to this bone,[5] but fractures may also occur spontaneously during exercise. Splint bone fractures can be open or closed, simple or comminuted, and localized in the proximal, middle or distal part of the bone (Figure 93-2). The horse presents with different grades of lameness, but it is usually severe in open, proximal fractures and moderate in distal fractures. The interval between the occurrence of the injury and the time of admittance at the clinic also has a significant influence on the lameness. The presence of swelling, pain, and heat is directly related to the extent of soft tissue damage. The definitive diagnosis can be obtained through a number of diagnostic imaging techniques; however, radiography is the most frequently applied modality. It is very important to take several radiographic projections and to

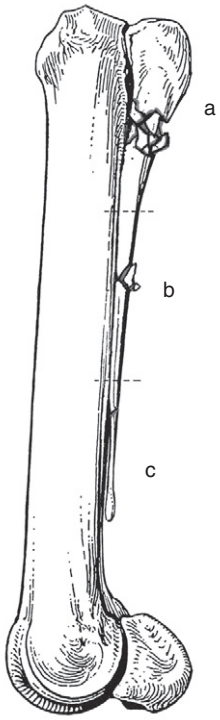

Figure 93-2. Classification of splint bone fractures: The bones can be divided into three equal parts: *a,* proximal; *b,* mid-body; and *c,* distal.

include the proximal articulation. Ultrasonography should be performed to assess concurrent injury to the suspensory ligament. Computed tomography, if available, can be very helpful for a definitive diagnosis in difficult cases (Figure 93-3). Involvement of the MCIII/MTIII affects the prognosis negatively. Complications like nonunion, osteomyelitis, and sequestrum or excessive callus formation are common when the fracture is not treated properly.[1]

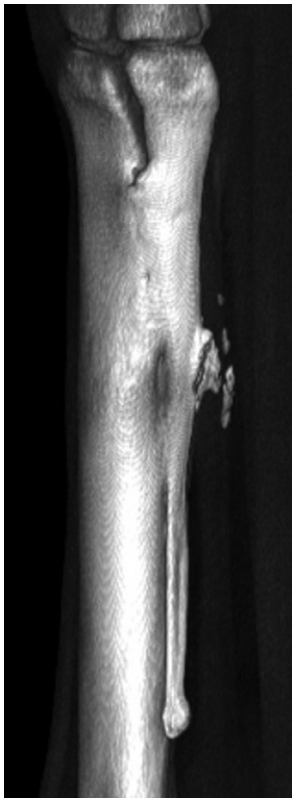

Figure 93-3. Longitudinal CT view of MCII and MCIII, demonstrating calcification near the splint bone.

Proximal Fractures

Proximal splint bone fractures are usually the result of a kick from another horse and therefore are frequently open and may be comminuted. They may become complicated by the development of osteitis, osteomyelitis, and/or bone sequestra. According to some studies, MTIV is the most commonly involved bone. MCIII/MTIII and the articulation between the splint bone and the carpal and tarsal bones should be examined carefully for fractures or fissures.

TREATMENT

Proximal splint bone fractures are the most difficult to treat and a variety of management techniques including surgical interventions have been described.[3,6-10]

Standing surgical intervention

Open multifragment proximal splint bone fractures are candidates for standing surgery, consisting of a thorough wound débridement under sedation and local anesthesia, with removal of loose fracture fragments and temporary installation of a drain.[1,11,12] Postoperative management includes antimicrobial and anti-inflammatory treatment in conjunction with support bandaging. Follow-up radiographs are taken 2 and 4 weeks later. If the wound has healed and the radiographs show that the splint bone is stable and the fracture is healing, no further treatment is necessary (Figure 93-4).[1] The patient should be maintained in a box stall for 1 month followed by 2 months of hand-walking exercise or small paddock.

In one report, 12 of 14 horses with open multifragment splint bone fractures returned to athletic function after conservative therapy.[11] Other authors reported that healing occurred in the majority of horses that were treated with only wound débridement and no additional stabilization or splint bone removal.[1] Another study compared conservative and surgical treatment of open multifragment fractures of MTIV and yielded comparable results with respect to return to full work and cosmesis, with conservative therapy being significantly less expensive and associated with less morbidity.[12]

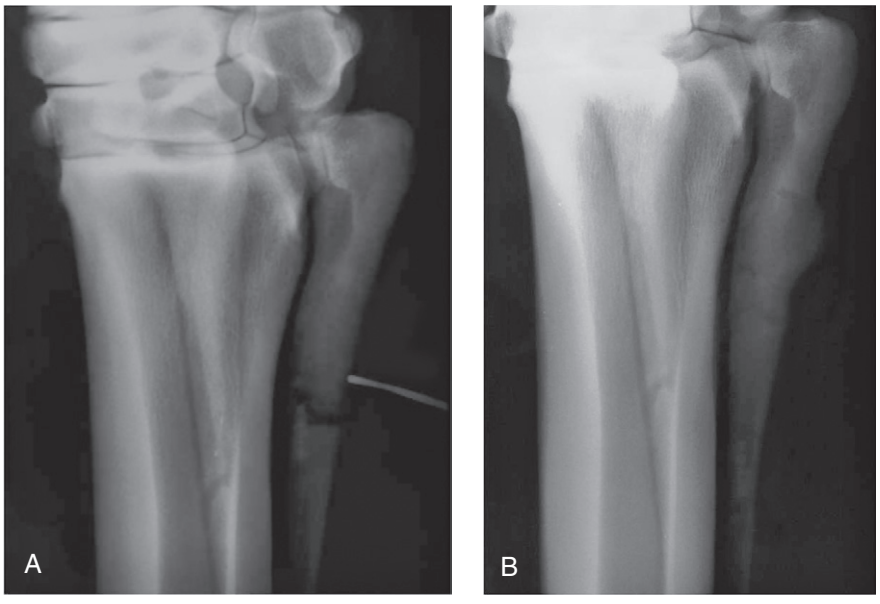

Figure 93-4. A, Proximal, multifragment, open splint bone fracture treated with standing wound débridement. **B,** Healing after 7 weeks.

Internal fixation

Removal of fracture fragments plus the residual distal splint bone is thought to provide a rapid return to function.[7] It is generally accepted that no more than the distal two thirds of the splint bone should be removed, with the exception of MTIV. In cases where more than two thirds of the splint bone has to be removed, internal fixation of the proximal fragment to MCIII/MTIII with the help of a small plate is suggested to maintain axial support.[9]

Because implantation of metallic implants bears a high risk of postoperative infection, particularly if the original injury was caused by external trauma, their use should be reserved for cases in which there is a high probability of luxation or subluxation of the proximal splint bone fragment. The other option and preferred technique for internal fixation is plate application in which the screws only engage the splint bone, providing the most ideal anatomic repair (Figure 93-5).[9] If the screws inserted through the plate holes cross the splint bone and penetrate MCIII/MTIII, horses often remain lame, so that the plate will need to be removed about 3 to 4 months postoperatively (Figure 93-6). Appropriate implants include 3.5 mm (narrow) dynamic compression plates (DCPs) or locking compression plates (LCPs), semitubular plates or reconstruction plates (Synthes Inc., Solothurn, CH). Principles of plate application do not differ from those of other fractures (see Chapter 76). The ligamentous attachments on the abaxial surfaces of the proximal splint bones make the palmar or plantar abaxial aspect a tension surface, making it the ideal location for plate application.[14] Screw fixation alone can be useful in selected cases, such as simple fractures with minimal displacement (Figure 93-7). However, this is generally associated with a relatively high rate of technical failure.[9]

Postoperatively no cast is needed. Horses should be kept in a box stall for 1 month, followed by walking exercise for an additional 2 months or turnout in a small paddock.

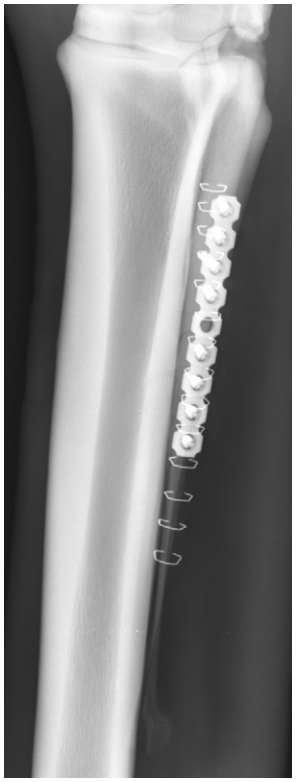

Figure 93-5. Radiographic image showing fixation of a proximal splint bone fracture using a 2.7-mm Unilock plate, with screws engaging only the splint bone.

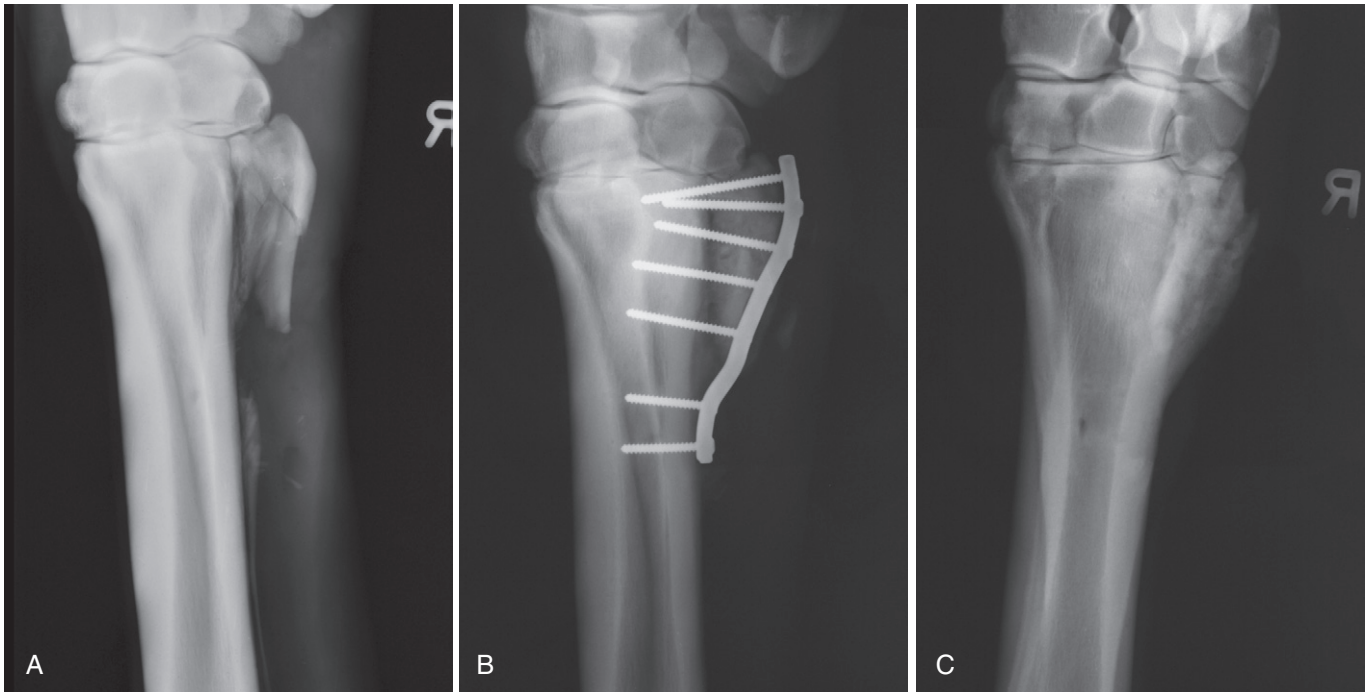

Figure 93-6. Radiographic images of an open proximal comminuted splint bone fracture. **A,** The fracture before repair showing a large gap between the proximal and the distal parts. The missing fragments were lost at the time of injury. **B,** Fusion of the splint bone to the cannon bone using a 4.5 mm DCP. **C,** Removal of the plate after healing of the fracture.

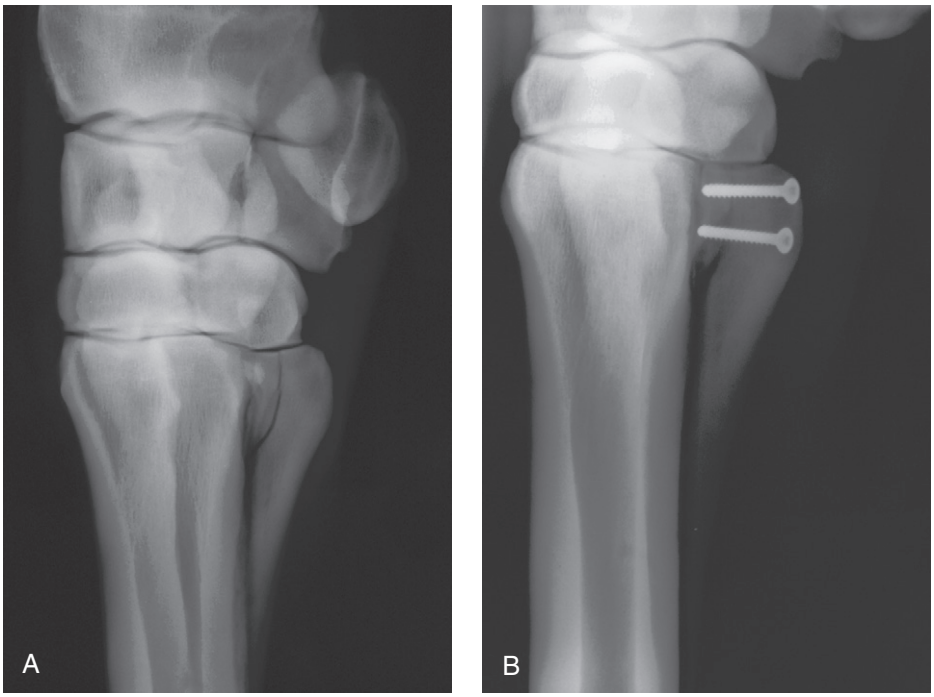

Figure 93-7. APLMO radiographic view. **A,** Proximal, articular, closed splint bone fracture; **B,** Fixation with two 3.5 mm screws.

Segmental ostectomy

A segmental ostectomy with removal of the injured sections of the splint bones can be performed in some complicated fractures composed of multiple small fragments.[10] The horse is usually anesthetized and placed in lateral recumbency; however, a standing procedure with the horse under sedation has also been described.[10] A linear skin incision is made immediately over the affected portion of the splint bone. Contaminated and necrotic tissues are débrided and any loose bone fragments, osseous callus, and sequestra are removed. The remaining proximal and distal portions of the splint bone are subsequently obliquely ostectomized with an osteotome or an oscillating saw. After flushing of the wound, subcutaneous tissue and skin are closed in two layers. In open fractures, a drain is placed in the wound for 2 to 3 days.

Firm pressure bandages are maintained for 2 weeks and horses are rested in a stall for 1 month followed by 2 months of hand-walking or small paddock exercise. Follow up radiographs are recommended to evaluate healing and stability of the proximal fragment.

In one study, three horses with open, proximal multifragment fractures treated with segmental ostectomy returned to normal activity within 8 weeks after surgery with excellent cosmetic results.[10]

Removal of the entire fourth metatarsal bone

Complete removal of the entire MTIV to treat open multifragment fractures of the proximal portion of this bone has been successfully performed.[3] In one report, five of eight horses returned to their intended use and two more were pasture sound.[3] Because of possible subsequent instability or luxation of the tarsometatarsal joint, a full-length hindlimb cast should be considered for recovery and for the first 4 weeks postoperatively (Figure 93-8).

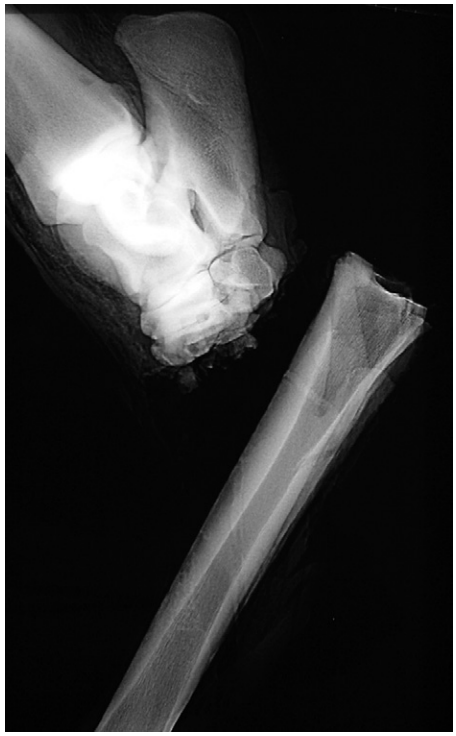

Figure 93-8. Radiographic image showing luxation of the tarsometatarsal joint that occurred during recovery after complete removal of MTIV.

PROGNOSIS

Prognosis for return to athletic function after treatment of proximal splint bone fractures depends on type and age of the fracture as well as additional involvement of soft tissues and MCIII/MTIII. Complicated fractures including open, comminuted, and

articular fractures have a good prognosis after standing surgical intervention without removal of the distal part of the bone or fixation of the fracture.[1,11] Plate fixation is recommended only when the proximal part of the splint bone has become unstable relative to MCIII/MTIII.[1] The prognosis after internal fixation of closed fractures is excellent.

COMPLICATIONS

Possible complications of standing surgical intervention are excessive callus formation, which can interfere with the suspensory ligament, nonunion, and instability of the proximal fragment.[7,13]

The major complication after internal fixation is infection, particularly if the original injury was caused by external trauma. Plate removal may be necessary after healing of the fracture if infection occurs. However, because the splint bones are exposed to limited load bearing, most fractures heal, even in the presence of infection.[14]

Possible complications after segmental ostectomy include instability of the proximal fragment, sequestration of the distal fragment, and excessive exostosis formation at the splint bone ends.[10]

Midbody Fractures

TREATMENT

Surgical management

Midbody fractures should be managed surgically with removal of the distal fragment of the splint bone, removal of the callus, and excision of the distal-most aspect (1 cm) of the remaining proximal fragment (Figure 93-9).[7] Alternatively, a segmental ostectomy can be performed (Figure 93-10).[10] Before closure of the wound, all fragments should be removed and the wound flushed thoroughly. In open fractures, a drain is placed in the wound for 2 to 3 days. This treatment ensures quick recovery of the horse and return to previous use.

Nonsurgical management

Conservative treatment is often chosen for financial reasons. Good candidates are horses that have chronic fractures with minimal callus formation and patients with nondisplaced fresh fractures.

PROGNOSIS

The prognosis for midbody splint bone fractures after surgical treatment is very good.[1] Thirteen horses with mid-body splint bone fractures treated with a segmental ostectomy returned to normal activity within 8 weeks after surgery with excellent cosmetic results.[10]

The outcome after conservative treatment is favorable, but the convalescent time is usually longer.

COMPLICATIONS

Complications after surgical treatment are rare and include exostosis formation at the splint bone and/or MCIII/MTIII, which leads to a longer convalescence. A rare complication, which can occur during removal of the distal splint bone, has been reported.[15] A horse treated by partial ostectomy of a splint bone fracture with an oscillating saw suffered an avulsion fracture of the plantar MTIII cortex during a fall 1 month postoperatively (Figure 93-11, *A*). The fracture originated at the horizontal saw cut that extended into the cortex of the MTIII, which occurred accidentally during the osteotomy.[15] A bone plate was applied to the plantarolateral aspect of the remaining splint bone with the screws extending into MTIII (see Figure 93-11, *B*). The fracture healed uneventfully.

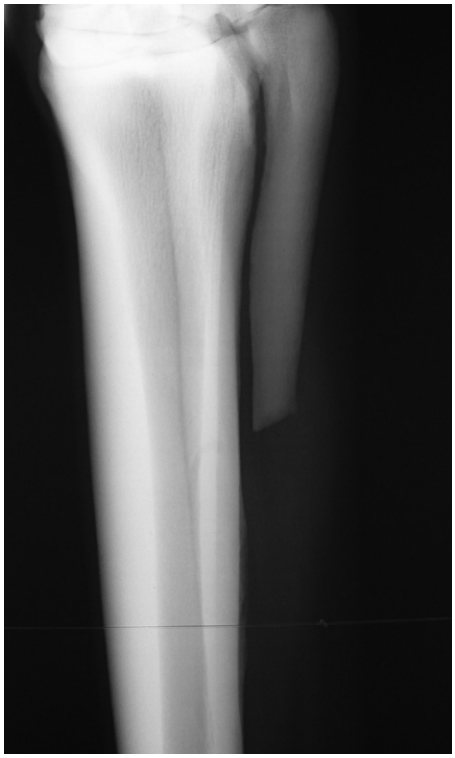

Figure 93-9. Postoperative image showing appearance after surgical removal of the distal half of the splint bone for treatment of a mid-body MTIV fracture.

A possible complication after conservative treatment is excessive callus formation, which can lead to secondary suspensory desmitis. Some cases require surgical removal of the fragment at a later time. Another possible complication is non-union, which most likely is the result of continuous intermittent traction exerted by the collateral ligaments.

Distal Fractures

Fractures of the distal third of the splint bone are usually simple in nature and occur most frequently at the narrowest part of the bone or immediately distal to the attachment of the interosseus ligament.[16] Distal fractures are very common and can result from external or internal trauma. Internal trauma, especially in the forelimb, occurs secondary to excessive stress with extension of the carpus during exercise or secondary to suspensory desmitis with loss of pliability of the suspensory ligament.

TREATMENT

Surgical management

Although distal splint bone fractures can be treated conservatively, surgical excision of the distal fragment improves the prognosis (Figure 93-12).[17] The surgery is technically simple and can be successfully performed under general anesthesia or with the horse standing under local anesthesia.[14] An incision is made directly over the bone, cutting down to the bone surface over the length of the fragment. The distal tip of the fragment is grasped with towel clamps or tissue forceps and elevated. The

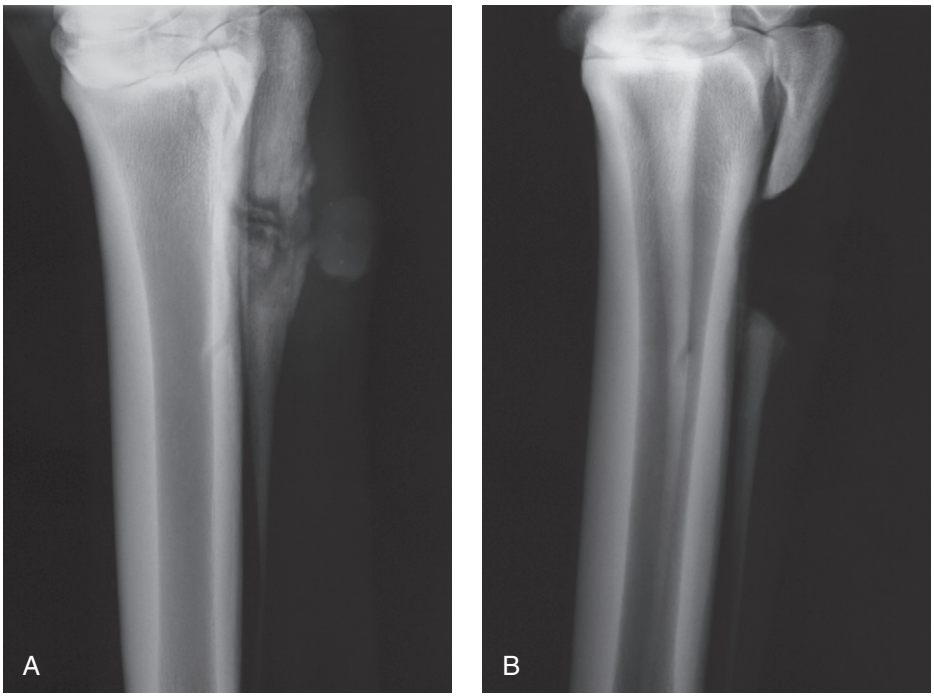

Figure 93-10. A, Radiographic image of an open, multifragment mid-body fracture of MTIV. **B,** Postoperative image showing the performed segmental ostectomy.

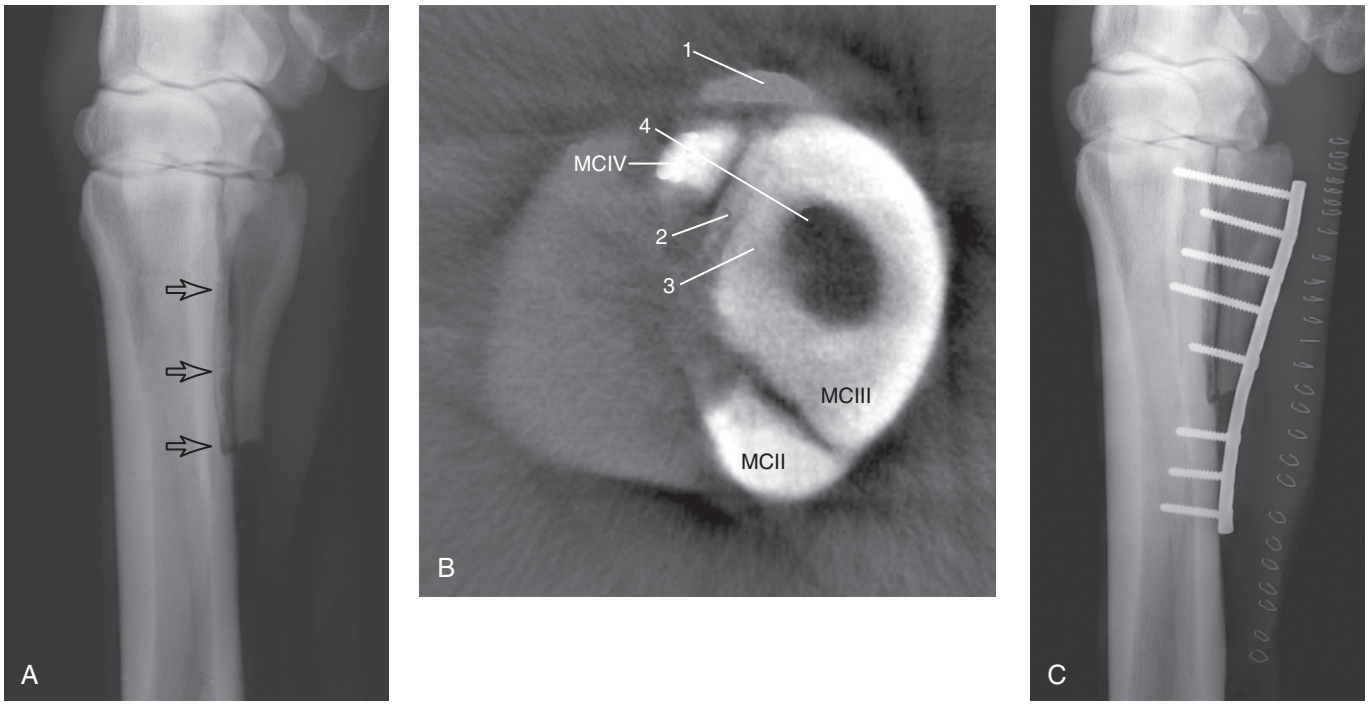

Figure 93-11. A, Avulsion fracture of the palmar MCIII cortex *(arrows),* which developed during a fall 1 month after partial ostectomy of a splint bone fracture with an oscillating saw. Note the horizontal saw cut in MCIII at the most distal *arrow.* **B,** Intraoperative cross-sectional reconstruction (Siremobile ISO 3D, Siemens Medical Solution, Erlangen, Germany) at the level of the fracture: *1*, Periosteal new bone fomation protecting the fracture; *2*, fragment in the lateropalmar cortex of *MCIII; 3*, intact lateropalmar cortex of *MCIII; 4*, direction of screw implantation through the plate. **C,** A 9-hole 3.5-mm DCP applied over the MCIV with the screws extending into MCIII.

attachments between the distal fragment and MCIII as well as the bandlike structure that extends distad from the distal end of the splint bone to the suspensory apparatus are severed with heavy curved scissors or a with a scalpel. As upward traction is applied on the fragment's extremity, sharp dissection is continued proximad to approximately 8 to 16 mm proximad to the fracture site. After the distal fragment and the fracture callus are separated from MCIII/MTIII, the splint bone with its periosteum is transected obliquely with an osteotome or oscillating saw approximately 1 cm proximal to the fracture. Before closure

of the wound, all fragments should be removed and the wound flushed thoroughly. Meticulous hemostasis is indicated to reduce the risk of postoperative excessive new bone formation at the distal stump of the bone. In open fractures, a drain is placed in the wound for 2 to 3 days.

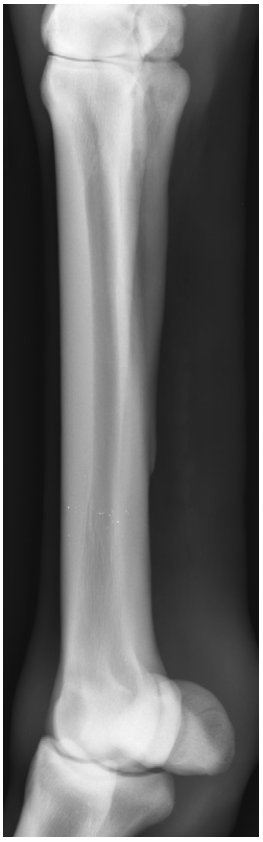

Figure 93-12. Postoperative image of a simple distal splint bone fracture after removal of the distal fragment.

PROGNOSIS

Prognosis depends to a great extent on the coexistence and degree of suspensory desmitis,[18] sesamoiditis, and/or metacarpophalangeal joint disease.[14] Thus, it is essential to preoperatively evaluate adjacent tissues by means of physical examination, radiographs, and ultrasonography. If no other structures are involved, prognosis after surgical removal of the distal splint bone is excellent.

COMPLICATIONS

A possible complication after conservative treatment is excessive callus formation, which could interfere with the suspensory ligament and lead to persistent lameness.[19]

Exostoses

Exostoses (splints) occur mostly in young, immature horses and develop between MCIII and the splint bones. Splints may occur at any level but most commonly develop 6 to 7 cm distal to the carpometacarpal joint and involve MCII (Figure 93-13, *A*). Possible causes for splints are direct trauma leading to subperiosteal hemorrhage and elevation of the periosteum, instability between MCIII and MCII, MCII fractures, or inflammation of the intercarpal ligament.[20] The last can result from too much work on hard surfaces or on a circle, especially in immature horses, or from conformation abnormalities (such as bench knees) or carpus varus deformities. Lameness is usually present in the acute stage, especially if the patient is lunged on hard surfaces, and tends to deteriorate with work. Horses develop a firm swelling at the level of the exostoses, which is warm and painful on palpation.

DIAGNOSIS

Radiography can be useful to determine the size and the activity of the exostosis. Oblique radiographic projections of the affected splint bone should be taken using soft exposures (see Figure 93-13, *B*). It is important to assess the axial part of the exostosis

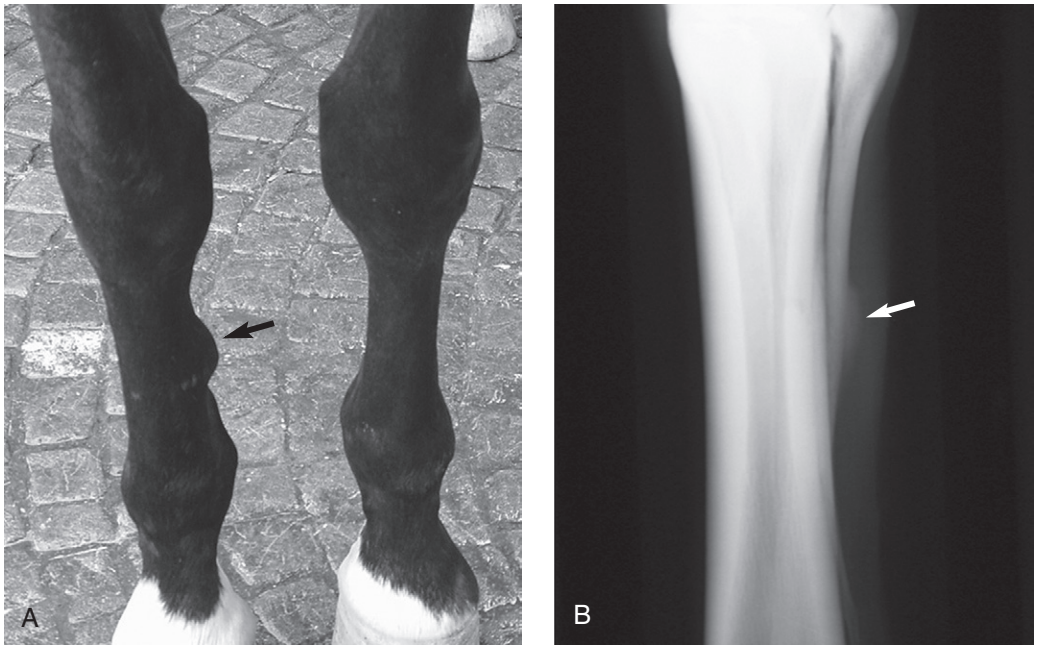

Figure 93-13. A, Photograph of a horse with splint exostosis between MCIII and MCII about 6 to 7 cm distal to the carpometacarpal joint *(arrow)*. **B,** Oblique radiographic projection of MCII with soft exposure showing the size of the exostosis *(arrow)*.

to determine if there is impingement on the suspensory ligament. These cases (hidden splints) are best diagnosed with dorsopalmar radiographs; in difficult cases computed tomography can be very helpful.

TREATMENT

Nonsurgical management

Lameness associated with a clinically active exostosis usually resolves with rest, ranging from 2 to 3 weeks to 2 to 3 months. Local application of dimethylsulfoxide may facilitate reduction of the swelling; infiltration of the exostosis with corticosteroids has a similar effect. In horses with severe exostoses, surgical removal of the exostosis and the distal part of MCII should be considered.

Surgical management

If the axial exostoses are impinging the suspensory ligament and the lameness does not resolve with rest, surgical removal of the exostosis should be attempted. The best technique is a simple linear incision over the exostosis and *en bloc* excision of the entire bone mass along the cleavage line between the new bone and MCIII.[14] This excision should include the overlying periosteum. An oscillating saw or a sharp-bladed osteotome is used to cut the excessive bone and return it to a normal contour. After hemostasis is achieved, the loose subcutaneous tissues are closed over the exposed bone surface, and the skin edges are apposed in routine fashion. In very large exostoses, a portion of the skin margin is excised to reduce dead space and the associated tendency for hematoma or seroma formation. The most critical aspect of managing cosmetic splint removals is postoperative bandaging. A tight bandage applying direct pressure over the surgery site followed by an outer padded compression bandage should be kept in place for 2 to 3 weeks to minimize the accumulation of blood or serum at the surgical site.[14]

Polydactyly

Polydactyly of the metacarpal or metatarsal region occurs occasionally and is the most frequently described congenital phalangeal malformation in the horse (Figure 93-14, *A*).[21] In 80% of the cases the supernumerary digit occurs at the medial aspect of the forelimb.[21] The etiology of polydactyly is unknown in the horse. Usually the digit represents an atavistic form of a regular small metacarpal/metatarsal bone. Other described forms are the teratogenic form, where the supernumerary digit evolved from a teratogenic splitting of the basipodial elements and all the phalangeal bones are affected, but the splint bones are often normal. The bilateral symmetric-inherited form represents yet another hereditary condition in poultry and dogs.[21] The diagnosis is based on clinical examination. A complete radiological examination of the involved metacarpal and metatarsal region and adjacent joints is required to evaluate the osseous abnomalities (see Figure 93-14, *B*). The supernumerary metacarpus or metatarsus and digit should be removed to improve cosmetic appearance and to prevent injury to the digit (see Figure 93-14, *C*).[21] The surgery should be performed under general anesthesia. An incision is made directly over the supernumerary digit starting at mid cannon bone and continuing distad up to the supplementary hoof. The supernumerary digit has extensor and flexor tendons, a suspensory ligament, and sesamoid bones like the other digits, as well as nerves and vessels. The vessels are ligated and transected together with the tendons as far proximad as possible. An osteotomy of the digit (usually MCII) is performed at its middle part with an osteotome or oscillating saw.

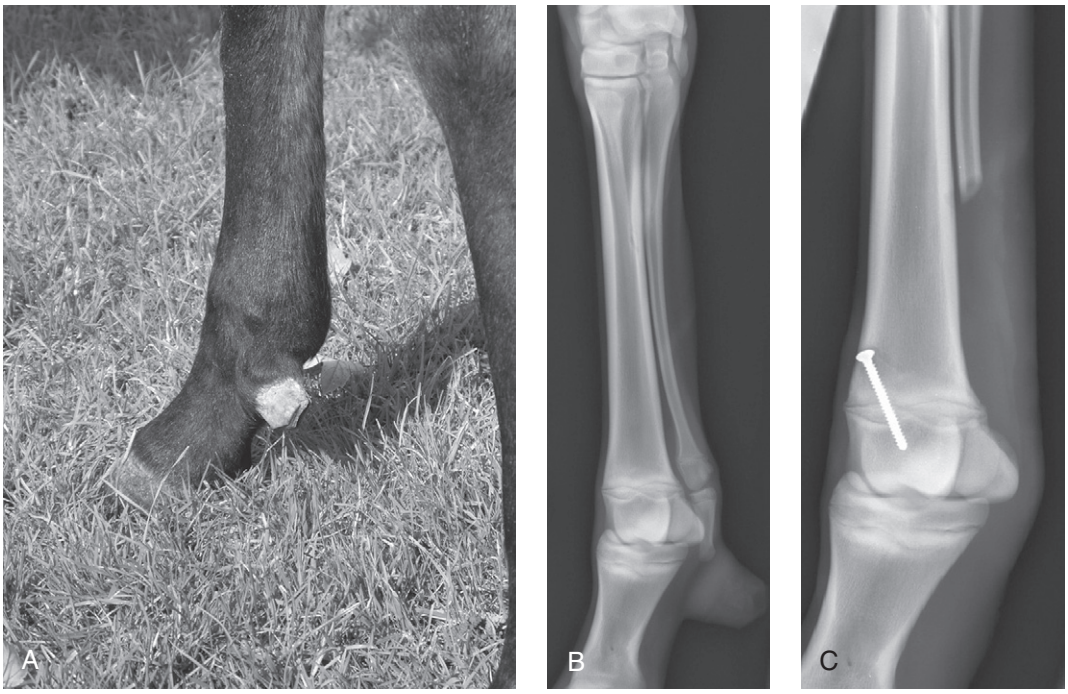

Figure 93-14. Polydactyly in a foal. **A,** The rudimentary medial (second) digit shown on the limb from the side. **B,** Oblique radiographic view depicting the rudimentary digit. **C,** Postoperative oblique radiographic view showing the resected distal half of MCII. Note the placement of a transphyseal screw on the lateral side of the distal MCIII for retarding growth, because the rudimentary digit led to a varus deformity of this bone.

Neoplasia

Fibro-osseous tumors of the appendicular skeleton are very rare in horses.[2] An ossifying fibroma, usually found primarily in the jaw of young animals, has been described in the distal part of the fourth metacarpal bone of a 13-year-old horse.[2] The horse showed neither lameness nor pain on palpation; after surgical removal of the distal third of the splint bone, including the tumor, wound healing occurred without complications. Eighteen months after surgery radiographs showed no recurrence of the mass.[2]

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CHAPTER 94

Carpus

Alan J. Ruggles

Carpal injuries in the horse occurs in all breeds but most often in racing breeds. Types of injuries include trauma from direct blows or kicks, fractures or subchondral bone injury from stress adaptive response, lacerations and septic conditions, and soft tissue injury to ligaments or tendons. Diagnostic procedures should be aimed at specifically identifying the site of the injury in the carpal region. The complicated anatomy of synovial structures in this region occasionally jeopardizes this goal. This chapter summarizes anatomy, diagnositic procedures, surgical considerations, and outcomes associated with carpal injury in the horse.

ANATOMY

The carpal region contains three joints: the antebrachiocarpal joint, middle carpal joint, and carpometacarpal joint. The middle carpal and carpometacarpal joints always communicate with each other. This fact is important when evaluating regional anesthesia of the proximal palmar metacarpal region. The palmar outpouchings of the carpometacarpal joint extend distad. Inadvertent puncture to this joint, and subsequent anesthesia of this joint and the middle carpal joint, may occur when performing regional anesthesia of the proximal metacarpal region.[1]

Injury of the carpometacarpal joint is mostly desensitized by intra-articular anesthesia of the middle carpal joint. However, it has been demonstrated that analgesia of the antebrachiocarpal and middle carpal joints is not specific. In one study, diffusion of mepivacaine between the antebrachiocarpal and middle carpal joints occurred in 84% of the tested limbs, and diffusion between the middle and antebrachiocarpal joints occurred in 96% of tested limbs.[2] Therefore, a positive response to intrasynovial anesthesia of one portion of the carpus might not specifically implicate that particular joint as the source of lameness. This information confirms clinical experiences in horses that block out variably in the carpal and proximal metacarpal region. In practical terms, consider evalaution of the whole region (carpus, carpal canal, and proximal metcarpus) when lameness is alleviated by regional or intra-articular anesthesia of the carpal region.

The bones that compose the carpus include the radius; the proximal row of carpal bones (radial carpal, intermediate carpal, and ulnar carpal bones); the distal row of carpal bones (second, third, and fourth carpal bones); the metacarpal (MC) bones II, III, and IV; the accessory carpal bone; and occasionally a first carpal bone (but rarely a fifth). The orientation of the carpal bones relative to themselves is maintained by their soft-tissue attachments, including the intercarpal ligaments,

collateral ligaments, fibrous joint capsule, and the palmar carpal ligaments. The extensor carpi radialis and common digital extensor tendons as well as their sheaths span the carpus dorsally and dorsolaterally, respectively. They are maintained in their position over the dorsal surface of the carpus by the extensor retinaculum located at the distal radius approximately at the level of the physeal scar. The lateral digital extensor tendon courses over the lateral side of the carpus, as does the tendon of the ulnaris lateralis. Palmar and medial to the palmar carpal ligaments is the carpal canal, a synovial structure in which the superficial and deep digital flexor tendons, median artery, and nerve are located as well as the accessory ligament of the superficial digital flexor tendon (ALSDFT). Immediately medial to the carpal canal is the tendon and tendon sheath of the flexor carpi radialis, which is an important surgical landmark for desmotomy of the ALSDFT.

Effusion of carpal joints at the dorsal aspect of the carpus is easily recognized if these structures harbor an inflammatory process. The lateral recesses of the antebrachial and middle carpal joints will also be distended and the effusion can be more easily found by applying pressure over the dorsum of the carpus to distend these recesses. The anatomic borders and clinical significance of these structures has been documented.[3-5]

Arthroscopic evaluation of the palmar aspect of the antebrachial and middle carpal joints can be accomplished for diagnostic procedures and for removal of osteochondral fragments. Areas that can be accessed via palmar approaches to the antebrachial and middle carpal joints include the caudal medial and lateral radius; the palmar aspects of the radial, ulnar, second, third, and fourth carpal bones; and the dorsal aspect of the accessory carpal bones. The palmar bundles of the medial palmar ligaments are easily recognized, but the palmar bundles of the lateral palmar intercarpal ligament are not accessible.[5]

The function of the bony and ligamentous structures of the carpus is to dissipate axial loading during weight bearing. The anatomic position of the carpal bones and the resultant transmission of forces to the intercarpal ligaments protect the weight-bearing surfaces of the carpal bones during exercise to prevent injury.[6] Loss of interosseous fibrous connections promotes instability, lameness, and osteoarthritis. The support of the cuboidal bones and the compressive stiffness of the hyaline cartilage are key to carpal function.

PATHOPHYSIOLOGY

Injury to the carpus most commonly occurs within the antebrachiocarpal and middle carpal joints. Injuries of the proximal metacarpus, which can involve the carpometacarpal joint, are discussed in Chapter 92. Osteochondral fragments develop at both the dorsolateral and dorsomedial aspects of the antebrachiocarpal joint. Injury of the middle carpal joint nearly always involves the medial aspect of the joint—that is, the radial facet of the third carpal bone, the distal aspect of the radial carpal bone, and occasionally the medial aspect of the distal intermediate carpal bone.

High-energy injuries, such as falls or kicks, to the carpus cause acute single load failure. The resultant injury often causes open or comminuted fractures and carpal instability. Additional details on stabilizing luxations of the carpus are found in Chapter 81. Single load injuries including luxations of one or more carpal joints are less common than those developing as a result of repetitive loading. Axial, cyclic loading of the carpus during training induces stress-adaptive remodeling of the carpal bones. Alterations in bone porosity, sclerosis, and associated stiffness can predispose the bone to fracture.[7]

Exercise induces regional changes in bone density, which can be associated with the characteristic location of carpal injury. In one study, Thoroughbred horses exercised on a treadmill had increased trabecular thickening and increased bone mineral density in the third and radial carpal bones, in areas where cartilage degeneration and fractures commonly occur.[8] Previous work had identified increased areas of bone stiffness in the radial facet of the third carpal bone.[7] Loss of compressive stiffness of the subchondral bone plate as a result of exercise, which is radiographically manifested by sclerosis, likely contributes to fractures of the carpus.

Likewise, cartilage injury has been correlated with underlying bone sclerosis.[9] One study demonstrated significant increases in the mean tissue density in the distal, dorsal aspect of the radius, the radial carpal bone, and the third carpal bone in response to exercise.[10] This correlates with areas of increased sclerosis seen on radiographs of trained horses and with predilection sites for carpal injuries in racing breeds. In addition, severity of sclerosis of the third carpal bone has been correlated with lameness in trained Standardbred horses.[11] Training has been shown to increase the thickness of the calcified cartilage zone. The effect of this thickening is unknown, but it might represent a protective reaction to reduce an abrupt stiffness gradient between hyaline cartilage and subchondral bone. Conversely, the thickening of the calcified cartilage zone relative to hyaline cartilage thickness, represented by tidemark advancement toward the periphery, could represent a pathologic process and predispose these areas of bone to failure.[12]

In some horses, the loss of compressive stiffness of the subchondral bone plate and thickening of the calcified cartilage zone can lead to cartilage injury and fracture. Osteochondral fragmentation of the distal radial carpal and proximal third carpal bones is associated with subchondral bone damage caused by repetitive loading. These locations are prone to reinjury following arthroscopic fragment removal and débridement, leading to a poor prognosis because of subsequent joint deterioration from cartilage debris and subchondral bone failure.

Injury to the intercarpal ligaments can occur and usually involves the palmar medial intercarpal ligament within the middle carpal joint. Injury to the collateral ligaments is usually associated with avulsion fractures of the proximal aspect of the vestigial metacarpal bones or distal radius. Injury to the extensor tendons usually develops in association with severe flexural deformities or lacerations. Injuries to the palmar aspect of the carpus do occur, but they are less common than dorsal injuries and have been associated with hyperextension or hyperflexion of the carpus during recovery from anesthesia. Pathology of the deep and superficial digital flexor tendons, fractures of the palmar aspect of the cuboidal bones or accessory carpal bone, and exostoses of the caudal aspect of the distal radius all can cause lameness. Lameness caused by structures within the carpal canal can be difficult to differentiate from lameness originating from other structures of the carpal region.

The role of conformation in carpal injury is complex and subject to personal opinion and anecdotal information. It is an area of intense concern in prepurchase examinations. One study found an association between offset carpi and lameness associated with the metacarpophalangeal joint.[13] The same study demonstrated a protective effect of carpal valgus on carpal injury.[13] These observations are consistent with my clinical

experience. Additionally, severe conformational abnormalities of foals can lead to malformation of the cuboidal bones and early osteoarthritis (see Chapter 86).

OSTEOCHONDRAL FRAGMENTS

Occurrence

Osteochondral fractures of the carpal bones, or carpal chips, are a common cause of lameness in horses. Racing breeds are particularly prone to carpal chip fractures, and the fractures are most likely to be a performance-limiting problem.

The occurrence of osteochondral fragmentation is a consequence of training and racing. In support of this, in Thoroughbred yearlings radiographed prior to sale, only 9 of 1121 horses (0.8%) had an osteochondral fragment in the carpus.[14] In racing Thoroughbreds and Quarter Horses, the distal aspect of the radial carpal bone is most often affected.[15] In another survey of osteochondral fragmentation in racing Thoroughbreds in Japan, 60% of the fractures occured in the antebrachiocarpal joint and 40% in the middle carpal joint.[16]

Additional articular damage associated with osteochondral fragments tended to be greatest in the middle carpal joint.[15] If the injury is located within the antebrachiocarpal joint, the distal lateral aspects of the radius and the proximal intermediate carpal bone are usually involved. If the middle carpal joint is involved, the trauma typically occurs at the distal radial carpal and proximal third carpal bones.[15]

Racing Standardbreds typically injure the carpal bones in the middle carpal joint. In a survey of 176 Standardbred horses undergoing carpal arthroscopy, only two antebrachial carpal joints had osteochondral fragments.[17]

Etiopathogenesis

Osteochondral fragments of the carpus are induced by trauma. The history usually includes a sudden onset of carpal swelling. Certainly, some carpal chips with sharp areas of demarcation and healthy subchondral bone represent acute single-event failures; however, degenerative bone and cartilage, compatible with chronic injury, most often are found both on radiographs and at surgery. Palmar carpal osteochondral fragments can be the result of hyperflexion injuries and may develop during recovery from general anesthesia.

In the third carpal bone, reduction in vascular channels can lead to ischemic necrosis of the subchondral bone.[7] At surgery, degenerative bone appears discolored and brittle relative to the surrounding healthy bone. In racing Standardbreds, the prevalence of lameness was lower, 6.7% in horses with mild versus 45.4%, in horses with severe sclerosis of the third carpal bone.[11] Resorption of the diseased bone predisposes to development of a fracture at the junction of the compliant and noncompliant bone.[18]

Diagnosis

Diagnosis is based on clinical signs, including lameness, painful response to carpal flexion, joint effusion, response to intra-articular anesthesia, and reduced performance. A complete set of well-exposed radiographs, including the tangential view of the distal row of carpal bones and flexed lateral projections, is needed. Occasionally, the tangential view of the proximal row of carpal bones is required for diagnosis. If a carpal chip is detected, radiographs of the contralateral carpus are recommended, because it is common for fragments to be present bilaterally (Figure 94-1).[15]

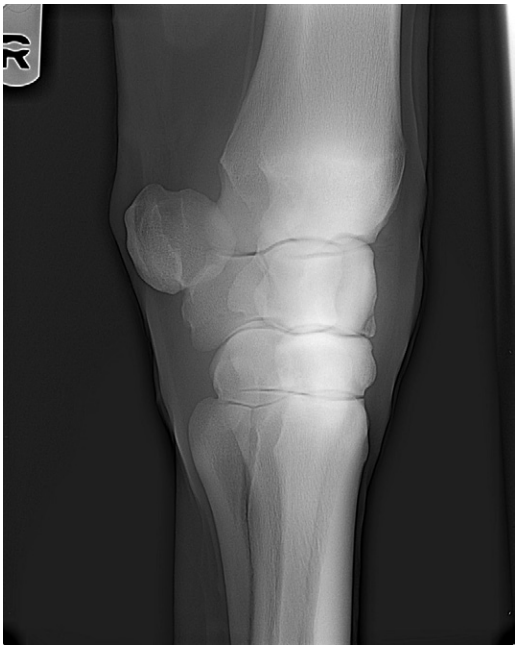

Figure 94-1. Dorsolateral to palmaromedial 45-degree oblique projection of a Thoroughbred with an osteochondral fragment of the distal medial aspectof the radial carpal bone.

When a carpal chip is identified radiographically, it is important to document it as the cause of lameness, particularly in nonracing breeds, because it can represent an incidental finding. Intra-articular anesthesia in most cases confirms the associated joint as the source of lameness. Some horses become lame in the contralateral forelimb after anesthesia because of the presence of bilateral carpal chips or another secondary lameness.

As mentioned before, local anesthesia of the proximal suspensory region may accidentally involve also the palmar recesses of the carpometacarpal joint, anesthetizing both the carpometacarpal and middle carpal joints. Therefore, the relationship between the palmar recesses of the carpometacarpal joint and the proximal suspensory ligament must be considered when applying local anesthesia in this area. Because of this, anesthesia of the middle carpal joint should be performed before the high palmar block if carpal lameness is suspected. Alternatively, the two-point high palmar block (see Chapter 72) is recommended for regional anesthesia of the proximal metacarpal region, because it prevents the anesthetic agent from entering the carpus.[1]

Using the high palmar block there is a risk of entering the carpal canal and desenstizing structures within the canal. Therefore caution is indicated in the interpretation of a positive local nerve block at the palmar/plantar aspect of the proximal MCIII/MTIII.

Case Selection

Most carpal chips are of clinical importance in racing breeds, and their removal under arthroscopic guidance is the treatment of choice. Factors that should be considered before chips are removed are the degree of lameness, the amount of degenerative change seen on the radiographs or lack thereof, previous intra-articular therapy (especially corticosteroids), racing or training history, and economic concerns. Horses with carpal

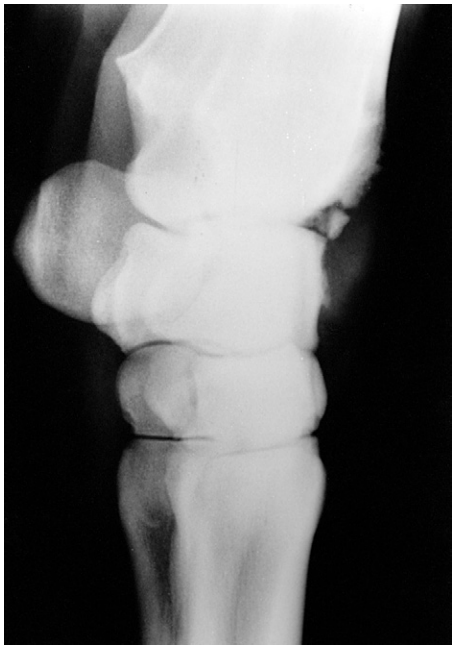

Figure 94-2. Dorsomedial to palmarolateral oblique radiograph of the carpus of a Thoroughbred with an osteochondral fragment of the distal lateral aspect of the radius. Note the enthesiophyte formation and subchondral lysis consistent with osteoarthritis.

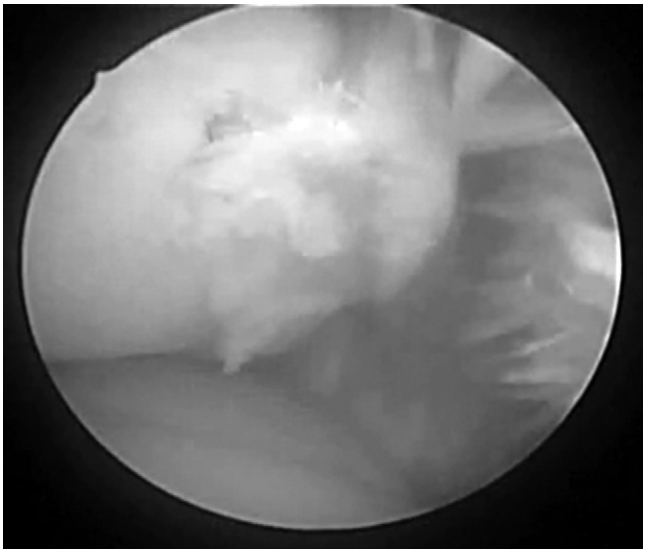

Figure 94-3. Arthroscopic view of the distal aspect of the radial carpal bone with moderate cartilage damage (grade II) and an osteochondral fragment. A greater than 30% loss of cartilage is associated with reduced postoperative performance.

chip fractures are usually sound or mildly lame when jogged in hand, if examined a few days after the injury. If moderate or severe lameness is present, other conditions are suspected, such as a carpal slab fracture, moderate-to-severe cartilage damage, intercarpal ligament injury, or sepsis.

The radiographic findings of enthesiophytosis and subchondral bone lysis are associated with cartilage and subchondral bone damage at surgery (Figure 94-2). In one study, subchondral lysis of the radial carpal bone was associated with both osteochondral fragmentation and additional cartilage injury.[19] In another study, horses with cartilage degeneration involving more than 30% of the articular surface of the affected bone (Figure 94-3) had reduced postoperative performance.[15]

Factors associated with reduced racing prognosis postoperatively include repeated joint injection, reduced response to joint injection, injury early in training, poor preoperative racing record, or previous carpal surgery. These factors, coupled with economic considerations, are important in the surgeon's case selection and the owner's willingness to have surgery performed.

Treatment

The horse is positioned in dorsal or lateral recumbency based on the site of the lesion and the surgeon's preference. If unilateral carpal arthroscopy is to be performed, I choose lateral recumbency with the limb containing the lesion(s) down. If bilateral carpal arthroscopy is planned, or if both sides of a joint are to be examined, I prefer to place the horse in dorsal recumbency with the limbs suspended from a rack or a ceiling mount. Antebrachiocarpal joint injury often occurs on both the lateral and medial side simultaneously, and thus dorsal recumbency is usually selected. Some surgeons always prefer either dorsal or lateral recumbency for all carpal surgeries.

The standard arthroscopy portals for the antebrachiocarpal and middle carpal joints are located between the common digital and extensor carpi radialis tendons (lateral portal) and medial to the extensor carpi radialis tendon (medial portal). Portal locations for instruments and the arthroscope are determined by the location of the lesion, with the arthroscope portal placed distant to the lesion.[20] The portals are made large enough to allow egress of fluid without its accumulation in the subcutaneous space (approximately 6 to 8 mm). Large portals should be avoided because adequate joint distention will be difficult to maintain. If gas is used as the arthroscopic medium, the portals should be very small and only allow access of the arthroscope and instruments. The versatility of arthroscopy allows additional nonstandard portals to be made depending on the site of the lesion.

When the osteochondral fragment is identified, it is partially loosened with an elevator if needed, grasped with Ferris-Smith rongeurs, and removed through the instrument portal by twisting the forceps back and forth while applying traction. Undermined cartilage flaps and diseased subchondral bone are removed with a curette. The lesion is débrided, minimizing the amount of cartilage removed, especially in weight-bearing areas of the joint. Débridement of partial-thickness cartilage lesions adjacent to the fragment is avoided unless the cartilage is detached from the underlying subchondral bone. An exception to this is partial-thickness cartilage injury overlying diseased subchondral bone (e.g., radial facet of the third carpal bone), which is typically débrided. Additional information on arthroscopy is found in Chapter 13.

Aftercare

The postoperative use of intra-articular or intravenous hyaluronan and intramuscular or intra-articular polysulfated glycosaminoglycan (Adequan) varies among surgeons. My standard postoperative instructions for routine osteochondral carpal arthroscopy consists of 2 weeks of stall rest, 2 weeks of stall rest

with hand-walking, and 2 weeks of pasture exercise, continued hand-walking, swimming physiotherapy, or a combination thereof. On occasion, a longer period of rest is given if substantial lameness or marked cartilage injury was present at the time of surgery. Bandaging is carried out until sutures are removed at approximately 12 days after the surgery.

Prognosis

A scale has been developed to grade cartilage injury observed at surgery. Grade I represents minimal articular cartilage fibrillation or fragmentation extending no more than 5 mm from the fracture site. Grade II represents articular degeneration, including up to 30% of the articular surface of the bone. Grade III indicates loss of 30% to 50% of the articular surface. Grade IV represents severe loss of bone and cartilage associated with the fracture.[15]

Postoperative results of carpal arthroscopy for osteochondral fragments depend on the degree of cartilage injury and the horse's previous racing success. Horses with minimal to no cartilage injury (grades I or II) have a 72.8% chance of returning to racing at a level equal to or better than that before injury.[15] If cartilage damage is greater than 30% of the visible surface of the affected bone (grades III or IV), only a 53.5% chance of return to racing at a level equal to or better than that before injury was found.[15] In a survey of carpal arthroscopy in racing Standardbreds, 74% returned to racing after surgery and 61% raced at or above their previous level.[17] In another survey from Japan, 82.6% of racing Thoroughbreds returned to racing after carpal surgery, which is better than results in North America. A likely reason for the discrepancy is the higher percentage of antebrachiocarpal joint injuries with little additional cartilage injury in the Japanese study.[16] It is my experience that cartilage injury is greater and the prognosis worse with chip fractures of the middle carpal versus the antebrachiocarpal joint.

SUBCHONDRAL LUCENCY OF THE THIRD CARPAL BONE

Etiopathogenesis

Subchondral lucency or crush fracture of the third carpal bone is a common injury in racing breeds. It might be a prelude to slab fracture of the third carpal bone. Repetitive loading of the third carpal bone during training and racing leads to increased sclerosis of the radial facet, degeneration of the articular cartilage, and in some cases, loss of support from the subchondral bone. Subchondral lucency significantly influences the degree of carpal lameness in Standardbreds.[21]

Diagnosis

Clinical signs include lameness, reduced performance, and mild-to-moderate effusion of the middle carpal joint. Affected horses improve with intra-articular anesthesia of the middle carpal joint. A well-exposed tangential (skyline) projection of the third carpal bone is required for diagnosis (Figure 94-4). Radiographic signs include both sclerosis and lysis of the radial facet of the third carpal bone. In one study of racing Standardbreds and Thoroughbreds that improved after intra-articular anesthesia of the middle carpal joint and showed minimal to no degenerative changes on radiographs, 80% had cartilage damage, an incomplete fracture, or a crush fracture of the radial facet of the third carpal bone.[22] Lameness has been associated with increased sclerosis of the third carpal bone in racing breeds.[11] Radiographs of the contralateral carpus should be taken because disease affecting both carpi is common.

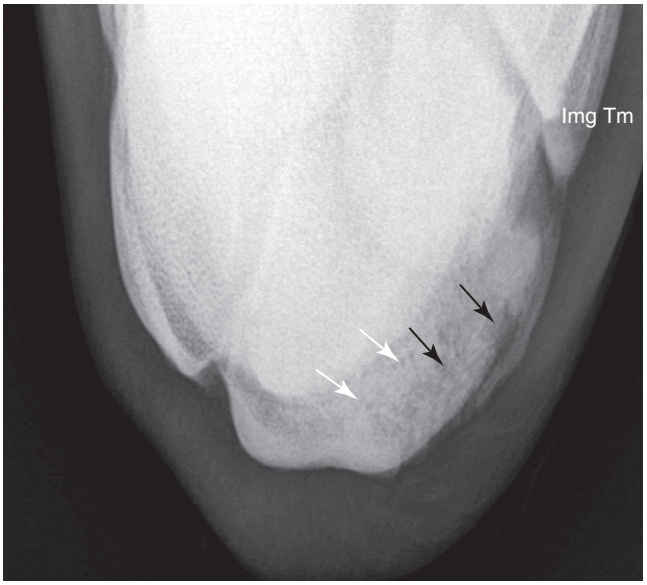

Figure 94-4. Tangential (skyline) projection of the left carpus of a 4–year-old Standardbred demonstrating sclerosis *(white arrows)* and lucency *(black arrows)* of the third carpal bone consistent with third carpal bone degeneration.

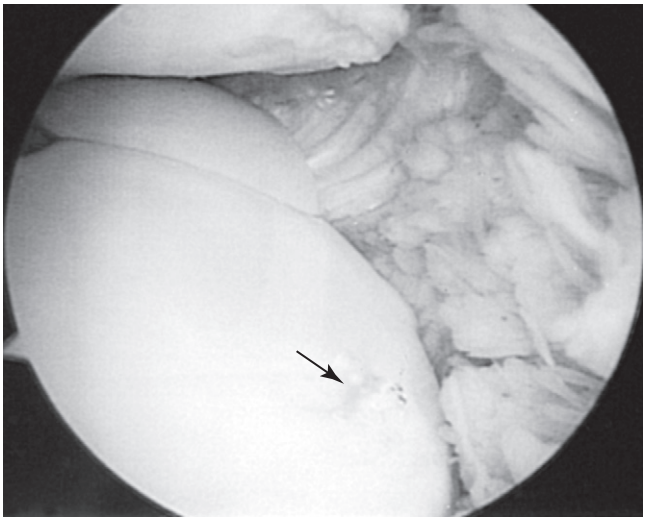

Figure 94-5. Arthroscopic view of the middle carpal joint of a 3-year-old racing Standardbred with a focal defect *(arrow)* in the weight-bearing surface of the radial facet of the third carpal bone. On a tangential radiographic projection, subchondral lucency of the radial facet was seen.

Treatment

Some horses respond to intra-articular therapy but many do not; therefore, arthroscopic débridement is the treatment of choice.[23] The articular cartilage often acquires partial- to full-thickness injury (Figure 94-5). Removal of the abnormal cartilage reveals brittle, yellow, diseased subchondral bone and

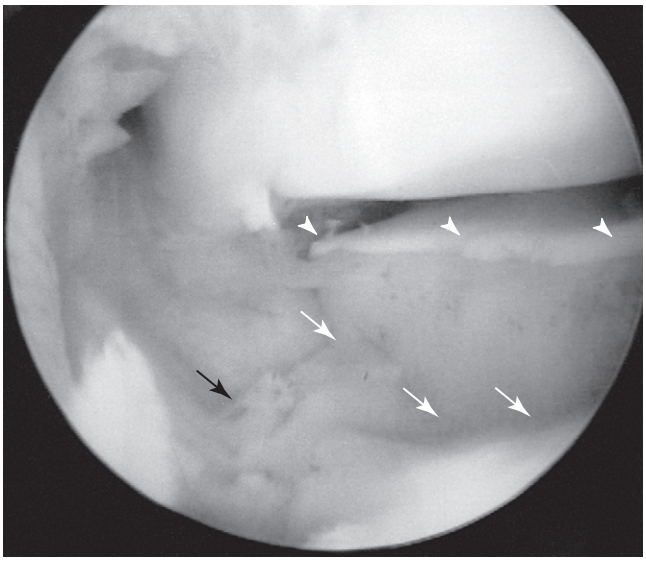

Figure 94-6. Postoperative appearance of the third carpal bone after débridement of degenerative cartilage and subchondral bone in a racing Standardbred. The diseased portion of the third carpal bone was resected until it was no longer in a weight-bearing position. *White arrowheads,* Line where the cartilage and bone were resected vertically. The normal cartilage and its thickness is seen. *White arrows,* Bottom of the verical resection of bone; *black arrow,* dorsal end of the resected cartilage.

occasional fractures. In some cases, the mechanical strength of the cartilage and bone is very poor, and the arthroscopic probe sinks into the subchondral plate. The abnormal cartilage and bone are completely removed. If a dorsal rim remains on the third carpal bone, it is typically removed (Figure 94-6). Postoperative management is identical to that of osteochondral fragments. Prognosis for return to racing for horses after arthroscopic débridement of such lesions is 89%.[24]

MEDIAL PALMAR INTERCARPAL LIGAMENT INJURY

Etiopathogenesis

Diagnosis of partial or complete disruption of the medial palmar intercarpal ligament (MPICL) has become possible because of the use of arthroscopy in equine surgery. The medial and lateral palmar intercarpal ligaments prevent dorsal displacement of the middle carpal joint.[23] The frequency of injury to this ligament ranges between 8.7% and 70%.[25,26] MPICL injury can occur alone or in conjunction with other types of carpal injury, including osteochondral fragments and cartilage damage.[25] One report found no association between intercarpal ligament injury (ICL) and cartilage injury.[27]

Injury to the lateral palmar intercarpal ligament (LPICL) has been described. It is important to differentiate this from lucencies in the ulnar carpal bone, which are generally viewed as incidental finidings. LPICL injuries are characterized by a small avulsion fragment at the origin of the ligament at the ulnar carpal bone.[28] A good prognosis for return to athletic soundness after arthroscopic removal has been reported.[29]

Diagnosis

Clinical signs of MPICL injury include moderate lameness and joint effusion. In acute cases, no radiographic signs specific to this injury are seen. In chronic cases, new bone formation on the distal dorsal aspect of the proximal row of carpal bones can be seen, which is related to a degree of joint instability. Acute ICL injury is suspected by observation of the clinical signs listed earlier, absence of radiographic abnormalities, and response to middle carpal joint anesthesia. Large avulsion fractures of the origin of the MPICL at the distal radial carpal bone are sometimes seen in displaced third carpal bone slab fractures. For this reason, dorsal to palmar radiographic projections should be included in cases of third carpal slab fractures. Ultrasonographic evaluation of the MPICL has been described in the standing horse.[30] Magnetic resonace imaging offers an advanced method of imaging soft tissue structures of the carpus and should be considered in horses with mild to moderate lameness that improves with desentization of the carpal region in the absence of a definite lesion on radiographic, ultrasonographic, and/or nuclear scintigraphy imaging.

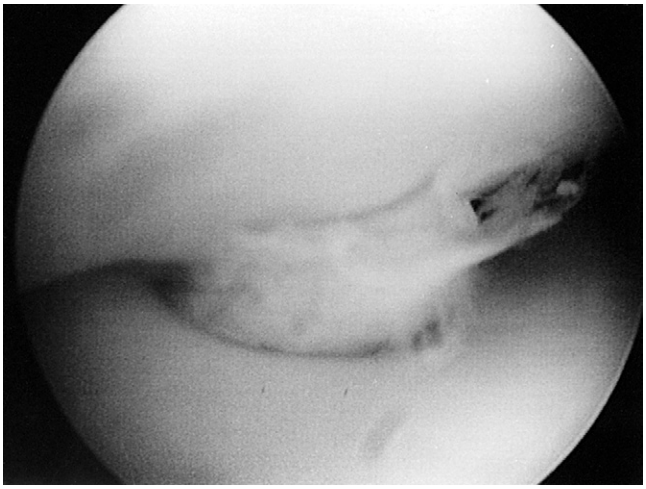

Figure 94-7. Arthroscopic view of the middle carpal joint of a racing Standardbred with a complete tear of the medial palmar intercarpal ligament. The lameness in this horse was isolated by intra-articular anesthesia of the middle carpal joint. The radiographs were normal.

Confirmation of the diagnosis is achieved through arthroscopic evaluation. Most lesions can be diagnosed through examination of the dorsal pouch but visual examination of the palmar pouch of the middle carpal joint may allow specific examination of the palmar bundle of the MPICL. Ligament disruption can be partial or complete (Figure 94-7). A grading scale of injury has been suggested, where grade I indicates rupture or fraying of a small number of fibers; grade II, rupture of up to one third of the ligament fibers; grade III, rupture of two thirds; and grade IV, complete rupture of the ligament. Mild (grade I) tears can be incidental findings at surgery and might not be associated with lameness.[27]

Treatment

No method of ligament repair is available, and the surgical procedure is limited to diagnosis, débridement of free strands of the ligament, removal of any associated avulsion fractures, and débridement of other lesions within the joint. Three months of stall confinement followed by 3 months of pasture turnout is recommended following surgery.

The prognosis is considered fair for partial tears (grades II and III) and guarded for complete tears (grade IV). In one study,

injury to the MPICL combined with subchondral bone damage was the best indicator of postoperative performance.[31] The presence of a complete MPICL tear or a mild tear with large subchondral bone defects was associated with a poor outcome.[32]

CARPAL SLAB FRACTURES

Etiopathogenesis

The third carpal bone is a common site for injury. Fracture types include osteochondral fragments, corner fractures, small and large frontal plane slab fractures, sagittal slab fractures, and comminuted fractures. Carpal slab fractures occur most often in the frontal plane of the radial facet of the third carpal bone.[32] Frontal plane slab fractures also can occur in the radial, fourth, and intermediate carpal bones.[33] Because the radial facet of the third carpal bone undergoes stress-adaptive remodeling during race training, repetitive loading consistently leads to fractures in this location.[32] Additionally, subchondral lucency, cartilage injury, and incomplete fractures are often diagnosed in this area.[15,18,20,22,23] Sagittal slab fractures can be displaced or nondisplaced. These fractures can go undetected unless a properly positioned and exposed skylne projection of the distal row of carpal bones is made. In addition, dorsopalmar and 45-degree oblique dorsomedial to palmarolateral projections are helpful when evaluating sagittal slab fractures of the third carpal bone.

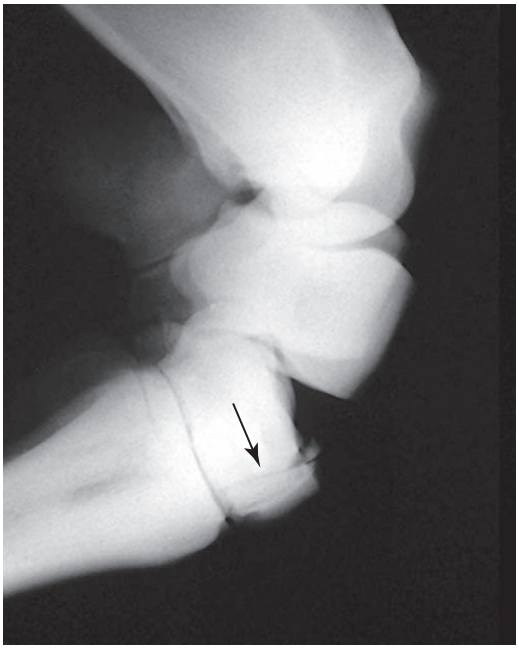

Figure 94-8. Lateral radiographic view of the carpus of a Thoroughbred with a minimally displaced, complete frontal plane slab fracture of the third carpal bone *(arrow)*. Notice the osteochondral fragment (trough lesion) at the proximal articular surface.

Diagnosis

Horses with complete slab fractures of the third carpal bone have moderate to severe lameness, are reluctant to flex the carpus, and exhibit joint effusion. The right forelimb is more often affected in Thoroughbreds, whereas both forelimbs are equally affected in Standardbreds.[34,35] Horses with incomplete slab fractures show a variable degree of lameness and usually exhibit little joint effusion.

Radiographs, including the skyline projection, confirm the diagnosis and aid in the choice of treatment. Fragmentation at the articular margin of the middle carpal joint, third carpal bone sclerosis, and lysis can be present, which usually indicate advanced disease. Diagnosis of incomplete fractures is more difficult because localizing signs can be absent. Careful lameness examination, diagnostic anesthesia, well-positioned and well-exposed radiographs, and occasionally nuclear scintigraphy are necessary for a diagnosis. After a diagnosis is established, radiographs of the other carpus are obtained because bilateral slab fractures or other carpal disease can be present. Bilateral slab fractures occur most commonly in Standardbreds.

Treatment

Treatment options for complete slab fractures include their surgical removal or surgical repair of the fracture with interfragmentary bone screws placed in lag fashion. Surgery is recommended for all horses, even those to be retired from racing. Conservative management of complete slab fractures often results in moderate to severe osteoarthritis. Removal of slab fractures is recommended when thin fragments (less than 10 mm thick) are present or if there is significant loss of articular cartilage associated with the fracture.

In many complete slab fractures, a loose wedge-shaped piece of bone and articular cartilage is found at the parent portion of the bone immediately behind the fracture plane (Figure 94-8). This fragment might not be obvious on the preoperative radiographs. The consequence of this pathology is that curettage of this diseased bone and cartilage results in a significant loss of cartilage and subchondral bone, forming a trough behind the fragment. In such cases, removal of the entire slab fracture may be elected. Alternatively, just the removal of the articular component of the slab combined with screw fixation of the remainder of the slab may be elected. If cartilage loss is minimal or if the slab fracture is more than 10 mm thick or incomplete, compression of the fracture fragments with one or more bone screws is preferred.

Successful management of incomplete fractures is possible with rest alone; however, some horses refracture when they return to racing and eventually require surgery. For this reason, compression of the fracture by placement of a cortex screw in lag fashion under arthroscopic guidance is the treatment of choice for most incomplete third carpal bone slab fractures in racing breeds.

Surgical Technique

FRAGMENT REMOVAL

Fragment removal is best accomplished through an arthrotomy approach. The extent of the fragment is assessed and the joint capsule attachment of the middle carpal and carpometacarpal joints are severed with a scalpel. The fragment is pared loose from its bed with a periosteal elevator and subsequently grabbed with a rongeur. Any remaining fibrous attachments are severed through rotating movements with the rongeur. Persistent tags are sharply transected with the scalpel. When loose, the fragment is removed and the arthrotomy is closed in three layers using routine technique.

Fragment removal under arthroscopic supervision is a time-consuming procedure and only encouraged for small slab fractures. Severing of the joint capsule attachments can be accomplished with a sharp periosteal elevator or an osteotome.

The difficulty in this procedure is the visualization and transection of the carpometacarpal joint capsule.

The postoperative management is identical to that of screw fixation (see later).

SCREW FIXATION

Internal fixation of frontal or sagittal slab fractures can be accomplished using arthroscopic or arthrotomy techniques. However, arthroscopy has considerable advantages over arthrotomy and is considered the treatment of choice. Arthroscopy provides a more complete examination of the joint and facilitates débridement of the fracture gap, but it is technically more difficult to properly orient the implant(s), and requires experience.[20,36]

In arthroscopic repair, the horse is positioned in dorsal recumbency or in lateral recumbency with the affected limb uppermost. The lateral portal is used to position the arthroscope for examination of the medial aspect of the middle carpal joint. Placing the arthroscope in the most lateral aspect of the dorsolateral arthoscopic portal aids in examination, reduction, and fracture fixation of frontal plane slab fractures. The medial portal is used to débride the fracture and should be made along the frontal plane fracture to aid in placing instruments within the fracture plane. The joint is held in extension because this position opens the fracture gap, which aids débridement.

The fracture hematoma and fragments of bone are removed via placement of instruments through the medial portal. Subsequent flexion of the joint reduces the fracture and allows assessment of anatomic reduction. Hypodermic needles are placed at the lateral and medial border of the fracture and along the articular surface of the third carpal bone in the middle carpal joint. An additional needle is placed into the carpometacarpal joint to allow assessment of the width of the third carpal bone. The locations of the hypodermic needles and the reduction of the fracture are confirmed arthroscopically (Figure 94-9). Care should be taken to confirm anatomic reduction of the slab fracture along its entire fracture plane. A 2.5-mm drill bit, oriented perpendicular to the dorsal surface of MCIII, is placed through a separate incision in the center of the fracture fragment. Its location is assessed radiographically or via image intensification (Figure 94-10). Standard AO technique is used to place a 3.5- or 4.5-mm cortex screw in lag fashion. During the placement of the marker bit and screw, it is important to maintain the carpus in flexion to provide adequate reduction.[20,36]

If the fracture is repaired through an arthrotomy, an incision is made into the middle carpal joint over the radial facet of the third carpal bone just medial to the extensor carpi radialis tendon.[37] The fracture line is identified and débrided. Orientation of the drill bit is easier with an arthrotomy compared with the arthroscopic technique. The drill bit should be positioned perpendicular to MCIII and in the center of the fracture fragment (if only one screw is required).

For most fractures 3.5-mm screws are used (Figure 94-11). The 3.5-mm screws have the advantage of easier placement of single or multiple screws because of their smaller size (Figure 94-12). There is reduced risk of splitting the fracture fragment compared with the 4.5-mm screws. The 3.5-mm screw also has a smaller head, which interferes less with the soft tissues over the carpus and is helpful when it is necessary to cross the screws in multiplane fractures (Figure 94-13). Because of these features, 3.5-mm screws are used in thinner slabs. The 4.5-mm screws are used for fractures that are large, typically involving both the radial and intermediate facets of the third carpal bone

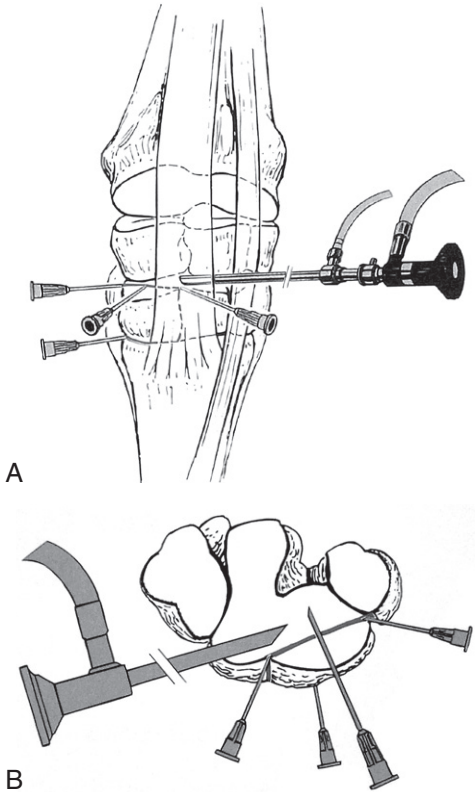

Figure 94-9. Dorsopalmar **(A)** and skyline **(B)** view of a schematic drawing of arthroscope and needle placement for the surgical repair for a third carpal bone slab fracture under arthroscopic guidance. One needle is placed on each end of the fracture, one in the carpometacarpal joint and one centered over the middle of the slab fracture in the middle carpal joint to serve as guides for the location and direction of screw placement.

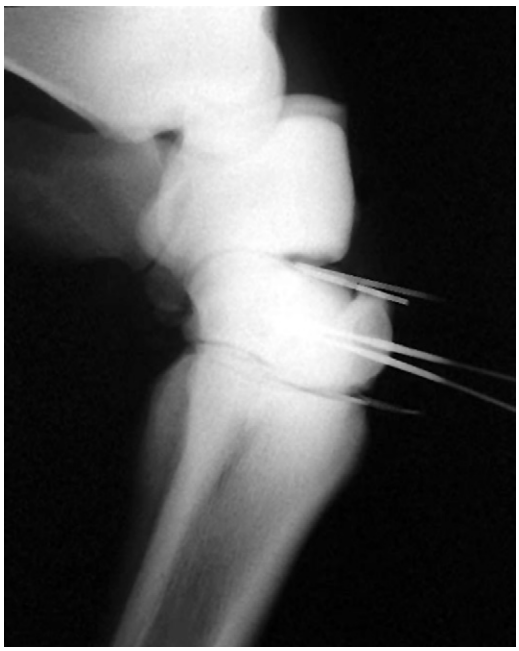

Figure 94-10. Intraoperative radiographic view of the carpus of a Thoroughbred during surgery for repair of a third carpal bone slab fracture. Notice the placement of the needles to identify the borders of the fracture and the marker drill bits to confirm proper placement and orientation of the implants.

or other carpal bones (Figure 94-14) or radial facet slab fractures that are greater than 15 mm in thickness. Alternatively the use of a headless, tapered screw (Acutrak) has been described for repair of slab fractures, although I have no experience with using this screw system.[38]

Careful intraoperative monitoring with radiographs or image intensification is required regardless of whether the repair was achieved through an arthrotomy approach or under arthroscopic supervision.

Sagittal slab fractures should be repaired with 3.5- or 4.5-mm screws, although 3.5-mm screws are preferred because of their small head size and reduced risk of interference of the screw head with the second carpal bone. These fractures are usually located on the medial aspect of the third carpal bone. Both arthroscopy and arthrotomy approaches can be applied with intraoperative monitoring to guide placement of the implant(s). The surgical approach depends on location of the fracture. Screw placement should avoid contact of the screw head with the second carpal bone, which can cause lameness (Figure 94-15).

The presence of additional carpal pathology is typical for cases of third carpal slab fracture. Aside from the previously mentioned trough behind the slab fragment, distal radial carpal bone chips, cartilage damage at different locations, and MPICL injury may be present.

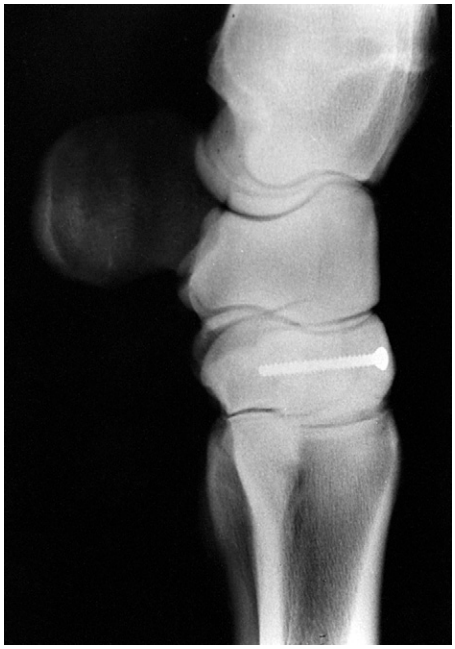

Figure 94-11. Lateral radiographic view after repair of a third carpal bone frontal plane slab fracture with a single 3.5-mm cortex screw placed in lag fashion.

Aftercare

Aftercare is routine, with bandaging for several weeks and a slow, gradual return to work. Most horses require approximately 4 months of recovery before returning to race training; however,

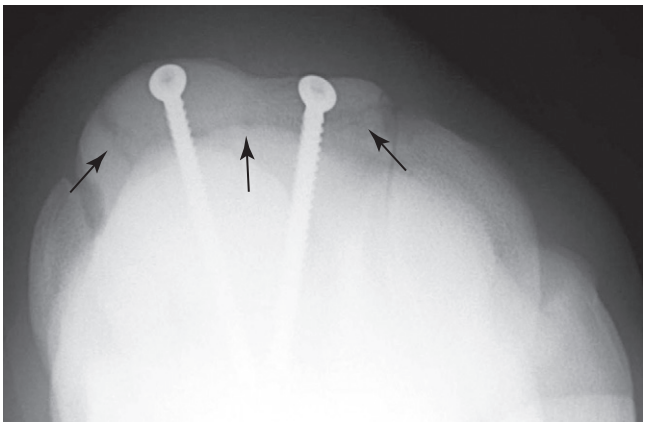

Figure 94-12. Tangential radiographic view of a third carpal bone of a Thoroughbred after repair of a frontal plane fracture *(arrows)* with two 3.5-mm cortex screws placed in lag fashion.

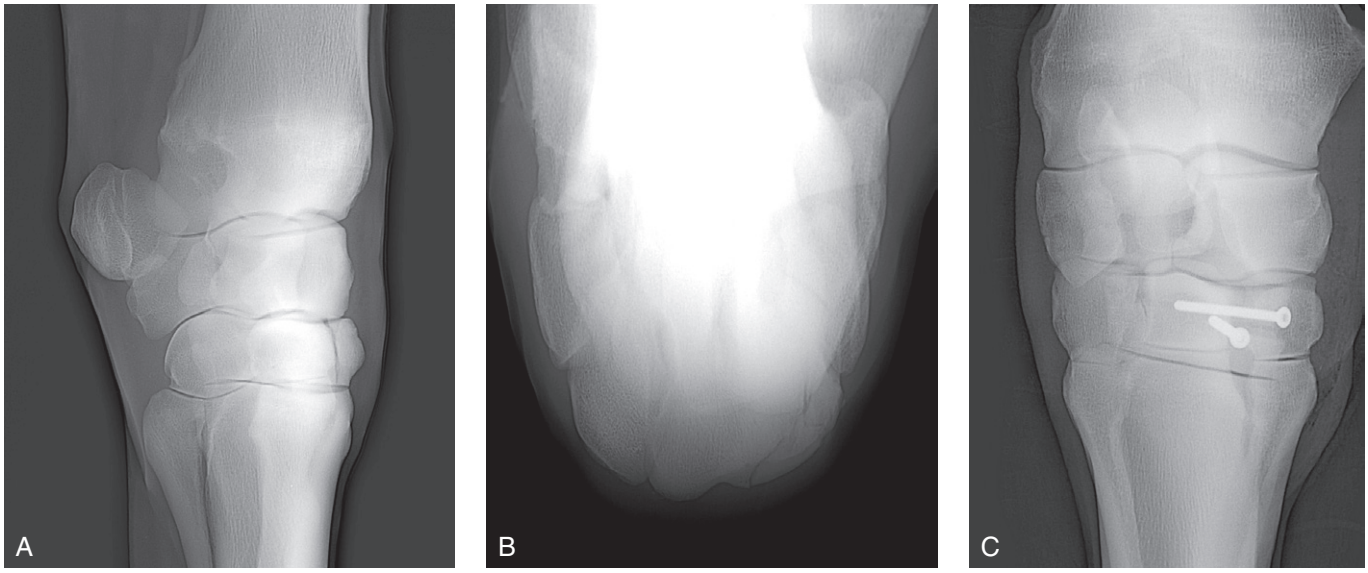

Figure 94-13. A, Dorsolateral to palmaromedial oblique radiographic views of a frontal and sagittal plane slab fracture of the radial carpal bone of a Thoroughbred. **B,** Skyline projection of the same horse. **C,** Dorsal to palmar projection of the same horse after placement of two 3.5-mm cortex screws in lag fashion to stabilize the multiple plane fracture.

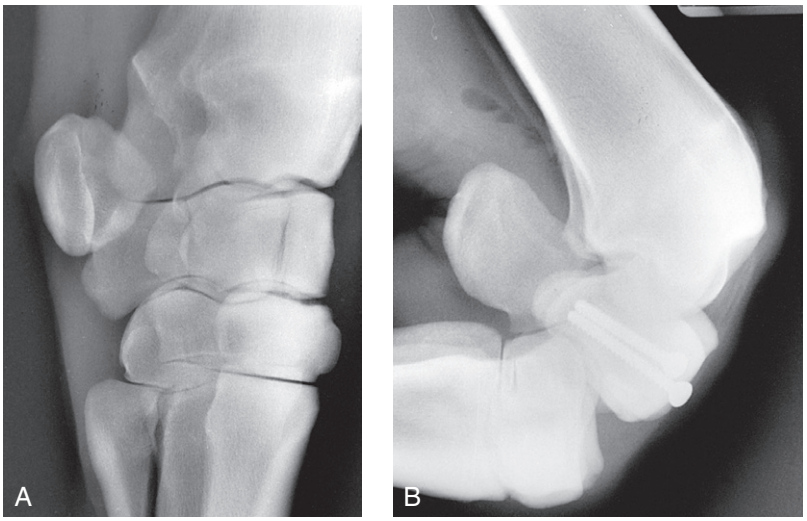

Figure 94-14. A, Dorsolateral to palmaromedial oblique radiographic view of a frontal plane slab fracture of the radial carpal bone of a Thoroughbred. **B,** Flexed lateral radiograph after repair of the radial carpal slab fracture using two 4.5-mm cortex screws placed in lag fashion.

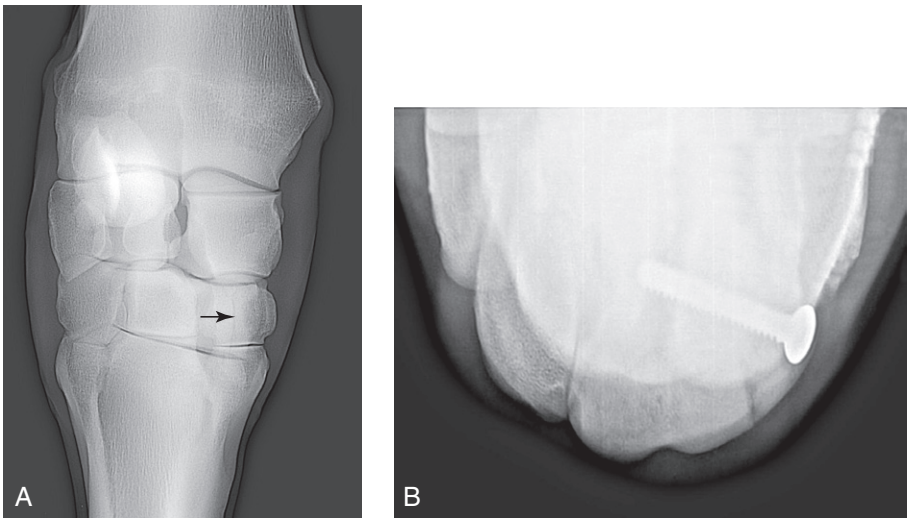

Figure 94-15. A, Dorsopalmar projection of a 3-year-old racing Thoroughbred with a sagittal plane slab fracture of the medial aspect of the third carpal bone *(arrow)*. **B,** Skyline projection of the third carpal bone after repair with a 3.5-mm cortex screw placed in lag fashion to compress the fracture.

additional time may be required depending on fracture healing. Prognosis differs among breeds, with 77% of Standardbreds and 65% of Thoroughbreds returning to racing after surgery.[34,35] Nondisplaced frontal plane slab fractures repaired with cortex screws placed in lag fashion have a good prognosis for return to function at or above the previous level.[39] Compression of the fracture by the screw coupled with minimal to no significant cartilage injury is significant in the outcome of such cases. Compression of sagittal slab fractures of the third carpal bone with bone screws is associated with an improved prognosis for return to racing soundness when compared to those treated nonsurgically.[40]

MULTIFRAGMENT FRACTURES

Etiopathogenesis

Multifragment fractures typically occur after kicks, falls, or collisions. Such injuries can also occur during racing. Non–weight-bearing lameness and carpal swelling are typical. These horses exhibit angular limb deformity and carpal hyperextension because of carpal collapse.

Diagnosis

Radiographs confirm the presence of multiple fracture lines involving several bones. Most of these fractures involve the medial aspect of the carpus and can include the radial, intermediate, second, and third carpal bones; the proximal vestigial metacarpal/metatarsal (splint) bones; or a combination thereof. The loss of support of the bone column on the medial aspect of the carpus typically causes a carpal varus deformity (Figure 94-16).

Treatment

First aid is aimed at reconstructing the bone column by placing palmar and lateral splints over a heavily padded bandage. The inherent instability and articular nature of the fractures usually makes external coaptation with casts or splints inadequate as the sole treatment, and often results in severe lameness in most horses, the development of pressure necrosis of the skin, and a high risk for developing laminitis or angular limb deformity in the supporting limb.

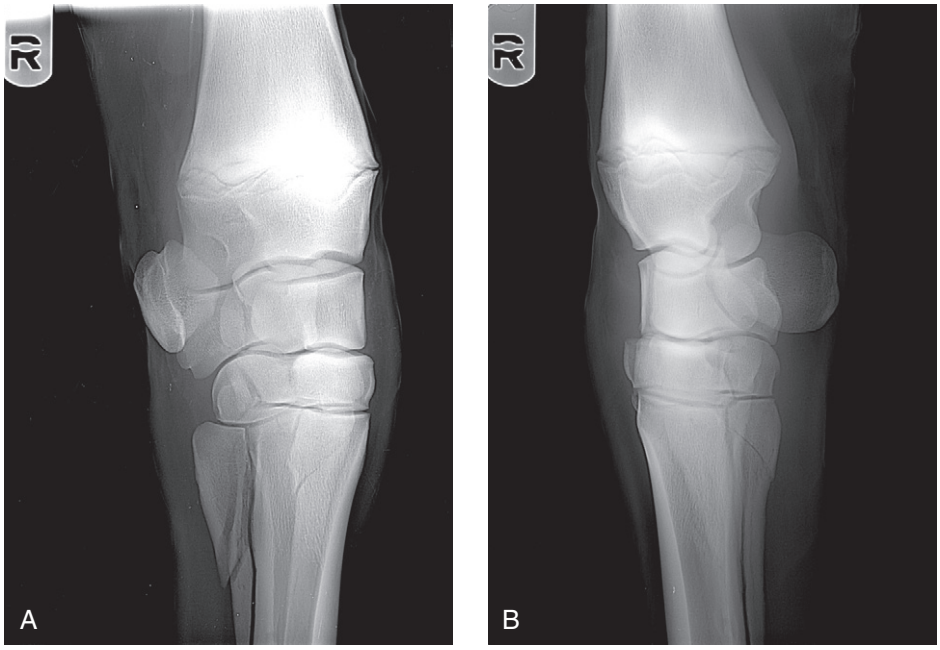

Figure 94-16. Dorsolateral to palmaromedial **(A)** and dorsomedial to palmarolateral oblilque **(B)** radiographic views of a Thoroughbred weanling with a comminuted fracture of the fourth carpal bone and fractures of the proximal aspect of the lateral and medial splint bones.

Realignment of the bone column by reconstruction of the carpal bones with screws is recommended. If the bone column cannot be re-established, carpal arthrodesis with bone plates should be considered (see Chapter 81). Any attempt to treat such an injury is a salvage procedure. For carpal reconstruction, an arthrotomy over the appropriate joint space is performed, and the carpal bones are reconstructed using 5.5-mm, 4.5-mm, and 3.5-mm screws. In some cases, arthroscopic reconstruction is possible but can be quite difficult in comminuted fractures.

Proper reduction of the fracture fragments is of paramount importance during the reconstruction of the bone column for weight-bearing and load sharing with the implants. Additionally, a reduction in the development of osteoarthritis is achieved. At surgery, additional fracture lines and fragments are often found, which complicate the procedure. Coaptation using a sleeve cast for 4 to 6 weeks is recommended postoperatively.

In cases of multicarpal bone fracture, the surgeon and owner should recognize that carpal arthrodesis may be necessary if articular damage is severe or the bony column cannot be reconstructed anatomically. Partial or pancarpal arthrodesis can be performed as needed, although partial carpal arthordesis is preferrred when possible because of the improved mobility and comfort. A variety of implants have been used for partial or pancarpal arthrodesis.[41-43] For a more in-depth description of different arthrodesis procedures, please review Chapter 81.

ACCESSORY CARPAL BONE FRACTURES

Etiopathogenesis

Fracture of the accessory carpal bone occurs in Thoroughbred horses that race over fences, but it can be seen after collisions or falls in any breed. Relative to other carpal injuries, accessory carpal bone fractures are rare, representing 2% of all carpal fractures.[44] In a survey of Thoroughbred yearlings radiographed prior to sale, accessory carpal bone fractures were found in 0.4% of the horses.[14] Because of its location on the palmar aspect of the carpus and its multiple ligamentous attachments, the accessory carpal bone undergoes intense loading during extension of the carpus.

The preponderance of frontal plane fractures is likely caused by avulsion of the bone by its palmar attachments during carpal loading. Other suspected causes of accessory carpal bone fractures include compression between the radius and MCIII and direct trauma. It is typical for a small degree of comminution to be present at the fracture site in frontal plane fractures.[44] Nonarticular chip fractures and comminuted fractures are occasionally diagnosed.[45,46]

Diagnosis

Acute, severe lameness is typical. The carpus can be held in a somewhat flexed position to reduce loading. Carpal canal effusion might be present, but carpal joint effusion might be absent unless there is an articular component to the fracture.

Diagnosis is confirmed through radiographic examination. In acute fractures, displacement is usually present, although nondisplaced fractures do occur. In nondisplaced fractures, another set of radiographs should be taken 10 to 14 days after injury. Nuclear scintigraphy and, if available, computed tomography are helpful in defining the fracture configuration in detail. In chronic fractures, fragment edges are indistinct and rounded off.

Treatment

Treatment options include conservative management or surgical reconstruction and compression with bone screws or plates.

Nonsurgical Management

Nonsurgical management has been successful. Stall rest for 3 months followed by turnout at pasture for 3 to 9 months is recommended. Of 19 horses with accessory carpal bone

fractures, 17 were treated conservatively with rest for 3 to 6 months and two had internal fixation. All 11 horses available for follow-up treated conservatively were sound, and 7 had returned to racing.[44] A recent study evaluationg racing performance of horses suffering from articular fragments revealed that the number of starts was not affected compared to maternal siblings but the earnings in 2- and 3-year-old racehorses were significantly lower.[47]

Surgical Management

Surgical repair with bone screws has been most often attempted in simple vertical fractures. Because of the location, the concave shape of the accessory carpal bone, and the narrow space for error in screw placement, attempts at surgical repair are infrequent. These difficulties, coupled with the low incidence of the injury, make the decision between conservative and surgical management difficult. In one study, vertical fractures were created experimentally, followed by treatment with immediate or delayed repair with bone screws or conservative management.[48] Three horses treated conservatively were still lame 6 months later, whereas all horses that had been surgically treated were sound at 6 months. However, wound infection and screw breakage were reported complications. Another clinical study reports success with surgical management of vertical fractures with bone screws.[46]

In a case report, the application of two four-hole 2.7-mm dynamic compression plates (DCPs) perpendicular to the vertical fracture plane resulted in solid bone union within 3 months and return of the horse to full athletic use.[49] The plate counteracts the tensile forces that displace the fracture, and the compression exerted across the fracture plane aids healing. The same result was achieved with a cervical fusion plate (Synthes Inc.) applied to the lateral surface of the fractured accessory carpal bone in another case (Figure 94-17).[49] The use of LCP technology or of the Unilock system (Synthes Inc.) would have application to this fracture repair as well.

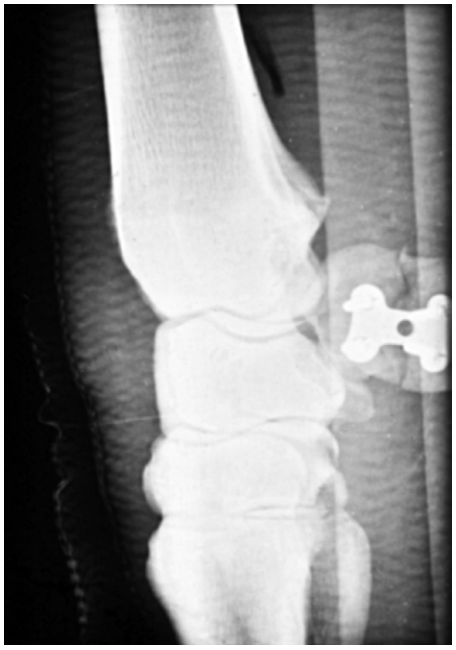

Figure 94-17. Lateral radiographic view of a 5-year-old Thoroughbred with a vertical fracture of the accessory carpal bone that was repaired with a cervical fusion plate. (Courtesy J. Auer, Zurich, Switzerland.)

Prognosis

Although conservative management of accessory carpal bone fractures can result in complete return to athletic soundness in fractures, complete bone union is usually not present. In chronic fractures, the potential for carpal canal syndrome exists and should be evaluated during the examination.[50] Because surgical management of simple vertical fractures has the potential for returning the horse to athletic soundness with minimal callus formation and good bone union, it is the currently recommended treatment.

CARPAL HYGROMA

Etiopathogenesis

Carpal hygroma is characterized by a subcutaneous swelling over the dorsum of the carpus. It is usually associated with a history of trauma to the region. Although the swelling is usually not infected, the potential for sepsis exists after drainage or injection, or both. The management of hygroma is discussed in detail in Chapter 82.

DISTAL RADIAL EXOSTOSIS

Clinical Signs

Exostosis of the distal radius is a rare cause of lameness in horses. The development of exostosis on the caudal border of the distal radial physis induces a tenosynovitis of the carpal canal and irritation and trauma of the deep digital flexor tendon. Exostosis is differentiated from osteochondroma by its location and by histologic examination. Osteochondromas are present on the caudal border of the distal radial metaphysis typically 2 to 4 cm proximal to the distal radial physis. Exostoses are devoid of hyaline cartilage remnants, which are present in osteochondromas.[51] Despite their histologic and location differences, osteochondromas result in the same clinical signs described later for distal radial exostoses and are treated the same. Some exostoses are radiographically inapparent and diagnosis is aided by ultrasonography, which may reveal tendon injury as well.

Regardless of the origin of the mass, the resultant tenosynovitis caused by both conditions results in both a weight-bearing and a swinging-limb lameness. The horse often carries the affected limb slightly abducted at the trot, and the lameness generally worsens with carpal flexion. Occasionally, the gait can appear somehwhat bizarre and almost dysmetric in the affected limb. Effusion of the carpal canal can be difficult to detect without careful inspection of the lateral aspect of the limb in the distal third of the forearm. Lameness is improved after intrasynovial anesthesia of the carpal canal. Consideration of the relationship of the proximal metacarpal structures and the carpal canal should be taken into acount as entry into the carpal canal while performing high palmar anesthesia is possible (see earlier).[1] Nuclear scintigraphy of the distal caudal radius may reveal focal intense uptake of radiopharmaceutical.

Radiography confirms the presence of the exostosis on the caudal aspect of the distal radial physis (Figure 94-18). Exostoses are occasionally seen as an incidental finding on survey radiography. Clinically significant exostoses generally have a

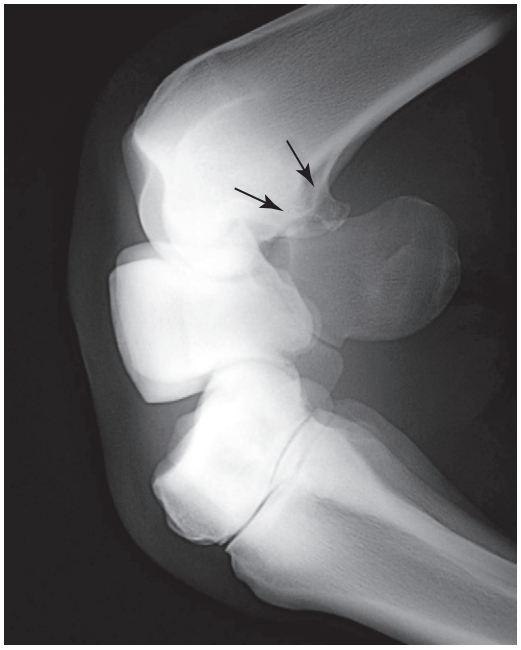

Figure 94-18. Flexed lateral radiographic view of the carpus of a 7-year-old Thoroughbred with an exostosis of the caudal aspect of the distal radial physis *(arrows)*. When these lesions extend more than 1 cm into the canal, they frequently are a cause of lameness.

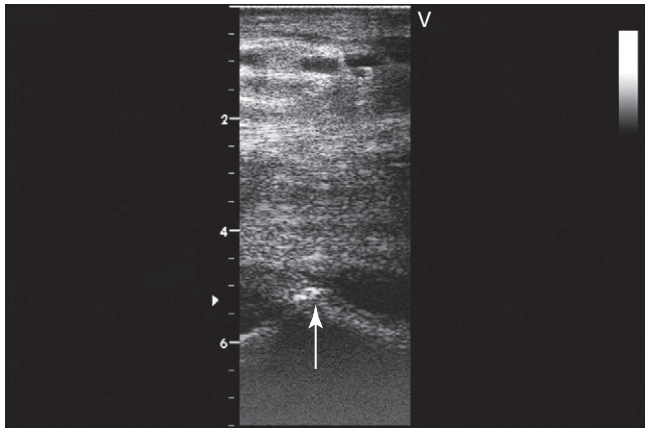

Figure 94-19. Transverse, caudal ultrasonic image of the distal radius of a 10-year-old Warmblood mare revealing fluid within the carpal canal and a caudally projecting exostosis *(arrow)* from the distal radius. This exostosis was not radiographically apparent.

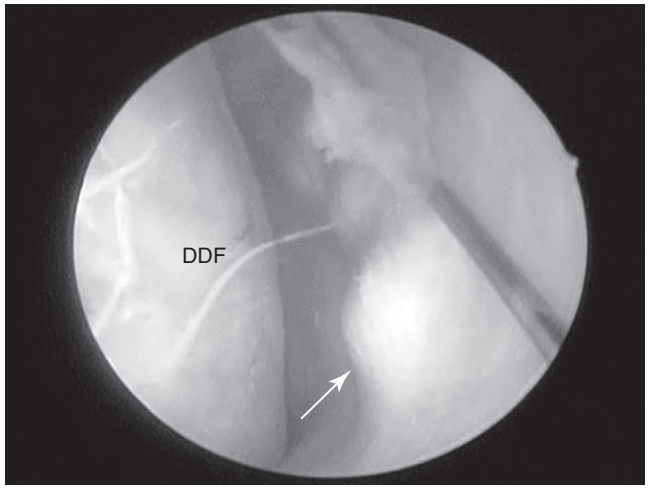

Figure 94-20. Endoscopic view of the mare in Figure 94-19, revealing the exostosis *(arrow)* (just below the hypodermic needle), dorsal to the deep digital flexor tendon *(DDF)*. There is evidence of tendon fibrillation and fibrin within the joint.

protrusion of at least 10 mm from the caudal aspect of the radius into the carpal canal, although potential "radiographically silent" exostoses can cause lameness as well. Ultrasonagraphic examination of the caudal radius and carpal canal may demonstrate radiographically inapparent exostosis, tendon injury, or carpal canal inflammation. It is important to confirm the exostosis as the cause of lameness by intrasynovial anesthesia prior to medical or surgical management (Figure 94-19).

Treatment

Nonsurgical Management

Nonsurgical management of carpal canal tenosynovitis involves intrasynovial injection of hyaluronan and corticosteroids. In my experience, if the response to therapy is incomplete or short-lived, surgical removal of the exostosis is the treatment of choice to remove the underlying cause of the tenosynovitis.

Surgical Management

Distal radial exostoses can be removed via carpal canal endoscopy, which is the method of choice.[52,53] The horse is positioned with the affected limb positioned uppermost or in dorsal recumbency and the forelimb and carpus prepared for aseptic surgery. The carpal canal is distended with lactated Ringer's solution and the arthroscopic cannula is placed between the lateral digital extensor and the ulnaris lateralis tendons through a stab incision at a level of the the distal radial physis.

The carpal canal is entered and a standard 4-mm 25-degree arthroscope is used for the examination. There is ample room within the carpal canal, and the arthroscope is usually positioned to examine the exostosis and any associated deep digital flexor tendon injury without difficulty. A needle is used to ascertain the proper location for the instrument portal distal to the arthroscopic portal (Figure 94-20). A 5-mm osteotome is used to loosen the exostosis from its bed. Care should be taken to separate the exostosis from the radius as close to its base as possible and not to completely dislodge it. The mass is grasped with a Ferris-Smith rongeur and removed. Large proliferations are best removed by extending the stab incision longitudinally. Alternatively the exostosis can be removed piecemeal through the stab incision using Ferris Smith ronguers. The associated bed can be further débrided with a rongeur, curette, or osteotome. On occasion the bony prominence must be detected by probing through the fibrous lining of the carpal canal onto the distal radius. Examination of the soft tissue structures and evaluation for adhesion formation is important during the endoscopy to determine prognosis. Release of the carpal flexor retinaculum can also be accomplished via endoscopic guidance if constriction of the carpal canal is suspected.[54]

The canal is lavaged and the fibrous layer of the tendon sheath or subcutaneous tissue and skin is closed in separate layers. Intraoperative radiographs are recommended to confirm the complete removal of the exostosis. Two-layer closure of the endoscopy and instrument portals is recommended in tendon sheaths to prevent leakage of synovial fluid, which occurs more commonly than after arthroscopic procedures. A full limb bandage is used for recovery from general anesthesia. Postoperative recommendations include box stall confinement for 4 weeks and pasture turnout for 4 to 8 weeks thereafter.

For additional information on this condition, please review Chapter 95.*

Prognosis

Prognosis after removal of distal radial exostoses is good; however, prognosis depends on concurrent flexor tendon injury or adhesion formation, which may be present. In a clinical report, 9 of 10 horses returned to full athletic function after endoscopic removal of the exostosis. The remaining horse became sound after further intrasynovial medication with hyaluronan.[45]

ANGULAR LIMB DEFORMITIES

Angular limb deformities of the carpus occur commonly in foals. Valgus deformity is more common than varus deformity. Angular limb deformities of the carpal region can be caused by physeal dysplasia, joint laxity, and cuboidal bone hypoplasia or malformation.

Methods of treating angular limb deformities of the carpal region include splints, bandages, casts, growth retardation, and, most commonly, periosteal elevation and transection. Treatment varies depending on the age of the horse and the cause of the deformity. Treatment options are discussed in detail in Chapter 86.

LUXATION (SUBLUXATION)

Etiopathogenesis

Carpal luxations are traumatic events caused by falling with one limb wedged in a fixed position, as a result of a being kicked or hit by a vehicle. Other causes are speculative because often the animal is found in the pasture with an obvious luxation whose origin was not observed.

Diagnosis

The animal might be presented with an obvious deformity in the carpus resulting in an abnormal position of the limb distal to the carpal region. In this instance, the diagnosis is obvious from radiographs (Figure 94-21). Alternatively, the animal might bear weight on the limb but show a marked lameness. Standard radiographs might show some small bone chips in the periarticular region as the only abnormality. If the luxation is in association with skin trauma, crepitus is recognized in the periarticular soft tissues. A definitive diagnosis of joint involvement with the wound is made through arthrocentesis and stress radiographs under general anesthesia.

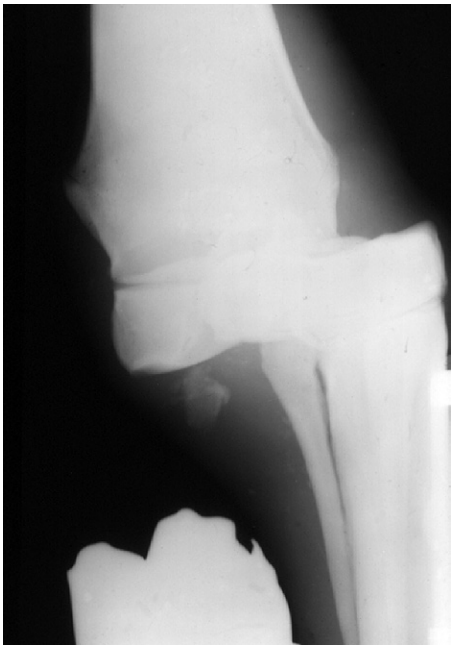

Figure 94-21. Craniocaudal radiographic view of the distal radius of a horse with complete luxation of the middle carpal joint. Note that the proximal and distal rows of carpal bones overlap and are rotated, resulting in a lateral view of the distal limb. A bone fragment is recognized below the intermediate carpal bone, indicating that an avulsion fracture of the palmar aspect of the intermediate carpal bone has occurred. (Courtesy J. Auer, Zurich, Switzerland.)

Treatment

Luxations may be managed conservatively or through surgical intervention.

Nonsurgical Management

Conservative treatment involves cast application for 3 months with two or three cast changes. During this time, the ruptured periarticular soft tissues heal by fibrosis. Depending on which joint is involved, decreased carpal flexion is the result. Careful management of the convalescent period helps in restoring a useful animal. Coaptation should only be considered if the bone column is intact and the bone column does not collapse when the horse is bearing weight.

Surgical Management

Surgical management through partial or pancarpal arthrodesis is an option and is discussed in Chapter 81. Proximal fractures of the second or fourth metacarpal bone may cause subluxation of the carpometacarpal joint. Small plates to repair these fractures are recommended to overcome the tension forces of the collateral ligaments, even in small foals. My experiences with coaptation or screw fixation alone have been disappointing. Plate fixation of proximal fractures of the small metacarpal bones is discussed in detail in Chapter 93.

*An editorial decision was made to discuss this condition in both chapters because slightly different aspects are elucidated in addition to good pictorial material.

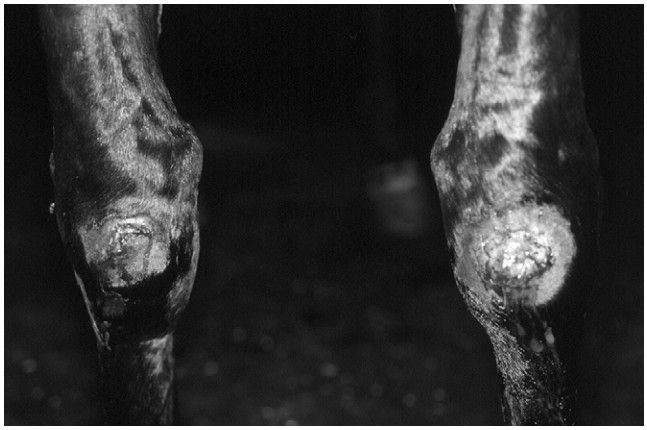

Figure 94-22. A Warmblood horse admitted for treatment of coronation, which occurred during a riding accident. (Courtesy J. Auer, Zurich, Switzerland.)

CORONATION (COURONNEMENT)

Etiopathogenesis

Coronation refers to a severe soft-tissue injury of the dorsum of the carpus that occurs after a fall. It is characterized by the loss of skin over the carpus, and in more severe cases, the wound involves the synovial sheaths of the extensor carpi radialis, common digital extensor tendons, or one of the carpal joints.

Diagnosis

Physical signs of injury are obvious, with a flap or degloving wound present over the dorsum of the carpus (Figure 94-22). Entry into the tendon sheaths or joints might be obvious, but puncture wounds might go unnoticed.

If synovial involvement is suspected, a needle should be placed into the carpal joint or tendon sheath at a site distant from the wound after aseptic preparation and the structure distended with sterile saline to detect any leakage through a skin defect. Care must be taken to avoid entry through

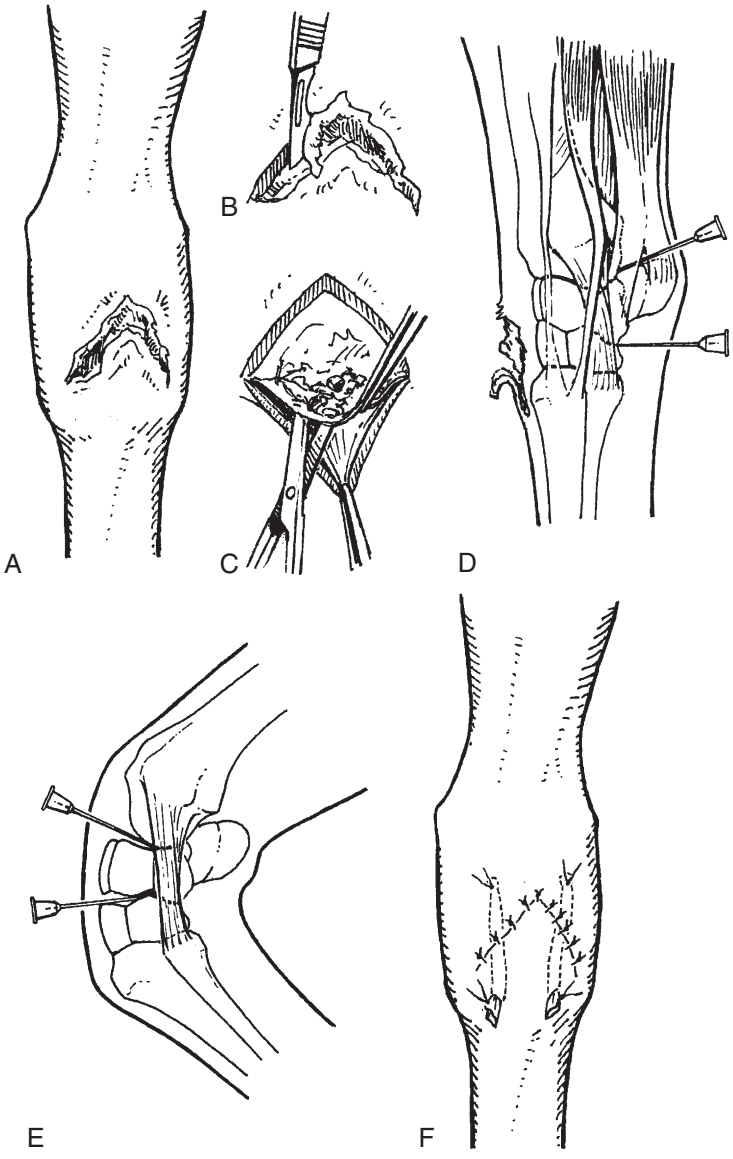

Figure 94-23. Treatment of a coronation injury. **A,** Appearance of the injury. **B,** A 1- to 2-mm rim is sharply removed around the skin defect. **C,** Careful débridement of the subcutaneous tissues aids in determining if the joint is involved as well. **D,** Needles placed from the palmar aspect of the carpus into the joints for joint lavage allows verification of any potential joint involvement. **E,** Alternatively, the needles may also be inserted from the dorsolateral or dorsomedial aspect of the carpus. **F,** After installing one or two drains, the skin is closed in routine fashion.

traumatized or infected tissue to prevent inoculation of the synovial structure with bacteria. For the tendon sheath, a proximal location is chosen, and for the carpal joints, the palmar recesses may be used. Any draining fluid obtained is collected and submitted for cytologic examination and culture and susceptibility testing. Occasionally, the extensor carpi radialis or common digital extensor tendon is ruptured as well.

Radiographs should be obtained after cleaning the wound to assess bone injury and identify additional debris, especially gravel. Contrast radiography can be used to identify communications between the synovial structures and the wound. The use of ultrasonography is limited because of subcutaneous emphysema from the wound.

Treatment

Treatment consists of management of the wounds and any involved synovial structure. Wound management and management of synovial infections are discussed in Chapters 26, 27, and 85, respectively. Some specific issues of management of these wounds deserve mentioning here.

I prefer to close acute wounds into synovial structures after lavage and intrasynovial antimicrobial therapy unless severe contamination exists. A 1- to 2-mm rim of skin is sharply dissected along the skin defect, followed by careful sharp surgical débridement of the deeper tissues (Figure 94-23, *A*, *B*, and *C*). In cases where the joint capsule is opened as well, intra-articular lavage is mandatory. This can be accomplished through needles placed from the palmar aspect of the carpus (see Figure 94-23, *D*) or from the dorsolateral or dorsomedial aspect of the carpus (see Figure 94-23, *E*). The joint capsule is subsequently closed using simple interrupted sutures of a size 0 or 2-0 absorbable suture material.

After implantation of one or two drains the skin is closed in routine fashion (see Figure 94-23, F). Delayed wound closure is sometimes necessary to identify the extent of injury and allow débridement. The use of splints, cast bandages, and casts is usually necessary to provide immobilization for wound healing. Supportive therapy (such as repeated joint lavage), anti-inflammatory agents, and systemic, regional, and/or intrasynovial antimicrobial agents varies with the type and severity of the wound.

Horses can return to full athletic activity after injuries that involve superficial structures. Wounds that involve the tendon sheaths, joints, or carpal bones have a reduced prognosis, which is determined by the extent of the wound and use of the horse. Prognosis is improved with prompt (less than 24 hours) treatment of the open joint. Even wounds that have had significant loss of tissue over the dorsum of the carpus can heal and provide a satisfactory outcome if the synovial structures respond appropriately.

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CHAPTER 95

Radius and Ulna

Jeffrey P. Watkins

The equine antebrachium consists of the radius and ulna in conjunction with the muscles that flex and extend the carpus and digit. The radius is the major weight-supporting bone in the antebrachium. The olecranon, by serving as the insertion site of the triceps muscle, acts as the lever arm during elbow and carpal extension. The primary indications for surgical intervention in this area are caused by traumatic events that result in fracture of either the ulna or the radius.

FRACTURES OF THE ULNA

Fractures of the ulna are relatively common in foals and adult horses. A wide variety of fracture configurations occur, and most are amenable to open reduction and internal fixation. The treatment of choice is fracture fixation using a bone plate applied as a tension band. Provided complications are not encountered, the prognosis is favorable for a return to full function.

Anatomy

The ulna has two components, the body and the olecranon process (or more simply, the olecranon). The body of the ulna extends distad from the level of the proximal radial physis. It is triangular in cross section and tapers distally, essentially ending in the mid-diaphyseal region of the radius. It is closely adherent to the radius, and in adult horses, it is fused to the caudal cortex of the radius distal to the interosseous space. Functionally, it serves as the distal attachment of the lever arm through which the triceps muscle functions.

The olecranon is found proximal to the body of the ulna. It is concave medially and has a proximal tuberosity for muscle attachment. Tendinous insertions on the olecranon tuberosity, principally by the triceps brachii muscle, provide elbow extension during locomotion and in doing so, impart weight bearing. Equally important, by extending the elbow during standing, the triceps apparatus allows extension of the carpus through engagement of the stay apparatus of the forelimb. The olecranon is also an important component of the cubital joint, with articular cartilage present in the proximal aspect of the trochlear notch. In the distal aspect of the trochlear notch there is a synovial fossa, which is devoid of articular cartilage.

Foals have a physis in the proximal olecranon. Because the separate center of ossification of the physis is not involved in formation of a joint, it is termed an *apophysis*. Although growth at the olecranon physis is responsible for the ultimate size of the olecranon tuberosity, disturbances of physeal growth

following injury or fracture repair are well tolerated and do not result in disability. However, because growth occurs at the proximal radial physis, the ulna must be able to elongate during rapid growth of the foal along with the radius to maintain congruency between the humeral condyle and the trochlear notch of the olecranon.

Pathogenesis

A direct blow from a kick or a fall causes most fractures of the ulna. A common history includes injury during halter training of weanling foals. Often the fracture occurs when the foal rears up and falls onto its side. The body of the ulna, or more commonly the olecranon, fractures, and fragments are displaced during contraction of the triceps muscle when the foal jumps to its feet.

Several classification systems for ulnar fractures have been published, but to prevent confusion, only the one that I use is listed here (Figure 95-1).[1] The most common configuration in horses younger than 1-year of age is the type 1b fracture, where the caudal one third to one half of the physis is disrupted, propagating the fracture into the metaphysis, and finally exiting into the proximal aspect of the trochlear notch near the anconeal process (Figure 95-2).[2] Less commonly, the fracture breaks out the cranial cortex of the olecranon proximal to the anconeal process and remains nonarticular (also type 1b). Occasionally, comminution occurs at the anconeal process.

Other configurations include physeal separations (type 1a), most common in neonates; transverse fractures entering the trochlear notch near its midpoint (type 2); distal olecranon–proximal ulnar body fractures, which usually have an oblique configuration traversing the bone in a proximal and cranial direction to enter the distal aspect of the trochlear notch (type 5) and are most commonly encountered in horses older than 1 year of age[3]; and comminuted fractures (type 4). Type 3 fractures—nonarticular, nonphyseal fractures of the olecranon extending from the caudal cortex to the cranial cortex proximal to the anconeal process—are rare.

The degree of soft tissue injury varies greatly. Contusions resulting from a kick are often accompanied by a skin wound at the site of impact. These wounds might communicate directly with the fracture, and if the fracture contains an articular

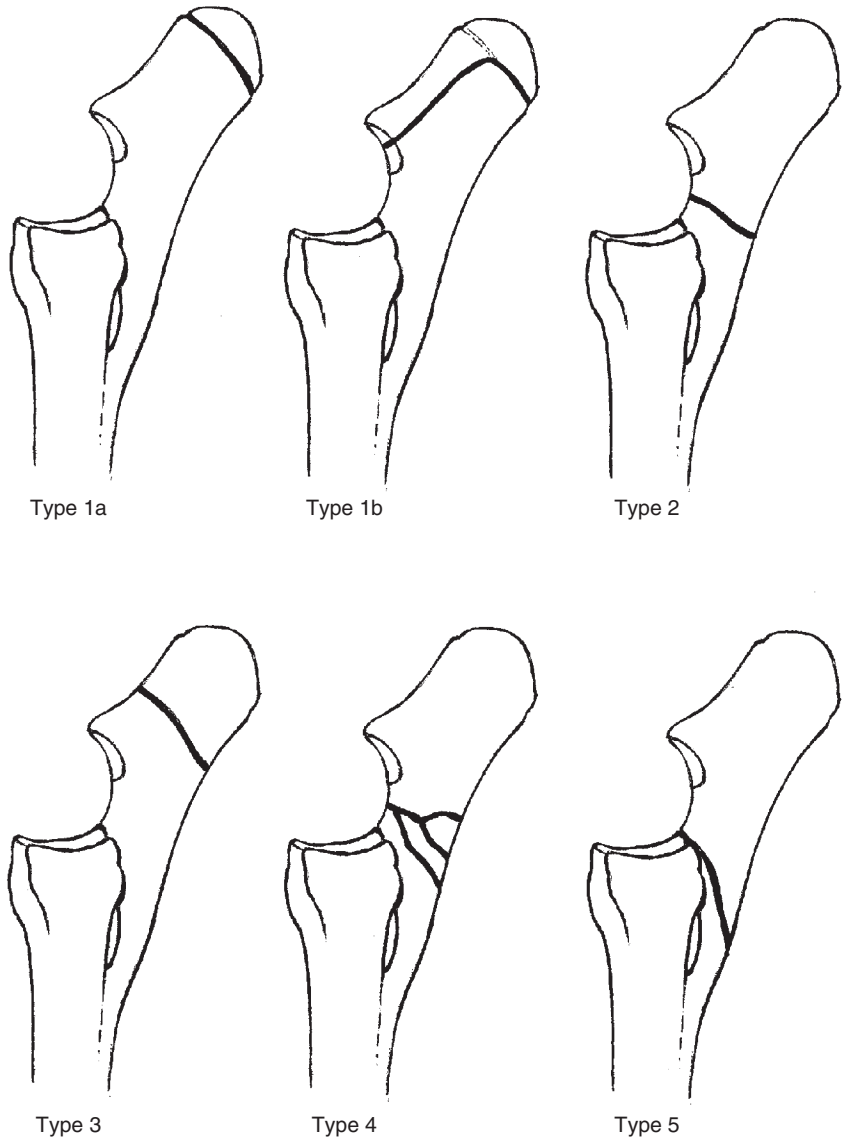


Figure 95-1. Classification scheme for ulnar fractures: *Type 1a,* Fracture along the apophyseal physis; *Type 1b;* fracture staring out along the apophyseal physis and then breaking into the metaphyseal part of the olecranon process and finally entering the joint; *Type 2,* simple fracture through the body of the olecranon process with articular involvement; *Type 3,* simple fracture through the body of the olecranon process without articular involvement; *Type 4,* multifragment fracture of the olecranon process with or without articular involvement; *Type 5,* distal oblique ulnar fracture with marginal articular involvement.

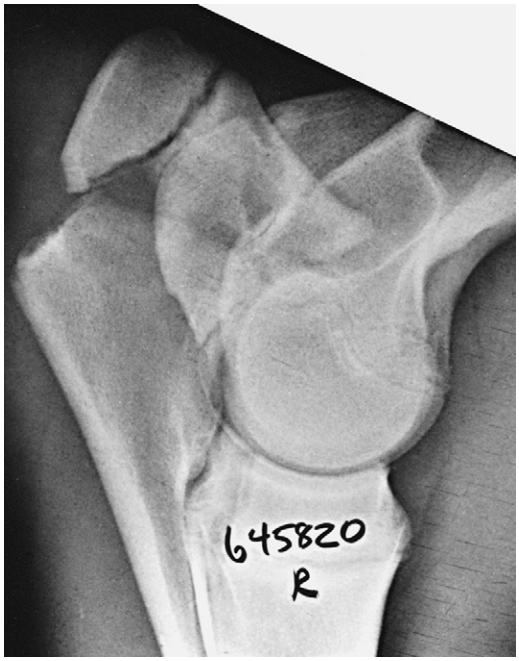


Figure 95-2. Mediolateral radiographic view of a displaced type 1b, articular olecranon fracture.

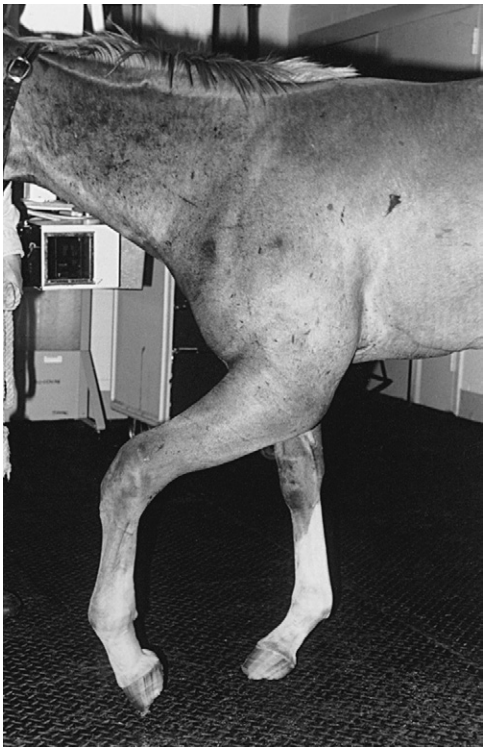


Figure 95-3. Photograph demonstrating the typical dropped elbow stance in a foal with a displaced olecranon fracture.

component, joint sepsis can develop secondary to bacterial colonization of the fracture and subsequent extension into the joint cavity. Displacement of the fracture is another indicator of the degree of soft tissue damage. Marked displacement indicates disruption of the aponeurosis of the ulnar head of the deep digital flexor muscle laterally and the ulnar head of the flexor carpi ulnaris muscle medially.

Diagnosis

Lameness, accompanied by a dropped elbow, is typical on presentation. However, with nondisplaced fractures typical of type 5 fractures, lameness may be the primary presenting complaint. When the fracture becomes displaced, the affected limb will have a dropped-elbow appearance, because the carpus cannot be fixed in extension. Depending on the amount of displacement, the elbow might appear only slightly dropped, with the carpus held in flexion, the limb placed slightly craniad, and the toe touching the ground. With severe displacement, marked dropping of the elbow along with carpal flexion is evident (Figure 95-3). The differential diagnosis for a dropped elbow stance includes ulnar fracture, humeral fracture, and radial neuropathy.

Soft tissue swelling is usually not severe in most ulnar fractures and is localized to the caudolateral aspect of the proximal antebrachium. This is distinct from humeral fractures, in which the swelling is typically much greater and best visualized by standing in front of the horse and comparing the relative thickness of the humeral regions on affected and unaffected sides. Pain on deep palpation and manipulation of the olecranon aids in localizing the fracture.

Definitive diagnosis is based on radiographic documentation of the fracture. Best detail of the area is obtained with a medial to lateral projection and the beam obliqued slightly cranial to caudal. When the affected limb is extended craniad, complete visualization of the cubital joint and olecranon is possible, and the fracture configuration and location can be determined. With type 1b and type 4 fractures, it is important to visualize the anconeal process to delineate comminuted fragments. A cranial to caudal radiograph is necessary to detect concurrent injury to the proximal radius (Figure 95-4). A contrast arthrogram determines the extent of communication between a soft tissue wound and an articular fracture.

Treatment

First Aid

Initial care for horses with fracture of the ulna accompanied by a wound should include appropriate wound therapy consisting of removal of hair at the wound margins and careful cleansing and débridement, followed by application of a sterile bandage to prevent further contamination before surgery. Systemic broad-spectrum bactericidal therapy should be initiated immediately.

It is important to ascertain the extent of soft tissue trauma. Fortunately, many wounds encountered with olecranon fractures are superficial and do not markedly affect therapy or prognosis. However, deep wounds that communicate with the fracture, particularly articular fractures, dictate immediate and aggressive therapy. Therapy for open articular fractures includes administration of intra-articular antimicrobial drugs. In addition, antimicrobial-impregnated polymethyl methacrylate (PMMA) beads placed into the depths of extensive soft tissue wounds may reduce bacterial numbers in the wound prior to open reduction and internal fixation of the fracture.

Many horses with displaced fractures cannot engage their stay apparatus and therefore are unable to support weight on

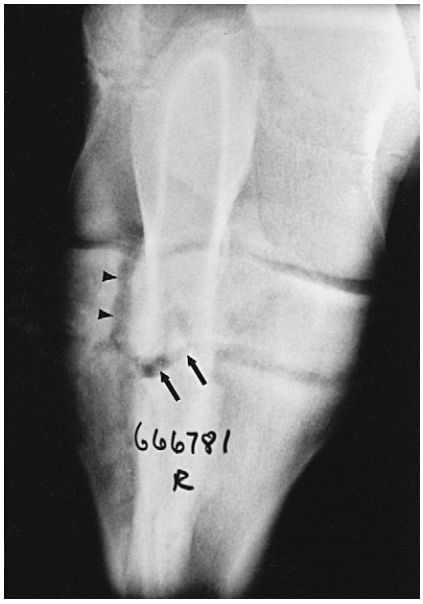


Figure 95-4. Craniocaudal radiographic view demonstrating a Salter-Harris type III fracture of the proximal radius *(arrowheads),* which occurred in conjunction with a fracture of the ulna *(arrows).*

the affected limb. If so, a splint is applied when wound therapy is complete. Although it is not possible to splint the fracture per se, splinting the carpus in extension allows weight to be supported on the affected limb. In horses with a droppped elbow secondary to a displaced olecranon fracature, splinting the limb in extension markedly reduces anxiety and minimizes fatigue of the contralateral limb before internal fixation.

The splint should consist of rigid material applied over a well-padded full-limb bandage. Splints are fashioned from schedule 40 polyvinylchloride (PVC) pipe; an appropriate length is cut from a 15-cm (6-in) diameter pipe and split longitudinally, using one third of the pipe's circumference. The splint should extend from the ground to the proximal antebrachium and is affixed to the bandage with nonelastic adhesive tape. Additional padding at the proximal aspect of the splint prevents soft tissue damage near the fracture site. Futher information on first aid is found in Chapter 73.

Nonsurgical Management

Conservative therapy consisting of strict stall confinement has been advocated for nondisplaced, nonarticular fractures of the olecranon, especially type 5 fractures.[1,4] If the patient does not bear full weight on the limb, a full-limb splint is applied. Functional soundness was reported in 7 of 10 horses with type 5 fractures managed conservatively.[4] Poor results are achieved following conservative therapy for other fracture configurations.[1]

Horses with nondisplaced, nonarticular fractures that are managed conservatively have a prolonged convalescent period because of the instability of the fracture. Healing is slow, and lameness is protracted. Although type 5 fractures do not violate the articular surface of the cubital joint, they communicate with the synovial cavity. Inflammatory mediators released into the joint from the fracture may have detrimental effects on the articular cartilage and lead to degenerative articular changes. More importantly, synovial fluid entering the fracture line may disrupt healing, leading to development of a pseudarthrosis. Despite the inherent risk of surgery and anesthesia, surgical treatment is preferable, and the primary determinant for choosing conservative therapy over surgical therapy is economics.

Surgical Management

Open reduction and internal fixation is the treatment of choice for articular or displaced fractures of the ulna. By using the tension-band principle, stable fixation of nearly all fracture configurations can be achieved with minimal complications and a favorable prognosis. Tension-band fixation can be accomplished using a plate applied to the caudal aspect of the ulna or by using tension-band wire or cable fixation.

The surgical approach to ulnar fractures has been described.[5] It is advisable to curve the skin incision proximad to avoid the point of the elbow. When possible, wounds should also be avoided when approaching the fracture. After the skin and subcutaneous fascia have been incised, deep dissection between the ulnaris lateralis muscle and ulnar head of the deep digital flexor tendon exposes the caudal and lateral aspects of the ulna.

Distally, the ulna is exposed for plate application by subperiosteal dissection. Proximally, the caudal and lateral aspects of the olecranon are closely invested in the dense fibrous connective tissue aponeuroses of the ulnar head of the deep digital flexor and flexor carpi ulnaris muscles. Sharp dissection of the fibrous connective tissue attachments from the olecranon is necessary for plate application. Additional dissection exposes the proximal aspect of the olecranon tuberosity for fixation of type 1 and 3 fractures. In these cases, the insertion of the triceps is sharply divided in the sagittal plane parallel to the longitudinal fiber bundles and dissected abaxially at their insertion on the olecranon far enough to accommodate the width of the plate.

After the bone is exposed, the fracture hematoma is evacuated and the fracture line débrided of all fibrin or, in chronic cases, fibrous tissue. Assessment of the bone at this time might reveal hairline cracks in the ulna that might not have been evident radiographically. If the anconeal process is comminuted, the fracture is opened and the fragment is removed through the fracture gap. An alternative technique involves cranial dissection along the lateral aspect of the ulna until the joint can be palpated and penetrated with a short stab using closed blunt scissors. Finger palpation allows identification of the anconeal fragment and its subsequent removal. In displaced fractures, reduction is accomplished by grasping the olecranon process medially and laterally with a large pointed reduction forceps and applying caudal and distal traction with the limb held in extension and pulled caudad.

A soft aluminum template can be used to provide a model for plate contouring. The plate is positioned to allow screw engagement of a minimum of five cortices on either side of the fracture. Usually a narrow limited-contact dynamic compression plate (LC-DCP), a dynamic compression plate (DCP), or a locking compression plate (LCP) is used for fixation. In large patients or for more comminuted fractures, a broad LC-DCP, DCP, or LCP may be chosen. In adult horses with comminuted fractures, double plate fixation with a caudal and a lateral plate may be necessary. A study evaluating 18 ulna fractures in 16 patients treated with one or two LCPs, revealed results similar to those with the use of DCPs.[6] Infection was still the main factor influencing the outcome and complete healing.

The appropriately contoured plate is applied by positioning one screw proximal and another screw distal to the fracture with the fracture held in reduction. It is important to apply the plate to the caudal border and not err to the caudolateral aspect to prevent the screws from exiting through the medial aspect of the olecranon tuberosity proximally and penetrating the medial cortex of the radius distally. The first cortex screw is placed in the proximal fragment using the neutral drill guide through a compression hole. This screw is not tightened completely, and the plate is displaced distad before placing the second cortex screw in the distal fragment using the load drill guide. Tightening these two screws compresses the fracture and brings the plate into contact with the underlying bone. Overcompression of minimally displaced fractures with comminution at the articular surface can result in fragment displacement. Therefore, screws should be applied in the neutral position with comminuted fractures.

At this point in the repair, an intraoperative lateromedial radiograph should be obtained to assess the fixation (Figure 95-5). The intraoperative radiograph can be used to evaluate the adequacy of reduction and screw position and as a guide for directing and gauging the approximate length of the remaining screws.

If further compression is needed, an additional cortex screw on either side of the fracture can be placed in the load configuration. Before tightening these additional load screws, the previously placed screw in the same fragment should be loosened slightly. The remainder of the cortex screws are placed in the neutral position. If an LCP is used, one or two locking screws are placed on either side of the fracture with another locking screw positioned at either end of the plate. If needed, all but two screws (to achieve axial compression) can be locking head type (Figure 95-6).

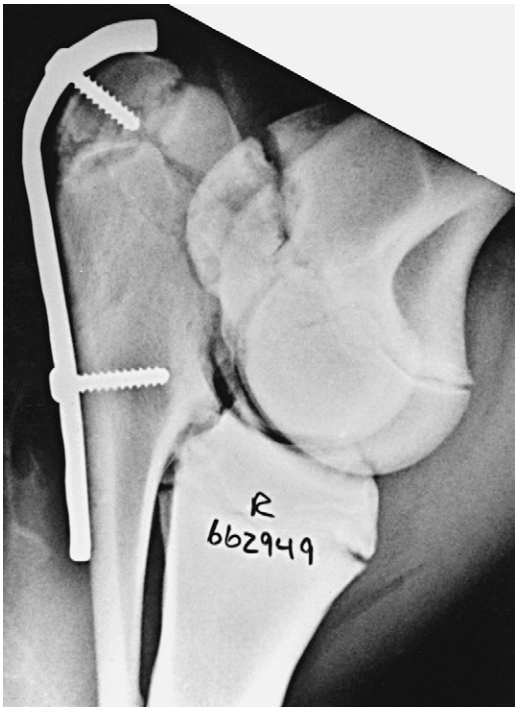


Figure 95-5. Intraoperative lateromedial radiographic view documenting fracture reduction and plate application by means of a proximal and distal cortex screw. The remaining cortex screws are placed in lag fashion through the plate into the cranial cortex of the olecranon in a type 1b fracture.

Screw placement in the proximal fragment must avoid penetrating the concave medial cortex. By aligning the drill bit parallel to a Steinman pin inserted along the medial aspect of the olecranon process in a cranial direction, the surgeon is assured that the drill hole will exit at the cranial border of the bone, ensuring solid fixation. Screws at the level of the trochlear notch should not penetrate into the joint.

Distally, screws should not engage the caudal cortex of the radius in foals. If the body of the ulna is transfixed to the radius, growth of the proximal radial epiphysis forces the anconeal process into the humeral condylar notch, and subluxation of the cubital joint develops, resulting in elbow dysplasia. The age at which it is safe to engage the caudal cortex is not well defined. In one study, there was growth at the proximal radial physis with relative displacement of the ulna until 18 months of age.[7] However, in another study in which the radius and ulna were transfixed, it appeared that subluxation was not a clinical problem in foals that were 7 months old or older at the time of surgery, even though subluxation was evident radiographically and at necropsy.[7]

Therefore, I recommend that engagement of the caudal cortex of the radius be avoided in foals younger than 1 year of age. If not, careful monitoring is necessary, and either the implants or the screws engaging the radius should be removed as soon as the fracture has healed (although subluxation can occur even after the implants are removed).[8] If subluxation occurs, it can be managed with an osteotomy of the body of the ulna at a level distal to the joint (Figure 95-7).[9] By not stabilizing the osteotomy, the ulna can adjust into a normal position during the postoperative period. Another alternative is active proximal displacement of the proximal fragment through flexion and extension of the joint and subsequent bridging of the osteotomy gap with a narrow plate.

The proximal location of types 1 and 3 fractures challenges the fixation, because the small size of the proximal fragment limits the number of screws available for purchase. With a type 1b fracture, the plate is contoured over the dorsal aspect of the apophysis to allow purchase with three short screws. The fourth and usually the fifth screw in the plate are placed in lag fashion across the metaphyseal portion of the fracture to engage the cranial cortex of the olecranon (Figure 95-8). Purchase in the cranial cortex of the olecranon adds substantial strength to the fixation.

Compression at the fracture line improves fracture reduction; this is particularly important for type 1b fractures that enter the articulation. Care is taken with type 1b fractures to avoid overcompressing the caudal aspect of the fracture using a DCP, because the cranial aspect of the fracture can become displaced. In these fractures, the initial screw is placed in the apophysis through the second plate hole, and another screw is placed in the sixth or seventh hole of the plate. These screws are not completely tightened, allowing the fracture to be held in reduction while the lag screws are placed across the metaphyseal fracture. When the lag screws are tight, the remainder of the fixation is routine.

Prognosis

The prognosis following plate fixation of ulnar fractures is favorable. Success, defined as fracture union, occurred in 13 of 19

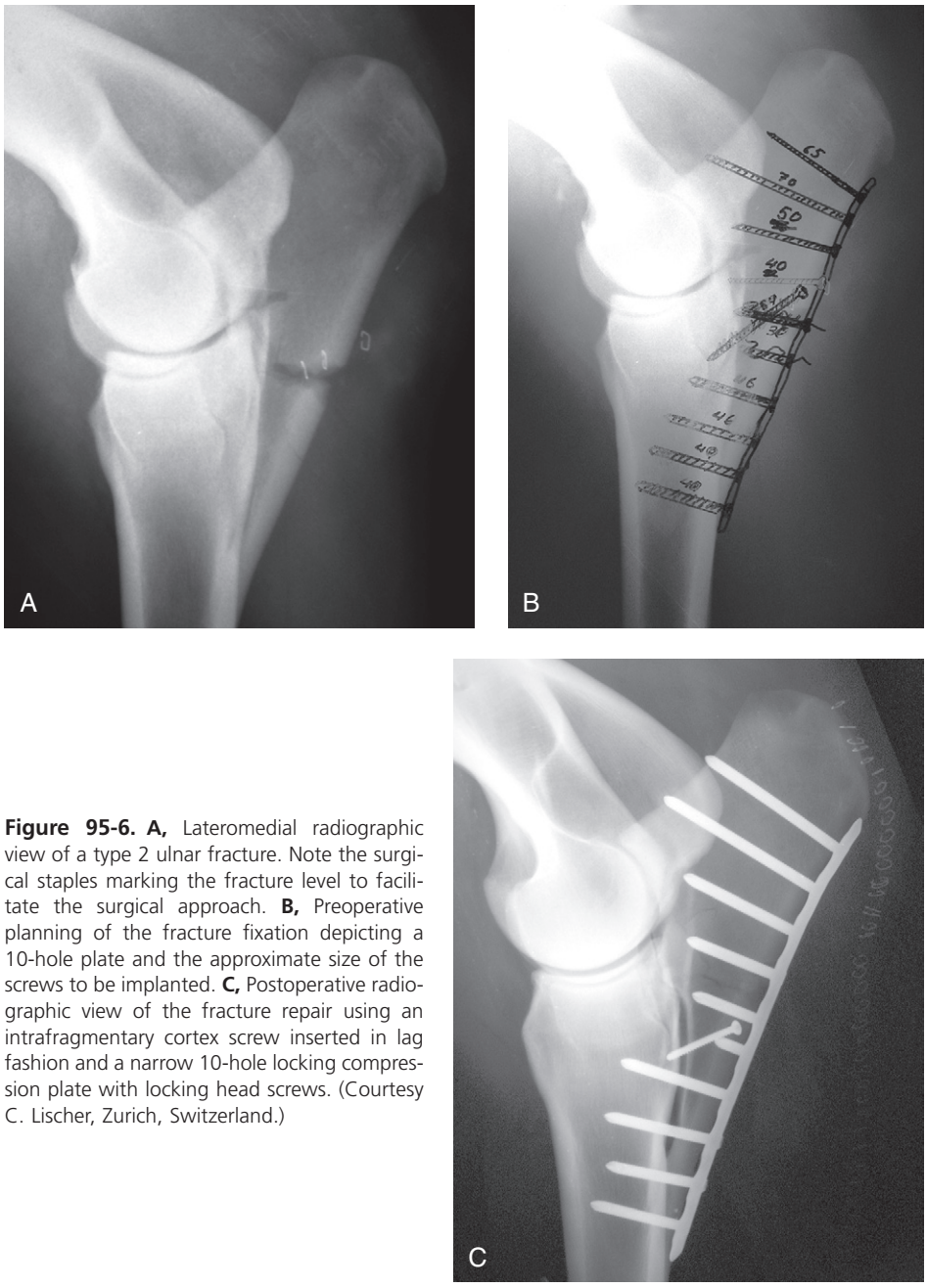


Figure 95-6. A, Lateromedial radiographic view of a type 2 ulnar fracture. Note the surgical staples marking the fracture level to facilitate the surgical approach. **B,** Preoperative planning of the fracture fixation depicting a 10-hole plate and the approximate size of the screws to be implanted. **C,** Postoperative radiographic view of the fracture repair using an intrafragmentary cortex screw inserted in lag fashion and a narrow 10-hole locking compression plate with locking head screws. (Courtesy C. Lischer, Zurich, Switzerland.)

patients (68%), with a return to full function in 11 of the 13 cases.[1] Another study reported return to full function in 16 of 21 patients (76%).[10] Type 2 fractures were the most common configuration encountered in both studies. When both patient populations were combined, full function returned in 27 of 34 patients (79%). Type 4 fractures were the second most common configuration in these reports, but only two of seven patients returned to full function. In a retrospective analysis of 17 adult horses with comminuted articular fractures (type 4), long-term follow-up was available for eight, and five were sound enough for riding or training.[11]

In another retrospective study of 77 olecranon fractures, type 1b fractures were the most common configuration in horses younger than 1 year of age.[2] Twenty horses underwent plate fixation as previously described (see Figure 95-8). Fracture healing occurred in 19 of the 20 repaired fractures. Sixteen horses were available for long-term follow-up, and 13 (81%) were reported to be sound. Twelve horses were older than 2 years of age at the time of follow-up, and nine of these were performing their intended purpose. The remaining four horses, which were not yet 2 years of age, were all reported to be in training for their intended use without evidence of complications related to their fracture. In this case series, there were no catastrophic fixation failures, and other complications were minimal. Furthermore, four horses with comminution at the anconeal process did not appear to be negatively effected by removal of the anconeal process fragment. Type 5 fractures were the most common fracture in horses older than 1 year of age, and in those cases, 19 of 20 patients that were repaired by plate fixation had a successful outcome. Follow-up was

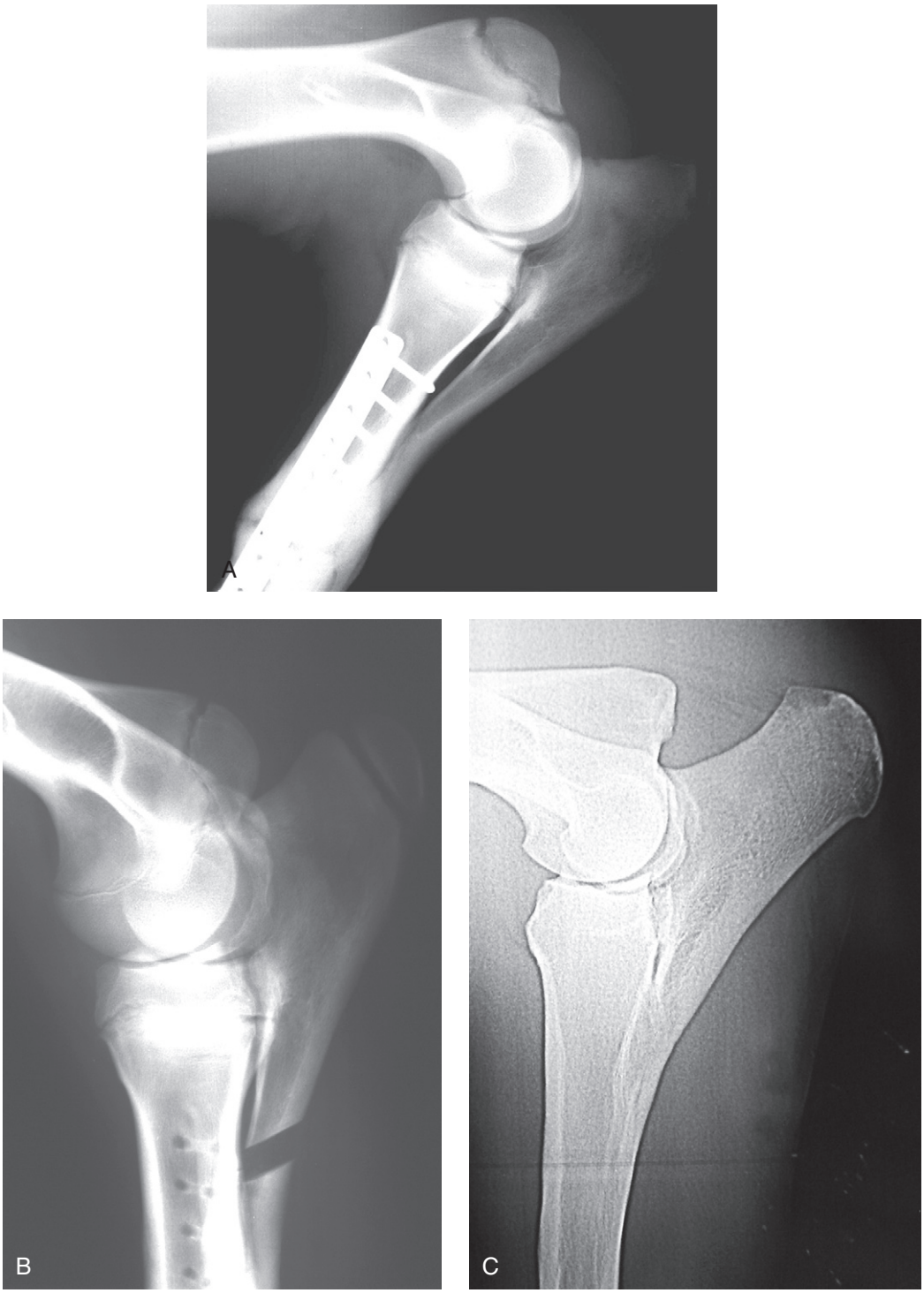


Figure 95-7. A, Follow-up radiographic views of a mid-diaphyseal radius fracture in a foal treated with bone plates. The fracture shows progressive healing; however, there is a subluxation of the elbow joint. **B,** An ulnar osteotomy was performed 10 days prior to taking this radiograph, which resulted in correction of the subluxation and restoration of the normal joint anatomy. An osteotomy gap of 10 mm developed. **C,** The 6-year follow-up of the same animal included a radiographically normal configuration of the joint without osteoarthritis and a sound animal. (Courtesy G. Bodo, Budapest.)

unavailable for 5 horses. Time since surgery in 13 horses was at least 2 years in 13 patients, with 11 of these horses performing their intended use.[3]

Tension-band wire fixation has been used to manage olecranon fractures in horses. In 22 horses managed with tension-band wiring, fracture union was achieved in 18 cases, and 13 of 17 horses returned to athletic function.[12] Patients are candidates for tension-band wiring if they weigh 250 kg or less. When fractures are in close proximity to the physis, either pins or screws are positioned cranial to the apophysis and directed

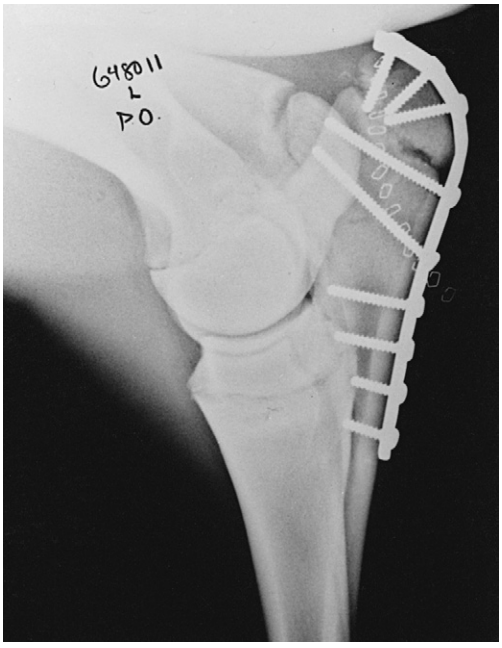


Figure 95-8. Postoperative mediolateral oblique radiographic view of a repaired type 1b articular ulnar fracture. Note the two long interfragmentary screws reaching to the cranial edge of the olecranon process.

distad into the ulnar body. Distal fractures are repaired with wires alone or in conjunction with lag screws. Fractures in foals and weanlings are repaired with at least two or three 1.2-mm diameter wires, whereas four to six 1.5-mm diameter wires are used in older horses.

The advantages of tension-band wiring compared to plate fixation are less-expensive equipment, less risk of screws entering the joint space or engaging the caudal cortex of the radius, less risk of fracture of the apophysis because screw holes in the proximal fragment are not necessary for fixation, and, with more distal fractures, less tissue dissection required.

Complications

Complications of repair of ulnar fractures are infection, fixation failure, tendon contracture, support limb varus deformity, suspensory apparatus fatigue, and support limb laminitis.

Infection is most likely following repair of open, contaminated fractures. However, fractures with substantial soft tissue trauma also have an increased risk. Methods of reducing the infection potential include the use of perioperative systemic antimicrobial agents and, in high-risk patients, local application of antimicrobial-impregnated PMMA, usually as beads or in some cases as plate-luting material. If the fracture becomes infected, but the fixation is stable, a successful outcome is likely. However, the convalescent period is substantially prolonged, and lameness often persists until the infection is resolved. Resolution of infection requires implant removal after the fracture has healed. Infection in the presence of instability at the fracture invariably results in failure. Articular fractures that become infected have the additional risk of joint sepsis, which substantially worsens the prognosis.

Fixation failure can occur secondary to fracture of the apophysis in proximal olecranon fractures, especially with type 1a fractures in neonates, in which the apophysis consists of an ossified nucleus surrounded by a relatively wide rim of hyaline cartilage. Following plate fixation of proximal fractures in older foals and adults, failure can result from fracture of the proximal fragment through the screw holes.[12] However, this complication did not occur following plate fixation of 20 patients with type 1b fractures.[2] Implant failure also can occur from using wire techniques, especially in large foals and adults.[12] Therefore, fractures in large foals and especially in adults deemed at risk for implant failure are candidates for plate fixation. It is important to note, that the proximal end of the plate should extend to the level of the physeal scar of the olecranon to minimize the risk to failure at the proximal aspect of the plate.

At the distal aspect of the plate, where the ulna is positioned along the caudal aspect of the lateral cortex of the radius, screws should be angled toward the midline to avoid the lateral cortex of the radius. Use of locking head screws in the distal aspect of the plate may not be indicated as they have to be inserted perpendicular to the plate, which could weaken the bone and result in a fracture of the radius, especially if the large 5-mm screw is used (Figure 95-9).[6]

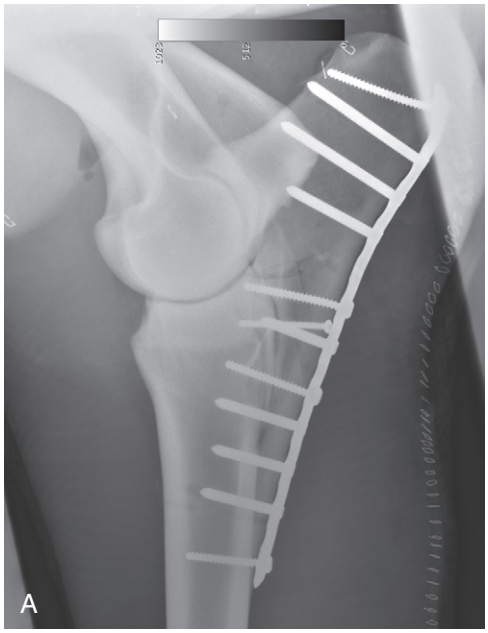
Prolonged lameness for any reason results in a number of compensatory complications. Persistent lameness or increasing lameness in the postoperative period indicates fracture instability, infection, or both. With articular fractures, osteoarthritis secondary to incongruent joint surfaces or prolonged, severe synovitis causes persistent lameness. In foals, persistent lameness often results in varus deformities of both the carpus and the metacarpophalangeal (MCP) region, as well as fatigue of the suspensory apparatus with hyperextension of the MCP joint in the contralateral limb. The ipsilateral limb can develop a flexural deformity of the carpus or MCP region, or both. In adult horses, ipsilateral flexor contracture may develop, but support limb laminitis is the most likely and potentially devastating complication in the contralateral limb.

FRACTURES OF THE RADIUS

In horses, radial fractures are not as common as ulnar fractures. When they occur, a wide variety of fracture configurations is possible. For simple, displaced radial fractures, open reduction and internal fixation can be expected to produce a good to excellent outcome in horses weighing less than 250 kg. Fixation of radial fractures in adult horses, however, poses a substantial challenge. Although success has been reported, the prognosis for an adult horse with a completely displaced radial fracture is unfavorable.[13]

Anatomy

The radius is the major weight-supporting bone of the antebrachium. Proximally, it articulates with the humerus and the ulna and forms the cubital joint. Distally, along with the proximal row of carpal bones, it forms the antebrachiocarpal joint. Proximally, the biceps brachii inserts on its cranial surface, and distally, the tendons of the digital extensor muscles pass through their respective tendon sheaths as they traverse the carpus en route to the digit. Medially, the radius lacks overlying muscle and is covered only by skin and subcutaneous fascia. The bone is strongly curved in the sagittal plane and has a distinct cranial bow. Biomechanically, this places the caudal cortex under



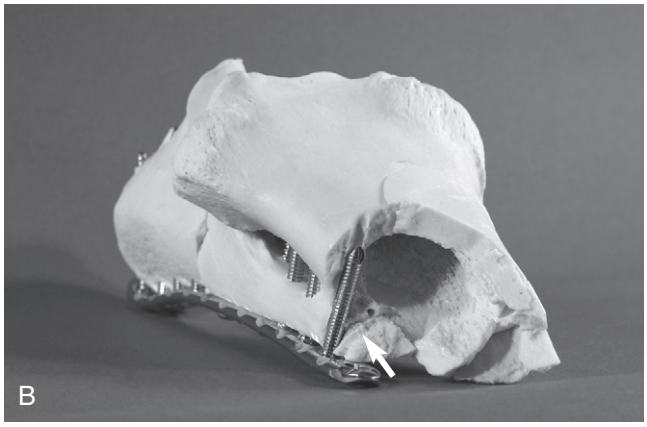


Figure 95-9. A, Immediate postoperative radiographic view of an ulnar fracture repaired with a 3.5-mm cortex screw applied in lag fashion and a 12-hole narrow LCP. Note immediately distal to the most distal locking head screw a drillhole crossing the entire radius is visible. **B,** Specimen of the fractured radius from the horse shown in **A** 7 days after surgery. The most distally located 5-mm locking head screw was positioned in the medial cortex of the radius. The drill hole prepared through the DCU portion of the same combihole (which was abandoned because the locking head screw) is visible *(arrow)*. The locking head screws must be inserted perpendicular to the plate, leading to the introduction into the medial cortex. The combination of the empty drill hole next to the locking head screw served as a stress riser, leading to a radius fracture. (Courtesy A. Fürst, Zurich, Switzerland.)

substantial compression. The cranial and craniolateral aspects of the radius are loaded in tension.

In foals, a proximal and a distal physis are present. These physes, along with their respective epiphyses, are subjected to compressive forces. Therefore, fractures involving these physes are classified according to the Salter-Harris scheme. Growth at these physes is responsible for the ultimate length of the bone and thereby affects limb length and conformation. Disproportionate growth at the distal radial physis is a common cause of carpus valgus limb deformity.

Pathogenesis

Most radial fractures result from external trauma. In foals, entrapment of the limb proximal to the carpus can result in proximal radial physeal fractures. However, direct blows to the antebrachium are the most common cause of fracture in both foals and adults. Often, the cause of the fracture is a kick from another horse.

Commonly, the fracture is comminuted, regardless of the patient's age. In adults, comminution almost always accompanies either a spiral or an oblique fracture of the diaphysis. The degree of comminution varies from single or multiple large butterfly fragments to multiple small fragments. Incomplete or complete nondisplaced fractures of the radius often occur in adult horses that have been kicked. These injuries may be witnessed as a solid blow to the distal medial aspect of the antebrachium. A skin wound may be evident.

Because the fracture is not displaced and an instability is not readily apparent, radiographs are occasionally omitted from the initial evaluation. Because the horse remains noticeably lame, the fracture is detected later, when it becomes unstable or radiographs are taken because the lameness fails to resolve. Therefore, radiographic examination is recommended in all cases in which there is evidence of a kick injury to the antebrachium.

Occasionally, if the initial radiographs are taken immediately after the incident, the fracture cannot be detected. In these cases stall confinement until a suspected fracture is confirmed or eliminated is recommended (see "Treatment," later). Follow-up radiographs taken 5 to 7 days after the injury allow identification of the fracture lines. The degree of fragmentation accompanying comminuted fractures in foals is less than that of comminuted fractures in adults. Fracture configurations in foals vary. Mid-diaphyseal transverse fractures result from a cranial blow to the antebrachium. A lateral blow results in an oblique fracture of the proximal metaphysis. Physeal fractures occur at both the proximal and the distal radial physes. Displacement with Salter-Harris type I and type II fractures of the proximal physis is accompanied by fracture of the ulna. If the radial metaphysis displaces craniad, injury to the radial nerve is possible. Occasionally, Salter-Harris type III fractures are encountered in the proximal epiphysis.

Diaphyseal fractures of the radius are at high risk of becoming open secondary to skin penetration by the sharp fracture fragments on the medial aspect of the antebrachium. Abduction of the distal limb displaces the fracture into the relatively sparse covering of soft tissues medially and predisposes to skin penetration. In addition, there is often substantial injury to the musculature covering the dorsal and lateral aspects of the antebrachium, accompanied by severe soft tissue swelling and hematoma. The degree of soft tissue injury accompanying radial fractures makes them highly susceptible to infection at the fracture site.

Diagnosis

Complete displaced radial fractures are easily identified on physical examination. Non–weight-bearing lameness accompanied by instability and crepitation in the antebrachial region is pathognomonic for these injuries. The patient usually holds the limb with the carpus and MCP joint flexed and drags the toe. Attempts at weight bearing result in valgus angulation at the fracture. Soft tissue swelling is usually evident. Wounds

or secondary penetration of the soft tissues at the medial aspect are often present. Horses with incomplete fractures or with nondisplaced complete fractures do not demonstrate instability or crepitation. However, they are usually lame, and in many cases a wound is evident at the site of traumatic impact.

Radiographic evaluation confirms the presence of a fracture. Multiple views, including oblique projections, are advisable to delineate the severity of the fracture and to assist in determining a course of action and rendering a prognosis. It is important to delineate the extent and location of fragmentation in comminuted, completely displaced fractures. Nondisplaced fracture lines usually emanate proximad and distad from the displaced fracture. Occasionally, these are evident only after the fracture is surgically exposed, but complete radiographic evaluation before attempting internal fixation determines the feasibility of repair and the best method for managing the fracture.

Fractures with multiple small fragments that are likely to preclude anatomic reconstruction should be identified. This is especially important when these fragments are located along the caudal cortex, which must be reconstructed for repair to be successful.

Treatment

First Aid

As with ulnar fractures, wounds associated with a displaced fracture of the radius require immediate attention. There is a high probability that wounds at the medial aspect of the antebrachium communicate directly with the fracture. Initial management should include appropriate hair removal, cleansing, and topical and systemic antimicrobial therapy.

Immediate immobilization of a displaced fracture is of paramount importance. With closed fractures, appropriate external coaptation can prevent fragments from displacing and the fracture from opening. Sedation is necessary for applying a bandage and splint with an unstable radius fracture. When the patient is controlled, and after appropriate wound therapy in open fractures, a Robert Jones bandage is applied extending from the hoof to as far proximal on the antebrachium as possible.

The bandage should be applied in layers to increase rigidity. A caudal splint is applied that extends from the ground to the level of the olecranon process. A second lateral splint extends from the ground to the level of the mid-scapula. The portion of the splint extending proximal to the bandage should contact the brachium when the limb is directly beneath the body (Figure 95-10). This provides counterpressure to prevent the limb from abducting with weight bearing. Nonelastic adhesive tape or fiberglass casting tape is used to affix the splints to the bandage. More information on bandaging and first aid for fracture patients can be found in Chapters 17 and 73.

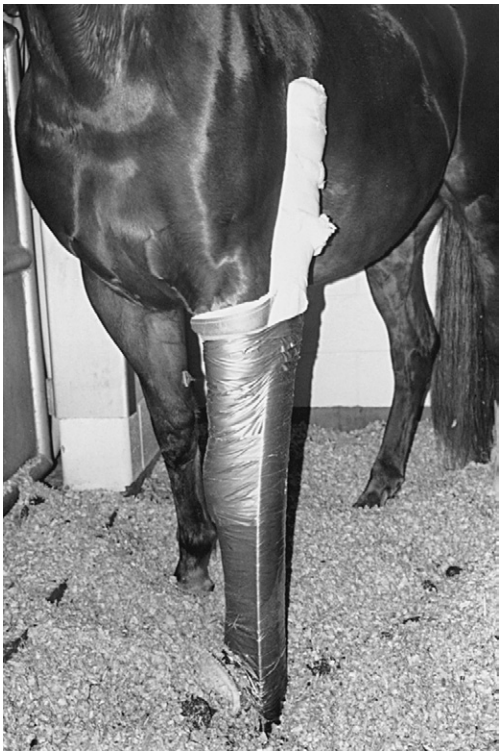


Figure 95-10. Photograph of appropriate external coaptation of a radial fracture in an adult horse before surgical repair.

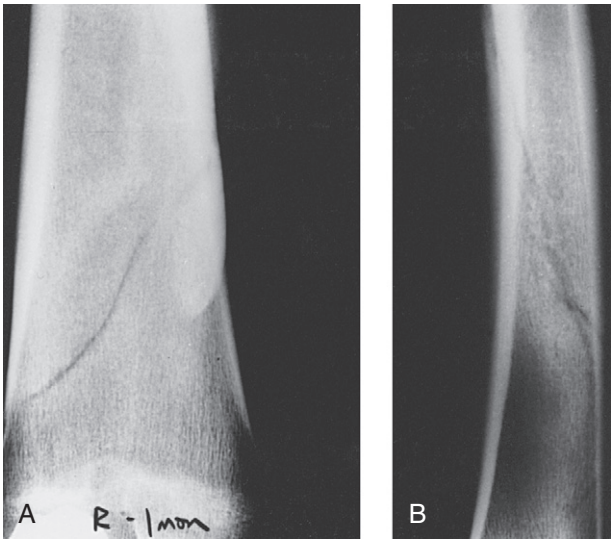


Figure 95-11. Craniocaudal **(A)** and lateromedial **(B)** radiographic views of a nondisplaced fracture of the radius in an adult horse secondary to a kick injury.

Nonsurgical Management

Incomplete and nondisplaced complete fractures are candidates for conservative therapy (Figure 95-11).[14-16] In one report, incomplete radial fractures became complete, displaced fractures in only 3 of 10 horses managed conservatively.[16] Predicting success based on the radiographic appearance of the fracture was not possible. An important aspect of therapy was preventing recumbency to reduce the likelihood of displacement secondary to forces experienced during standing up (see Figure 76-1).

Phenylbutazone should be used judiciously to reduce inflammation and encourage limited weight bearing on the fractured limb. However, care should be taken to avoid providing substantial pain relief, which could result in overuse of the fractured limb.

The horse is confined until there is evidence of advanced fracture healing. This usually requires a minimum of 3 to 4

months. During the last 30 days of confinement, a program of gradually increasing hand-walking is recommended. Access to free paddock exercise is allowed thereafter. An additional 30 days of paddock exercise is recommended before the horse is gradually returned to its intended function.

Surgical Management

ADULT HORSES

Open reduction and internal fixation of displaced radial fractures in adult horses is a monumental task that provides only a limited chance of survival.[13] In many cases, the fracture configuration and degree of soft tissue trauma preclude any reasonable likelihood of success. Even with fractures amenable to fixation, the biomechanical forces acting on the bone–implant construct place the fixation at risk for failure. Even the strongest, most stable construct has a high potential either to fail catastrophically during recovery or to fatigue and fail before fracture union.

Fixation with two plates is necessary; one plate is positioned cranially and the second plate is applied to either the medial or the lateral cortex. One of the plates should be a dynamic condylar plate, with the condylar screw placed in the smaller fracture segment. The second plate should be a broad 4.5-mm LC-DCP, DCP, or ideally a 5.5-mm LCP. As many 5.5-mm cortex and 5-mm locking head screws as possible are implanted, and every opportunity to provide compression across fracture lines is used (Figure 95-12). The entire length of the radius is spanned by the plates, and the plates should be offset to avoid ending at the same level in the transverse plane. The second plate is placed lateral distally and twisted proximad to end on the craniolateral aspect of the proximal metaphysis, lateral to the insertion of the biceps brachii tendon on the radial tuberosity. The use of the human, slightly curved, 4.5-mm LCP allows application over the entire lateral side of the radius without twisting the proximal aspect of the plate (see Figure 76-39). Plate luting with antimicrobial-impregnated PMMA is recommended if LC-DCPs and DCPs are used. Cancellous bone grafts should be incorporated into the fixation to promote healing. Information on the implantation techniques of plates can be found in Chapter 76.

FOALS

Diaphyseal fractures in foals, other than simple transverse middiaphyseal fractures, are double plated as well. Implant selection depends upon the size of the foal, the fracture configuration, and the location. A broad LC-DCP, DCP, or LCP is usually applied to the cranial cortex, and in most cases, 5.5-mm cortex screws and 5-mm locking head screws are used throughout its length. The second plate may be either a broad or narrow LC-DCP, DCP, or LCP, fixed with a majority of 4.5-mm cortex screws. However, nearest the fracture and at the ends of the plate, 5.5-mm cortex or 5-mm locking screws should be inserted. The lateral plate is applied as described for the adult horse earlier (Figure 95-13). The use of the slightly curved human LCP can be used as an alternative in the foal as in the adult.

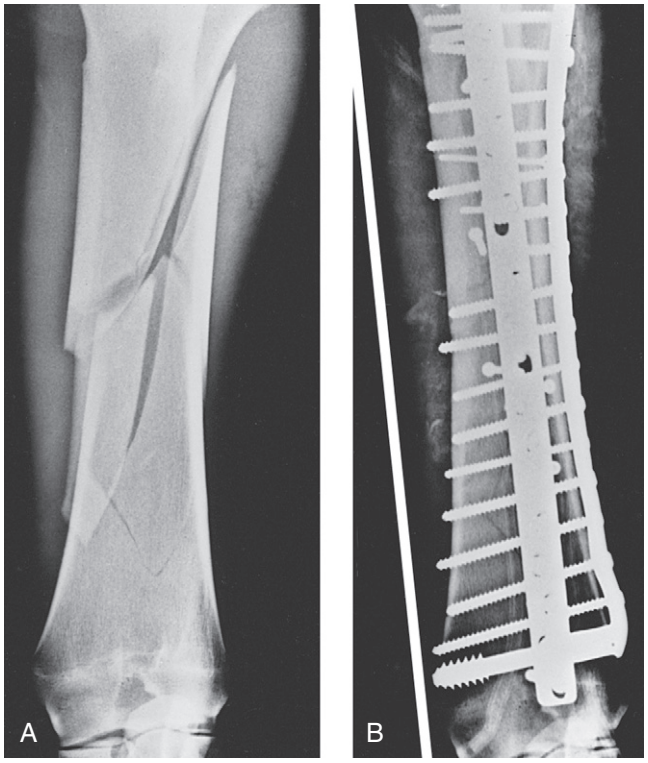


Figure 95-12. Craniocaudal radiographic view of a comminuted radial fracture in an adult horse before internal fixation **(A)** and after application of a dynamic condylar screw plate applied to the lateral cortex and a broad dynamic compression plate applied to the cranial cortex **(B).** Most screws are 5.5-mm cortex screws. Note that the plates span the length of the radius.

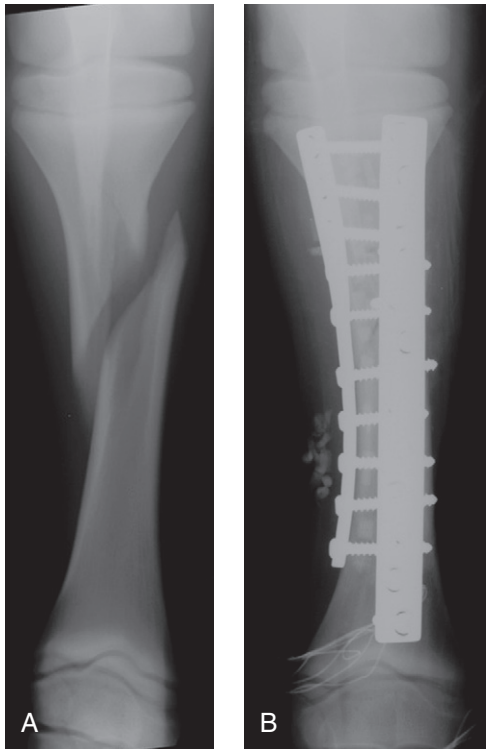


Figure 95-13. Craniocaudal radiographic view of a proximal metaphyseal radial fracture in a foal before internal fixation **(A)** and after application of a broad dynamic compression plate (DCP) to the cranial cortex and a narrow DCP applied to the lateral cortex distally and spiraled cranially at its proximal aspect **(B).**

Multifragment (comminuted) fractures

In foals with multifragment fractures, the repair is as described for adults, with the exception of implant selection. A second broad LC-DCP, DCP, or LCP is used in place of the dynamic condylar screw plate. I prefer to place the second plate laterally, unless the configuration of the fracture dictates medial placement. Lateral placement, although technically more demanding, avoids positioning the implant subcutaneously and, particularly in fractures that are open medially, avoids direct communication with the wound. In most cases, both implants can be placed using a lateral approach between the extensor carpi radialis and common digital extensor muscles. If the lateral plate is extended to the distal end of the radius, placement of the distal screws may require separate stab incisions lateral to the common digital extensor tendon, especially if an LCP is used.

Mid-diaphyseal fractures

Mid-diaphyseal transverse fractures in small foals can be repaired with a single broad LC-DCP, DCP, or LCP applied to the cranial cortex, provided the caudal cortex can be anatomically reconstructed. The strong cranial-to-caudal bending moment of the radius makes this a stable fixation in foals weighing less than 250 kg. With single-plate fixation, it is advisable to use 5.5-mm cortex screws throughout the entire length of the plate. An LCP is preferred , and 5-mm locking head screws should be placed on either side of the fracture and at the ends of the plates. Compression of the LCP onto the bone surface must be accomplished with cortex screws or the push-pull device (see Chapter 76). Although precise bone-plate contact is less important when using an LCP, close apposition to the bone surface improves the plate's mechanical performance, which is very important in management of this fracture.

Proximal physeal fractures

Fractures involving the proximal physis are less common than diaphyseal or metaphyseal fractures. They are candidates for internal fixation, with special considerations.

Type III fractures are rare, and conservative therapy is successful when they are nondisplaced. However, anatomic reconstruction of the articular surface and compression across the fracture is the treatment of choice for displaced fractures. This is accomplished with screw fixation using lag technique alone or in combination with tension band wire fixation.

Salter-Harris types I and II fractures of the proximal physis are accompanied by ulnar fractures (Figure 95-14, *A*). Plate fixation of the ulnar fracture, with screws engaging both cortices of the radius where possible, combined with a laterally applied narrow plate with the most proximal screw in the radial epiphysis, is recommended (see Figure 95-14, *B*). In small foals, transphyseal screw and wire fixation in place of the plate on the lateral aspect of the radius can be used.

As mentioned before, transfixation of the ulna to the radius can result in elbow subluxation, but growth at the radial physis is minimal when the germinal layers of the growth plate are injured and the implants bridge the physis, restraining growth. The apparent disparity in bone length between limbs does not appear to have an adverse effect on gait.

If the metaphyseal portion of the fracture displaces craniad (Figure 95-15), the radial nerve can be traumatized, resulting in low radial nerve paresis. Difficulty in active extension of the digit is the result. Most foals learn to place the foot on the ground appropriately, and the paresis usually resolves, but in the immediate postoperative period a splint may be needed to keep the digit in extension.

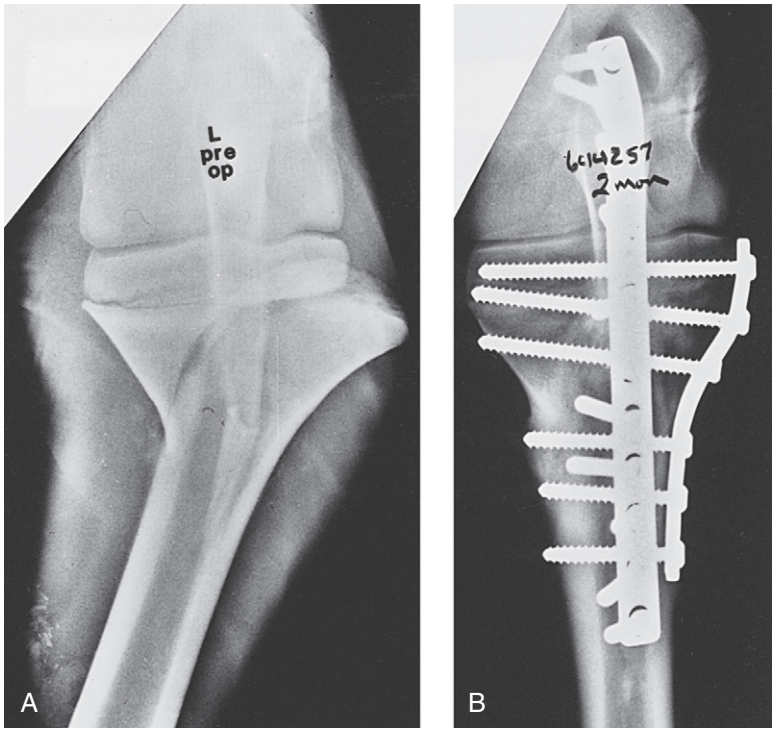


Figure 95-14. Craniocaudal radiographic view of a Salter-Harris type II fracture of the proximal radius prior to repair **(A)** and after the application of a narrow dynamic compression plate (DCP) to the lateral aspect of the proximal radius and a narrow DCP applied to the ulna with screws transfixing the ulna to the radius **(B).**

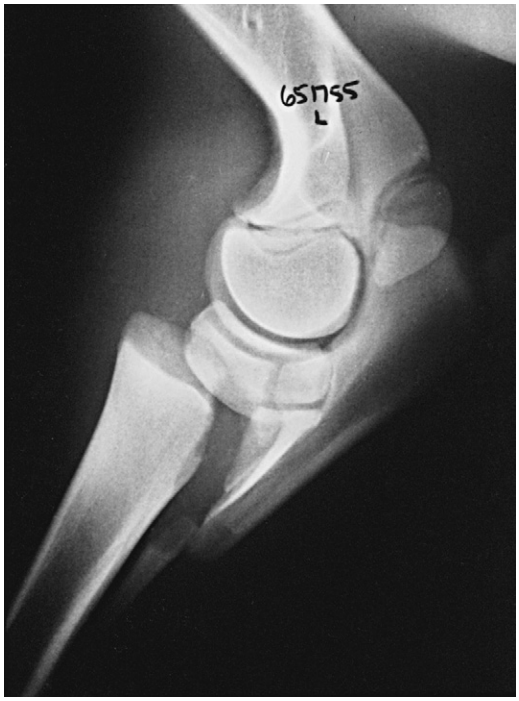


Figure 95-15. Lateromedial radiographic view of a Salter-Harris type II fracture of the proximal radius accompanied by an ulnar fracture with cranial displacement of the distal fragment, resulting in temporary radial nerve paresis.

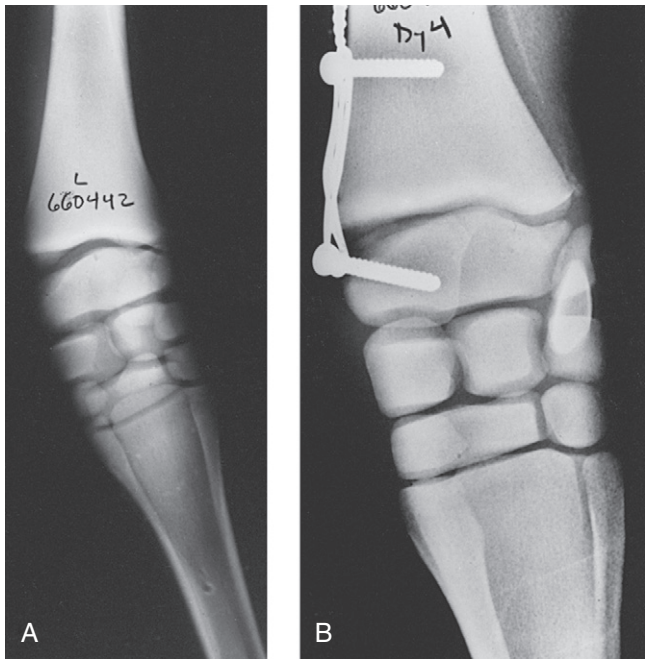


Figure 95-16. Craniocaudal radiographic view of a foal with a Salter-Harris type I fracture of the distal radius, before repair **(A)** and after repair with a transphyseal bridge using screws and wires **(B).**

Distal physeal fractures

Fractures of the distal physis are less common than fractures of the proximal physis. Salter-Harris type I physeal fractures result in displacement at the medial aspect of the physis (Figure 95-16, *A*). The fractures occur in neonates and are treated by transphyseal bridging across the medial aspect of the physis. A T-plate or screws and wires can be used (see Figure 95-16, *B*). The implants are removed when the fractures are healed at approximately 4 to 6 weeks.

Prognosis

Adult horses with displaced radial fractures have an unfavorable prognosis. In rare instances, successful internal fixation can be achieved.[13,15,17,18] In one report, internal fixation was attempted in six adult horses, none of which survived.[19] In another case series, nine of 15 horses that were presented for radial fracture had attempted internal fixation, and only two were discharged from the hospital.[13] However, with careful case selection and improved methods that increase the strength and stability of the bone–implant construct, the success rate can be improved. The substantial expense associated with fixation, combined with the high risk of failure, often dissuades owners from pursuing repair.

However, displaced radial fractures in foals, particularly if there is no comminution and when the fracture is closed, have a favorable prognosis. Physeal fractures and transverse fractures of the mid-diaphysis have an excellent prognosis.[19] In six of seven foals with mid-diaphyseal transverse fractures, and in two other foals, one with Salter-Harris type I and another with a type II proximal physeal fracture, the fractures healed. Similarly, fixation of proximal oblique fractures in 3 of 4 foals had an excellent outcome.

Complications

Complications following repair of radial fractures are the same as for any equine long bone fracture repair. Infection, instability, failure of fixation, and support limb complications are the most common. The propensity for radial fractures to be open medially substantially increases the risk of infection. Even in closed fractures, the degree of soft tissue trauma and the lack of muscle covering of the medial aspect of the radius increase the potential for infection. The technical difficulty of the repair often prolongs surgery time. The quantity of implants used for repair significantly contributes to postoperative infection.

Fixation failure is the most common fatal complication. Catastrophic failure is a major risk during recovery from anesthesia, particularly in adult horses. Long surgery time and the condition of the patient at the time of anesthesia play major roles. Attention to the patient's physiologic status before and during anesthesia, along with a controlled recovery, can reduce the incidence of failure (see Chapter 21). The technical difficulty of the fixation dictates that an experienced surgical team be assembled. Every effort is made to minimize surgery time.

Fatigue failure of the implants is not uncommon. This is especially a problem in adult horses, where the high loads experienced by the bone–implant construct cause micromotion and cycle the implants. The greater the instability, the greater the probability of fatigue failure. Bone-to-bone contact is imperative at the caudal cortex. If that is not achieved, cyclic loading fatigue failure is inevitable (Figure 95-17). Methods to reduce the likelihood of fatigue failure include using the strongest implants available and plate luting except when LCPs are used.

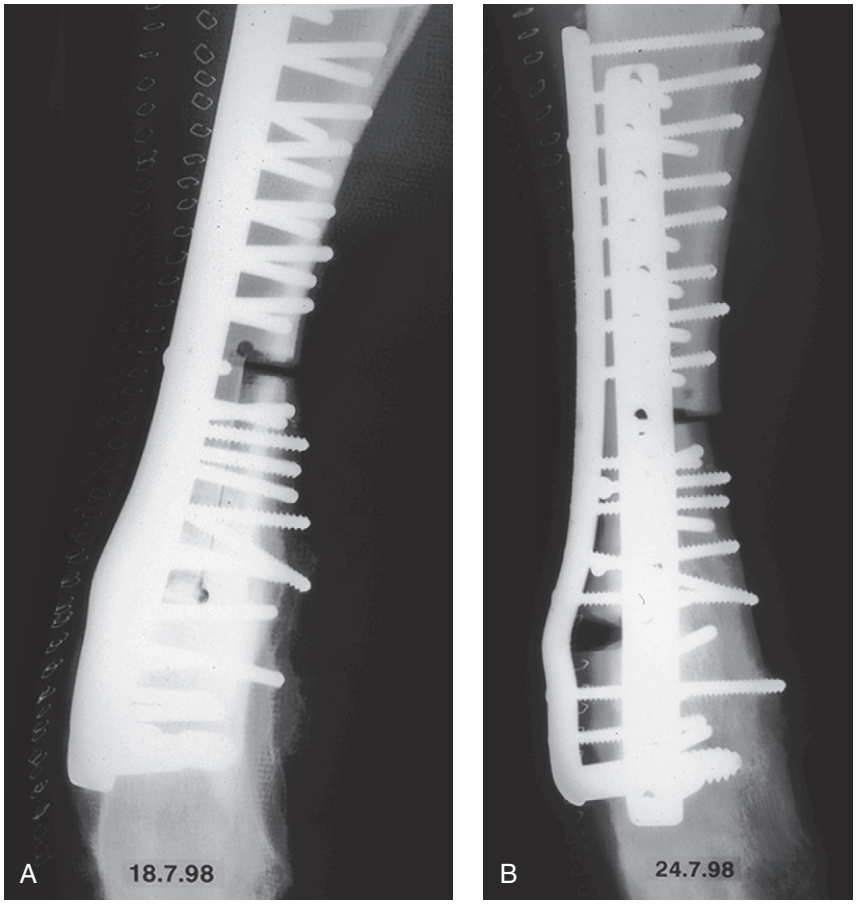


Figure 95-17. A, Lateromedial radiographic view of corrective step osteotomy performed in the frontal plane of an adult Warmblood suffering from a severe varus deformity of the radius following abnormal healing of a radial fracture. Note the gap at the caudal osteotomy site. **B,** A 1-week follow-up radiograph showing narrowing of the gap at the caudal cortex and widening of the gap cranially. Screw loosening can be seen at the level of the proximal osteotomy in the lateral plate. Two days later, the dynamic condylar screw (DCS) plate broke at the level of the osteotomy, resulting in humane destruction of the animal. (Courtesy J. Auer, University of Zurich.)

Anatomic reconstruction of the fracture, especially at the caudal cortex, is an absolute necessity.

To prevent support limb complications, an early return to some weight bearing on the fractured limb is imperative and best accomplished by restoring stability to the fracture site and preventing infection. In foals, the primary concern is for varus limb deformity and fatigue of the suspensory apparatus, resulting in MCP joint hyperextension. To reduce these complications, a foot plate with an extended heel and lateral bearing surface is applied. Support bandages are not advocated in foals because they tend to promote MCP joint hyperextension rather than prevent it. In adult horses, support limb laminitis is a major risk. Appropriate foot support, such as a heart bar shoe, may aid in reducing the frequency of support limb laminitis. Support wraps and judicious use of phenylbutazone are recommended in adults during the postoperative period.

OSTEOCHONDROMA

Osteochondromas originate from the dysplastic growth of aberrant growth cartilage. They are located adjacent to a physis and occur most commonly in adult horses at the caudal aspect of the distal radius.[20-22] Solitary osteochondromas have been reported in two foals: in one, the calcaneus was affected, and in the other, the distal palmar aspect of the middle phalanx was affected.[23,24]

Osteochondromas are exostoses composed of trabecular bone contiguous with cancellous bone of the adjacent medullary cavity. There is a characteristic cartilage cap composed of hyaline cartilage undergoing endochondral ossification. The histologic characteristics are identical to those of multiple cartilaginous exostoses of horses. However, the genesis of the lesion in the latter condition is hereditary. To date, a hereditary basis for solitary osteochondroma has not been identified.[25]

Pathogenesis

Causes of solitary osteochondroma include displacement of a growth cartilage fragment that rotates and grows transverse to the longitudinal axis of the bone, an aberrant nest of subperiosteal growth cartilage, and herniation of growth cartilage through a perichondral defect. With continued growth, the expansile nature of the lesion impinges on adjacent structures and results in lameness. Osteochondromas of the caudal distal radial physeal region are located in the carpal canal and cause tenosynovitis. They also impinge on the deep digital flexor tendon as it passes through the area.[20-22] Bony projections that are not true osteochondromas, referred to as *physeal remnant spikes,* have been noted to cause the same clinical syndrome (see Chapter 94).[25,26]

Diagnosis

Horses with distal radial osteochondromas or physeal spikes are presented with effusion in the carpal canal. Lameness is mild

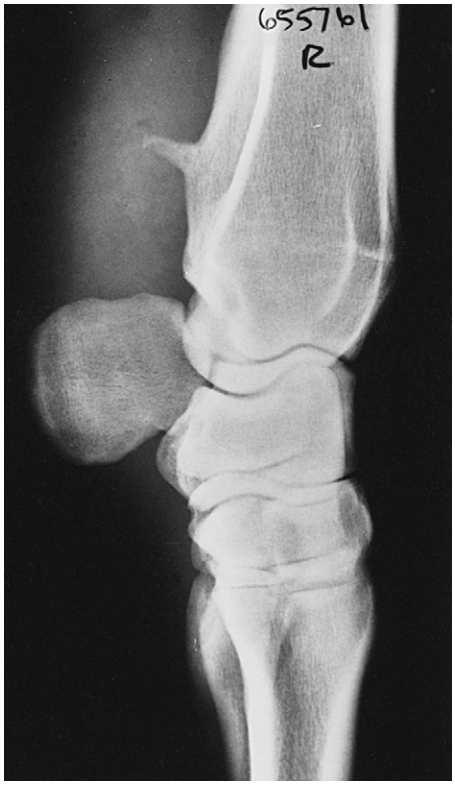


Figure 95-18. Lateromedial radiographic view demonstrating a solitary osteochondroma of the caudal aspect of the radius proximal to the physeal scar.

and intermittent. Pain is evident on direct palpation and is induced by carpal flexion. In some cases, the bony projection can be identified by deep palpation. Radiography reveals the characteristic bone growth on the palmar aspect of the radius adjacent to the physis (Figure 95-18). There is a wide variation in the size and degree of ossification of the lesion. Ultrasonography can aid in determining the presence of deep digital flexor tendinitis. However, the absence of ultrasonographic changes does not preclude the presence of superficial injury to the tendon due to impingement by the osteochondroma or physeal spike.

Treatment

Instillation of corticosteroids into the tendon sheath temporarily resolves tenosynovitis in most instances. However, clinical signs usually recur. Surgical excision of the bony projection is curative and is the treatment of choice. Although excision via an open approach to the lateral aspect of the carpal canal was originally reported, removal under arthroscopic guidance is currently advocated.[18,21,22]

With the affected limb positioned uppermost, the arthroscope is placed into the proximal aspect of the carpal canal through a stab incision located 3.5 cm proximal to the level of the physeal scar between the lateral digital extensor and ulnaris lateralis muscles.[22] Although a medial approach has been described, the surgeon is less likely to injure the median artery and vein from a lateral approach. Furthermore, the lateral approach makes it technically easier to manipulate the scope and instruments without interference from the opposite limb.

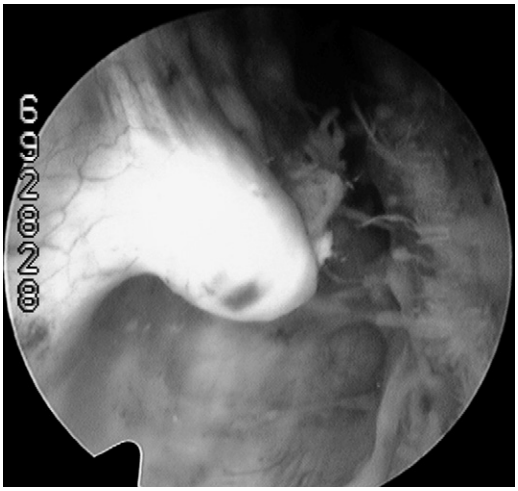


Figure 95-19. Endoscopic view of caudal radial osteochondroma before its removal. Note the injury to the deep digital flexor tendon on the right side of the photograph caused by the osteoochroma.

When the arthroscope is in the carpal canal, the sheath is distended and the osteochondroma or physeal spike is readily identified (Figure 95-19). The bony projection's attachment to the underlying bone is severed with a small osteotome positioned through a separate stab incision distal to the scope portal, and the bony projection is removed. Release of the carpal flexor retinaculum can also be accomplished via endoscopic guidance if chronic distention of the carpal canal is diagnosed as a complicating problem. The bone bed is curetted and the sheath is lavaged before closure of the skin incisions. Postoperative care is routine.

For additional information on this condition, please review "Distal Radial Exostosis" in Chapter 94.*

Prognosis

Horses are reported by their owners to be sound for their intended purpose following removal of the osteochondroma or physeal spike.[20-22,25,26] Distention of the carpal canal resolves with time. With chronic carpal canal distention, resolution of the effusion following osteochondroma removal can require additional medical therapy, including repeat local corticosteroid injection.

SUBCHONDRAL CYSTIC LESIONS

Subchondral cystic lesions have been reported in the proximal epiphysis of the radius. Although they are infrequent, they have been noted as a cause of lameness.[27,28] Like subchondral cysts in other locations, the definitive pathogenesis remains elusive, with developmental and traumatic causes proposed. Affected horses vary in age but are most commonly young individuals that are in athletic training or competition. The degree of lameness is variable, but it can be acute and relatively severe. With rest, lameness usually dissipates, only to return after activity is resumed.

*An editorial decision was made to discuss this condition in both chapters because each describes slightly different aspects aside from the common parts.

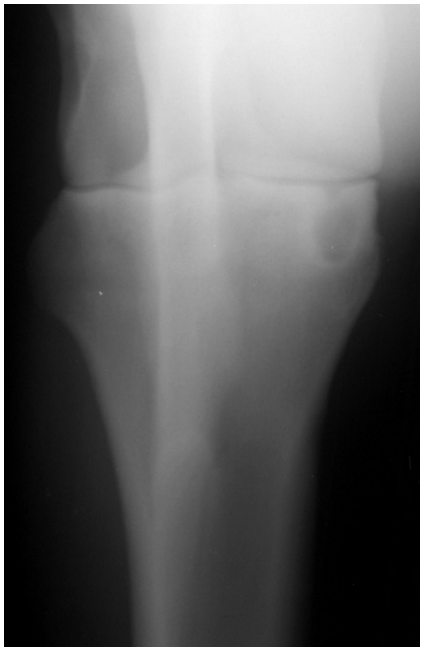


Figure 95-20. Craniocaudal radiographic view demonstrating a subchondral cystic lesion in the medial aspect of the proximal epiphysis of the radius.

Clinical findings that aid in localizing the lameness to the cubital joint are lacking. However, intra-articular anesthesia of the affected joint significiantly reduces or eliminates the lameness. Radiographic examination delineates the lesion, most typically in the medial aspect of the epiphysis (Figure 95-20). Options for treatment include intra-articular therapy and rest.[27,28] If lameness persists, an extra-articular surgical approach can be performed, allowing either débridement, intralesional corticosteroid injection of the cyst, or filling of the defect with an osteoinductive material. A potential complication of débridement via the extra-articular approach is fracture through the cyst.[27] Additional information on subchondral cystic lesion is found in Chapter 89. At the time of this writing, too few cases have been treated in a consistent fashion to reliably predict the outcome of management.

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CHAPTER 96

Shoulder

Lisa A. Fortier

Orthopedic abnormalities of the scapulohumeral (shoulder) joint are rare. Because shoulder pathologies are uncommon and relatively difficult to diagnose, treatment can be delayed, leading to an increase in secondary osteoarthritis. Further, there are very few large studies documenting treatment and outcome following specific injuries or treatments to guide the surgeon.

DIAGNOSIS OF SHOULDER LAMENESS

Localization of lameness to the shoulder region can be straightforward in cases when there is associated swelling or pain on palpation, but in many patients intra-articular or bursal anesthesia is necessary to confirm clinical examination findings. Horses with fractures of the shoulder region or luxation of the scapulohumeral joint are acutely, and initially, severely lame and have localized swelling. When the scapulohumeral joint is less severely affected, joint effusion cannot be palpated because of the overlying musculature, but a painful response can often be elicited following deep palpation of the notch between the cranial and caudal prominences of the greater tubercle of the humerus. Less-specific signs of shoulder lameness include shoulder muscle atrophy, pain on extension or flexion of the shoulder joint, a narrow ipsilateral hoof with a long heel, and, typical of forelimb lameness, signs of a shortened anterior phase of the stride. Nuclear scintigraphy can help localize the cause of lameness to the shoulder joint.

When the lameness is localized to the shoulder region, radiographs should be obtained. If bicipital bursitis or biceps tendinitis is suspected, an ultrasonographic examination should be performed.[1] Although not currently available for imaging equine shoulders, magnetic resonance imaging (MRI) is considered the method of choice for evaluating shoulder pain in people because it provides detailed information on all bone and soft-tissue components of the shoulder.[2]

Radiographs of the shoulder joint can usually be obtained in the standing horse, although higher-quality images are achieved when the horse is under general anesthesia because of the absence of motion artifact. The horse's leg is extended forward to center the shoulder joint over the trachea for a standard mediolateral or a more specific cranioproximal-craniodistal oblique view of the proximal portion of the humerus projection to diagnose long oblique fractures of the greater tubercle.[3] Normal survey radiographs do not exclude the shoulder as a cause of lameness, particularly in cases of osteochondrosis (OC) or other pathologies of the humeral head or glenoid cavity.[4-6] In cases where survey radiographs are normal and OC is suspected, positive-contrast radiography using 7 to 10 mL of a sterile water-soluble iodinated contrast medium should be performed.[7] Double-contrast radiography more clearly delineates the glenoid notch than positive-contrast radiography. To obtain a double-contrast image, 30 mL of air is injected following positive-contrast radiography.

INTRASYNOVIAL ANESTHESIA OR SYNOVIAL CENTESIS

When clinical examination findings do not definitively localize the lameness to the shoulder joint or bicipital bursa, diagnostic intrasynovial anesthesia can be useful. Local anesthetic (20 mL) is injected into the shoulder joint using an 18-gauge, 8.9-cm spinal needle inserted in the notch between the cranial and caudal prominences of the greater tubercle of the humerus. The needle is directed toward the elbow of the opposite limb, parallel to the ground, and is advanced until bone or cartilage is contacted. Extrasynovial deposition or leakage of local anesthetic solution out of the shoulder joint can block the suprascapular nerve, resulting in lateral subluxation of the scapulohumeral joint, thereby producing the clinical appearance of sweeney.[8] If the suprascapular nerve is inadvertently blocked, the horse should be placed in a box stall until the anesthetic wears off to prevent injury. In horses with intact cartilage that covers subchondral bone abnormalities, there might be minimal or no improvement in lameness following intra-articular anesthesia.

The bicipital bursa is entered approximately 4 cm proximal to the distal aspect of the deltoid tuberosity. An 18-gauge, 8.9-cm spinal needle is inserted between the biceps brachii muscle and the humerus and directed proximomedial to approximately 4 cm in depth, where 5 to 10 mL of local anesthetic is injected. Synovial fluid can usually be aspirated from both the shoulder joint and bicipital bursa to confirm intrasynovial needle placement and therefore deposition of local anesthetic or for synovial fluid analysis and culture in cases where infection is suspected.

DISORDERS OF THE SHOULDER REGION

Osteochondrosis

Osteochondrosis (OC) is reported in the shoulder joint less often than in other sites such as the tarsocrural or femoropatellar joint. Clinical signs usually appear in horses 4 to 12 months old, and the lesions can be bilateral. Clinical examination in combination with conventional or contrast radiography reveals that the majority of shoulder OC lesions in horses are of the osteochondritis dissecans (OCD) type (cartilage flaps). Typical radiographic signs include irregular areas of subchondral bone with radiolucent areas surrounded by sclerosis. Contrast radiography more clearly delineates the presence and extent of undermined cartilage flaps and aids in determining a more accurate preoperative prognosis than one based on plain radiographs alone (Figure 96-1). Subtle cartilage lesions might not be detected using current imaging modalities, and diagnostic arthroscopy should be used if clinical examination, intra-articular anesthesia, or nuclear scintigraphy implicates the shoulder joint as the source of lameness.[4]

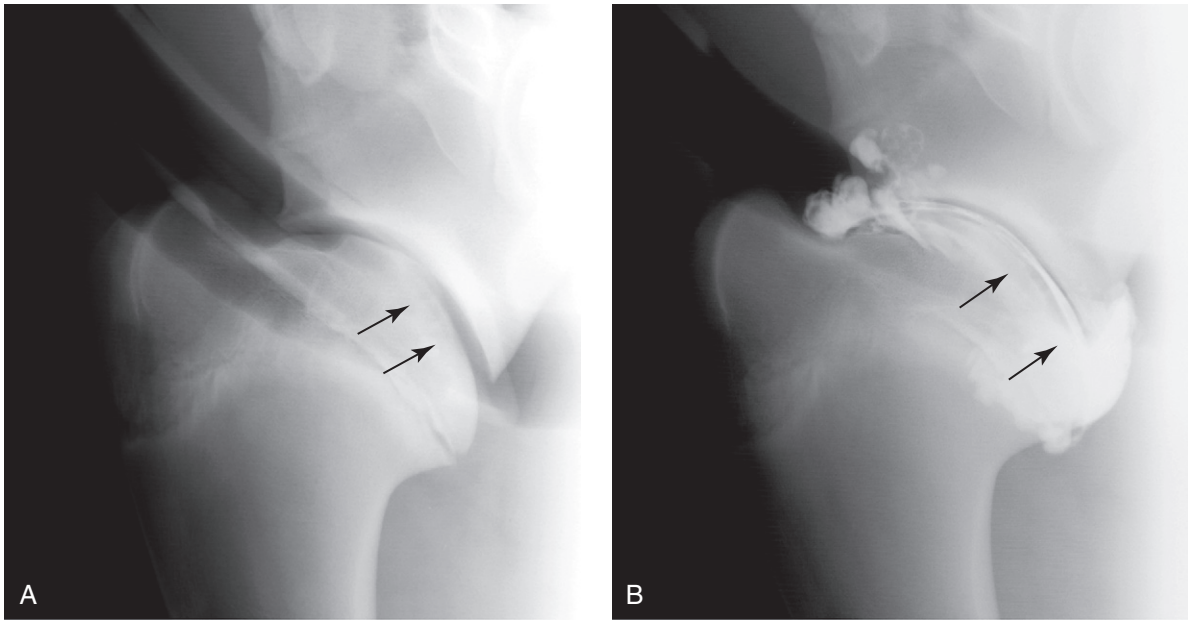

Figure 96-1. A, Mediolateral radiograph of the shoulder joint. A subchondral lucent region is present in the caudal humeral head *(arrows).* The humeral head in this region also appears flattened. **B,** A positive contrast arthrogram of the same shoulder joint depicted in **A.** Contrast material outlines the shoulder joint and further delineates the extent of subchondral bone lysis *(arrows).*

Secondary osteoarthritis (OA) develops rapidly in cases of shoulder OCD, and radiographs should be carefully scrutinized preoperatively for signs of OA, because the prognosis for future athletic function is poor in affected horses. Radiographic signs of shoulder OA include flattening of the humeral head and glenoid cavity, remodeling of the caudal rim of the glenoid, and subchondral bone sclerosis of the humeral head or glenoid cavity.[5]

Treatment

Arthroscopic surgery with débridement of loose cartilage flaps and curettage of abnormal underlying subchondral bone is the treatment of choice for shoulder OCD lesions. Repair of cartilage flaps using polydioxanone pins has been successful in select cases of OCD involving the femoropatellar and metacarpophalangeal joints and should be considered for cases of shoulder OCD.[9] Only cases with smooth, partially attached, unmineralized cartilage flaps should be considered for repair with polydioxanone pins.

Arthroscopy of the shoulder joint is technically more challenging than other joints. Lateral and craniolateral approaches have been reported.[10-12] The lateral approach is described here because it provides maximal visualization of the medial aspect of the humeral head and leaves the cranial portal (cranial to the infraspinatus tendon and proximal to the notch between the cranial and caudal prominences of the greater tubercle of the humerus) open for insertion of an egress cannula. The open cranial portal can also be used to introduce a large curved forceps, which can be engaged in the glenoid notch to distract the humeral head and gain access with the arthroscope to the medial side of the joint.

To perform shoulder arthroscopy, the horse is placed in lateral recumbency with the affected limb uppermost. The leg should be draped to allow adduction and traction during surgery to increase surgical exposure. The joint is distended with 60 mL lactated Ringer's solution using an 18-gauge, spinal needle inserted in the notch between the cranial and caudal prominences of the greater tubercle of the humerus (this site becomes the cranial portal). The arthroscope is inserted 1 to 2 cm caudal to the infraspinatus tendon and cranial to the teres minor muscles. A third site 2 to 4 cm caudal to the arthroscope portal can be used as an instrument portal. The optimal site for instrument portal entry is determined using an 18-gauge, spinal needle. If the lesions are extensive, or if they occur on the humeral head and glenoid cavity, more than one instrument portal might be required to complete the surgical débridement.

Cartilage flaps are removed and subchondral bone is débrided using several instruments including Ferris-Smith rongeurs, motorized resectors, periosteal elevators, and curettes (Figure 96-2). Lesions on the most medial aspect of the humeral head might be inaccessible. When débridement is complete, the joint is lavaged to remove cartilage and bone debris and the skin incisions are closed routinely.

Antimicrobials and phenylbutazone are administered perioperatively, and phenylbutazone is continued for the next 5 to 10 days. Horses are restricted to stall rest for 14 days, at which time hand-walking begins for 5 minutes per day. After 30 days of stall rest with hand-walking, horses are turned out for 4 to 6 months before exercise is resumed.

Prognosis

There are few reports on outcome of surgical or conservative treatment for shoulder OCD in horses, and all of them have limited case numbers so they should be interpreted with some caution. The most recent report indicates that the prognosis for return to soundness with or without surgery is poor with 25% (8/32) of horses returning to their intended use.[13] Of the 32 horses in the study, 18 were operated and 14 were treated con-

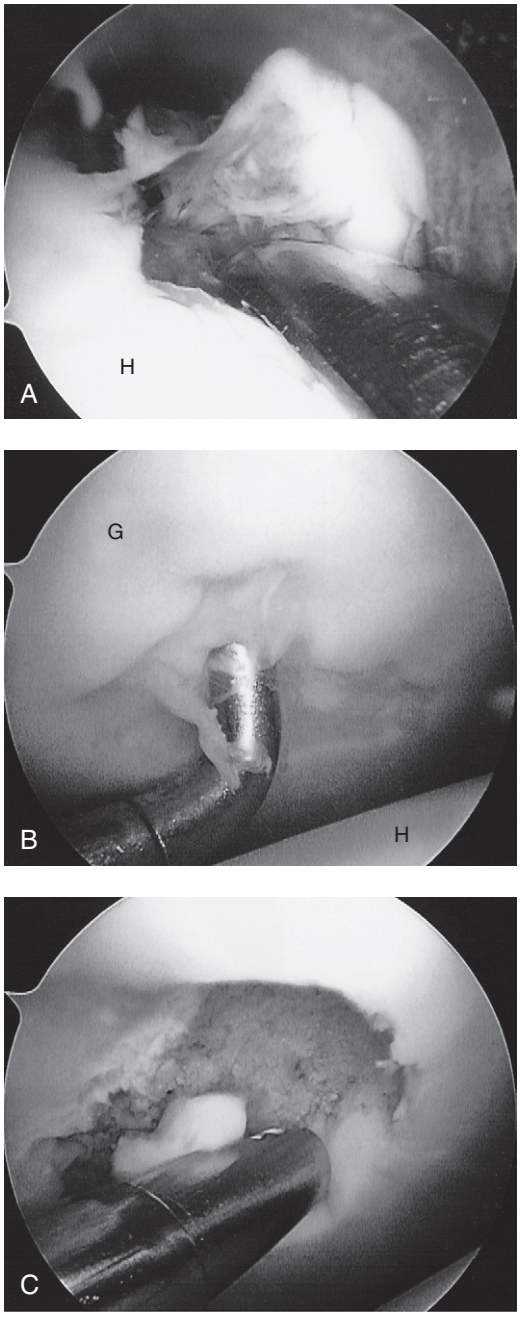

Figure 96-2. A, An arthroscopic image demonstrating elevation of an osteochondritis dissecans lesion of the humeral head *(H).* **B,** A probe is inserted into a cystic defect in the glenoid cavity *(G);* humeral head. **C,** Use of a motorized burr to débride a lesion in the glenoid cavity.

servatively. Only 15% of potential racehorses became sound for the intended use, and 67% of non-racehorses achieved their intended purpose. This study also suggested that there is no significant interaction between unilateral versus bilateral involvement or lesion severity. In two earlier studies enlisting a smaller number of horses intended for racing, the prognosis for return to athletic function following arthroscopic débridement was considered good, even for middle-aged horses with approximately 80% of horses achieving soundness.[4,14] The prognosis is considered to be poor when OA is detected on preoperative radiographs, although this has not been evaluated in a clinical series because most of these horses are not operated because of the poor prognosis. It should be noted that lesions identified during arthroscopy are often more extensive than suggested by radiography.[4,11,13]

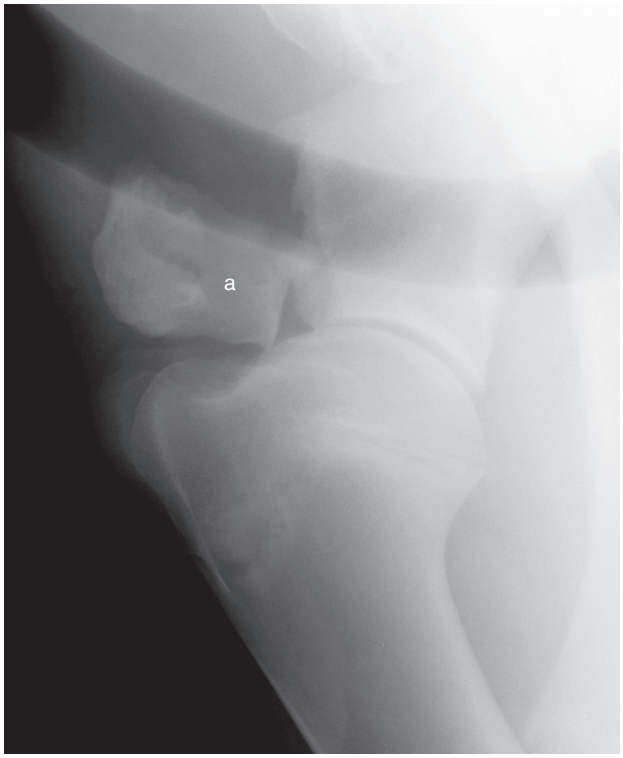

Figure 96-3. Mediolateral radiograph of the shoulder joint. A displaced fracture of the supraglenoid tubercle *(a)* is seen. The fracture fragment is displaced craniad and ventrad.

Fractures

Supraglenoid Tubercle

Supraglenoid tubercle fractures are usually simple, intra-articular epiphyseal fractures. These fractures occur most commonly in horses younger than 2 years of age and are either the result of direct trauma or are avulsion fractures caused by tension from the biceps tendon, which originates on the supraglenoid tubercle. Horses with supraglenoid tubercle fractures are able to bear weight, but they are variably lame and reluctant to fully extend the affected limb.

Careful visual inspection and palpation of the cranial aspect of the shoulder suggests a supraglenoid tubercle fracture. In chronic cases, shoulder muscle atrophy is present. Radiography of the shoulder is diagnostic. On a mediolateral radiographic projection, the fracture fragment is typically displaced in a cranioventral direction as a result of tension from the biceps brachii and coracobrachialis muscles, which originate on the supraglenoid tubercle (Figure 96-3). More recently, a cranioproximal-craniodistal oblique view of the proximal portion of the humerus has been suggested as a more reliable radiographic projection to diagnose long oblique fractures of the greater tubercle and should be included in a thorough radiographic series.[3] The radiographs should be carefully evaluated for signs of OA (described previously), which will decrease the prognosis for athletic function.

TREATMENT

Several treatment options are available, and the therapy chosen depends on the duration of the fracture and the intended use of the horse. Conservative management for all but the smallest fractures typically results in residual lameness and OA of the shoulder joint. Surgical options include repair of the fracture or removal of the fracture fragment. The goal of surgical repair should focus on restoring articular congruity of the glenoid cavity to prevent the development of OA. Therefore, if the fracture is chronic (longer than 1 week), enough bone remodeling will have occurred to preclude accurate reduction of the fracture, and the fragment should be removed. In acute fractures with involvement of a substantial (one third or greater) portion of the glenoid cavity, internal fixation should be considered to reconstruct the glenoid articular surface and provide maximal opportunity for future athletic performance. Acute fracture fragments that involve only a small portion of the glenoid cavity can be removed with no apparent impact on future athletic performance.

Fragment removal

The horse is placed in lateral recumbency with the affected limb uppermost. A 20-cm (8-inch) skin incision is made, beginning at the distal extent of the scapular spine, centering over the cranial aspect of the point of the shoulder, and extending distad over the deltoid tuberosity. The brachiocephalicus and supraspinatus muscles are separated and retracted; the brachiocephalicus muscle is retracted craniad and ventrad, the supraspinatus muscle is incised in the direction of its fibers directly over the tubercle, and the muscle is split and separated with self-retaining retractors to expose the fracture.[15]

At this point, repeated palpation will guide the surgeon to the shoulder joint and fracture fragment. Care should be taken to identify and preserve the suprascapular nerve, artery, and vein as they course across the neck of the scapula. The articular surface of the humeral head and glenoid cavity should be inspected for signs of cartilage damage, which, if present, will diminish the prognosis for athletic function. The fracture fragment is grasped with a large bone-holding forceps and excised using a combination of blunt and sharp dissection to detach the muscular and tendinous attachments. The dissection can be challenging, particularly on the medial aspect of the fragment because of lack of visualization.

Following removal of the fragment, the area is lavaged and closed in several layers to diminish dead space. If substantial dead space remains, a closed suction device should be used to prevent development of a postoperative seroma. Assisted recovery from anesthesia is recommended. Antimicrobials and phenylbutazone are administered perioperatively and continued until the there are no clinical signs of seroma formation.

Horses are restricted to stall rest for 60 days to allow time for the dead space to fill with fibrous tissue and the biceps to reattach. A carefully controlled rehabilitation program is important for these horses to regain strength and coordination in their shoulder joints. Percutaneous stimulation of the shoulder muscles during rehabilitation to minimize muscle atrophy should be considered. In humans, percutaneous muscle stimulation has been shown to minimize muscle atrophy after stroke or spinal cord injury. Typically, physical therapy begins with range-of-motion exercises and hand-walking for 5 minutes per day, followed by walking over ground poles and gradual increases in duration of exercise each day. Horses are not usually ready to return to training or to be turned out into a paddock for 6 to 12 months after surgery.

Fracture fixation

A variety of techniques have been described for internal fixation of supraglenoid tubercle fractures.[3,16-19] Tension from the biceps tendon is the primary force that needs to be neutralized when performing internal fixation. Partial or full transection of the biceps tendon to prevent tension on the fracture repair and facilitate internal fixation has been described, but it is not routinely recommended.[18]

The fracture site is approached as described previously for fragment removal. The fractured bone ends are débrided and the fracture is reduced using large bone-reduction forceps. The fracture is repaired using two or three 5.5-mm cortex bone screws placed in lag fashion. Care is taken to implant the screws in a somewhat diverging pattern to increase resistance against the axial tension on the implants. Additional support is provided by applying a figure-of-eight tension band between the supraglenoid tubercle and the cranial edge of the scapula using large-diameter (1.5-mm) wire (Figure 96-4) or a 1-mm cable.[3,16,17] Wound closure and immediate postoperative care are similar to that described for fracture fragment removal.

Following internal fixation, horses are confined to a box stall for at least 8 weeks, until postoperative radiographs indicate sufficient fracture healing. An intense rehabilitation program involving physical therapy and a controlled exercise protocol should be prescribed until shoulder musculature returns to normal, at which time training may be resumed.

PROGNOSIS

Fractures of the supraglenoid tubercle are rare and therefore there are few reports regarding surgical outcome. Return to athletic function depends on the amount of cartilage damage

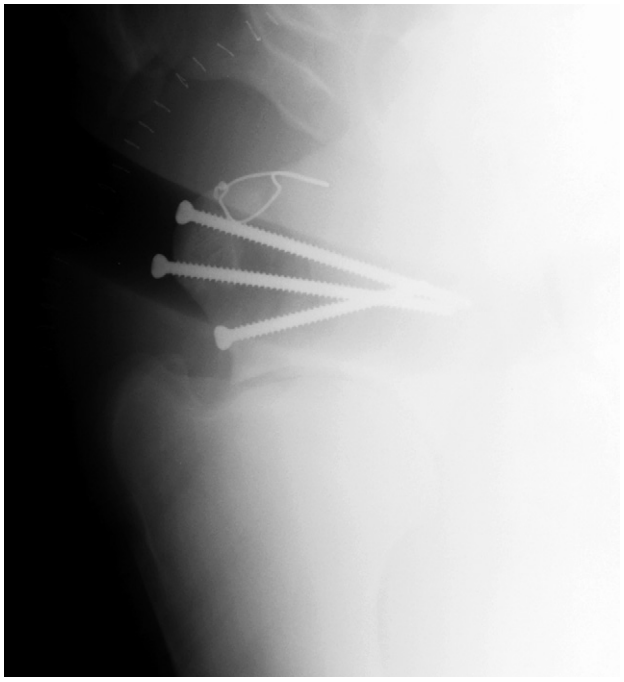

Figure 96-4. Internal fixation of a supraglenoid tubercle fracture using three 5.5-mm cortex bone screws and a tension band wire. (Courtesy Alan J. Nixon, Cornell University.)

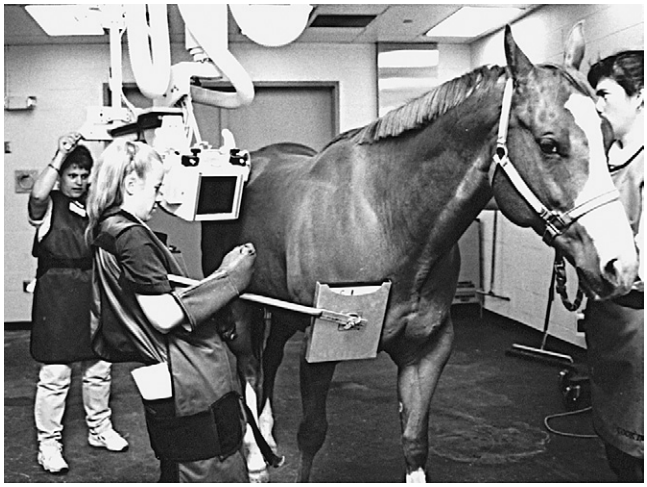

Figure 96-5. The position for obtaining a caudal proximolateral to cranial distomedial oblique (skyline) view of the lateral tuberosity of the proximal humerus is shown. The x-ray beam is directed from a caudal to a cranial direction.

present at the time of surgery and, in cases where the fracture fragment is removed, the amount of glenoid cavity removed with the fracture fragment. Both of these factors are assumed to be directly related to development of shoulder OA. The few reports that are available regarding supraglenoid tubercle fractures in the horse suggest that the prognosis for athletic function is fair to good following either fracture removal or internal fixation but poor with conservative treatment.[3,18,19] The most recent study indicates that 9 of 10 horses treated surgically and only 2 of 5 treated conservatively returned to athletic function.[3]

Miscellaneous Other Fractures

Fractures of the neck or body of the scapula or proximal humerus are rare but have been described.[20-22] These fractures are usually the result of trauma, and horses are variably lame.

The cranial and caudal aspects of the greater tubercle of the proximal humerus can fracture in different configurations. If the entire greater tubercle fractures transversely, the scapulohumeral joint can luxate. When clinical examination or nuclear scintigraphy suggests involvement of the greater tubercle, a caudal proximolateral to cranial distomedial oblique (skyline) radiograph of the proximal humerus should be obtained to diagnose the fracture (Figure 96-5). Depending on the size of the fracture fragment and the muscular attachments involved, the fragment may be treated conservatively, removed, or repaired using cortex screws placed in lag fashion.

Horses with complete fractures of the body of the scapula or scapular neck are unable to bear weight. A standing mediolateral radiograph with the affected limb pulled forward will reveal the fracture site. If the neck of the scapula is involved, function of the suprascapular nerve should be evaluated (see later). Internal fixation of complete or longitudinal fractures of the scapula has been described using internal fixation with multiple plates.[20,22,23] Application of a locking compression plate (LCP) cranial and caudal to the spine is the preferred technique (Figures 96-6 and 96-7). Incomplete fractures can be managed conservatively.[24]

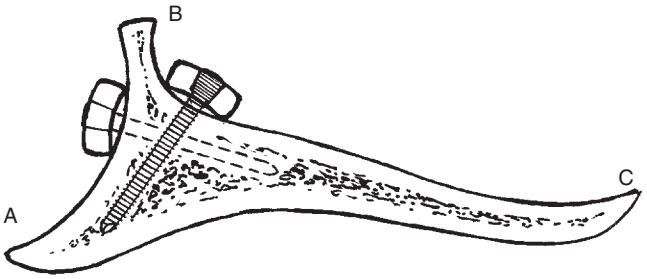

Figure 96-6. Crossectionoal view across the body of the scapula distant to the fracture. One LCP is applied to the cranial and caudal angle between the scapula and its spine. *A,* Cranial edge; *B,* spine; *C,* caudal edge.

Scapulohumeral Joint Luxation

Scapulohumeral joint luxations are rare in horses and are traumatic in origin. Because of their traumatic nature, concurrent fractures of the scapula or proximal humerus can occur.[25] The horses are non-weight-bearing and might have shoulder atrophy if the luxation is chronic. The humeral head can be palpated lateral, or less commonly cranial, to the scapula. Standing caudolateral to craniomedial oblique-view radiographs best demonstrate the luxation (Figure 96-8). The radiographs should be carefully evaluated for fractures of the humeral head or scapula, particularly involving the lateral rim of the glenoid.

Treatment

Closed reduction followed by scapulohumeral joint arthroscopy to evaluate the articular surfaces and remove cartilage debris resulted in return to sound performance in one case.[26] Closed reduction should be attempted whithin 24 hours and before an open approach is performed to preserve intact lateral musculature of the shoulder joint and minimize the potential for re-luxation of the shoulder joint postoperatively. Open reduction should be combined with some form of internal fixation such as scapulohumeral tension wires, although most types of internal fixation eventually fail.

The ultimate outcome for most horses with scapulohumeral luxation is shoulder OA and severe lameness, resulting in euthanasia. Arthrodesis of the scapulohumeral joint has been successful in one small (250 kg) horse and in Miniature Horses.[27-29] Arthrodesis is currently accomplished using a narrow LCP contoured to the cranial surface of the scapula and cranial aspect of the humerus.[29] For more information, review Chapter 81.

Prognosis

There are very few reports detailing repair of scapulohumeral joint luxation, but the prognosis for soundness following repair appears to be poor because of resultant crippling shoulder OA.

Pathologies of the Biceps Brachii and Infraspinatus Tendons and Their Associated Bursae

The biceps brachii muscle originates on the supraglenoid tubercle as a bilobed tendinous structure, and it inserts on the medial radial tuberosity. It passes over the proximal cranial aspect of

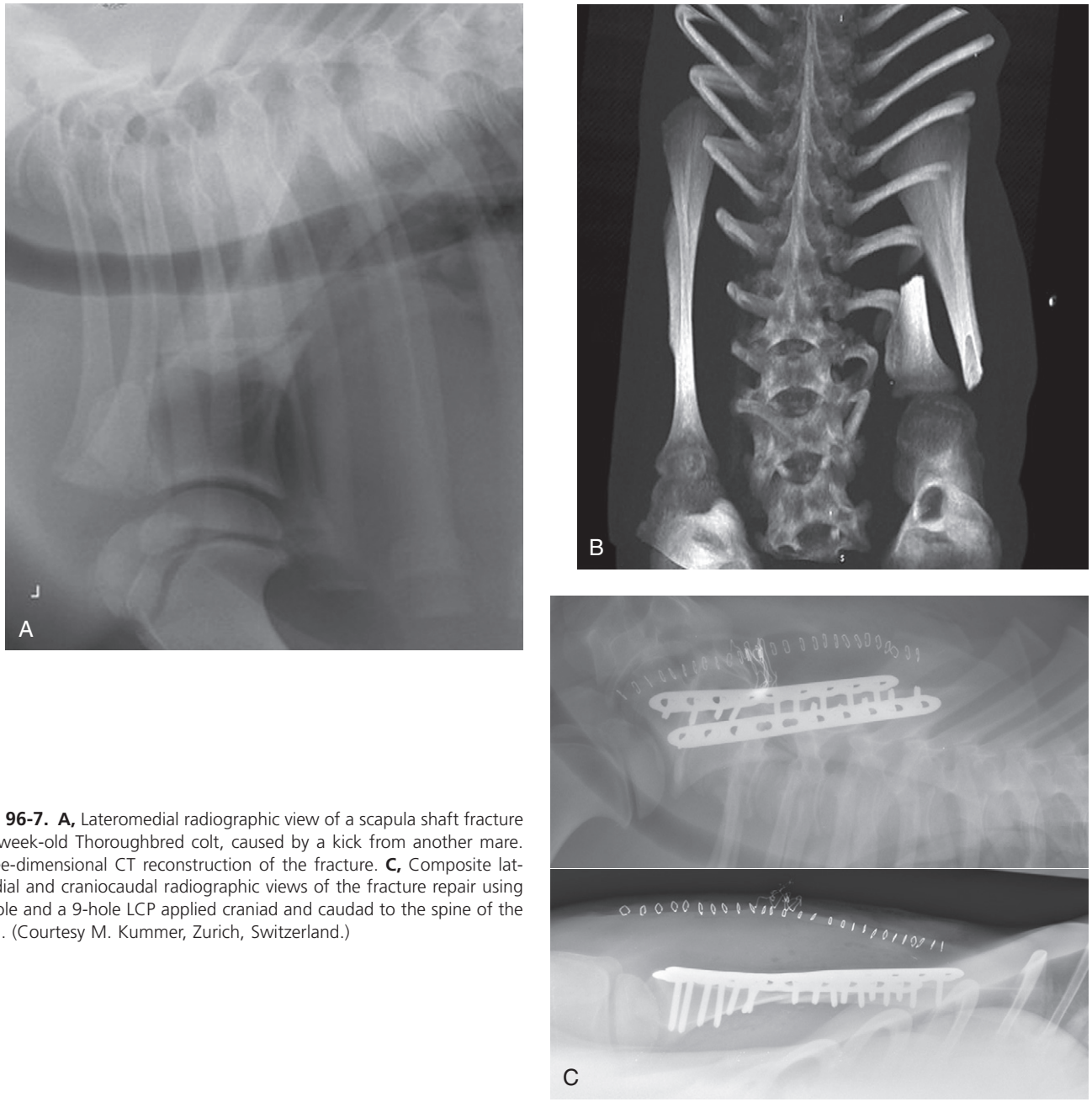

Figure 96-7. A, Lateromedial radiographic view of a scapula shaft fracture in a 3-week-old Thoroughbred colt, caused by a kick from another mare. **B,** Three-dimensional CT reconstruction of the fracture. **C,** Composite lateromedial and craniocaudal radiographic views of the fracture repair using a 10-hole and a 9-hole LCP applied craniad and caudad to the spine of the scapula. (Courtesy M. Kummer, Zurich, Switzerland.)

the humerus, where it is bound to the intertubercular groove by a tendinous part of the superficial pectoral muscle. It acts to flex the elbow joint and provides some stability to the shoulder. Passively, it limits elbow joint extension when the shoulder joint is in flexion, and it limits joint flexion when the elbow joint is extended.[30] The intertubercular (bicipital) bursa lies between the proximal biceps tendon and the humerus.[31]

Horses with bicipital bursitis or biceps tendinitis exhibit pain when the biceps is grasped and pulled laterally. Nuclear scintigraphy can be helpful in any of the vascular, soft tissue, or bone phases, but a definitive diagnosis requires intrasynovial anesthesia. After localizing the lameness to the biceps region, ultrasonographic and radiographic examinations of the biceps region should be obtained. Normal ultrasonographic morphology of the biceps tendon and bicipital bursa have been reported.[32] Caudolateral to craniomedial oblique radiographs of the proximal humerus are required to identify concurrent lesions involving the humeral tubercles (Figure 96-9).

Various pathologies have been reported in association with the biceps tendon or bicipital bursa including bursitis, tendinitis, ossifying tendinitis, medial displacement of the biceps tendon, and infectious bursitis.[33-39] Congenital hypoplasia of the minor tubercle of the humerus with subsequent medial luxation of the proximal tendon of the biceps brachii has also been reported in four mature horses.[40] Simple biceps tendinitis is managed conservatively, but surgical intervention should be considered for horses with idiopathic or infectious bursitis or with chronic bicipital bursitis.

The infraspinatus tendon extends from the infraspinatus muscle and courses over the lateral side of the shoulder joint

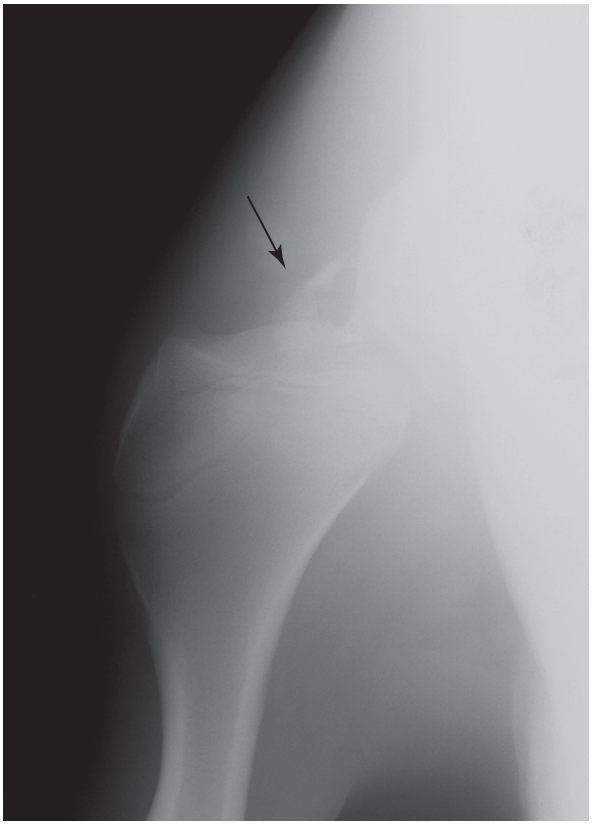

Figure 96-8. A caudolateral to craniomedial oblique view of the shoulder joint. A scapulohumeral luxation is present, with the humeral head located lateral to the glenoid cavity. A concurrent fracture, presumably originating from the glenoid, is present *(arrow)*.

and the caudal eminence of the greater tubercle to its insertion on the dorsolateral humerus. The infraspinatus bursa is located between the infraspinatus tendon and the caudal eminence of the greater tubercle. The normal ultrasonographic appearance of the infraspinatus tendon and bursa have been reported.[1] Injury to the greatere tubercle of the humerus and infraspinatus tendon with sepsis of the infraspinatus bursa has been reported in three horses.[41] In all cases, ultrasonography was more sensitive than radiographs for detecting osseous and soft tissue injuries. All three horses became sound following lavage of the bursa, removal of fracture fragments when present, and adminsitration of intrasynovial and systemic antimicrobials.

Treatment

ENDOSCOPY

Infection of the bicipital or infraspinatus bursa should be addressed using endoscopy and systemic antimicrobials. Endoscopy permits a thorough exploration of the infected bursa as well as a means of copious lavage and removal of adhesions. Synovial resection should be considered; however, it is controversial in cases of septic arthritis.

Endoscopy of the bicipital bursa in normal and infected bursae has been described.[38,42] The horse is positioned in lateral recumbency, with the affected limb uppermost. The bursa is distended with 100 mL lactated Ringer's solution as described earlier. The arthroscope is inserted into the distal aspect of the bursa through a skin incision placed 2 to 3 cm proximal to the deltoid tuberosity on the dorsolateral aspect of the humerus. The arthroscope is advanced proximomedially through the brachiocephalicus muscle, beneath the biceps brachii muscle, and along the cranial surface of the humerus. When the distended bursa is entered, and fluid escapes from the cannula, the obturator is removed and replaced with the arthroscope. The instrument portal site is predetermined by placement of a spinal needle in the proximal aspect of the bursa, lateral to the biceps tendon. The bursa is explored and any foreign debris and adhesions are removed.

Endoscopy of the infraspinatus bursa has also been described.[41] The horse is placed in lateral recumbency and ultrasonography is used to identify the cranial aspect of the bursa, where needles are inserted cranial to the infraspinatus tendon and 1 cm distal to the greater tubercles. The bursa is distended and an arthroscope is inserted into this region. Following placement of the arthroscope and further distention of the bursa, an instrument portal is identified with needle placement and established in the caudal bursa. Minimal manipulations, other than thorough exploration and extensive lavage, are possible in the bursa, and the incisions may need to be converted to an open approach for removal of fragments of the greater tubercle.

The type and duration of antimicrobial and anti-inflammatory medications administered depend on the presence or absence of sepsis. Similarly, postoperative rehabilitation and return to training or performance are related to the degree of tendinitis or bursitis.

BICEPS TENDON TRANSECTION

A combination of bicipital bursitis, biceps tendinitis, and humeral osteitis has been reported in horses.[33,35] This disease complex is thought to be a manifestation of chronic bursitis or tendinitis and can be infectious or traumatic in origin. Affected horses are severely lame and unresponsive to conservative therapy. Ultrasonography of the biceps bursa is consistent with bursitis; radiographs of the proximal humerus reveal osteolytic changes associated with the lateral tuberosity.

Complete transection of the biceps tendon results in a fair prognosis for return to soundness.[35] If purulent material is found within the biceps brachii tendon, then a tenectomy should be considered. The approach to the biceps tendon is the same as that described for removal of supraglenoid tubercle fractures. Postoperative management is also similar to that for removal of supraglenoid tubercle fractures because biceps tendon support is lost in both instances. There is, however, less dead space to be filled in by fibrous tissue.

Prognosis

There are too few reports of the various tendon or bursa problems to provide an accurate prognosis. As with most synovial structures, the prognosis for return to sound function ranges from guarded to good depending on the structures involved and the duration of clinical signs.

Suprascapular Nerve Injury (Sweeney)

The suprascapular nerve is typically damaged as the result of trauma to the cranial shoulder where the nerve courses across the neck of the scapula. The suprascapular nerve innervates the

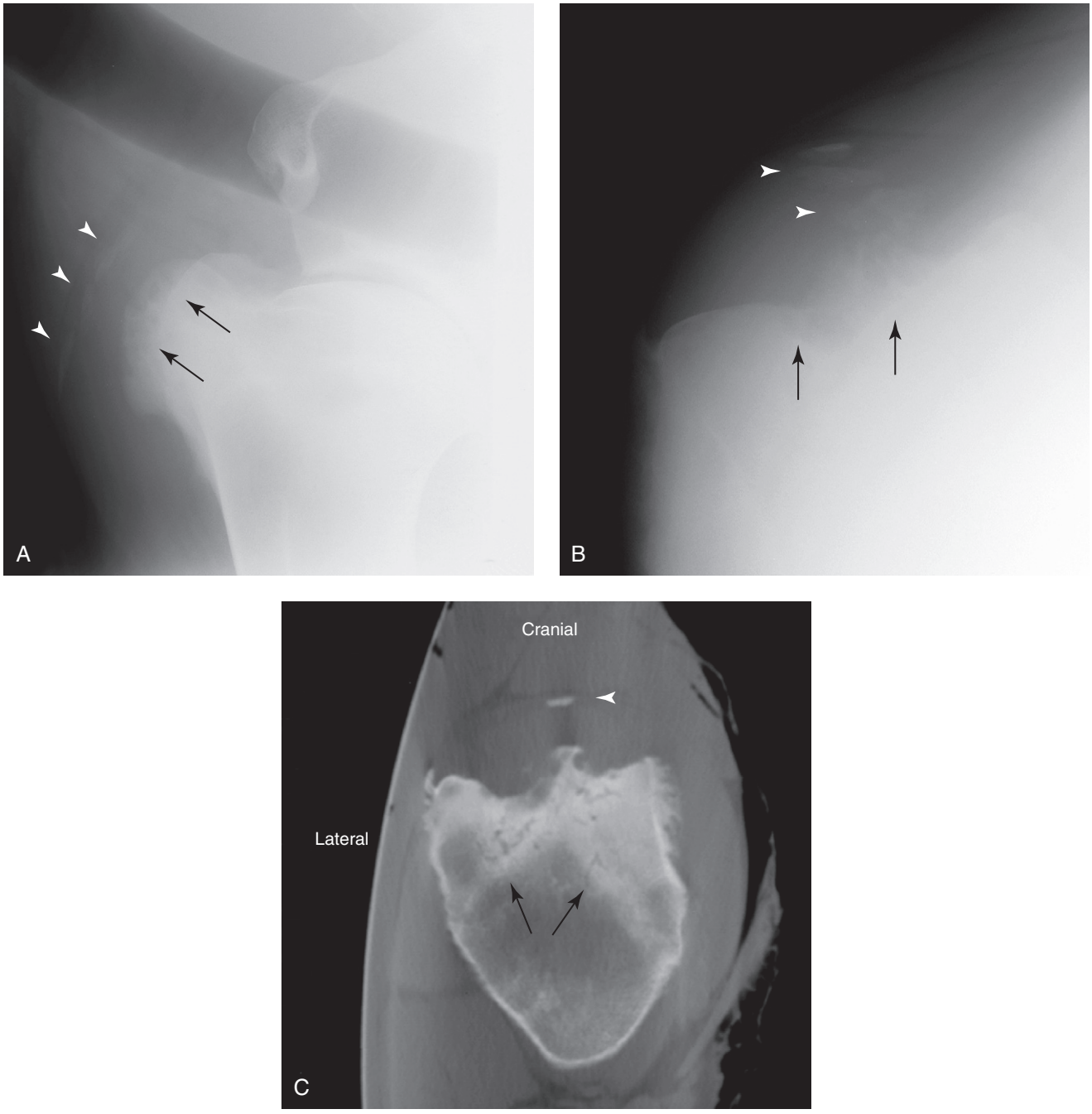

Figure 96-9. A, Mediolateral radiograph of the shoulder region in the intermediate tubercle of the humerus. Multifocal areas of irregular bone lysis *(black arrows)* can be seen. Cranial to the tubercles, there are linear foci of mineralization in the tendon of the biceps brachii *(white arrowheads).* **B,** Radiographic projection of the cranial proximal humerus. The lysis of the intermediate tubercle and mineralization of the bicipital tendon are confirmed and further defined in this radiographic projection. **C,** Postmortem transverse computed tomography evaluation of the affected shoulder region. The tubercles and medullary cavity of the cranial humerus are sclerotic *(black arrows).* There is a focal mineral density in the tendon of the biceps *(white arrowhead).*

infraspinatus muscle, which provides the majority of lateral support to the shoulder. As a result, horses with suprascapular nerve injury have a lateral motion or shoulder slip when bearing weight on the affected limb. This gait can be replicated by selective anesthesia of the suprascapular nerve, which suports the role of the suprascapular nerve in joint stability.[8]

If damage to the nerve is severe, the characteristic gait can be apparent immediately following injury, but more typically, a shoulder slip is not apparent for a few days to weeks after injury. Clinical signs do not indicate the degree of nerve damage, and there is no diagnostic method to determine the degree of recovery each horse will achieve.

Electromyography should be used to confirm that the suprascapular nerve, and not the brachial plexus, is the site of injury. Electromyography is only useful to evaluate nerve function from 7 days after injury on.

Treatment

NONSURGICAL MANAGEMENT

Nonsurgical management consisting of box stall rest alone results in a good return to function.[43] However, shoulder stability takes an average of 7 months to return. Surgical decompression of the suprascapular nerve can hasten the return of shoulder stability, diminish the amount of shoulder muscle atrophy, and return the horse to function sooner than that accomplished with nonsurgical therapy alone.[43,44]

The potential for a modestly decreased time to function needs to be weighed against the costs and risks associated with surgery, especially given the potential for postoperative supraglenoid tubercle or scapular neck fractures in horses following surgery.[45] A balance between the cost and benefit of each treatment is present in a staged approach consisting of nonsurgical management for 3 months, and if little improvement is noted, then surgical nerve decompression.

SCAPULAR NERVE DECOMPRESSION

The horse is positioned in lateral recumbency with the affected limb uppermost. A 25-cm (10-inch) skin incision is made over the spine of the scapula and through the cutaneus trunci muscle. The fascia covering the spine of the scapula is incised, and the brachiocephalicus muscle is elevated from the spine of the scapula while preserving the fascia deep to the supraspinatus muscle to protect the suprascapular neurovascular bundle. The nerve is elevated from the scapula, and a small amount of bone is rasped off the cranial margin of the scapula. Care is taken to maintain smooth edges of the bone to decrease potential postoperative nerve injury. A tendinous band that limits the movement of the suprascapular nerve can be palpated on the medial side of the scapula and covering the nerve. This band should be incised to maximize nerve decompression from scar tissue. Incisional closure is routine, with attention to obliteration of dead space.

Assisted recovery from anesthesia is recommended given the shoulder instability and potential for fracture of the supraglenoid tubercle. The potential for postoperative fracture through the notch created in the scapula exists for 6 weeks postoperatively. Antimicrobials and phenylbutazone are administered perioperatively, and low-dose phenylbutazone is continued for 3 to 5 days. The horse is confined to a box stall until shoulder stability is returned, typically 2 to 3 months. During stall confinement, muscle stimulation should be considered to minimize muscle atrophy. An intensive rehabilitation program then ensues to build shoulder strength prior to return to riding.

Prognosis

The prognosis for return to sound riding following conservative management or scapular nerve decompression is good.[44,45] Atrophy of the supraspinatus and infraspinatus muscles might not completely resolve in all cases.

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CHAPTER
97 Tarsus

Jörg A. Auer

ANATOMY

The bones that make up the tarsus include the tibia, the talus and calcaneus, the central tarsal bone, the fused first and second tarsal bone, the third and fourth tarsal bones, and the metatarsal bones (MTII, III, and IV) (Figure 97-1). The tarsal bones are maintained in intimate contact through their soft-tissue attachments, including the intertarsal ligaments, collateral ligaments, fibrous joint capsule, and plantar tarsal ligament. The medial and lateral collateral ligaments of the tarsus each consist of two ligaments: a long and a short ligament. The long collateral ligaments connect the distal tibia to the proximal metatarsal region, but they also have attachements to the talus and small tarsal bones. The short collateral ligaments originate from the medial or lateral malleolus, respectively, cranial to the origin of the long collateral ligaments, and attach to the talus and calcaneus. Although the long collateral ligaments of the tarsus are taut during tarsal extension, the short collateral ligaments are tense in flexion and loose in extension.[1,2]

The tarsus is composed of five joints: the tarsocrural (TC), proximal intertarsal (PIT), distal intertarsal (DIT), tarsometatarsal (TMT), and talocalcaneal (TCa) joints. Four of these five joints are almost immobile. Although micromovements do occur during ambulation and weight bearing, most of the movement of the tarsus is achieved in the tarsocrural joint.

The two distal joints are often injected for diagnostic and therapeutic purposes. In one study, therapeutic concentrations of methylprednisolone were measured in the DIT joint 6 hours after injection into the TMT joint, confirming that communication between the TMT and DIT joints existed in 10 of 10 sampled joints (100%).[3] In an earlier study, communication was found between the TMT and DIT joints in four of 57 tarsi (7%); in one horse (2%), communication between the DIT and PIT was established.[4] In seven horses (14%), communication between the DIT joint and the tarsal canal existed, pointing out the potentially inconsistent results that can be found after intraarticular anethesia.[4] Other studies in the literature cite the communication between the TMT and DIT joints at 7%[5] and 38%.[6] Based on these data, medications injected into the TMT joint can exert effects in the DIT joint, and this seems to be a common phenomenon after corticosteroid injections. However, for diagnostic purposes and to avoid a lack of response to therapeutic medications, the DIT joint should be injected at the same time. The TCa joint in horses is composed of three facets between the two bones: a facet at the medial aspect of the calcaneus and talus, an angulated coracoid facet at the lateral aspect of the calcaneus and talus, and a facet at the lateral aspect of the calcaneus (facing medially) and medial aspect of the talus (facing laterally).[7] A strong interosseus ligament in the central tarsal sinus maintains the two bones in close proximity. The special relationship of the three facets relative to each other prevents motion in the joint.[7]

Dorsally, the long digital extensor tendon spans the entire tarsal region and is maintained in position by the retinaculi. Just distal to the tarsal region, the lateral digital extensor tendon unites with the long digital extensor tendon. The strong fibularis tertius (peroneus tertius) and the tendon of the tibialis cranialis muscle course parallel to the long digital extensor tendon (Figure 97-2). The fibularis tertius tendon is penetrated by the tendon of the tibialis cranialis muscle, which then divides into three branches, the medial, middle, and lateral branches, before inserting on the central, third, and fourth tarsal bones as well as the proximal metatarsal region. The medial branch is better known as the cunean tendon, which at one time was thought to be responsible for the development of spavin. The fibularis tertius forms the cranial part of the reciprocal apparatus (Figure 97-3). The gastrocnemius muscle and tendon form the caudal part. The intact reciprocal apparatus ensures that when one of the major joints of the rear limb is flexed, all others flex.

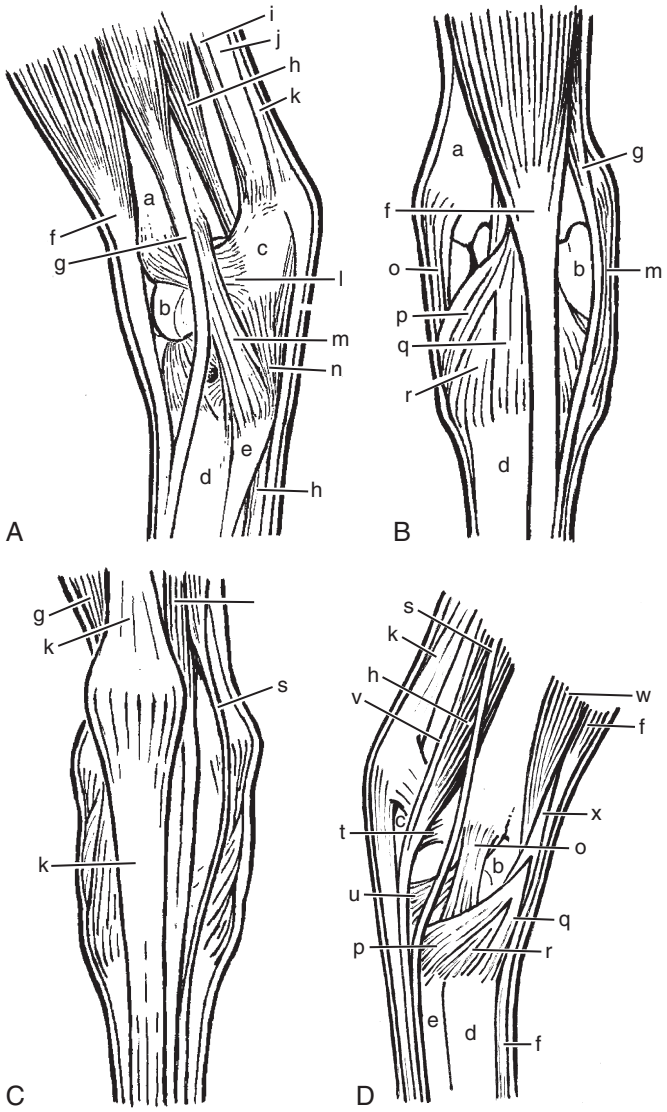

Figure 97-1. Graphic illustration of a lateral **(A),** dorsoplantar **(B),** plantarodorsal **(C),** and medial **(D)** view showing the anatomic structures associated with the tarsal region. *a,* Distal tibia; *b,* talus; *c,* calcaneus; *d,* third metatarsus (MTIII); *e,* MTIV; *f,* long digital extensor muscle and tendon; *g,* lateral digital extensor tendon; *h,* lateral digital flexor muscle (together with "v" form the deep digital flexor tendon); *i,* soleus tendon; *j,* Achilles tendon; *k,* superficial digital flexor tendon; *l,* short lateral collateral ligament; *m,* long lateral collateral ligament; *n,* plantar ligament; *o,* long part of the medial collateral ligament; *p,* medial part of the tibialis anterior tendon (cunean tendon); *q,* middle part of the tibialis cranialis tendon; *r,* branch of the fibularis tertius tendon; *s,* medial digital flexor tendon; *t,* medial plantar ligament between the talus and calcaneus; *u,* short part of the medial collateral ligament; *v,* tendon of the tibialis caudalis muscle; *w,* fibularis tertius muscle; *x,* main branch of the fibularis tertius tendon.

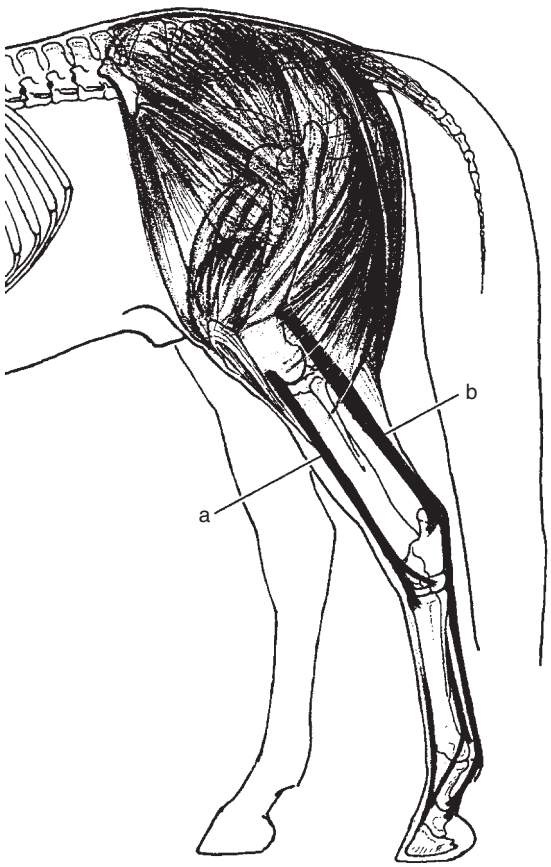

Figure 97-2. Correlation of the anatomic structures composing the reciprocal apparatus of the horse. *a,* The fibularis tertius tendon originates on the muscular fossa of the distal femur and inserts at the small tarsal bones as well as the third metatarsus (MTIII), whereas the lateral part courses toward the fourth tarsal bone; *b,* the gastrocnemius muscle originates from the plantar fossa of the distal aspect of the femur and with the Achilles tendon and unites with the soleus and accessory tendons before it inserts on the tuber calcanei.

Plantarly, the tendon of the superficial digital flexor muscle passes around the medial aspect of the gastrocnemius tendon and continues distad as the most plantar tendinous structure at the level of the tuber calcanei. The superficial digital flexor tendon is attached to the abaxial aspects of the calcaneus via the medial and lateral retinacula and continues distad to the digit. The voluminous intertendinous calcaneal bursa is located between the superficial digital flexor and the gastrocnemius tendon proximally and between the superficial digital flexor tendon and the long plantar ligament distally. A smaller bursa, the gastrocnemius calcaneal bursa, lies dorsal to the insertion of the gastrocnemius tendon on the tuber calcanei. These two bursae usually communicate, most commonly on their medial aspect. Another bursa is located between the subcutaneous tissue and the gastrocnemius tendon and is termed the *subcutaneous calcaneal bursa*. This bursa is usually a separate structure but can communicate with the intertendinous calcaneal bursa in some horses.[8] The tibialis caudalis and lateral digital flexor muscle both contribute to the deep digital flexor tendon that runs over the sustentaculum tali, embedded in the tarsal sheath. The tendon of the medial digital flexor muscle traverses the tarsal region medially and joins the deep digital flexor tendon in the proximal metatarsal region. All tendons pass over the tarsal region within a tendon sheath. The function of the tarsus is to transform axial loading during weight bearing from the cranioproximal to caudodistal oblique forces exerted along the tibia into vertically and distally oriented forces through the metatarsal area.

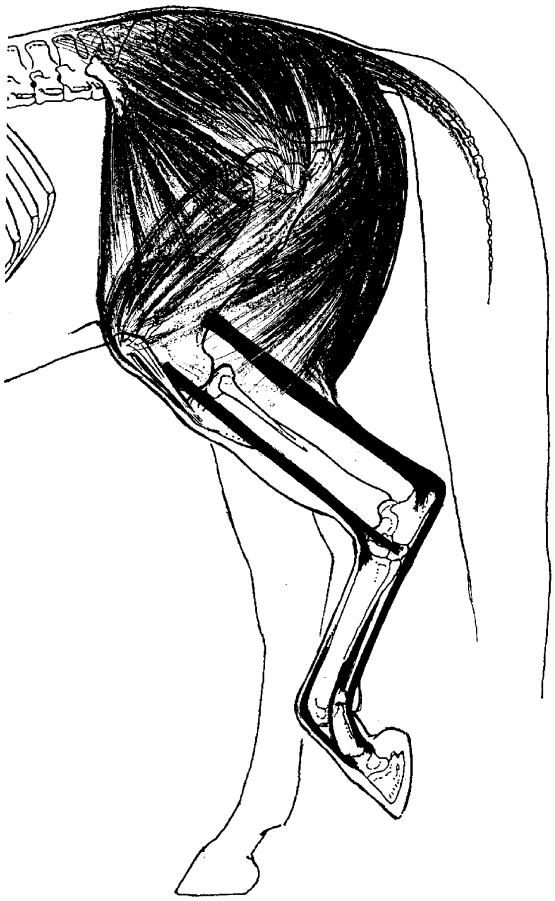

Figure 97-3. Illustration of the hind limb reciprocal apparatus demonstating that flexion of the tarsus or stifle results in flexion of all remaining joints in the hind limb.

ACQUIRED DISORDERS OF THE TARSAL REGION

Acquired disorders include fractures, osteoarthritis (OA), luxations, and tendon and ligament disorders. There is a large variability in presenting signs and diagnostic modalites needed to diagnose these disorders and subsequently manage them properly.

Osteoarthritis

Osteoarthritis is the most diagnosed acquired tarsal disorder causing lameness.[6] It is seen in three forms: one involves the distal tarsal joints (referred to as *bone spavin*), one is located at the talocalcaneal joint, and the other involves the tarsocrural joint (referred to as *high spavin* or *bog spavin* when joint distention is present).

Etiology

OA of the distal tarsal joints is the most common cause of hindlimb lameness in the horse.[9] The DIT and TMT joints are most often involved, but occasionally the PIT joint also is affected. A study in foals showed that bone density of the compact bone of the subchondral bone plate in the third tarsal bone reacts to variations in exercise at a very young age.[10] Low bone density resulting from a lack of exercise was reversed when exercise was introduced. Osteoarthritic radiographic signs (enthesiophytes) in the distal intertarsal joints were common at 5 months of age and increased in severity until 11 months of age. Although the clinical relevance of these abnormalities in foals is uncertain, they might be related to the development of osteoarthritis in this region later in life.[9]

Although the exact pathogenesis of distal tarsal OA is still unknown, compression and rotation of the distal tarsal bones, which occurs most forcefully at the gallop, can contribute to its development.[11] Dressage horses, western horses, pulling horses, and Standardbreds are prone to the disorder, which is associated with their type of athletic activity. Icelandic horses are often affected, which is possibly related to the mismatch of the horse and the rider and the gaits at which these animals are used. The rack is a fast four-beat gait during which the horses place their hind limbs extremely far forward, and this extreme type of gait can be responsible for the development of bone spavin in these horses. Poor conformation also is a contributing factor to this disorder: bowlegged and sickle-hocked conformation results in abnormal loading of the small tarsal bones and leads to the development of OA.

Crushing of the central or third tarsal bones in dysmature neonates because of incomplete ossification may eventually lead to the development of juvenile OA of the tarsus.

Other inciting causes of distal tarsal OA are septic arthritis, tarsal bone fractures, osteochondrosis, and excessive exercise.[9,12-14] OA also has been associated with increased intramedullary pressure within the cuboidal bones of the tarsal region.[15] Inciting causes for this rarely encountered tarsocrural and sometimes talocalcaneal joint OA include intra-articular fracture, osteochondrosis, septic arthritis, and collateral ligament damage.[16]

For most other tarsal disorders, external trauma, such as a kick from another horse or being hit by or running into an object, is the inciting cause. Repetitive trauma as it occurs during racing or competitive events in general can lead to the development of lameness.

Clinical Signs

The cardinal sign of a tarsal problem is lameness, which can vary depending on the severity, location, and acuteness of the disorder. Toe dragging is a common early sign that occurs because of a reluctance to flex the tarsus. Horses with OA often have a reduced foot flight arc, shortened cranial phase of the stride, gradual axial deviation in the flight path of the foot during the cranial phase of the stride, and a rapid abaxial deviation at ground contact.[17] Affected horses often show pain to back palpation, and many horses are presented for a primary complaint of back pain. However, in most cases, a weight-bearing lameness is noted.

Joint distention is not a typical sign of OA of the distal tarsal joints, because the tarsal retinaculum over these small joints prevents its detection. However, new bone proliferation at the dorsomedial aspect of the limb might be noticeable in chronic cases (Figure 97-4). A typical sign of a tarsocrural disorder is joint distention (bog spavin). However, as often noted in OCD, the swelling does not have to be painful to palpation.

An acute onset of a marked to severe weight-bearing lameness is a clear sign of trauma and is often associated with joint distention or swelling of the neighboring tissues, or both. Penetrating articular wounds often can lead to septic arthritis

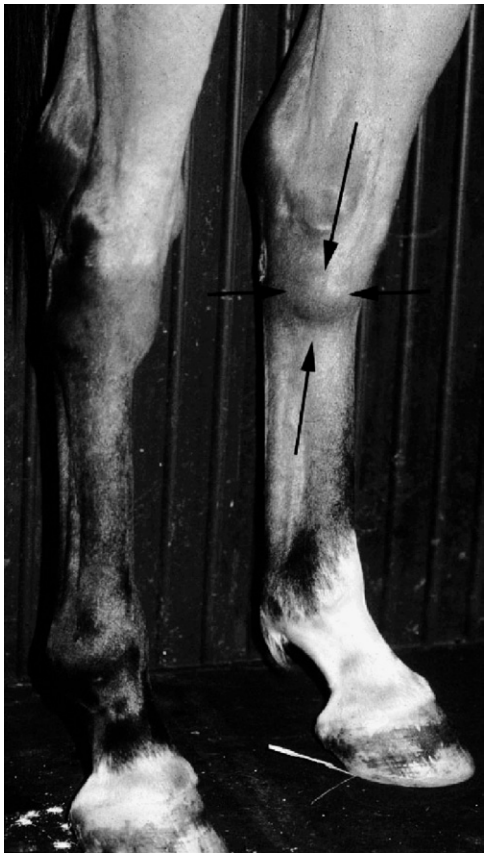

Figure 97-4. Marked enlargement at the dorsomedial aspect of the distal tarsal region *(arrows)* in a horse suffering from osteoarthritis of the distal two tarsal joints (bone spavin). Excessive new bone formation is responsible for this appearance.

with all the accompanying signs including heat, swelling, and pain.

Significant periosteal new bone formation and enthesiophytosis associated with a hopping gait in the rear legs are the distinctive clinical signs of juvenile OA. In severe cases of tarsal bone collapse, foals may show a "curblike" appearance in the distal tarsal region, which is associated with pain. For additional information on this condition, see Chapter 86.

Diagnosis

The diagnosis of tarsal disorders involves a routine lameness examination. In the presence of fractures and joint infections, the origin of which is often easy to localize, immediate attention should be given to the inciting cause, sidestepping the lameness examination.

Leading the horse in tight circles at walk in both directions can exacerbate the lameness on the inside limb. Some horses initially show a marked lameness, but with time they warm out of it. Often the horse exhibits a barely detectable unevenness at the time of presentation or no evidence of lameness at all because of bilateral pain. A history that includes deterioration in the horse's attitude suggests subtle hock pain, which does not produce overt lameness. Evaluation of the shoes and looking for uneven wear can lead the clinician toward the diagnosis. Palpation of the tarsal region can allow detection of enlargement over the dorsomedial aspect of the tarsus; however, most lesions do not cause pain upon digital pressure. Cunean bursitis and effusion of the cunean bursa are rare, but they can be detected by palpation. Flexion tests often exacerbate the lameness.

Intra-articular anesthesia is a valuable aid in localizing the problem. Because there is controversy in the literature regarding communication between the TMT and DIT joints, both joints should be anesthetized.[4,5,18-20] Significant improvement in lameness 10 minutes after intra-articular injection of 2 to 4 mL mepivacaine hydrochloride is diagnostic, but complete resolution of lameness might not occur because of intraosseous pain in the tarsal bones.[15] Larger volumes of anesthetic should not be used because of the potential for periarticular extravasation and contact with nonarticular tissues, including the sensory nerve supply from the distal limb as it passes through the tarsus.[18] In young racing Standardbreds, it might be advantageous to anesthetize the cunean bursa, because cunean bursitis is diagnosed in these animals.

The joint region to be injected is clipped and prepared as for aseptic surgery. Meticulous cleansing of the area without clipping of the hair (application of the antiseptic of choice, scrubbing for 5 minutes followed by removal of the antiseptic soap, repeated twice) has no higher risk of iatrogenic joint infection following arthrocentesis than clipping the hair prior to preparation and needle placement (see Chapter 72).[21,22]

The TMT joint is the easiest of the distal tarsal joints to inject (see Figure 72-13). The needle is inserted laterally, 0.5 cm proximal to the head of MTIV, in a dorsomediodistal direction at a 45-degree angle to both the sagittal plane and the ground until it makes contact with bone, usually at a depth of approximately 2 cm.[15] This technique results in the needle tip's passing deep, medial, and slightly distal to the palpable lateral aspect of the head of MTIV, positioning it near the articulation between MTIV and the fourth tarsal bone. The steep angle is necessary to position the needle tip sufficiently distal to avoid penetrating the DIT joint, which is located only slightly proximal to the head of the MTIV.

The DIT joint is injected medially in the space between the central tarsal bone, the third tarsal bone, and the fused first and second tarsal bones (Figure 97-5). The location for needle placement is found by palpation of the distal border of the cunean tendon, approximately 2 cm caudal to a vertical line extended distad from the medial malleolus.[13] Ideally, the needle is inserted to the hub at a 45-degree angle in a plantarolateral direction and parallel to the ground.

A dorsolateral approach to the DIT has been described (see Figure 72-13).[23] The injection site is located 2 to 3 mm lateral to the long digital extensor tendon and 6 to 8 mm proximal to a line drawn perpendicular to the axis of the third metatarsal bone through the proximal end of the fourth metatarsal bone. The needle is directed at an angle of approximately 70 degrees from the sagittal plane in a plantaromedial direction. In this report, the number of needle repositionings required to complete DIT joint centesis via the dorsolateral and medial approaches was not significantly different. I prefer this location because, in addition to having the same success rate as the medial approach, palpation of the landmarks is easy and the clinician is less vulnerable to getting kicked by the horse.[23]

When using the medial approach, full penetration of a 3-cm (1 inch) needle in small horses can result in the needle's passing completely through the DIT joint and into connective tissue deep to the joint capsule.[9] Also, the TMT joint can be entered

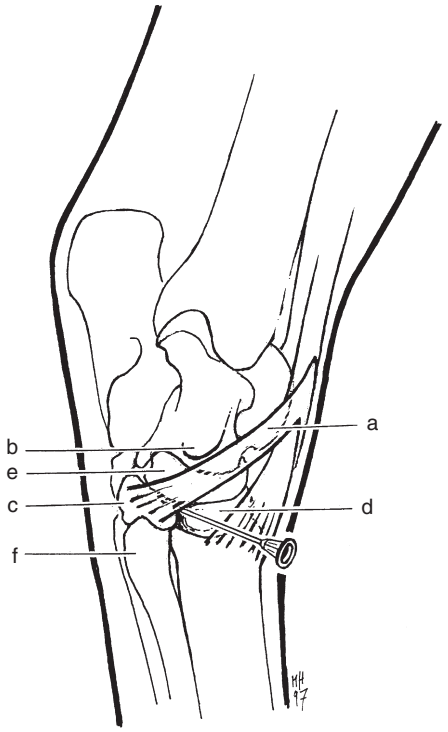

Figure 97-5. Graphic illustration of the injection site for the distal intertarsal (DIT) joint. The DIT joint is injected medially in the proximal aspect of the space that exists between the central tarsal bone *(e)*, third tarsal bone *(d)*, and fused first and second tarsal bone *(c)*. The entrance to this space can often be felt with firm palpation and is found at the distal border of the cunean tendon *(a)*, approximately 2 cm caudal to a vertical line extending distad from the medial malleolus. The location for needle entry can also be located along a line between the distal tubercle of the talus *(b)* and the palpable space between the second metatarsus (MTII) *(f)* and MTIII, where it intersects the cunean tendon.

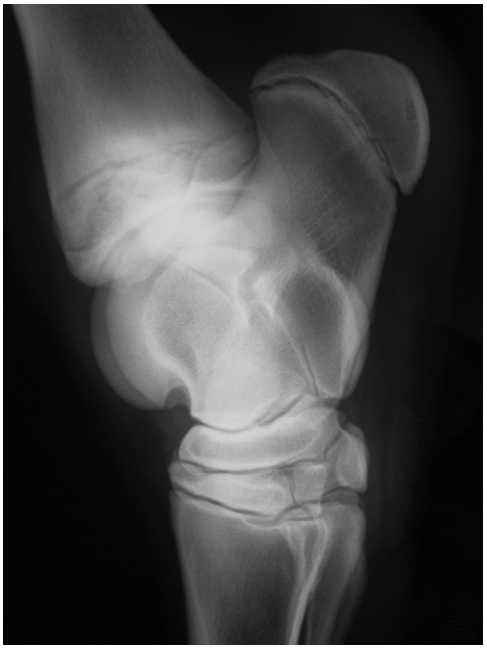

Figure 97-6. Lateromedial radiographic view of the tarsal region of a 4-month-old foal that was born with incomplete ossification. The central tarsal bone is partially collapsed dorsally. This foal is a good candidate to develop juvenile osteoarthritis.

inadvertently on the medial aspect of the tarsus while attempting to enter the DIT joint, because the TMT joint capsule is located just slightly distal to the DIT joint capsule.[7] The PIT joint is rarely injected alone, because it communicates with the tarsocrural joint, and this injection is not discussed here.

The tarsocrural joint, of all joints composing the tarsus, is the easiest to inject, especially when it is distended. The dorsomedial pouch of the joint can be easily palpated (see Figure 72-12). Taking care to avoid the saphenous vein, the needle is then inserted in a dorsocaudolateral direction, and depending on the degree of joint distention, 10 to 20 mL of synovial fluid is withdrawn prior to injecting 8 mL of mepivacaine hydrochloride.

Diagnosis is confirmed using standard radiographic views, including oblique projections. OA is commonly a bilateral condition; therefore, radiographs of both tarsal regions are recommended. Because some changes might be subtle, it is important to take good-quality radiographs. Lameness associated with the distal tarsal joints can exist with little or no radiographic abnormalities. Conversely, extensive radiographic changes might be present that are not associated with pain or lameness. Therefore, it is important to locate the site of lameness before obtaining radiographs. Fractures are best confirmed with good-quality radiographs. In rare cases, such as fractures of the central or third tarsal bones, this imaging technique may not afford a diagnosis.

Juvenile OA is easily diagnosed with the help of radiography (Figure 97-6). Dorsopalmar and lateromedial views are adequate for the diagnosis. Despite the fact that this problem is usually encounterd bilaterally, taking radiographs of one tarsus is usually enough to confirm the diagnosis.

Ultrasonography also can be used to arrive at a diagnosis as long as the lesions are peripheral. In-depth knowledge of the tarsal anatomy is a prerequisite if this imaging technique is used. Scintigraphy has been used to localize lesions not visible on radiographs, especially in early cases where radiographic changes are not yet apparent.[24,25] A recent study showed that ultrasonography can be a valuable asset in diagnosing dorsal tarsal soft tissue lesions, such as tendon pathology or distended distal tarsal joints.[26] Computed tomography can be applied in the tarsus and provides valuable details for an exact diagnosis, especially in cases of hidden new bone formations and fractures. This technique is gaining in popularity. A comparison of radiography, nuclear scintigraphy, and magnetic resonance imaging (MRI) for diagnosing specific conditions of the equine distal tarsal joints revealed a low sensitivity for radiography because a high percentage of horses had normal or inconclusive radiographs despite pathologic changes that were identified with MRI.[27] Conversely, nuclear scintigraphy correlated well with the location of lesions found with MRI in all horses in this study.

Treatment

OSTEOARTHRITIS OF THE DISTAL INTERTARSAL JOINTS (BONE SPAVIN)

Management of OA includes corrective shoeing, systemic and intra-articular anti-inflammatory medications, shock wave therapy, chemical and surgical arthrodesis, and cunean tenectomy.

Corrective shoeing

Horses with bowlegged conformation profit from corrective shoeing in which the medial hoof wall is shortened and a special horseshoe with a wider, thicker outside branch is applied. The application of lateral wedges between shoes and hoof wall decreases tension on the medial patellar ligament and pressure on the medial side of the tarsal joint. However, the metacarpophalangeal joint experiences considerably more stress.[28] A shoe with a lateral extension is used to unload the medial aspect of the tarsal region by altering the foot-ground weight distribution and foot orientation during stance.[29] A study revealed that proper conditioning also can have a protective effect against bone spavin and lateral luxation of the patella.[28]

Extracorporal shock wave therapy

The systems used to apply extracorporal shock wave therapy in equine medicine are of the middle-energetic (0.2 mJ/mm^2) to high-energetic (0.4 mJ/mm^2) form. Therapy is generally applied in two or three sessions at approximately 3-week intervals. For treatment of soft-tissue calcifications, intervals between 3 and 6 weeks are used. The skin overlying the target tissue is clipped or shaved. During application of the shock waves, the horse is sedated. Depending upon the system used, a session takes 10 to 25 minutes.

Some promising results have been achieved in the management of OA of the distal intertarsal joints.[30] Originally it was thought that the focused shock waves would stimulate bone remodeling, increase blood flow to bone-tendon interfaces, stimulate cellular metabolism, and provide short-term analgesia.[31] Several studies have been conducted to evaluate these effects , especially after some studies indicated a rapid decrease in lameness following treatment.[32] An experimental study comparing the effects of shock wave therapy on cutaneous nerve function to the effects of a local nerve block and sedation discovered that there were no effects on cutaneous sensation distal to the treated region for at least 3 days after shock wave application.[33] Another study conducted on horses suffering from proximal palmar metacarpal/plantar metatarsal pain and using an instrumented treadmill to determine the effect of shock wave therapy showed a slight but not significant gait improvement.[34] However, there was improvement in the forelimb parameters, indicating a certain degree of analgesia. Taking into account the results of the different studies, the ban imposed on the use of extracorporeal shock wave therapy in the days immediately preceding a competition seems justified, even if they are difficult to enforce.

Medical management

Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used as adjunctive medication during the convalescent period after surgery, but as a sole treatment for distal tarsal OA, they are only palliative. As long the medication is administered, the lameness might be reduced or even absent, but as soon as administration is stopped, the lameness returns.

Steroid treatment of OA involves intra-articular injection of long-acting corticosteroids into the involved joints (indicated only after diagnostic intra-articular anesthesia renders the animal free of pain or convincingly reduces the lameness). The TMT and DIT joints of both limbs can be treated simultaneously. With simultaneous treatment, the dose per joint should be reduced, because the total dose per horse should not exceed toxic levels (see Chapter 79 for specific drug dosages).

After injection, horses are rested for 2 or 3 days, following which work can be resumed. Most horses respond favorably to injection and are free of pain for several months. Aseptic technique is very important to avoid inducing a postinjection flare or septic arthritis. A sudden onset of lameness within a few days after the injections should be managed immediately with joint lavage under general anesthesia. Oral anti-inflammatory and systemic antimicrobial therapy is indicated to maintain a reasonably comfortable patient during management of this complication.

Lameness usually recurs after a few weeks to months and can be managed by additional corticosteroid injections. The pain-free interval shortens with repeated injections. In such patients, surgical or chemical fusion should be considered to maintain a useful horse.

A recent survy among equine paractitioners revealed that 70% included corticosteroids in their treatment regimen of tarsal OA. Most respondents combined cortiocosteroids with another medication, such as high molecular weight hyaluronan (59%) or amikacin (57%) (see below).[35] Triamcinolone acetate was the most frequently used drug in high-motion joints, whereas methylprednisolone acetate was most commonly used in low-motion joints.[35] Of the respondents, 44% used 18 to 40 mg of triamcinolone acetate as the total body dosage.

Hyaluronan and polysulfated glycosaminoglycan (PSGAG; Adequan) are used to improve the joint environment and reduce inflammation in high-motion joints (see Chapter 79). The above-mentioned survey also revealed that hyaluronan and PSGAGs were the most commonly used noncorticosteroid therapeutics, and they were primarily used prophylactically.[35] Although hyaluronan was frequently combined with corticosteroids (see earlier), PSGAGs were primarily injected IV.[35] It is my opinion that these drugs can provide temporary relief for distal tarsal OA. However, if the patient is to be used in athletic competition, return of lameness is inevitable. Therefore, these drugs do not have a place in the management of OA of the distal tarsal joints, especially if costs are considered.

A recent study showed that the combination of hyaluronan, chondroitin sulfate, and *N*-acetyl-D-glucosamine (Polyglycan) produced significant improvement in lameness and disease progression after intraarticular injection in an experimental carpal OA model in horses.[36] These results can also be applied to the tarsus.

Chemical fusion of the DIT and TMT joints by intra-articular injection of sodium monoiodoacetate (MIA) was introduced as an alternative to surgical arthrodesis for horses clinically affected with distal tarsal OA. MIA blocks a specific enzyme pathway in chondrocyte metabolism, resulting in chondrocyte death, cartilage necrosis, joint collapse, and fusion of the distal tarsal joints.[37-40]

Horses receiving MIA should be premedicated with phenylbutazone (4.4 mg/kg IV every 12 hours), beginning 12 hours before treatment. Injection with MIA should be preceded by contrast arthrography of each joint, because communication between the DIT and PIT joints and communication between the TMT and PIT joints has been documented (Figure 97-7).[21]

For a detailed description of the technique please review Chapter 81. Because of the inherent danger associated with communication with other joints, this technique has never gained widespread acceptance.

The use of intraarticular injections of ethyl alcohol has also been studied.[41,42] Ethyl alcohol functions as a neurolytic agent

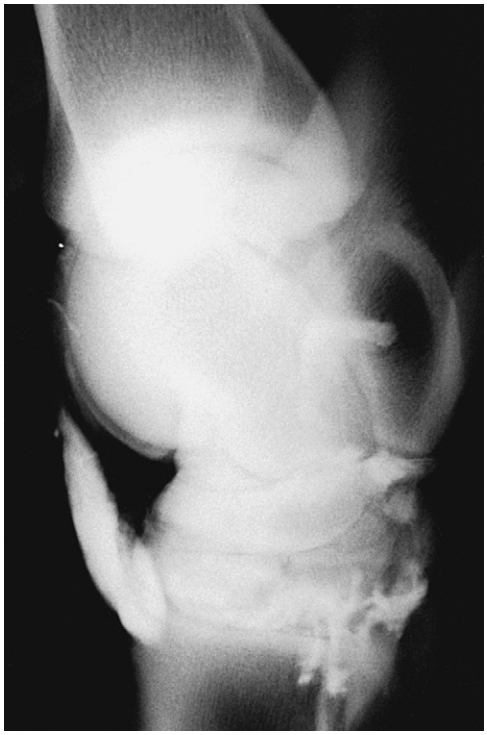

Figure 97-7. Positive contrast arthrogram after injection into the TMT joint, showing communication of the tarsometatarsal joint with the proximal intertarsal and tarsocrural joints and the extensor digital sheath.

that leads to sensory innervation blockade at the intraarticular level.[41] The same potential complications accompany this technique as with MIA injections.. This technique is described in detail in Chapter 81.

Although both techniques of chemical fusion are economical, it must be kept in mind that chemical fusion is a time-consuming procedure that bears the risk of inducing undesired effects in other structures not intended to be involved, such as the talocrural joint. The experimental data are promising; however, these techniques have failed to achieve consistently favorable clinical results.

Surgical management

Cunean tenectomy is a relatively simple procedure performed with sedation and local anesthesia. Cunean tenectomy can improve lameness by decreasing rotational forces on the tarsus that occur when the obliquely oriented cunean tendon tightens.[31] This surgical treatment for acute OA is controversial. In instances when distal tarsal OA is chronic and bone proliferation under the tendon is excessive, this technique can provide some pain relief. The procedure is usually performed bilaterally.

The surgical site is anesthetized by an inverted V-shaped line block spanning from the chestnut to the dorsal aspect of the saphenous vein. A 4-cm vertical skin incision is made over the cunean tendon approximately 5 cm dorsal to the chestnut, where the tendon is easily palpable. A 4-cm section of tendon is removed with a No. 15 scalpel blade, and the skin is sutured. A compression bandage is applied and changed as needed until the sutures are removed at 14 days after surgery.

The Wamberg technique complements cunean tenectomy with gridlike incisions over the medial aspect of the tarsus down to the bone. The purpose of these incisions, performed at 3.5 mm intervals, is to complete neurectomy of the region. This procedure has been abandoned.

Aftercare of cunean tenectomy consists of stall rest for 12 days with limited daily hand-walking. After the sutures are removed, the amount of hand-walking is increased gradually through the twenty-first postoperative day and includes walking over poles. Three weeks after surgery, riding starts over poles, and full work resumes at 6 weeks. This exercise regimen is important for preventing formation of restrictive scar tissue between the severed tendon ends and the cunean bursa.

The efficacy of cunean tenectomy has been debated, but there are no controlled clinical studies or experimental evidence in the literature to either support or refute the validity of the procedure. An owner survey of 285 cases of bone spavin treated by cunean tenectomy showed that most owners (83%) believed that lameness and performance improved after cunean tenectomy and that they would have the procedure performed again.[43] Today, cunean tenectomy as a sole procedure is rarely performed. It is, however, performed together with an arthrodesis using bone plates (see below and Chapter 81).

Laser facilitated ankylosis using neodymium: yittrium aluminum garnet (Nd:YAG) or diode[44,45] laser to destroy articular cartilage has also been performed. The theory behind this technique is that by superheating and vaporizing synovial fluid, chondrocyte death should follow. Clinical studies are currently not available, but experimental studies in sound horses demonstrated that laser application promotes partial ankylosis of the DIT joint within 5 [44] to 12 months.[45] However, in an experiemental study, diode laser–facilitated ankylosis resulted in significantly less fusion of the distal tarsal joints when compared to MIA injection or transarticular drilling (see below).[45]

Arthrodesis of the distal tarsal joints can be achieved using different techniques.[17,46-51] Most procedures involve radiographically guided removal of cartilage by local drilling across part of the joint space. Bone plates, intra-articular stainless steel cylinders, transarticular screws placed in lag fashion, and cancellous bone grafts have been used to improve stability and hasten fusion after drill removal of cartilage and subchondral bone, but none of these treatments appears to offer a substantial advantage over drilling alone.

Joint stability and pain relief can be obtained after bony bridging across only a small portion of the involved articular surface, which act as "spot welds." Techniques have to be developed to provide better stabilization over a larger articular area, similar to the technique used for proximal interphalangeal arthrodesis (see Chapter 81), where a dorsal plate is supplemented by two screws that provide transarticular compression in the palmar region. However, in most of the currently used techniques, only the plate bridges the joints to be arthrodesed, and transarticular screws are not used. For more information on screw fixation for OA, please review Chapter 81. However, I favor parallel transarticular drilling to promote fusion of the distal tarsal joints without internal fixation.

Articular drilling is performed bilaterally on both the DIT and TMT joints as a single procedure. The technique is described in detail in Chapter 81. Drilling too deeply can cause penetration of the tarsal canal, resulting in unnecessary periosteal reactions or profuse hemorrhage if the perforating branch of the cranial tibial artery is inadvertently traumatized. Avoiding these complications is preferable, but they appear to have no effect on the success of the procedure.

Aftercare

Horses can be lightly ridden 3 to 4 weeks after surgery. Administration of anti-inflammatory drugs maintains a reasonable level of comfort during the process of fusion. Most animals are free of lameness within 4 or 5 months of the procedure, but others show persistent lameness. Judgment on the outcome of the procedure is reserved until 12 months after surgery.

Prognosis

The prognosis for horses with distal tarsal OA treated with surgical arthrodesis is favorable, because most horses return to full function when the affected tarsal joints fuse.[17] Return to soundness over a 3- to 12-month period is reported in 66.7% to 85% of horses undergoing arthrodesis of the DIT and TMT joints.[47,49] Involvement of the PIT joint carries a less-favorable prognosis because of its consistent communication with and concurrent involvement of the TC joint.[49]

The prognosis following chemical fusion of the distal tarsal joints with MIA is favorable. Most horses show dramatic improvement in the original level of lameness within 1 week after treatment, even though fusion might not be apparent radiographically for 3 to 6 months (Figure 97-8).

Thirty-nine horses that had OA and were treated with intra-articular MIA were evaluated.[38] Treatment was considered a short-term success when there was simultaneous documentation of radiographic fusion and soundness. At 1, 3, 6, and 12 months, success was achieved in 4 of 26 (8%), 15 of 27 (56%), 21 of 26 (81%), and 27 of 29 (93%) horses, respectively. Long-term evaluation showed that 16 of 16 (100%) horses had radiographic fusion of the treated distal tarsal joints, and 12 of 16 (75%) were free of lameness and the treatment was considered successful. OA of the PIT and TC joints was encountered in 4 of 39 horses (10%) and was diagnosed between 1 and 4 years after MIA treatment as acute severe lameness and swelling of the TC joint.[38] Each horse was treated with MIA before the potential for communication between the DIT and PIT joints was recognized, and none received contrast arthrograms. It is presumed that such a communication resulted in contact of MIA with cartilage of the PIT and TC joints.

Another study revealed that in 50% of normal horses injected with 70% ethanol were radiographically fused at 4 months post injection and in 15 of 16 horses (94%) were fused after 12 months.[37]

OSTEOARTHRITIS OF THE TALOCALCANEAL JOINT

The TCa joint has a curved contour, which is best recognized on lateromedial radiographs. The medial, lateral, proximal, and interosseous talocalcaneal ligaments support it.[1]

Osteoarthritis of the TCa is rarely described (Figure 97-9).[16,52] However, two reports have renewed the interest in this disorder.[7,53] The clinical signs are not specific and resemble those of distal tarsal OA. Radiography and scintigraphy have been very beneficial in diagnosing the problem.[7,54]

Conservative therapy is usually unrewarding.[53] Stall rest, pasture exercise, and intra-articular corticosteroids with or without hyaluronan or PSGAG did not bring about consistent improvement of the lameness.

Surgical treatment involving partial tibial and fibular neurectomy cannot be recommended because of the poor results.[53] Osteostixis also did not result in improvement of the lameness.[54] The best results were achieved with arthrodesis of the TCa joint (see Figure 81-22).

Three techniques are described, all applying cortex screws across the joint.[7,53] One technique involves implanting three 5.5-mm cortex screws across the joint from the plantarolateral aspect of the calcaneus into the talus.[53] Lameness improved in

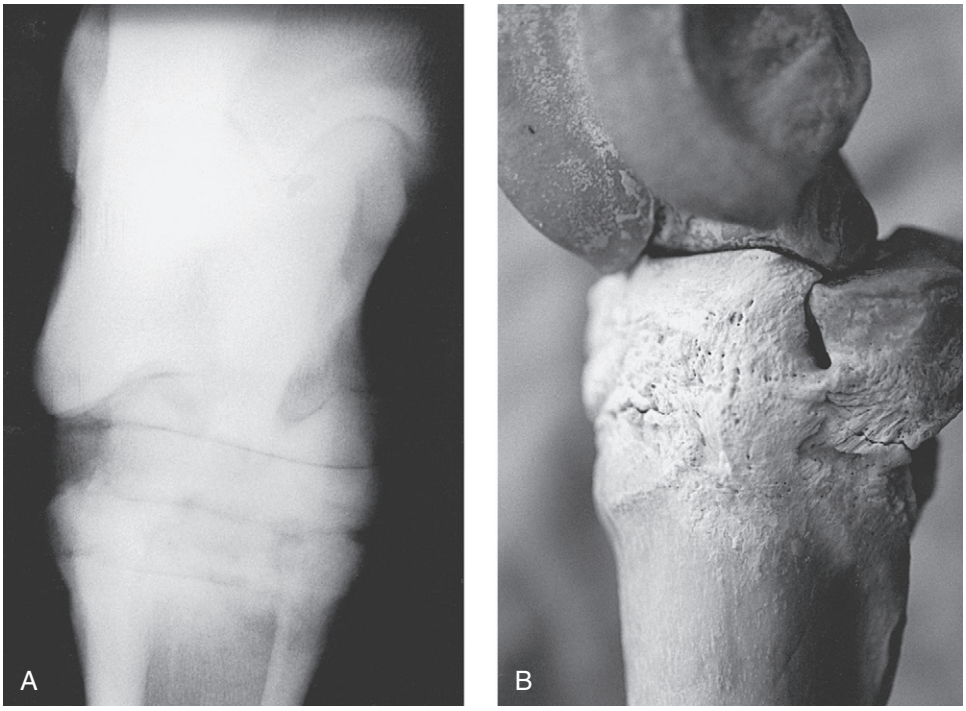

Figure 97-8. A, Dorsoplantar radiographic view demonstrating fusion of the tarsometatarsal and distal intertarsal joints 6 months after injection with sodium monoiodoacetate. **B,** Gross anatomic specimen of the same horse after it was euthanized 1 month later for an unrelated reason. Solid bone bridging is obvious in certain areas.

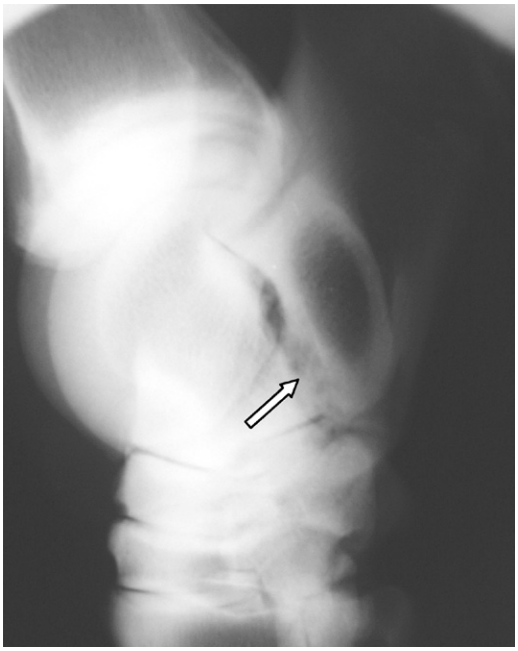

Figure 97-9. Lateromedial radiographic view of osteoarthritic changes located in the talocalcaneal joint *(arrow)*. (Courtesy R. Smith, Royal Veterinary College, London.)

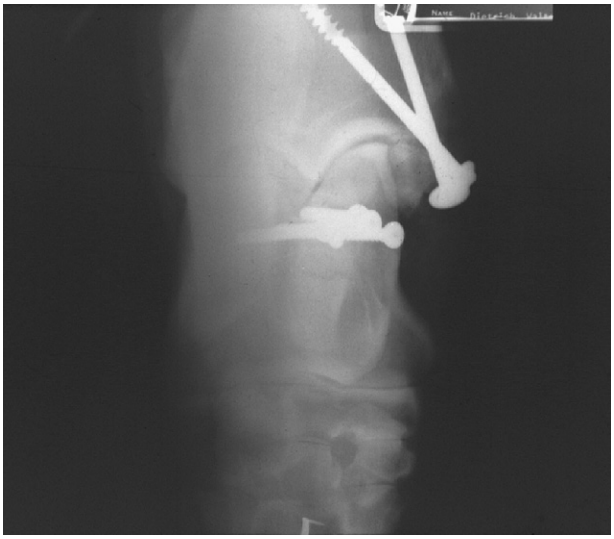

Figure 97-10. Dorsoplantar radiographic view of fixation of a medial malleolar fracture with two long cancellous screws and washers. Additionally, large fragments off the lateral trochlear ridge of the talus were reattached through three cortex screws, which were countersunk below the articular surface.

all six horses subjected to this treatment. The other report described two techniques.[7] In a pony, three parallel 5.5-mm cortex screws were implanted in lag fashion from a dorsomedial arthrotomy approach to the talus across the medial talocalcaneal articular facet into the calcaneus. Successful fusion was achieved. In the other horse, two 5.5-mm cortex screws were implanted as described and two additional identical screws were inserted from the plantarolateral aspect of the calcaneus into the talus.[7] Successful fusion was achieved, allowing the horse to return to full daily work 1 year later. Additional information is found in Chapter 81.

Fractures

Fractures of the tarsus, although rare because of the dense supporting structures that cover the bones in this area, occur when a significant external impact, such as a kick from another horse, is sustained in the tarsal region. Twisting and shearing of the tarsus while the lower limb is fixed can result in disruption of collateral and periarticular ligaments and subsequently lead to fractures. Twisting of the tarsus while the lower limb is flexed can lead to lateral maleolar fractures, which occur as avulsions because of the action of the short part of the collateral ligament.

Distal Tibia

Fractures of the distal tibia often involve the medial malleolus, and these arise from external trauma, which results in avulsion of the medial malleolus from the tibia that is rarely accompanied by rupture of the medial short collateral ligament.[55,56] Fractures of the lateral malleolus can occur as avulsions and usually involve the short lateral collateral ligaments.[25,55] Avulsion fractures of the lateral malleolus are often comminuted. Arthroscopy can be used to remove fragments involving the medial malleolus; however, because of the soft-tissue coverage and intimate association with the joint capsule or lateral collateral ligaments, or both, lateral malleolar fragments can be removed more easily through an arthrotomy into the dorsolateral pouch of the TC joint or through a direct approach over the fracture through the short lateral collateral ligament, or a combination of these techniques under arthroscopic guidance. If arthroscopy is attempted alone, the portals need to be placed into both the dorsoateral and plantolateral pouches and the fragment might have to be divided into smaller pieces for removal using rongeur or an osteotome and mallet.

Fragments larger than 3 cm are best reattached to the parent bone using 3.5-mm cortex screws placed in lag fashion.[12,57] Large avulsion fractures of the medial malleolus can be repaired with one or two 4.5- or 5.5-mm cortex or cancellous screws placed in lag fashion (Figure 97-10). The incisions are closed using routine technique. A full-limb cast is applied for recovery from anesthesia and maintained during the initial postoperative period, if fixation rather than removal has been elected.

Conservative management of nondisplaced distal tibial malleolar fractures is possible. Surgical management, either by removal of the fragment or by repair of the fracture in correct anatomic position, has a favorable prognosis for return to previous performance levels in one report.[30] However, an unfavorable to guarded prognosis was given in another report.[11] A total of 4 to 6 months of rest is needed after surgery, particularly if fracture fixation was used as the surgical treatment.

Trochlear Ridges of the Talus

Fractures of the talus are acute injuries and are almost exclusively the result of external trauma, such as a kick from another horse. The clinical signs vary considerably, depending on comminution, size, and displacement of the fracture. Significant effusion of the TC joint is seen with most fractures of the talus, and response to tarsal flexion is moderate to severe. Standard

radiographic projections should be supplemented with flexed lateromedial and flexed lateromedial oblique projections and skyline views of the trochlear ridges.[13] The distal aspect of the lateral trochlear ridge and the proximal aspect of the medial trochlear ridge (see Figure 97-7) are most often affected.[29,58,59]

Arthroscopic removal of small trochlear ridge fractures is generally the preferred treatment.[60,61] The arthroscope portal for removing trochlear ridge lesions can be either dorsomedial or dorsolateral. The dorsolateral portal offers better visualization of the distal aspect of the lateral trochlear ridge, but the standard dorsomedial portal is preferred because triangulation is better and the surgeon can carry out a more thorough exploratory examination of the joint from a medial approach. The instrument portal is always dorsolateral and is usually slightly distal to that used for distal tibial lesions. Osteochondral defects or cartilage flaps might extend proximad along the lateral trochlear ridge, necessitating extension of the limb during surgery. Some surgeons prefer an arthrotomy approach directly over the lesion.[12,60]

Large trochlear ridge fractures can be repaired with interfragmentary compression using several screws (Figure 97-11).[57] The fracture can be approached either through arthroscopy or an arthrotomy performed lateral to the ridge. After the fragment is identified and reduced, a glide hole is drilled perpendicular to the fracture plane from the dorsal aspect of the ridge. After preparing the thread hole, a deep countersink depression is prepared in the articular surface to accept the entire screw head. Therefore, it is better to use multiple 3.5-mm cortex screws, which have smaller heads, than to use 4.5- or 5.5-mm cortex screws. In large fragments, an adequate amount of solid bone is present to permit fixation. Anatomic reconstruction is a must if fixation is attempted. If this cannot be achieved, it is best to remove the fragment immediately to prevent the development of OA. Alternative fixation techniques include absorbable polydioxanone pins and cannulated screws.[57]

Spurs or fragments associated with the distal end of the medial trochlear ridge of the talus (dewdrop lesions) are usually incidental findings and are not an indication for surgery, because they are usually extra-articular (Figure 97-12).[13,55] Arthroscopic removal has been described in the literature, although the technique is difficult because of extensive soft-tissue covering.[62] For removal of these fragments, both the arthroscope and instrument portals are made in the dorsomedial pouch of the tarsocrural joint, with the instrument portal placed distal and slightly medial to the arthroscope.

Fragments that originate from the proximal aspect of the TC joint are best removed arthroscopically (Figure 97-13). With the horse in dorsal recumbency, the limb is positioned in flexion. With the joint distended, the arthroscope portal is made in the center of the caudolateral pouch of the TC joint. This approach provides good visualization of the most proximal aspect of the talocalcaneal articulation,[60] the proximocaudal aspect of the lateral trochlear ridge, the caudal aspect of the intermediate ridge of the tibia, and the medial trochlear ridge. Access to affected portions of the trochlear ridge can be accomplished as the degree of flexion is modified. The instrument portal is made in the caudomedial joint pouch using needle and visual arthroscopic guidance.

Fractures of the distal lateral or medial aspect of the body of the talus occur rarely. Extreme angular forces need to be involved to cause such a fracture (Figure 97-14, *A*). The animals are almost always non–weight-bearing lame and the tarsus is diffusely swollen. Radiography confirms the diagnosis. Conservative therapy in most cases is unrewarding. The horse shown in Figure 97-14 was readmitted for treatment after a year of limited pasture exercise because of continued lameness and pronounced joint distention of the tarsocrural and proximal intertarsal joint (Figure 97-14, *B*). The fracture was subsequently repaired with 3.5-mm cortex screws in lag fashion through an approach directly over the fragment. The lameness and joint

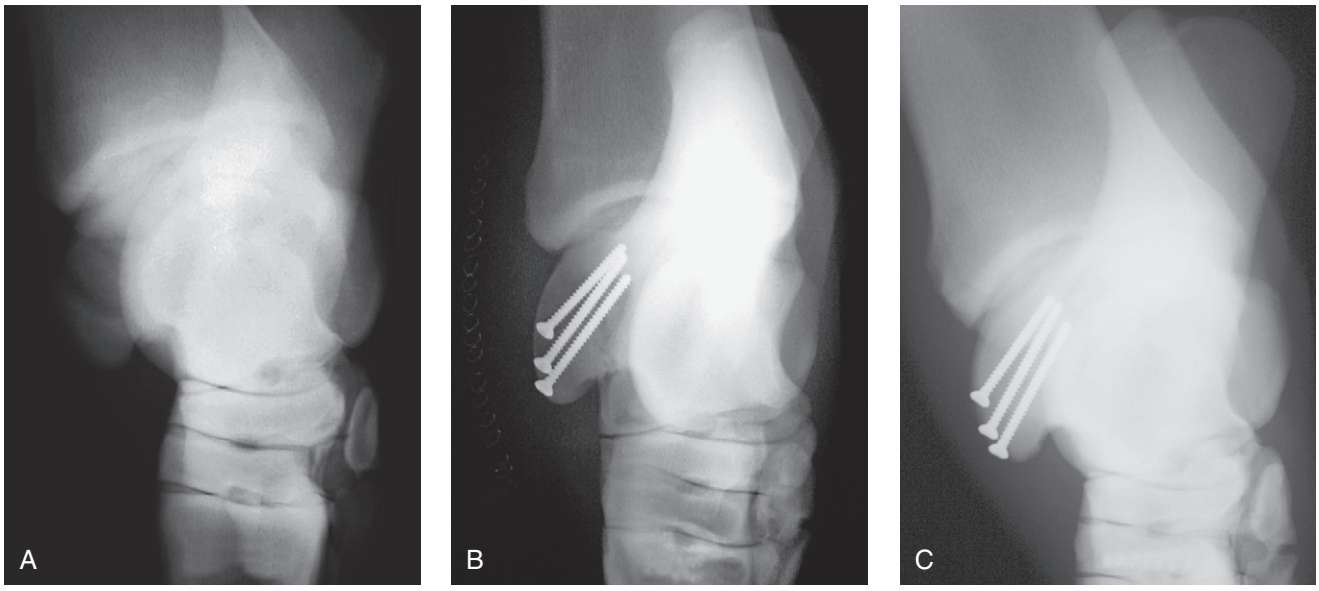

Figure 97-11. A, A large, slightly displaced slab fracture of the lateral trochlear ridge. **B,** The fracture was repaired with three 3.5-mm cortex screws inserted perpendicular to the fracture plane. The screws were inserted from the articular surface, and the screw heads were countersunk below the surface. **C,** A two-year follow-up revealed no osteoarthritis in the joint. The horse was successfully competing in show jumping events. (Courtesy C. Lischer, University of Zurich.)

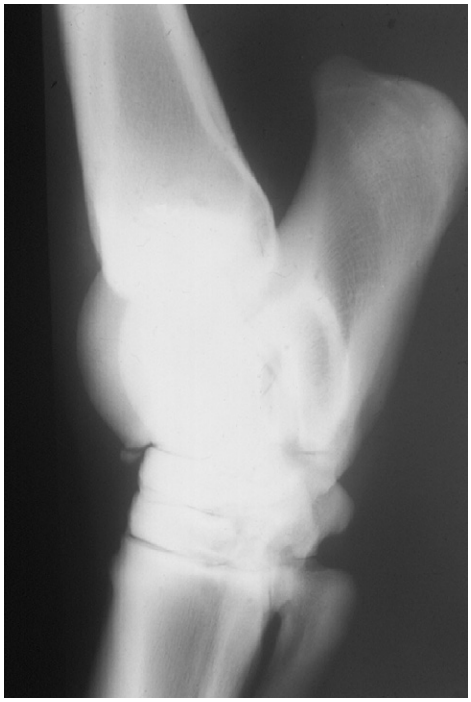

Figure 97-12. Dorsolateral–plantaromedial oblique radiographic view showing a teardrop lesion or fragmentation originating from the distal aspect of the distal medial trochlear ridge of the talus. This finding is usually incidental and rarely has any clinical significance. (Courtesy C. W. McIlwraith, Colorado State University.)

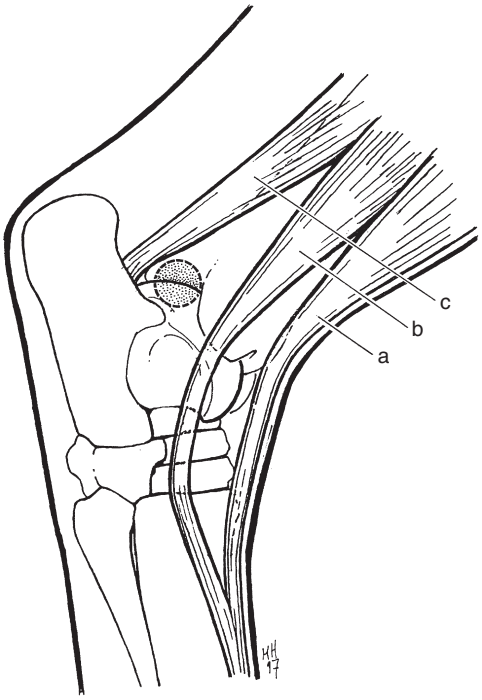

Figure 97-13. Graphic illustration of the arthroscope portal site for examining the plantar pouch and visualizing the plantar aspect of the trochlear ridges of the distal tibia. The arthroscope is placed contralateral, and the instrument is placed ipsilateral to the lesion. *a,* Long digital extensor tendon; *b,* lateral digital extensor tendon; *c,* deep digital flexor tendon.

distention resolved 4 months postoperatively and the horse was returned to show jumping.

Recovery after surgery of the trochlear ridges of the talus takes 8 to 10 weeks after arthroscopic removal of small fragments and 6 to 8 months or more if fracture repair is undertaken. Prognosis after fracture removal is favorable if soft-tissue damage at the time of injury is mild, secondary changes in the joint are minimal, and the fragment is shorter than 3 cm.[57]

Sagittal Fractures of the Talus

Sagittal fractures of the talus are rare.[55,63] The biggest challenge is correct assessment because radiographically they are difficult to evaluate , especially with complicated fracture configurations (Figure 97-15). Computed tomography is the imaging technology best suited for a detailed diagnosis, which is a prerequisite for proper screw placement. The intraoperative use of three-dimensional fluoroscopy (Arcadis orbic 3D) is a good alternative (Figure 97-15, *B*). The medial fragment is usually smaller, and fractures can be repaired with two or three 4.5- or 5.5-mm cortex screws placed in lag fashion. In special configurations, the screws may have to be inserted from the articular surface, making deep countersinking necessary to bury the entire screw head in the trochlear ridge (Figure 97-15, *C*). The fracture can be approached from either the medial or lateral side.[57,63] Arthroscopic visualization during the procedure is useful in assisting anatomic reduction of the fracture. Prognosis is favorable if anatomic reduction can be achieved.[57] Severely comminuted fractures of the talus are associated with severe soft-tissue trauma and destabilization of the TC joint and are inoperable in most cases.[57]

Calcaneus

Calcaneal fractures are often open and can be complicated by osteomyelitis, septic calcaneal bursitis, sequestration, and chronic drainage. Major fractures of the calcaneus can result in displacement of the superficial flexor tendon or can involve the sustentaculum tali (Figure 97-16). An open wound and local infection can eventually extend into the tarsal sheath. Lameness is usually severe with calcaneus fractures, and diagnosis is straightforward. In addition to standard projections, stressed radiographs can provide important information for diagnosis and management. Skyline views are useful for evaluating the tuber calcis and sustentaculum tali.

Small fragments distant from the calcaneal bursa can be removed through a direct approach or left to heal conservatively if they are not infected, but larger fragments should be repaired by screw fixation using lag technique. Transverse fractures of the shaft of the calcaneus require application of one or two narrow 4.5-mm plates or one broad plate placed on the plantarolateral aspect of the bone.[20] The selection of dynamic compression plates (DCPs), limited-contact dynamic compression plates (LC-DCPs), or locking compression plates (LCPs) depends upon availability and financial considerations. The superficial digital flexor tendon is reflected medially to allow placement of the plate, and the horse is recovered and maintained for 2 to 4 weeks in a full-limb cast that is removed with the horse standing. Removal of the implants after fracture healing can be helpful, but the overall prognosis to return to full performance for this type of fracture is guarded.[57,64,65]

Fractures involving the calcaneal bursa in the presence of an open wound from a kick usually have a poor prognosis for

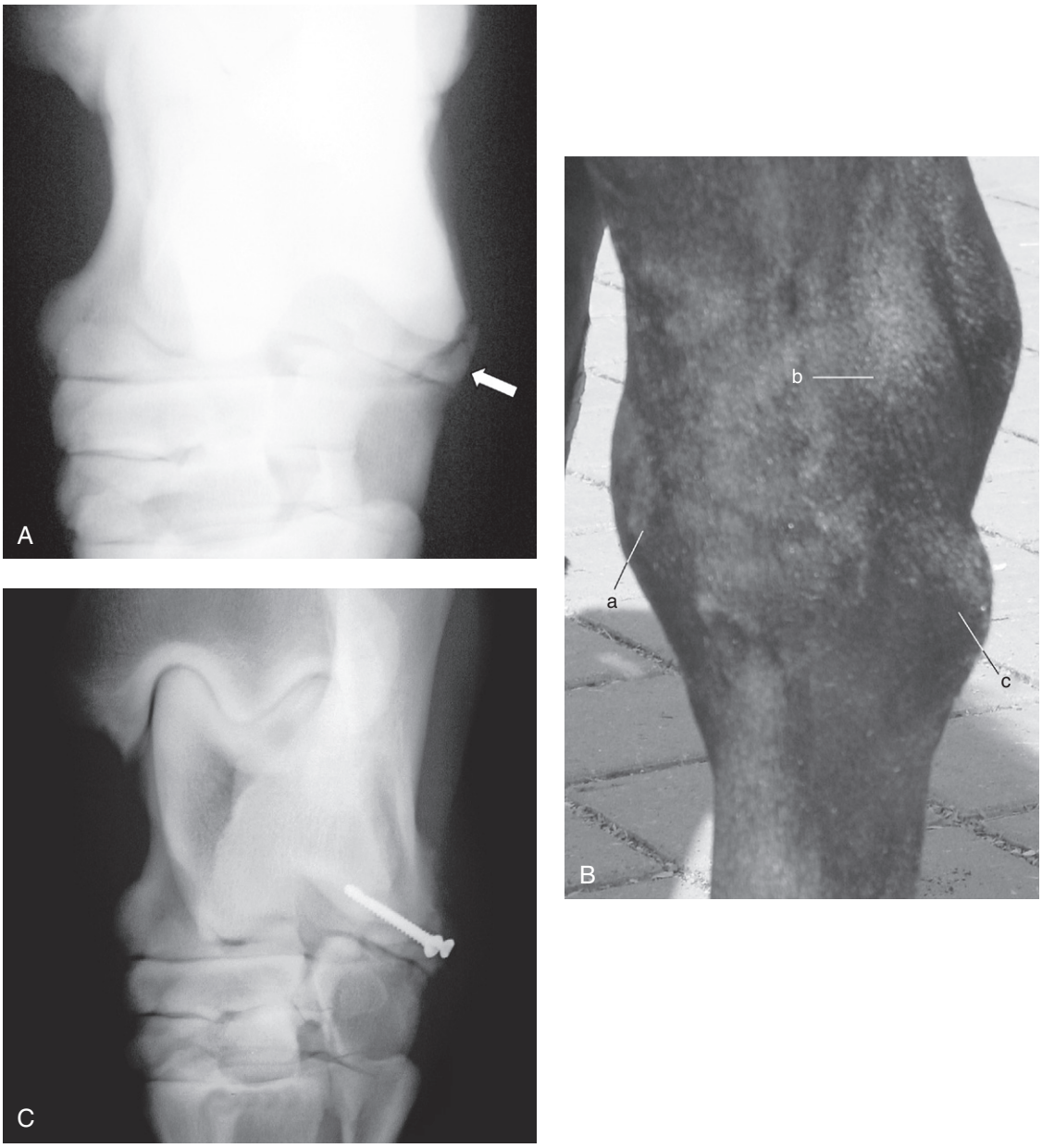

Figure 97-14. A, Avulsion fracture of the lateral aspect of the distal talus with proximal intertarsal (PIT) articular involvement. The horse was managed conservatively with rest for 1 year. **B,** Marked joint distention visible in the tarsocrural and proximal intertarsal joints. *a,* Dorsomedial pouch; *b,* plantarolateral pouch; *c,* lateral aspect of the proximal intertarsal joint. **C,** Postoperative radiographic view showing the fixation of the fracture with two 3.5-mm cortex screws. Six months later, the horse competed successfully in show jumping events.

future soundness. Invariably the bursa gets infected, and eventually the patient has to be destroyed for humane reasons. Aggressive treatment including bursoscopy should be implemented as soon as possible after the injury has occurred and includes meticulous débridement of the wound, removal of fragments, curettage of the fracture bed, lavage of the bursa, broad-spectrum antibiotics for 3 weeks, and placement of the limb in a cast for 3 weeks. Subsequently, a half-shell cast should be applied for an additional 3 weeks, followed by a Robert Jones dressing, provided the horse shows no signs of infection. Infection of the calcaneal bursa is the limiting factor when determining the prognosis in most cases. One study listed a prognosis for survival of 44%,[66] which is rather optimistic in my experience. If signs of infection are present, the wound needs to be evaluated immediately and the owner should be informed of the complication and the potentially grave prognosis. If a decision is made at that time to euthanize the patient, massive costs can be avoided.

Chip Fractures of the Sustentaculum Tali

Fractures of the sustentaculum tali occur rarely. These injuries are caused by acute trauma, mainly in the form of kicking injuries. The skin can be perforated, resulting in communication with the tarsal sheath of the deep digital flexor tendon. The animals are acutely lame and, depending on the duration of the

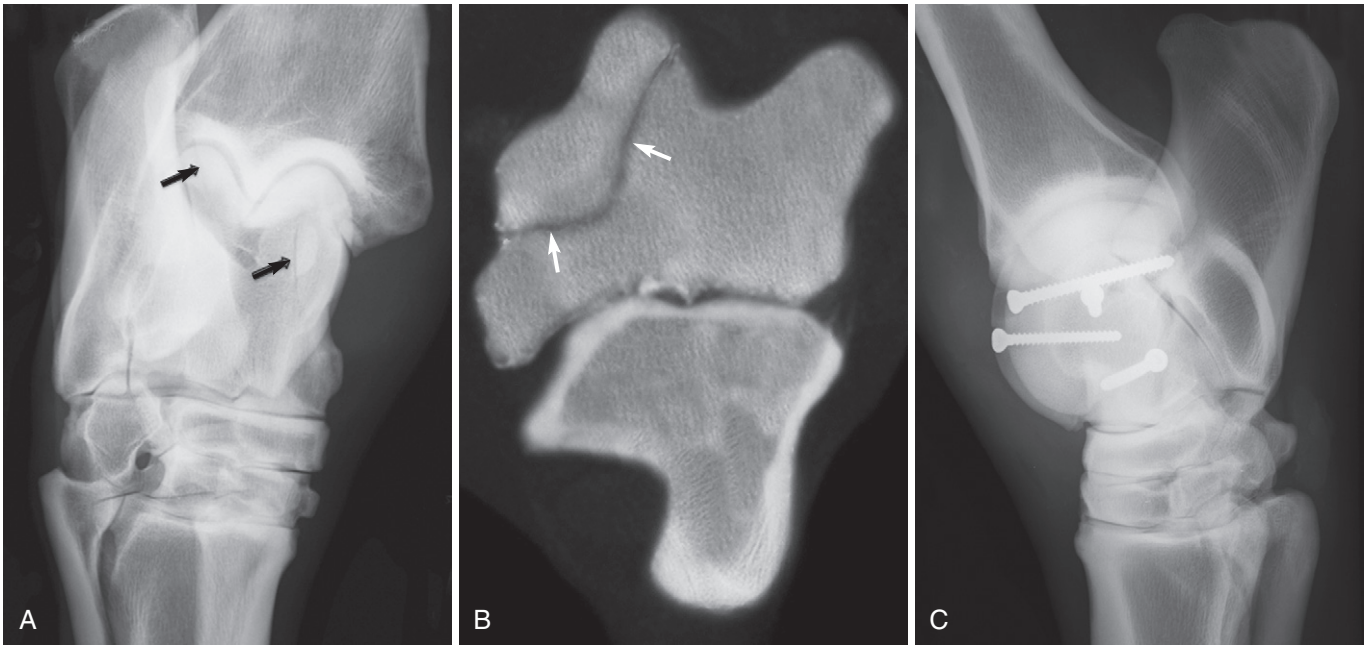

Figure 97-15. A, Dorsolateral-plantaromedial oblique radiographic view of the tarsus of a horse that sustained a sagittal talus fracture. Only two faint fracture lines are visible *(black arrows)* and this does not allow assessment of fracture configuration. The other standard radiographic views were inconclusive. **B,** Transverse intraoperative view generated with the Arcadis orbic three-dimensional C-arm depicting the configuration of the fracture (picture enhanced for better representation *[white arrows]*). **C,** Postoperative lateromedial radiographic view of the tarsus showing the four cortex screws inserted at right angles to each other. Note the screws inserted through the trochlear ridges were countersunk below the articular surface. (Courtesy A. Fürst, Zurich, Switzerland.)

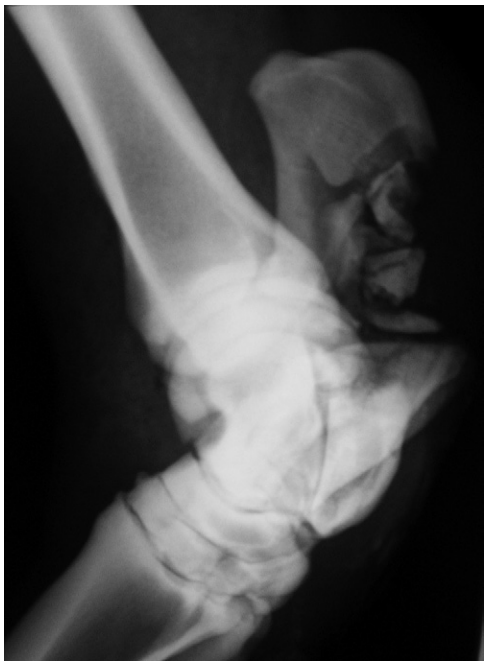

Figure 97-16. Lateromedial radiographic view of the tarsal region of a horse that sustained a mutlifragment fracture of the talus inflicted by a car accident. The mare was euthanatized. (Courtesy A. Fürst, Zurich, Switzerland.)

injury and possibly associated tendon sheath infection, a grade IV lameness may be present.

Diagnosis is by a radiographic examination using multiple views. The proximodistal flexed view (skyline) best delineates the sustentaculum tali (Figure 97-17). Analysis and culture of the tendon sheath fluid is indicated. Treatment should be initiated immediately, especially when the tendon sheath is penetrated. Chip fracture removal can be achieved with tenovaginoscopic technique, although one study suggests that many of the bony lesions might be outside of the tendon sheath and are best approached directly.[67] Flushing of the tendon sheath with copious amounts of fluids is indicated after chip removal and careful curettage of the fracture bed. The open technique of tendovaginotomy and fragment removal represent another surgical approach.

Conservative management is contraindicated, especially in open injuries. Involvement of the sustentaculum is a key parameter in the decision for surgery. If the fracture involves a significant amount of the flexor surface, reattachment with cortex screws may be considered, especially if the tendon sheath was not penetrated at the time of injury. If treatment is delayed, proliferative changes can develop, leading to chronic lameness with a poor prognosis for future usefulness of the horse.

With immediate, effective treatment, a guarded prognosis for return to athletic use can be given. Neglected cases have a grave prognosis. Chip fracture removal can result in postoperative displacement of the deep digital flexor tendon, but this is rare.

Fractures of the Small Tarsal Bones

Slab fractures of the central or third tarsal bones occur after racing or other high-speed events. Fractures of the third tarsal bone are more common. Lameness is severe initially, but it diminishes over several weeks. Marked response to hock flexion persists over time, and persistent effusion of the TC joint is noted if the central tarsal bone is involved. When the central

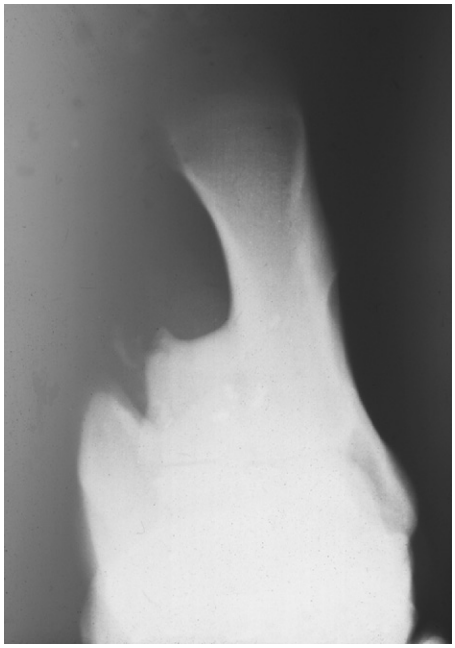

Figure 97-17. Flexed proximodistal (skyline) radiographic view of the sustentaculum tali showing a chip fracture. The fracture was the result of a kicking injury to the medial aspect of the tarsus.

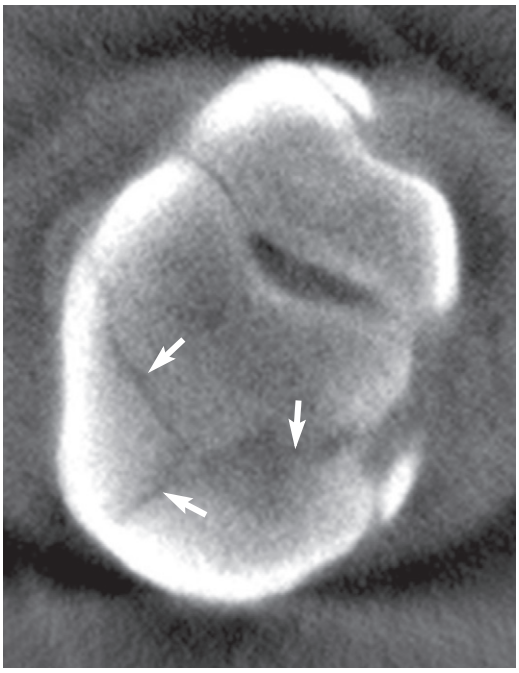

Figure 97-18. Computed tomographic cross section of the central tarsal bone showing a Y-fracture *(arrows)*. (Courtesy A. Fürst, Zurich, Switzerland.)

tarsal bone is fractured, intra-articular anesthesia of the TC joint partially alleviates lameness, but it can take 30 to 45 minutes for maximal effect. Diagnosis of central and third tarsal bone fractures is confirmed with standard radiographic views, but can require multiple oblique projections because central tarsal bone fractures are often complex and comminuted.[68] Radiographic confirmation of these fractures can be difficult. Nuclear scintigraphy and computed tomography are helpful when available (Figure 97-18).[63,69]

Screw fixation of slab fractures of the central and third tarsal bones is the treatment of choice (Figure 97-19). The horse is placed in dorsal or lateral recumbency, and the fracture is compressed using one or two 3.5- or 4.5-mm cortex screws placed in lag fashion through stab incisions using intraoperative radiographic or fluoroscopic control. Conservative treatment (prolonged stall rest) has been successful, and some horses return to athletic use. However, treatment by screw fixation offers the best prognosis, because conservative management usually results in OA.[54,68,70,71] Horses are rested for 4 to 6 months after surgery.

Residual lameness can persist after either surgical or conservative management and eventually requires intra-articular medication, chemical or surgical arthrodesis (see Chapter 81), or screw removal after fracture healing.

Luxations

Luxations usually occur at either the TMT joint or the PIT joint, but they can occur in any of the three distal joints and occasionally involve the TC joint. Lameness is severe, with significant soft-tissue swelling, palpable instability, and crepitus. Luxations and subluxations of the tarsus might reduce spontaneously, but they can still be demonstrated with stressed dorsoplantar radiographs.

Luxations of the tarsus can be associated with fractures. The presence of fractures generally decreases the chances for a successful treatment. Open reduction and internal fixation is mandatory if fractures are present.

Luxations of the distal tarsal joints are best treated primarily with surgical arthrodesis, including débridement of the articular cartilage through an incision (if open, then on the open side of the joint), cancellous bone grafting, and joint stabilization with either transarticular cortex screws placed in lag fashion or bone plates (see Chapter 81).[19] Horses with good temperaments and subluxations can recover for breeding purposes with only stall rest if they are kept tied on an overhead wire or in a rescue net to prevent them from lying down. Horses with luxations of the distal tarsal joints might return to soundness after joint fusion or arthrodesis, but luxation of the TC joint has a poor prognosis for athletic soundness because of joint and ligament damage.[11,72]

Tarsal luxation is diagnosed as an acute traumatic event. The animal is grade IV lame and might show an angular limb deformity in the tarsal region. However, lack of angular limb deformity does not rule out a luxation, because some luxations spontaneously reduce. Diagnosis is derived from palpation and a radiographic examination depicting the exact location of the injury. In cases of reduced luxation, stress views should be taken either standing or under general anesthesia.

Tarsal luxation in the absence of major fractures can be treated with a full-limb cast for 3 months, with several cast changes during that time. This treatment is associated with the usual complications of prolonged cast applications, including pressure sores and cast disease.

Surgical arthrodesis of the TMT, DIT, and PIT joints has achieved superior results. The skin incision extends from the top of the calcaneus to the mid-MTIII on the plantarolateral aspect. The subcutaneous tissues are separated to expose the bone. The head of MTIV is trimmed with a chisel. An alternative

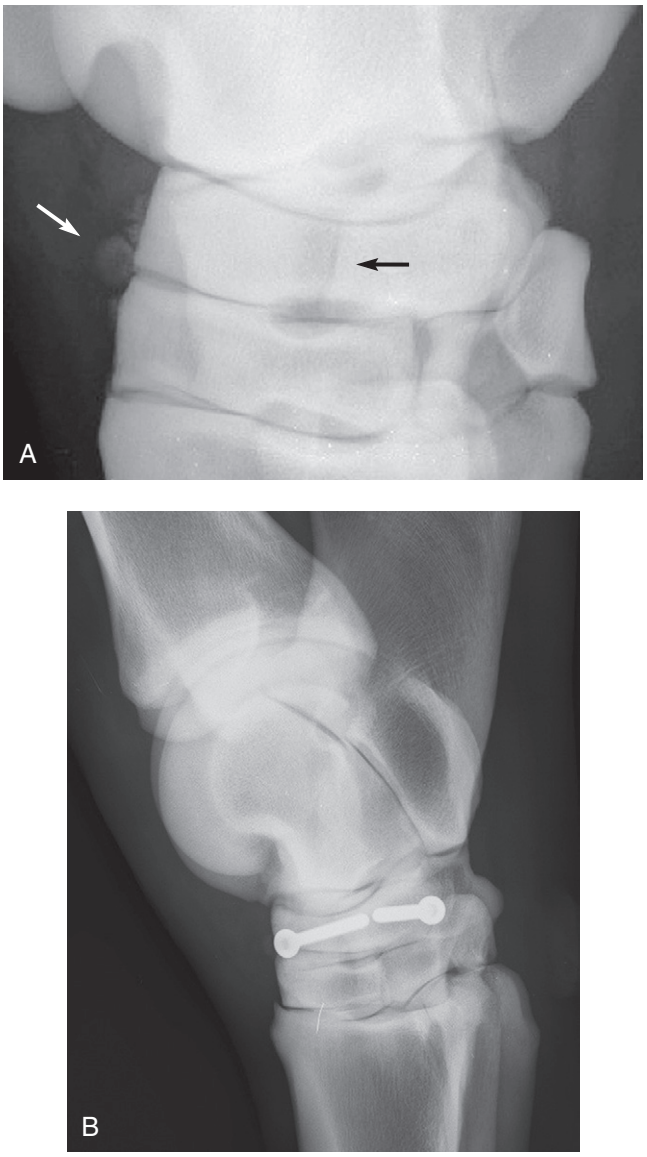

Figure 97-19. A, Preoperative lateromeial radiographic projection of the tarsal region of the same horse shown in Figure 97-15. One clear fracture line can be seen in the central tarsal bone *(black arrow).* Additionally, some small fragments are visible dorsal to the central tarsal bone *(white arrow).* The actual fracture configuration as shown in Figure 97-15 could not be appreciated radiographically. **B,** Postoperative lateromedial radiographic view showing the cortex screws inserted across the main fractures. (Courtesy A. Fürst, Zurich, Switzerland.)

approach involves removing the entire MTIV (see Chapter 81). A 12-hole broad plate is contoured to the bones and applied with 5.5-mm cortex screws (see Figure 97-17). The 5.5 mm LCP, which has superior rigidity to other plates and the added benefit of locking technology, is the recommended plate of choice. Solid purchase of each screw in healthy bone is of great importance with this fixation. Therefore, the direction of each screw through the corresponding plate hole has to be carefully selected using radiographic control. It is important to insert several oblique transarticular screws across the luxated joint to provide additional stability.

After the subcutaneous tissues and skin are closed, a full-limb cast is applied for the initial 2-week period, after which a Robert Jones bandage is applied for 2 to 3 additional weeks. Hand-walking can be started after 2 months, and light riding can be resumed after 3 months. Full recovery is expected, barring no postoperative complications, such as infection or implant breakdown.

Tendon Disorders

Luxation of the Superficial Digital Flexor Tendon

Superficial digital flexor (SDF) tendon luxations are seen in three forms in decreasing frequency: lateral displacement with disruption of the medial retinaculum, medial displacement with disruption of the lateral attachment, or splitting of the SDF tendon with a portion lying on either side of the tuber calcis.[6,33,73,74] The luxations can be partial or complete, with partial luxations causing more discomfort. The etiology can be direct trauma or excessive strain during partial flexion.[72]

Diagnosis of acute SDF tendon luxation can be difficult because of swelling, but palpation and observation during flexion and extension usually allow a diagnosis. Ultrasonography is the diagnostic imaging technique of choice. Sometimes dislocation occurs only during motion, and the tendons maintain appropriate alignment when the limb is at rest. In more chronic cases of permanent luxation, the metacarpophalangeal joint can be hyperextended during weight bearing compared to the other limb. This occurs when the SDF tendon bypasses the tuber calcis in a course that is in a straighter line, which makes it functionally too long.

Partial or intermittent dislocations can heal with stall rest, with or without the benefit of a bandage or full-limb cast. Rest for 4 to 6 months is necessary.[74] Despite a clinical report claiming that conservative management allows a successful return to athletic competition, the general thought is that surgical repair is mandatory for return to athletic function.[36,66,75-77]

The horse is placed in dorsal or lateral recumbency to allow exposure of the damaged side of the tarsal retinaculum. After débridement of the edges, horizontal mattress sutures are preplaced on both sides of the defect well back from the defect margin. The ruptured ends are subsequently apposed and closed with interrupted horizontal mattress sutures of size 0 monofilament absorbable material. A synthetic mesh graft is sutured over the defect using the pre-placed sutures.

Securing the retinaculum to the calcaneus at the original site may be problematic in cases where only a minimal amount of retinaculum is attached to the calcaneus. The use of anchor or suture screws ensures solid fixation (Figure 97-20).

Support of the repair can be provided by placement of bone screws into the calcaneus opposite the tear to provide a buttress against repeated dislocation.[73]

After the incision is closed, the limb is placed in a full-length cast for recovery. Confinement for 4 to 6 months is necessary to allow sufficient fibrosis.

Prognosis with surgical management diminishes as the anticipated level of performance increases. Prognosis for athletic soundness with conservative management is guarded for complete displacement, but it is favorable for cases involving partial rupture of the retinaculum. Medial dislocations have a better prognosis than do lateral displacements.[76]

Displacement of the Deep Digital Flexor Tendon

Luxation or displacement of the deep digital flexor (DDF) tendon is a rare congenital anomaly.[11] In this condition, the DDF tendon is located axial to its normal position and not

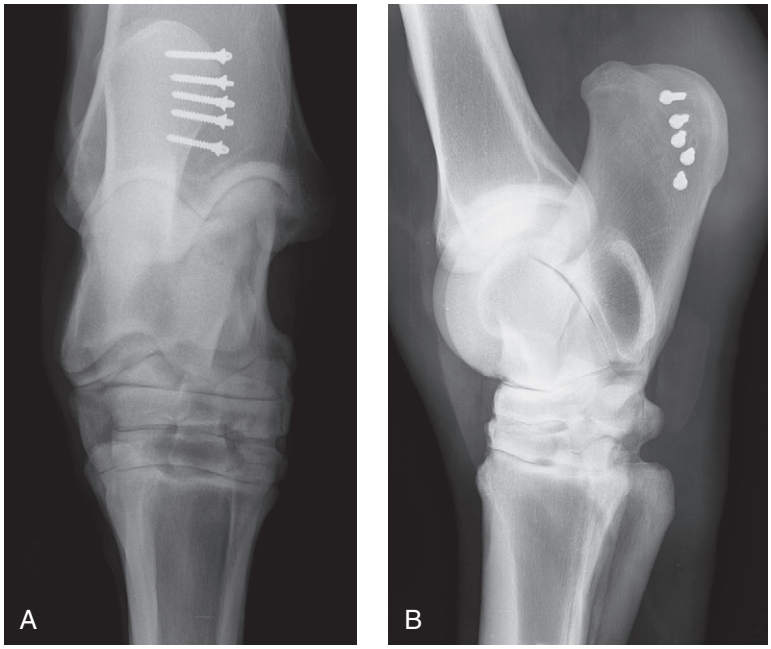

Figure 97-20. Dorsoplantar **(A)** and lateromedial **(B)** radiographic views of the tarsal region showing suture screws inserted into the lateral and proximal aspect of the calcaneus to facilitate the repair of the ruptured retinaculum. (Courtesy A. Fürst, Zurich, Switzerland.)

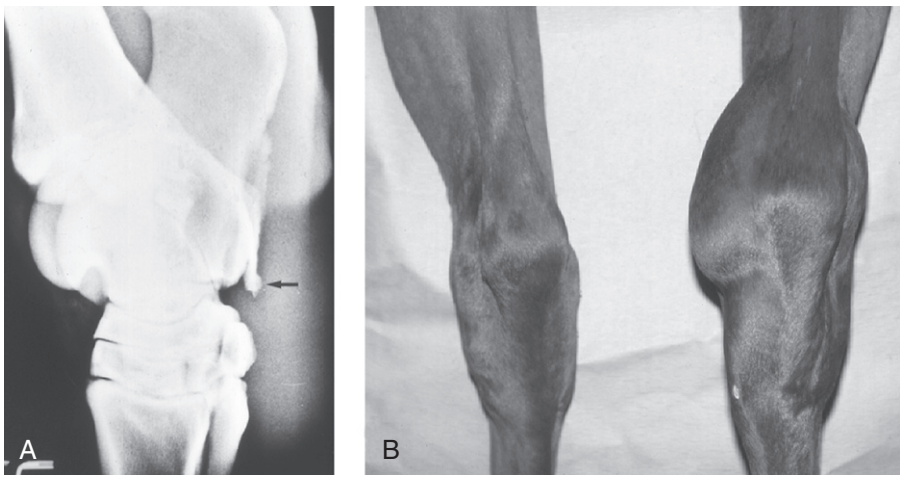

Figure 97-21. A, Lateromedial radiographic view showing osseous proliferation of the distal aspect of the sustentaculum tali. These lesions are associated with chronic irritation and inflammation of the sheath of the deep digital flexor tendon. **B,** Massive distention of the sheath of the deep digital flexor tendon medially in the tarsal region of a 6-year-old gelding. (**A,** Courtesy R. Welch.)

within the groove formed by the sustentaculum tali. The malpositioned DDF tendon exerts a medial pull on the distal limb, and the first recognizable clinical sign is a varus deviation of the tarsus noted at 2 to 4 weeks after birth. Diagnosis is based on palpation, ultrasonography, and skyline radiography of the tarsus that demonstrates hypoplasia of the sustentaculum tali. Surgery to replace the DDFT in its normal position and augment the sustentaculum tali has been recommended[11] but does not seem feasible, because it is very unlikely that the foal will grow up to be a functional athlete.

Tenosynovitis of the Deep Digital Flexor Tendon

Chronic irritation and tenosynovitis of the DDF tendon can be a sequela to sustentaculum tali injury. Radiographically, new bone proliferation (Figure 97-21, *A*) can be seen in the region, leading to tendon sheath distention (Figure 97-21, *B*) and possible ossification. Contrast tenography can provide valuable information on the pathology present within the sheath.

Treatment of these problems involves tenoscopic exploration of the pathology and removal of any proliferative changes. Tendovaginotomy represents an alternative approach. However, chronic degenerative changes of the sustentaculum tali and overlying DDF tendon have a guarded prognosis for resuming normal work. After the skin incision is closed in routine fashion, a pressure bandage is applied and maintained for 3 weeks. Intratendovaginal hyaluronan 1 week postoperatively is indicated.

Avulsion of the Gastrocnemius

Rupture of the gastrocnemius muscle occurs occasionally in foals and young horses. The disorder can be caused by external trauma or excessive exercise in the field, possibly associated with slipping or sudden unexpected turns. The diagnosis is straightforward, because the reciprocal apparatus is not

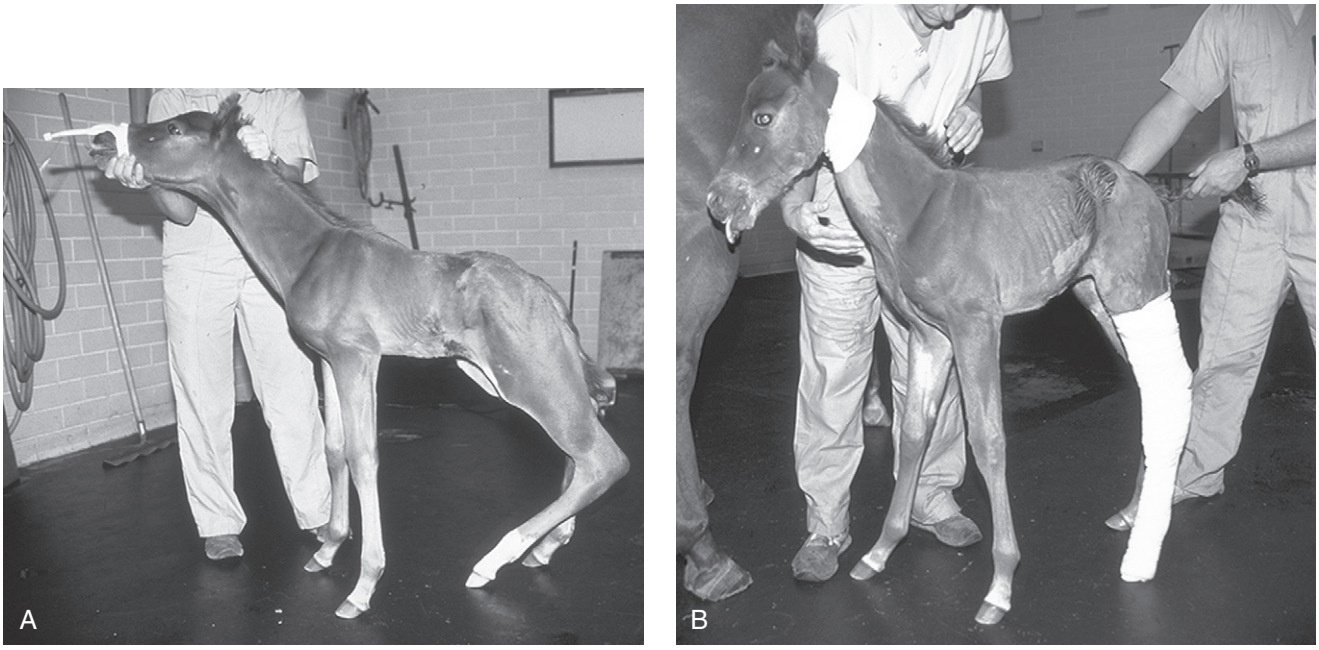

Figure 97-22. Three-week-old Quarter Horse foal with an avulsion of the gastrocnemius muscle. **A,** Weight bearing results in marked flexion of the tarsus and hyperextension of the stifle. **B,** Application of a tube cast over the tarsal region allows the foal to bear weight.

functional. As the animal places weight on the limb, the tarsus collapses distad, and the stifle joint becomes hyperextended (Figure 97-22, *A*).

Ultrasonography usually finds the gastrocnemius tendon intact but the origin of the muscle avulsed from the distal and caudal aspect of the femur. The avulsion can be complete or partial. Partial avulsion causes only slight dropping of the tarsus during weight bearing.

Management involves maintenance of a full-limb Robert Jones bandage with an incorporated splint for several weeks. An alternative treatment is application of a tube cast over the tarsal region (Figure 97-22, *B*) for 3 weeks, followed by a Robert Jones bandage for an additional 3 to 5 weeks.

Both partial and complete avulsions have a favorable prognosis for a competitive future. Complications include all problems encountered with long-term casting and bandaging of the tarsal region.

In a retrospective study, the medical records of 28 foals admitted because of disruption of the gastrocnemius tendon were reviewed.[78] Seventeen of these foals (61%) presented with a concomitant illness. Foals without a concomitant illness were more likely to achieve race training or to start a race ($p = 0.04$). Twenty-three of 28 foals were short-term survivors (discharged from the clinic). Of this population, 82% of the foals were able to achieve race training or start a race.[78]

DEVELOPMENTAL DISORDERS OF THE TARSAL REGION

Osteochondrosis

Etiology

Osteochondrosis (OC) in the equine tarsus occurs most commonly in the distal intermediate ridge of the tibia (Figure 97-23, *A*), followed by the lateral trochlear ridge of the talus (Figure 97-23, *B*), the medial malleolus of the distal tibia, the medial trochlear ridge of the talus, and the lateral malleolus of the distal tibia.[79-81] Subchondral cystic lesions (see Chapter 89) have been identified in the distal tibia and talus, but they are considered unusual.[82] These lesions also have been identified in the cuboidal bones of the distal tarsus and are associated with an inflammatory reaction.[12,83]

Osteochondrosis results from failure of endochondral ossification or loss of blood supply to immature cartilage and subchondral bone and can be influenced by various factors, including exercise, hormone imbalances, nutrition, heredity, and conformation (see Chapter 88).[75,84-89] Heritability estimates suggest that 25% to 52% of the variation in occurrence of OC in the hock can be attributed to genetic factors.[90-94] Others conclude that distal tibial lesions arise from biomechanical trauma early in life or represent supernumerary ossification centers that are not part of the OC complex.[80,95] One study documented 11 of 77 foals with obvious distal tibial fragments that were radiographically normal by 8 months of age.[96]

Diagnosis

Signs of tarsocrural OC are usually associated with the onset of training.[93,97] Synovial distention is the most common manifestation of the condition, sometimes followed by mild lameness (identified in 66 of 144 affected limbs).[91] Pain on flexion of the tarsus is usually mild, and synovial fluid analysis is rarely indicated. Four standard radiographic views of the tarsus should be taken, but flexed views may also be useful to demonstrate loose fragments or osteochondral defects in the caudal aspect of the joint. Bilateral lesions involve the distal tibia 20% to 45% of the time.[88,98] Distal lesions of the tibia vary dramatically in size, ranging from small fragments less than 5 mm in diameter to involvement of the entire intermediate ridge. OC lesions can be identified arthroscopically without radiographic evidence of

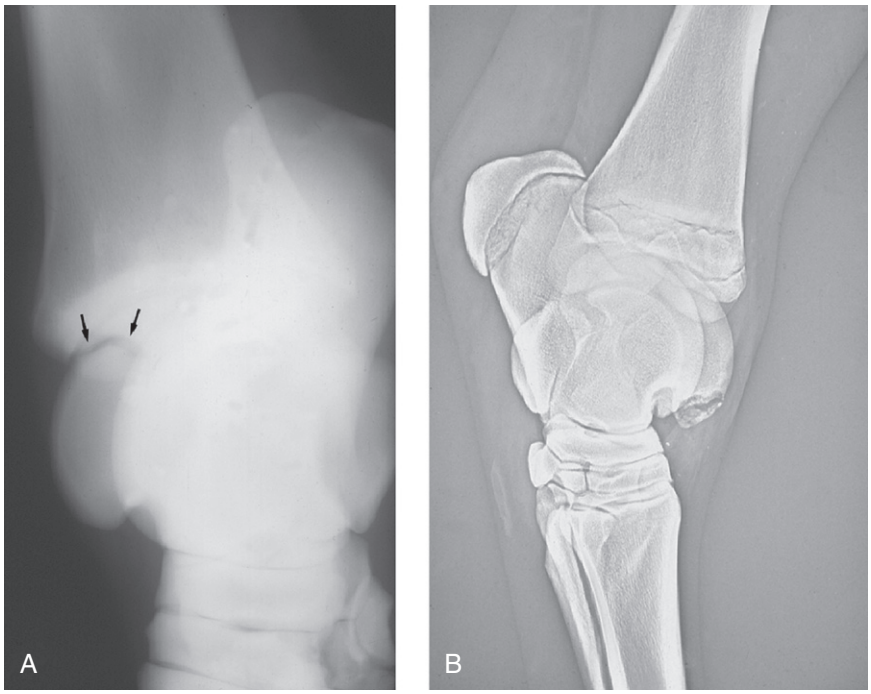

Figure 97-23. A, Dorsomedial-plantarolateral oblique (DMPLO) radiographic view of the tarsus showing a distal intermediate ridge of the tibia osteochondral (OC) lesion *(arrows).* **B,** DMPLO xeroradiographic view of the tarsus showing a lateral trochlear ridge OC lesion. (Courtesy C. W. McIlwraith, Colorado State University.)

their presence. These horses might or might not be presented with synovial effusion.[79]

Treatment

Arthroscopic removal of tarsocrural OC fragments is the treatment of choice, particularly if clinical signs of effusion or lameness are noted. Horses without clinical signs associated with radiographic evidence of OC can develop osteoarthritis of the TC and PIT joints and linear erosions along the side walls of the trochlear groove of the talus later in life.[4] Arthroscopic examination of the TC joint in mature horses with OC often reveals fibrillation of articular cartilage. These findings suggest that early surgical intervention is warranted to minimize secondary joint changes.[12]

DISTAL INTERMEDIATE RIDGE OF THE TIBIA

The horse is placed in dorsal recumbency with the limb in slight flexion. The arthroscope portal is made on the dorsal aspect of the dorsomedial pouch of the tarsocrural joint, centered over the pouch (Figure 97-24). Placement of the arthroscopic portal too far medial makes it difficult to pass over the medial trochlear ridge, and placement too proximal or too distal also will jeopardize visualization. It is best to identify the saphenous vein before or after synovial distention and avoid it.

After the joint is distended, a stab incision is made through the synovial membrane, and the arthroscopic sleeve is inserted into the joint with a conical obturator. The limb is placed in 90 degrees of flexion, the arthroscope is inserted, and the joint is explored (Figure 97-25).

The location of the instrument portal can be identified by passing the arthroscope laterally over the proximal aspect of the exposed lateral trochlear ridge into the dorsolateral pouch and palpating the tip through the skin. This procedure is also useful to demonstrate a vessel in the area by backlighting it with the

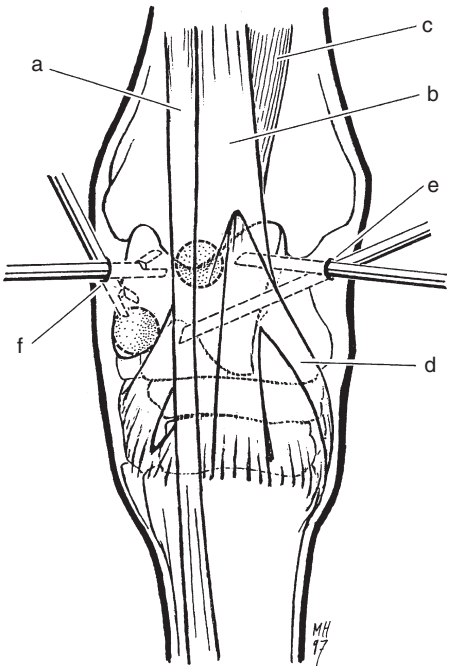

Figure 97-24. Graphic illustration of arthroscope and instrument portals for removing distal intermediate ridge OC lesions and lateral trochlear ridge OC lesions. These portals are also useful for diagnostic arthroscopy of the dorsal aspect of the tarsocrural joint and for removing fragments from the proximal intertarsal joint. *a,* Long digital extensor tendon; *b,* fibularis tertius; *c,* tibialis cranialis; *d,* medial branch of the tibialis cranials or cunean tendon; *e,* dorsomedial arthroscopic portal; *f,* dorsolateral instrument portal.

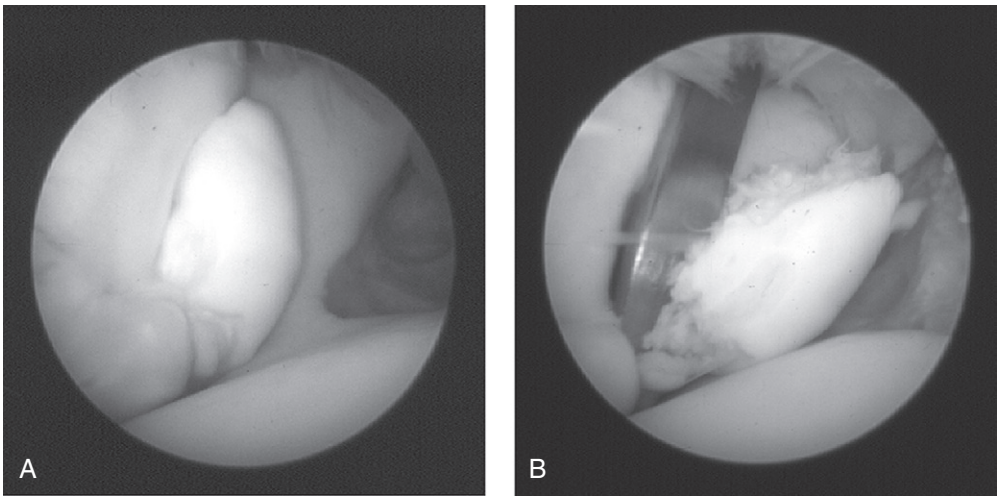

Figure 97-25. A, Arthroscopic view of a distal intermediate ridge (OC) lesion. **B,** Partial separation of the OC fragment from the distal tibia with the help of a periosteal elevator. These lesions are attached in most cases to the tibia by fibrous connective tissue.

arthroscope when the surgery room lights are dimmed. Alternatively, a needle can be inserted through the dorsolateral pouch and advanced until it makes contact with the lesion.[16] Insertion of a hypodermic needle at the intended site allows the exploration of the range of motion possible with the instrument.

When the ideal location is determined, a skin incision is made along the needle. A No. 11 scalpel blade is advanced into the joint along the needle, and a small incision is made into the synovial membrane. The needle and scalpel are removed and immediately exchanged with the arthroscopic probe. Intermediate ridge fragments may be loosely attached and elevated with the probe or may be firmly attached to the distal tibia, requiring partial separation with a periosteal elevator (Figure 97-25, *B*) or an osteotome and mallet. Care should be taken not to damage the articular cartilage on the lateral trochlear ridge with the elevator or osteotome. After elevation, fragments are grasped with rongeurs, rotated to divide any remaining attachments, and removed. The surgeon should grasp fragments so that the minimum diameter is presented at the instrument portal. Increasing the size of the instrument portal is often necessary for removal. After the fragments are removed, the OC bed is examined for remaining tissue and curetted until firm subchondral bone remains in the site.

Occasionally, fragments of the intermediate ridge detach from the parent portion of the bone and become located in the distal end of the joint on preoperative radiographs (Figure 97-26). Because these fragments can be free within the joint cavity or can pass into the PIT, removal can be problematic. If the fragments are free, they fall to the most distal aspect of the joint (which is into the dorsal aspect of the pouch [proximal] when the horse is in dorsal recumbency), especially if fluids are used to distend the joint. If fragments are located in the PIT, they can be left alone with minimal consequences or removed using a probe (see later). (The arthroscope can be passed into this joint through the normal communication just axial to the medial trochlear ridge.) Gas distention of the joint can aid in locating the fragment; however, if it cannot be localized intraoperatively, radiographs or fluoroscopy should be used to localize it. The instrument portal is selected in a place where the fragment can be accessed, and the fragment is removed.

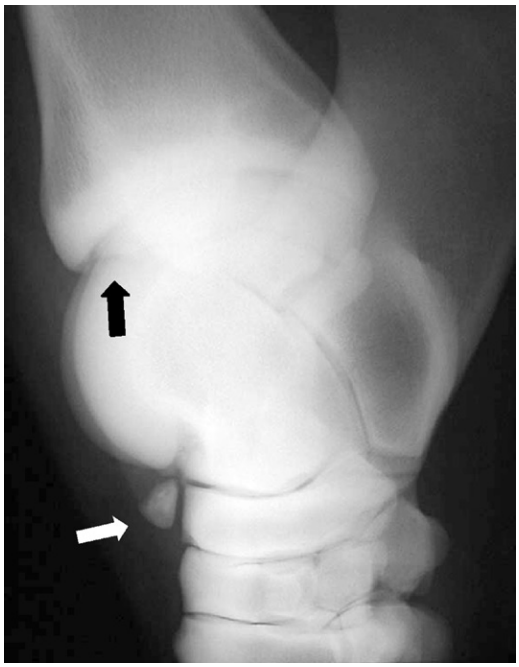

Figure 97-26. Lateromedial radiographic view of a tarsal region showing a loose fragment at the distal aspect of the joint cavity *(white arrow).* It is most likely located in the PIT. The origin of the fragment is the distal intermediate ridge of the tibia *(black arrow).*

MEDIAL MALLEOLUS

The arthroscope portal for medial malleolus lesions is made more ventrad (more distad on the limb) than for intermediate ridge lesions to minimize the risk of accidentally pulling the arthroscope out of the joint during visualization.[16] The instrument portal (also ventromedial) is identified by prior insertion of a needle. Techniques for removing fragments are similar to those used for other lesions in the TC joint.

LATERAL TROCHLEAR RIDGE

The arthroscope portal for removing lateral trochlear ridge lesions can be either medial or lateral. The lateral portal offers better visualization of the distal aspect of the lateral trochlear ridge, but the standard medial portal is preferred because triangulation is better and the surgeon can carry out a more thorough exploration of the joint from a medial approach.[16] The instrument portal is always lateral and is usually slightly distal to that used for distal tibial lesions. Both distal intermediate ridge and lateral trochlear ridge lesions can be treated with a single instrument portal when found concurrently within a single joint (see Figure 97-24). Osteochondral defects or cartilage flaps can extend proximad along the lateral trochlear ridge, necessitating extension of the limb during surgery. Some surgeons prefer an arthrotomy approach directly over the lesion, but it is not the recommended approach.[11,32]

PROXIMAL INTERTARSAL JOINT

Loose fragments, usually shed from the intermediate ridge of the distal tibia, can lodge in the dorsal pouch or dorsal joint capsule of the PIT joint (see Figure 97-26).[33] Although rarely of clinical significance, these fragments can be removed, if they are loose, by arthroscopically inserting an instrument into the pouch to blindly grasp the fragment or manipulate it into the TC joint for removal. Because of their limited clinical significance and the relative difficulty of finding and removing the fragments, it has been recommended that they be removed only if this can be easily accomplished during surgery for the primary lesion of the TC joint.[99]

Aftercare

Stab incisions are closed with skin sutures only. Bandages are applied and maintained for 14 days after surgery and are changed as necessary. Sutures are removed 10 days after surgery. Exercise is restricted to hand-walking for 4 to 8 weeks, and training can be resumed in 8 to 16 weeks, depending on the severity of the lesion and the amount of synovial distention before and after surgery. Postsurgical administration of phenylbutazone, hyaluronan, or PSGAG is useful to help resolve effusion, particularly in horses with prolonged synovial distention before surgery. Synovial effusion of the tarsocrural joint can recur within a few days after turnout, and this can be resolved by returning the horse to stall rest under bandage and administering NSAIDs for 10 days.

Prognosis

It is generally accepted that the prognosis for athletic soundness is improved with surgery compared to conservative management.[75,80,87,100,101] Seventy-seven percent of horses with OC of the tarsus raced successfully or performed their intended function following arthroscopy.[79] However, the prognosis decreases when articular cartilage degeneration or erosion is noted at the time of surgery. In one study, resolution of synovial effusion after arthroscopy was higher in racehorses (83%) compared to nonracehorses (74%), and it was more likely with lesions of the distal tibia compared with other locations.[79] There was no significant relationship between resolution of the effusion and successful performance outcome.[79]

Reports on the effects of OC on racing performance are contradictory. Although one study found no difference in the racing performance of Standardbreds demonstrating OC lesions compared with a radiographically normal control population,[102] another study reported that horses with OC of the TC joint had significantly fewer starts and lower earnings compared with horses without radiographic changes.[103] A third study found that the number of racing starts and race earnings for horses with surgically treated tarsocrural OC was reported to be lower than for age-matched siblings,[104] but a fourth study found that horses treated arthroscopically for OC of the distal tibia performed no worse than did their matched controls.[105] Despite these reports, I recommend surgical treatment of this condition in all types of horses to prevent the development of OA in the TC joint.

Angular Limb Deformities

Angular limb deformities of the tarsus are seen less often than in the carpus or forelimb fetlocks, but they are diagnosed and treated in a similar fashion. Tarsus valgus is by far the most common presentation and can be caused by ligamentous laxity, malformation or incomplete ossification of the cuboidal bones of the tarsus, physeal dysplasia, or uneven growth of the distal tibial physis. A complete discussion of the etiology, diagnosis, and treatment of angular limb deformities can be found in Chapter 86.

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CHAPTER 98

Tibia

Lawrence R. Bramlage

ANATOMIC CONSIDERATIONS

The osseous structures of the gaskin region of the horse include the tibia and fibula. The tibia is a strong tubular bone that has a triangular-shaped cross section proximally that changes to an oval shape oriented lateromedially as it courses distad.[1] The tibia is the major weight-bearing bone of the gaskin and transfers forces from the femur to the talus. The tibial crest, located at the proximal cranial end of the bone, contains a deep muscular sulcus, which is filled by the cranial tibial and long digital extensor muscles. The proximal articular surface of the tibia has a flat table surface with a centrally located intercondylar eminence. The distal articular surface has an undulating shape and contains two deep sulci oriented in caudomedial-to-craniolateral direction, which articulate with the trochlear ridges of the talus. For details on the complex soft tissue attachments between the tibia and both the femur and the tarsus, see Chapters 97 and 99.[1]

The fibula is a rudimentary bone of variable shape and completeness. In most cases the fibula is incomplete and consists of several centers of ossification that can be mistaken for fibular fractures.[1] Rarely, an atavistic complete fibula is identified radiographically in a pony or Miniature Horse foal (see Figure 86-21). The pathology associated with this condition creates limb deformities and tarsal osteoarthritis (see Chapter 86). Other than traumatic injury, this is the only condition in which lameness is associated with the fibula.

Other than lacerations, the most common injuries of the tibial region are fractures. Stress fractures in the tibia in training horses are the most common. In addition, traumatic fractures of the tibia in the horse occasionally occur. Two common types

of unstable tibial fractures are seen: the proximal physeal fracture and the diaphyseal fracture.

CLINICAL DISORDERS OF THE TIBIAL REGION

Tibial Stress Fractures

Etiology and Diagnosis

Tibial stress fractures are most frequently diagnosed in unraced Thoroughbreds in training.[2] Horses with stress fractures of the tibia have acute marked lameness that often resolves quickly between exercise periods and then reappears with the next strenuous exercise.[2] Pain can rarely be elicited with palpation and it is not practical to use diagnostic local anesthesia, other than to eliminate other causes of lameness. The diagnosis is made radiographically or with nuclear scintigraphy.[2] A discrete fracture is rarely seen in tibial stress fractures. Most often there is endosteal and periosteal callus, if the stress fracture is of sufficient duration.[2] The lack of significant callus in the presence of severe reoccurring lameness is an indication for nuclear scintigraphy. Nuclear scintigraphy is diagnostic if marked focal disphyseal or metaphyseal uptake is identified. Stress fractures only occur in heavily trained athletes. They are most commonly diagnosed in the distal metaphysis (Figure 98-1) or the caudal diaphysis (Figure 98-2), but proximal posterior lateral stress fractures are not unusual (Figure 98-3).[2]

Treatment

Treatment is cession of exercise. Stall rest with hand walking is allowed until the horse can trot sound in hand.[2] At that point, it is safe to allow paddock exercise. Occasionally acute stress fractures become complete fractures, even in the absence of significant radiographically identifyable fractures. The use of anti-inflammatory medication greatly increases the chances of complete fracture and so should be avoided. If horses are restricted and the lameness is not mitigated with medication, most horses protect the limb sufficiently to allow healing. Healing takes a minimum of 2 months. Follow-up radiographs are taken 60 to 90 days after diagnosis. Soundness

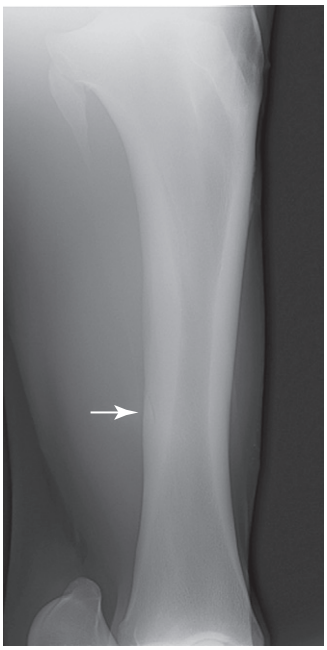

Figure 98-2. An APLMO radiographic view of a tibia with a mid-tibial stress fracture. The stress fracture can actually be seen in this horse *(arrow)*, which is unusual because callus normally obscures the fracture.

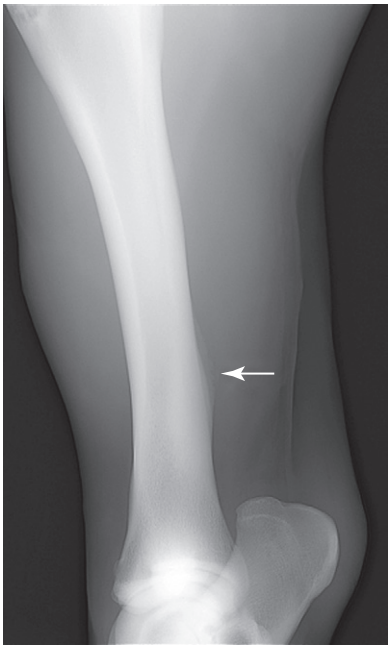

Figure 98-1. An anteroposterior lateromedial oblique (APLMO) radiographic view of the tibia showing the periosteal and endosteal callus *(arrow)* that accompanies a caudolateral tibial stress fracture.

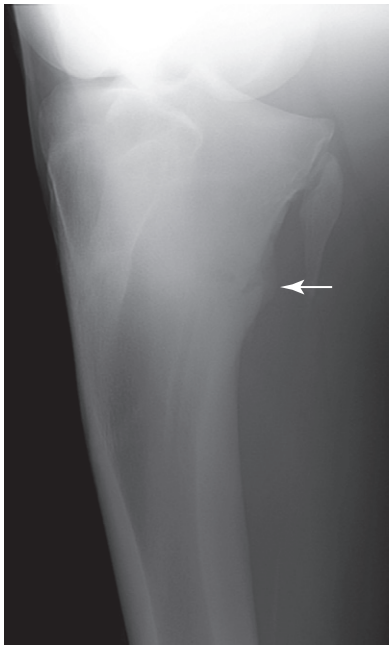

Figure 98-3. An APLMO radiographic view of the proximal tibia, which shows a chronic stress fracture of the proximal caudal tibia with associated callus *(arrow)*.

and radiographic healing/remodeling of the callus indicate the horse can resume training.

Fissure Fractures of the Tibia

Clinical Signs and Diagnosis

In most cases, fissure or incomplete fractures result from a kick from another horse or blunt impact from a solid object. Horses with incomplete fractures or with nondisplaced complete fractures do not demonstrate instability or crepitation. However, they are usually lame, and in many cases, a wound is evident at the site of traumatic impact.[3]

Usually, radiographic evaluation confirms the presence of a fracture (Figure 98-4). Multiple views, including oblique projections, are advisable to delineate the severity of the fracture and to assist in determining a course of action.

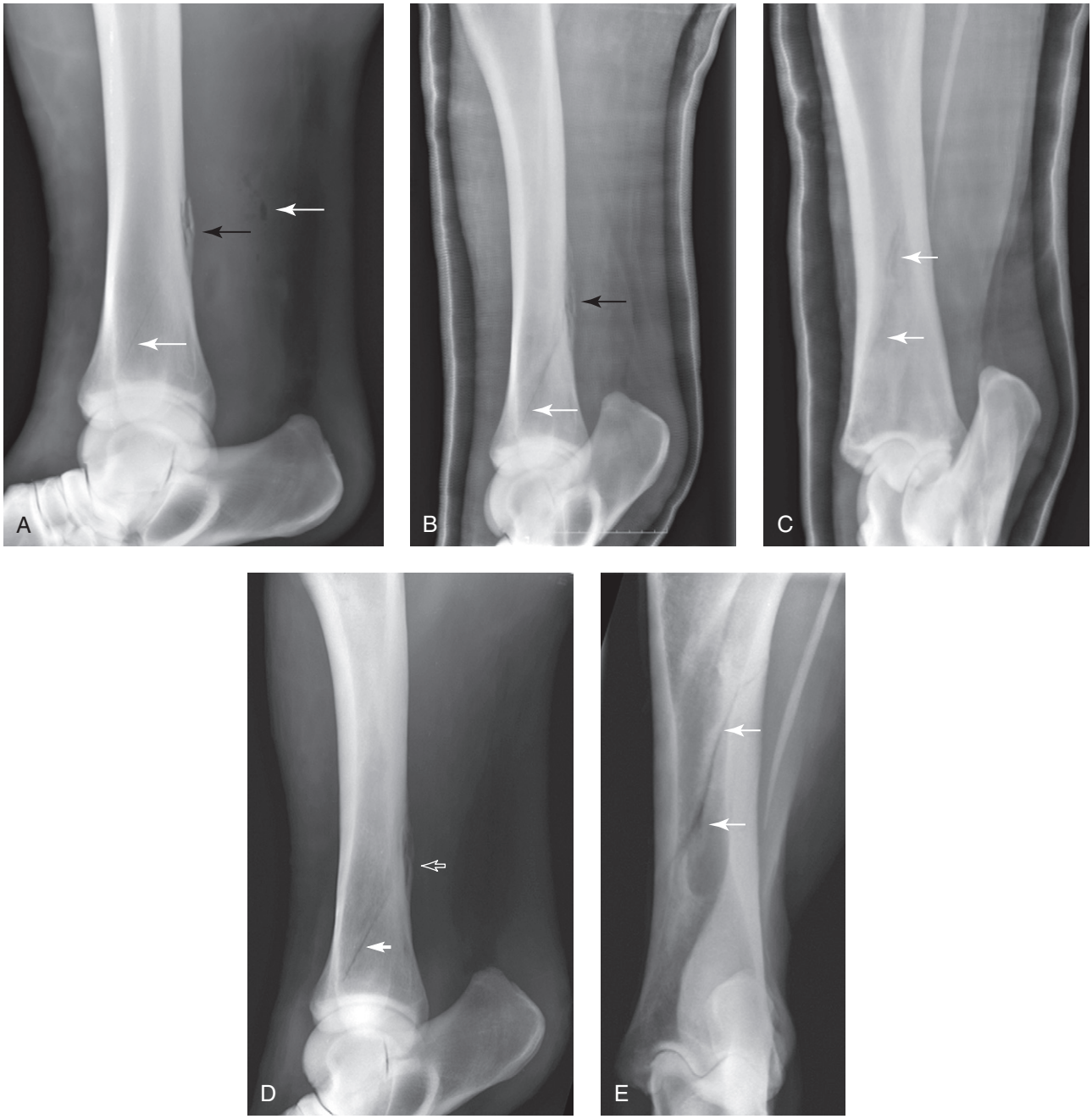

Figure 98-4. A, Lateromedial radiographic view of a traumatic fissure fracture in the distal tibia *(lower white arrow).* Some small fragments are visible at the caudal aspect of the bone *(black arrow).* There are air pockets in the soft tissues *(upper white arrow).* **B,** Thirty-day follow-up lateromedial radiographic view. The limb was placed in a fiberglass cast. The original fissure fracture is still visible *(white arrow)* and some remodeling is taking place at the caudal aspect of the tibia *(black arrow).* **C,** An APMLO radiographic view taken at the same time as **B** shows a proximal propagation of the fissure fracture *(arrows).* **D,** Sixty-day follow-up lateromedial radiographic view of the fissure fracture showing marginal healing distally *(white arrow)* but bone bridging the caudal cortex of the tibia *(open arrow).* **E,** A caudocranial radiographic view taken at the same time as **D** shows additional proximal propagation of the fissure fracture *(arrows)*. The horse was discharged from the hospital at this time and made a full recovery. (Courtesy M. Kummer, University of Zurich.)

Treatment

Incomplete and nondisplaced complete fractures are candidates for conservative therapy.[3] In one report, incomplete tibial fractures became complete displaced fractures in four of 10 horses using conservative management.[3] One of these fractures resulted from a kick to the stifle region. Therefore, only this region was radiographed and no lesions were detected. The horse was found 16 days later with a fractured tibia.[3] Because an important aspect of therapy is preventing recumbency to reduce the likelihood of displacement secondary to forces experienced during standing up, detection of incomplete fractures is necessary for successful outcome (see Figure 76-1).

Phenylbutazone should be used judiciously to reduce inflammation and encourage limited weight bearing on the fractured limb. However, care should be taken to avoid providing substantial pain relief, which could result in overuse of the fractured limb.

If a wound is present, it should be explored and treated using routine wound care. The indication for bandages is dictated by the configuration of the fracture and the extent of the wound. Parenteral antimicrobial treatment is indicated in horses presenting with a wound. Incomplete and fissure fractures of the distal metaphysis of the tiba may profit from a cast, which is best applied in the sedated standing horse in a weight-bearing position.

The horse should be confined until there is evidence of advanced fracture healing (see Figure 98-4, *B* through *E*). This usually requires a minimum of 3 to 4 months. During the last 30 days of confinement, a program of gradually increasing hand-walking is recommended. Access to free paddock exercise is allowed thereafter. An additional 30 days of paddock exercise is recommended before the horse is gradually returned to its intended function.

Proximal Physeal Fractures

The Salter-Harris type II proximal physeal fracture of the tibia is the most common tibial fracture diagnosed in foals (Figure 98-5, *A*). It most often occurs when the foal is kicked from the lateral aspect on a weight-bearing rear limb. The soft tissue of the medial proximal tibial physis ruptures because of the tension created on the medial aspect of the limb when the traumatic bending force occurs. The fracture propagates across the tibial physis from medial to lateral approximately two thirds the width of the bone, at which point the medial bending force on the distal limb distracts the physis, changing the fracture plane orientation from transverse to vertical, leaving the lateral physis intact and a triangular metaphyseal fragment attached to the epiphysis (see Figure 98-5, *A*).

With this fracture, the periosteal soft tissue is normally intact laterally but disrupted medially. Occasionally, complete disruption of the soft tissue surrounding the physis occurs, rendering the fracture more unstable and creating more displacement.

Preoperative Considerations

The biomechanical force that creates the fracture is a tension force medially; therefore, fracture instability is best neutralized by re-establishing support on the medial aspect of the limb. This requires an implant that maintains the proximal tibial epiphysis in a stable position on the metaphysis. Because normal bone formation, as part of bone growth, is already active in the growing physis, development of the vascular support system for fracture healing is unnecessary. Therefore, the fracture heals rapidly if it is stable, normally in 3 to 4 weeks.

Screw fixation, pinning, and external fixation have been described for treating these fractures, and plate fixation has been used on the lateral aspect of the tibia.[4-8] The biomechanically most stable fixation re-establishes the tension support on the medial aspect of the limb, a function that was previously provided by the soft tissue of the physis. If the lateral soft tissue is intact, stability is relatively easy to obtain. If the epiphysis is completely disrupted from the metaphysis, more elaborate fixation with more implants is required. The most helpful adjunct to the medial plate is the addition of a tension band to stabilize the tibial crest. It will neutralize the distracting forces of the quadriceps muscle that is transmitted across the stifle joint to the tibial crest via the patella and the patellar ligaments' attachment to the tibial crest.

Although this fracture is usually closed, soft tissue damage occurs medially at the site where implants must be inserted. The close implant-to-skin location makes sepsis more difficult to avoid. Broad-spectrum antimicrobial agents and anti-inflammatory medications are administered preoperatively.

Treatment

The treatment of choice for proximal physeal fractures of the tibia in foals is medial plate fixation.[7] The foal is positioned in either dorsal or lateral recumbency. When it is placed in lateral recumbency with the injured limb down, downward pressure on the proximal tibial metaphysis facilitates reduction. The opposite rear limb is flexed, abducted, and tied vertically to prevent it from interfering with surgery on the medial aspect of the injured limb.

The approach to the proximal aspect of the tibia for medial plate application is along the caudal margin of the tibia. The soft tissues are elevated craniad, and the plate is placed on the medial aspect of the tibia. The close apposition of the tibia to the surface of the skin with no interposed muscle on the medial aspect of the limb complicates wound healing if the skin incision lies directly over the implant. Therefore, a slightly curved incision is initiated distally on the caudomedial aspect of the limb and extended proximad across the proximal medial tibia. The proximal tibial physis is thin, and screws must be inserted sufficiently proximal to the growth plate to allow purchase in the bone but without endangering the articular surface. Placement of long needles in the plane of the proximal tibial physis permits orientation to the plane of the physis during drilling for screw placement.

Gentle traction on the physis and elevation of the metaphyseal fragment separates the fracture plane and allows careful débridement. Removal of small fragments and blood clots permits accurate reduction of the physis. Damage to the growth cartilage of the physis is normally induced by the metaphysis near where the fracture plane changes from transverse to vertical. Evaluation of the physeal cartilage provides information on the likelihood of survival of proximal tibial growth plate function. The amount of damage depends on the time from injury to surgery and the adequacy of preoperative immobilization.

After the debris is removed from the fracture, the soft tissue must be elevated along the medial aspect of the proximal tibial physis. If this soft tissue is not elevated, it becomes trapped between the metaphyseal fragment and the physis, preventing

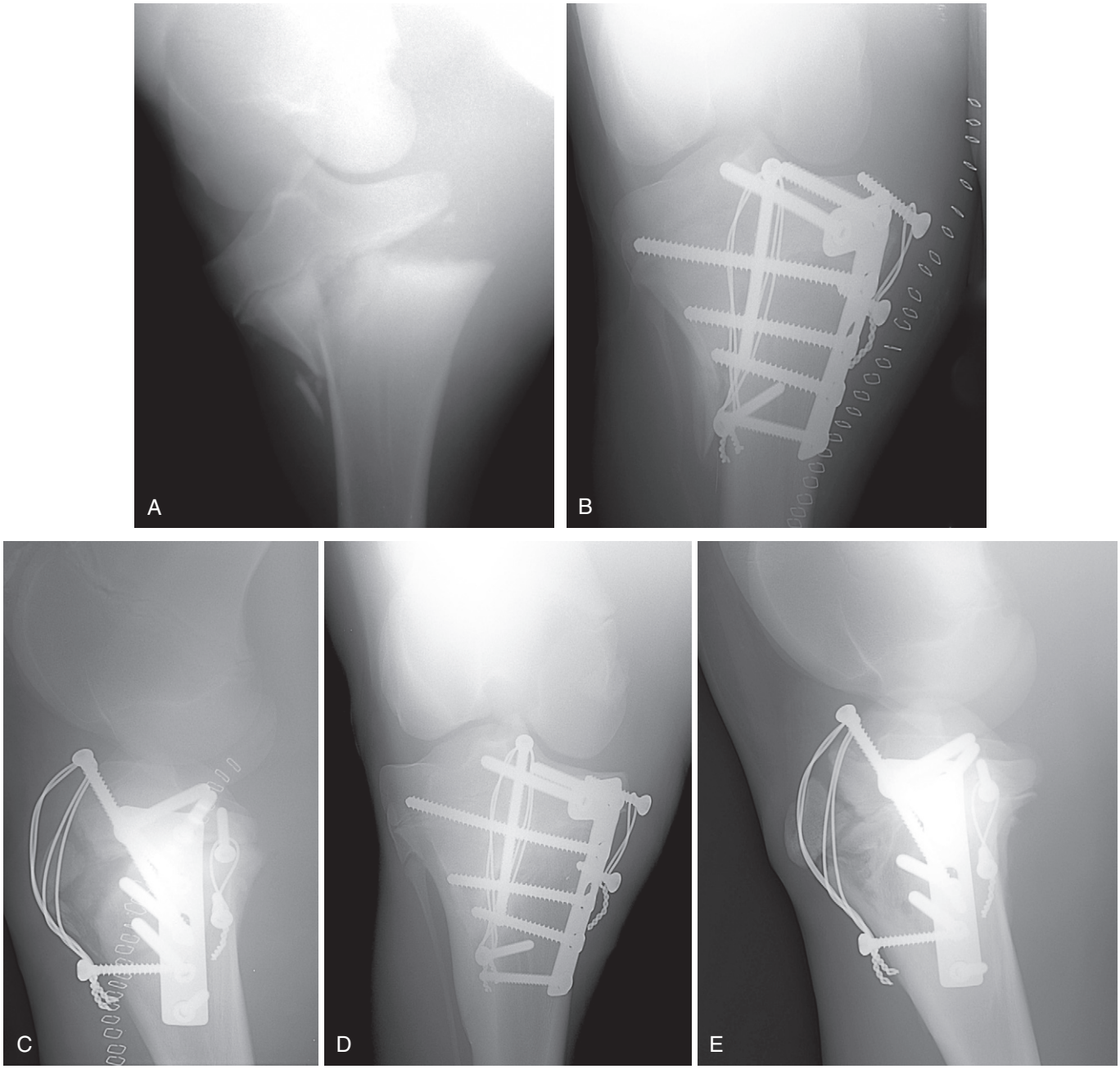

Figure 98-5. A, Craniocaudal radiographic view of a typical Salter Harris type II fracture of the proximal tibia. Craniocaudal **(B)** and lateromedial **(C)** postoperative radiographic views show the repair using a medially placed screw and wire tension band and a four-hole right angle "L" plate. A cranially placed screw and wire tension band was used to stabilize the tibial crest. Craniocaudal **(D)** and lateromedial **(E)** radiographic views of the healed fracture 8 weeks postoperatively. Note that the physis has closed.

complete reduction. Manual downward pressure on the medial aspect of the proximal tibial metaphysis places the intact soft tissue on the lateral aspect of the limb under tension and reduces the fracture. Reduction can be maintained by the placement of one 4.5-mm cortex screw in the epiphysis and one in the metaphysis. A figure-of-eight tension band wire is tightened between the two screws, maintaining reduction during placement of the primary implants if the stability is tenuous (Figure 98-5, *B* and *C*).

Soft tissue dissection is carried to the bone surface on the metaphysis, but the soft tissue on the epiphysis should not be disturbed. A four-hole T-plate, right angle "L" plate, five-hole broad limited-contact dynamic compression plate (LC-DCP) or locking compression plate (LCP), is used for fixation. (T-plates and right-angle "L" plates are named according to the number of vertical holes in the shaft; for example, a four-hole T-plate contains two horizontal holes and four additional holes in the vertical shaft.[7]) The two transverse holes are positioned over the epiphysis closer to the caudal aspect of the proximal tibia, halfway between the femorotibial joint surface and the physis. A right-angle "L" plate with the proximal (short) limb of the plate facing craniad and the long limb of the plate distal along the tibia gives the most diaphyseal contact without the plate overhanging the metaphyseal cortex (see Figure 98-5, *B* and *C*). The

two proximal screws are inserted across the width of the epiphysis. The screws should be oriented as much as possible parallel to the caudal cortex in a transverse plane, not perpendicular to the plate. The use of 5.5-mm cortex screws is ideal in the epiphysis. The distal screws in the T-plate are inserted into the metaphysis after using the tension device or are placed in the load position to achieve axial compression. If an LCP is used, two locking-head screws are placed in the epiphysis first, followed by axial compression of the fracture using a 5.5-mm cortex screw in the load position and finally implantation of the remaining locking head screws in the metaphysis. It is desirable to pass at least one of the plate screws across the proximal metaphysis into the lateral metaphyseal fragment that remains attached to the epiphysis. This increases the stability of the lateral aspect of the fracture (see Figure 98-5, *B* and *C*).

After the plate is in place, a second screw and wire construct can be applied to increase stability on the medial aspect of the bone, but the most stable fixation is achieved with a tension band wire used to stabilize the tibial crest, which always remains attached to the epiphysis. A 5.5-mm cortex screw is placed from proximal to distal through the epiphysis and into the metaphysis to act as the proximal anchor for the tension-band wire. A distal screw in the metaphysis or a hole through the cranial aspect of the metaphysis is used for the distal anchor of the tension band. One or two strands of 1.5-mm wire are subsequently placed in figure-of-eight fashion and twisted to tighten the construct (see Figure 98-5, *B* and *C*). Alternatively, a 1-mm cable can be substituted for the wire. The proximal screw is left above the bone surface under the patellar ligaments and not tightened down to the surface of the bone to allow removal at a later date.

When fixation is complete, the soft tissues are closed in two layers using routine technique. Closed suction drainage is optional. A stent bandage is sutured over the incision to reduce tension on and protect the incision. The area is difficult to bandage, but some compression of the soft tissue to reduce the hematoma can be obtained if elastic tape is used.

Aftercare

External coaptation is not used.[7] The foal is assisted to its feet during recovery from anesthesia. Broad-spectrum antibiotic therapy along with anti-inflammatory treatment is continued until the swelling has subsided. The condition of the soft tissues postoperatively determines the length of antibiotic treatment. Foals are confined to the stall for 4 weeks, after which time radiographs are taken. If there is any indication of postoperative complications, radiographs are taken earlier. The foal should be bearing weight on the limb within 1 or 2 days and should begin walking soundly within 7 to 10 days. Absence of soundness indicates developing complications.

If the 4-week follow-up radiographs indicate healing, exercise is increased over the next month to unrestricted exercise by 8 weeks. If no complications are encountered and the physis has not closed, the implants can then be removed by 8 weeks to allow the physis to resume growth if growth potential remains (Figure 98-5, *D* and *E*).

Complications

Three major complications are commonly encountered with this fracture: failure of the fixation, sepsis, and wound dehiscence. Failure of the fixation most commonly occurs by loss of purchase within the epiphysis. Failure seldom occurs from implant loosening in the metaphysis or by disruption of the implants themselves. In the rare case that a fissure fracture is not diagnosed initially, it may lead to a shaft fracture distal to the implants (Figure 98-6). Postoperative sepsis is possible, as with any equine fracture. A third complication that indirectly results in sepsis is dehiscence of the wound. The skin and subcutaneous tissue on the medial aspect of the limb are often traumatized by the sharp edge of the proximal tibial metaphysis if the duration from fracture to treatment was prolonged or if postinjury first aid was not adequate.[7] Sepsis does not necessarily mean failure if stability is maintained and as long as the infection is appropriately treated with drainage and antibiotics.

Prognosis

The prognosis after repair of proximal physeal fractures is favorable, but the smaller the foal, the better the prognosis.[4-7] The preoperative condition of the soft tissues, the amount of soft tissue stability on the lateral aspect of the limb, the ability to reduce the fracture, and the security of the proximal epiphyseal implants all determine the outcome. Sufficient healing of the fracture and resolution of the injury to allow athletic activity are anticipated in a majority of cases. Closure of the proximal tibial physis usually occurs, but it is not a major complication (see Figure 98-5, *D* and *E*). The proximal tibial physis of the horse does not close as readily as the physes of other animals or other sites, and the horse compensates for decreased limb length in spite of considerable damage if closure does occur.

Diaphyseal Fractures

Diaphyseal fractures are the second most common fracture of the tibia.[9] Most diaphyseal fractures have a spiral configuration and many are comminuted (Figure 98-7, *A* and *B*). The fracture is created when the forces of loading, which in the tibia create considerable torque, are combined with an excessive external force. Diaphyseal fractures can occur in animals of any age. Appropriate first-aid therapy is necessary to minimize secondary soft tissue damage, as with any fracture (see Chapter 73). The trauma required to fracture a tibia often creates an open fracture, which greatly decreases the chances of successful repair.

Preoperative Considerations

Usually, diaphyseal fractures of the adult tibia are high-energy, catastrophic injuries accompanied by extensive comminution and skin penetration.[9] For these reasons, attempts at repair are seldom practical, because of the very poor prognosis. However, tibial fractures in foals usually have less comminution and have a better chance of remaining closed, and repair has a greater chance of success.

Because traction on the distal limb does not reduce a tibial fracture—in fact, it causes overriding because of the reciprocal apparatus—hanging the limb in tension is not helpful. Placing the injured tibia down with the horse in lateral recumbency impedes surgical manipulation of the diaphyseal fracture and necessitates a medial surgical approach, where little soft tissue covers the bone and implant. Therefore, the horse is best positioned in dorsal recumbency with minimal traction on

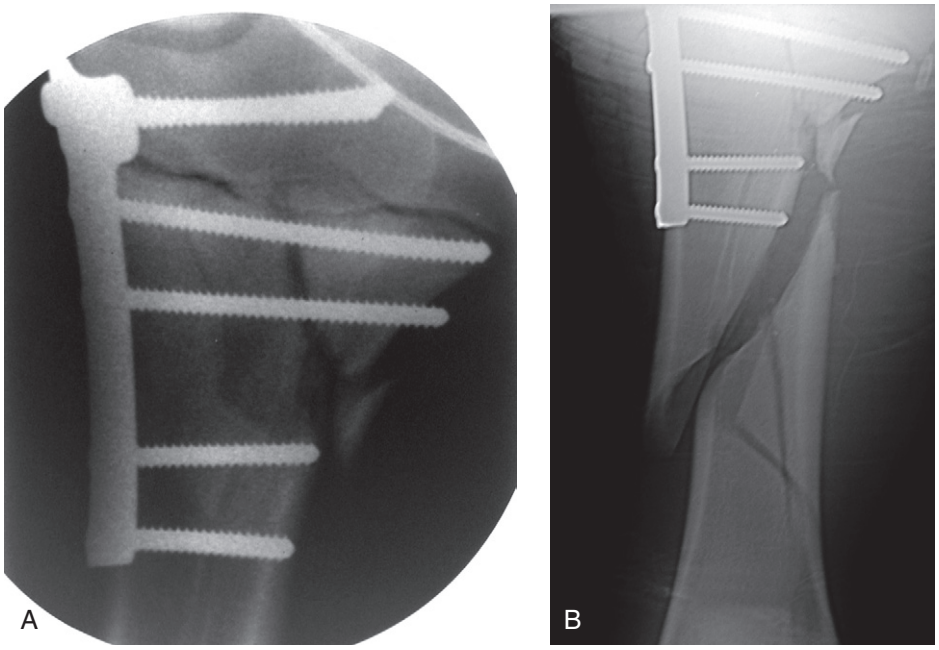

Figure 98-6. A, A craniocaudal intraoperative fluoroscopic view of a proximal tibial physeal fracture repaired with a four-hole T-plate. This fracture was more severe than a routine proximal tibial physeal fracture and required a more involved fixation. The metaphyseal component is comminuted and extends more than half the width of the bone. In addition, there is only one screw that is anchored in healthy bone distal to the comminuted fracture. This fracture probably required two implants and more distal fixation to resist the biomechanical instability the comminution created. **B,** The craniocaudal follow-up radiograph taken the next day because the foal was non–weight-bearing on the limb. A catastrophic bone failure occurred during the night. The fracture initiated at the distal end of the original fracture and spiraled distally, leading to catastrophic failure of the bone. The owner did not want proceed with treatment and the foal was humanely destroyed. (Courtesy J. Auer, University of Zurich.)

the limb or in lateral recumbency with the injured tibia uppermost.

Approaches to the tibia for diaphyseal fracture repair must take the anatomy into consideration. The cranial tibial artery is located on the cranial lateral aspect of the tibia. The lateral outpouching (sulcus muscularis) of the lateral femorotibial joint compartment extends distad, surrounding the long digital extensor tendon on the proximolateral aspect of the tibia. The tenuous soft tissue on the medial aspect of the tibia must be protected and the closely attached digital extensor tendons distally near the tarsocrural joint must be avoided.

Three surgical approaches are available: the medial approach directly over the tibia without muscle cover, the lateral approach between the long digital extensor and the cranial tibial muscles, and the cranial approach, in which the incision is made over the cranial tibial muscle.[9] The cranial approach over the muscle is the most versatile.

Deep dissection is extended slightly mediad allowing the muscle and soft tissue to be retracted as one unit laterad, permitting a cranial approach to the proximal tibial shaft. The cranial approach eliminates the necessity of dealing with any blood vessels, because all the vasculature is elevated with the cranial tibial muscle. Some surgeons prefer to place the implants on the surface of the periosteum. If this approach is elected, the vessels need to be protected if extra-periosteal dissection is used. Implants can be placed on the medial and lateral aspects of the bone through this incision, eliminating the need for two incisions, as are required with the medial and lateral approaches. The biomechanics of weight bearing of the tibia dictate that two plates be used and that one plate is best placed craniolaterally on the tension surface of the bone, spiraling distad to the cranial tibia (Figure 98-7, *C* and *D*).[10]

Broad-spectrum antimicrobial agents and anti-inflammatory medications are administered preoperatively.

Treatment

The incision is initiated craniolaterally along the lateral patellar ligament, extended to the tibial crest, directed craniad over the cranial tibial muscle to the distal aspect of the limb, and curved slightly mediad as the tarsocrural joint is approached. The incision is carried through the skin and subcutaneous fascia until the cranial tibial muscle is encountered. The cranial tibial muscle is elevated laterad, exposing the lateral surface of the bone.

Positioning of the implant at the most proximal aspect of the tibia in proximal metaphyseal and mid-diaphyseal fractures necessitates entering the femorotibial joint through the sulcus muscularis, which surrounds the long digital extensor muscle. Normally, the long digital extensor and cranial tibial attachments are avoided by separating them, which allows placement of the implant near the proximal physis or physeal scar. Working on both sides of the cranial tibial muscle allows placement of the plate without damaging the muscle.

Fracture reduction is accomplished by traction on the distal tibia and appropriate reconstruction of the bone. The repair begins by reattaching comminuted fragments to the parent bone with cortex screws placed in lag fashion to create a two-fragment fracture. The fracture is reduced to a proximal and distal fragment, and the plate is contoured to mimic the curve

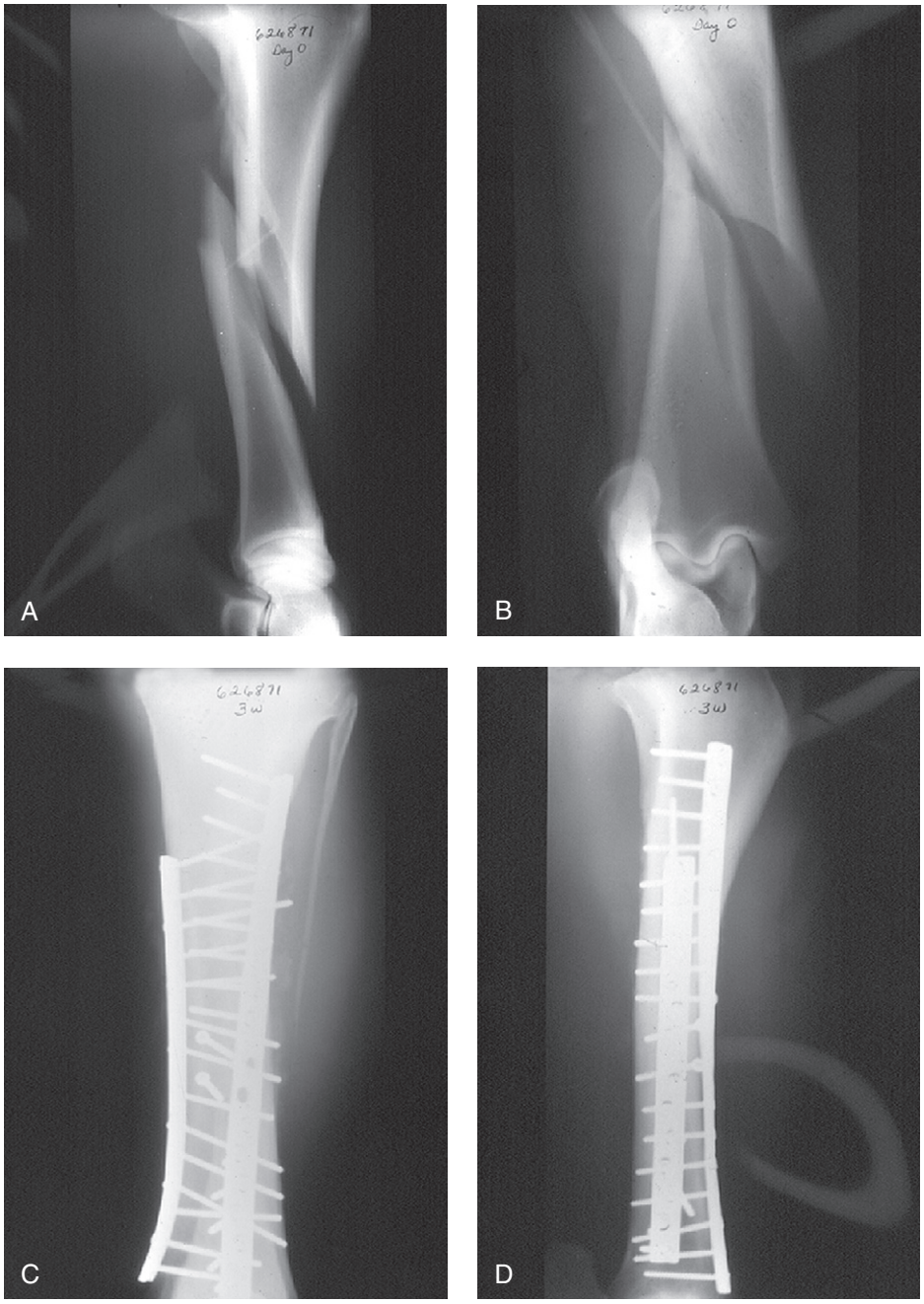

Figure 98-7. Lateromedial **(A)** and craniocaudal **(B)** radiographic views of a spiral comminuded displaced mid-shaft tibial fracture in an adult horse. Craniocaudal **(C)** and lateromedial **(D)** radiographic views of the repaired fracture using two interfragmentary 4.5-mm cortex screws inserted in lag fashion and two staggered broad 4.5 mm DCPs, 3 weeks after surgery.

of the tibia from craniolateral proximally to cranial distally (see Figure 98-7, *C* and *D*). The use of dynamic condylar screw (DCS) plates or 5.5-mm LCP constructs is desireable, especially in older foals and the rare adult horse undergoing treatment. The screws are inserted in the order dictated by the individual fracture, which differs from case to case. Care must be taken to cross any fracture plane with screws placed in lag fashion and to create interfragmentary as well as axial compression during application of the plate for the most stable fixation.

When the craniolateral plate is in place, the craniomedial plate is applied. It is contoured from craniomedial to medial and positioned in an envelope under the skin and subcutaneous tissue with or without elevation of the periosteum medially. It is occasionally necessary to flex and abduct the limb to gain access to the medial aspect of the tibia. The medial plate is positioned to avoid the screws of the lateral plate. Interfragmentary and axial compression is created as needed. Both plates can be loaded to distribute the axial compression circumferentially around the bone. Plate luting has been advocated for repair of this fracture to increase stability and fatigue life of the implant construct and should be considered for tenuous fixations.[9] After placement of the plates, a suction

drain is inserted, exiting proximolaterad, and attached to a suction reservoir.

Closure involves the fascia of the cranial tibial muscle, subcutaneous tissue, and skin. Release incisions can be made in the skin over the cranial tibial muscle to relieve tension on the closure if swelling has increased the diameter of the limb and endangered the closure.

Postoperatively, no cast is used. Cast immobilization increases the load on the implants because the cast cannot extend proximal to the stifle in the horse and the stifle joint cannot be immobilized. Stent bandages are sewn over the incision. The horse is allowed to recover from anesthesia unimpeded by bandages; bandages are placed after the horse is standing. The horse is always assisted in recovery; it should be helped to regain the standing position in one attempt, if possible. Bandaging is used as indicated during the postoperative period.

Aftercare

Antibiotics and anti-inflammatory drugs are continued for a prolonged period because tibial fractures are accompanied by considerable soft tissue damage. The health of the soft tissue

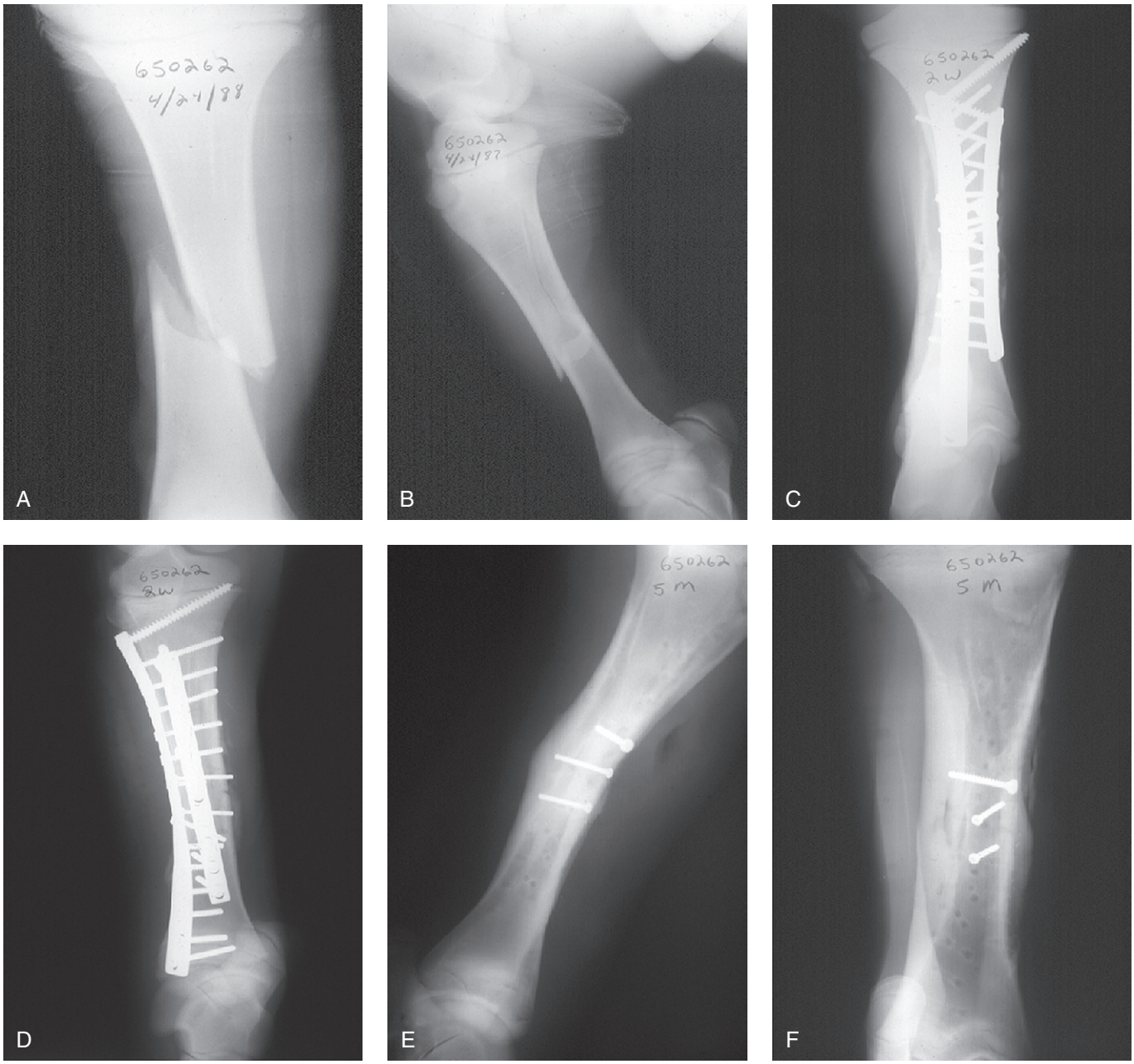

Figure 98-8. Craniocaudal **(A)** and lateromedial **(B)** radiographic views of a slightly displaced short oblique mid-shaft tibial fracture in a foal. Craniocaudal **(C)** and lateromedial **(D)** 2-week postoperative radiographic views of the repaired fracture, which used three interfragmentary cortex screws applied in lag fashion and a 14-hole broad 4.5-mm DCP placed craniolaterally and a narrow 10-hole 4.5-mm DCP placed medially. Lateromedial **(E)** and craniocaudal **(F)** 5-month postoperative radiographic views of the healed fracture after plate removal because of infection of the implants and drainage. The three cortex screws were covered by bone and, therefore, were left in place.

and the postoperative use of the limb by the patient dictate the length of antibiotic and anti-inflammatory therapy.

Stall rest is maintained, with no exercise allowed for 6 to 8 weeks, depending on the age of the horse. Foals are often radiographed at 6 weeks, whereas follow-up radiographs are taken at 8 weeks in adults to ascertain whether bridging of the fracture has occurred. Postoperative use of the limb is the most accurate assessment of stability of the fracture. If use of the limb is not attained or diminishes, radiographs are taken.

Complications

The most common complication with tibial fractures is breakdown of the fixation because of bone or implant failure. Tibial fractures are most often comminuted, and the torsional force applied to a fractured bone that contains multiple drill holes often causes propagation of the fracture into additional sites. A single acute overload generally causes the bone to fail; cyclic loading in an unstable construct will cause the implants to fail. Complications also include postoperative sepsis because of primary infection or breakdown of the incision. If the fixation remains stable, the fracture will heal even when contaminated and infected (Figure 98-8, *A* through *F*). With any major long bone fracture, all complications that preclude comfortable weight bearing eventually result in failure of the contralateral limb, which results in euthanasia.

Prognosis

Simple fractures in adult horses can be repaired successfully with internal fixation (see Figure 98-7, *A* through *D*); however, this fracture configuration is the exception rather than the rule. Most diaphyseal tibial fractures are comminuted and accompanied by massive soft tissue damage. Therefore, the prognosis for tibial fractures is unfavorable in adults. In foals, however, internal fixation of tibial fractures can be successful when the fracture is immobilized and the implants protect the bone enough to allow healing (see Figure 98-8, *A* through *D*).[11] The prognosis is guarded even in foals, but success in healing of the fracture and dismissal of the horse postoperatively is attainable in selected fractures (see Figure 98-8, *E* and *F*). Athletic activity is undertaken if the surgical procedure and stabilization proceed without complications.

Distal Physeal Fractures

Fractures of the distal epiphysis occur rarely. Diagnosis is routine, and treatment involves external coaptation with a

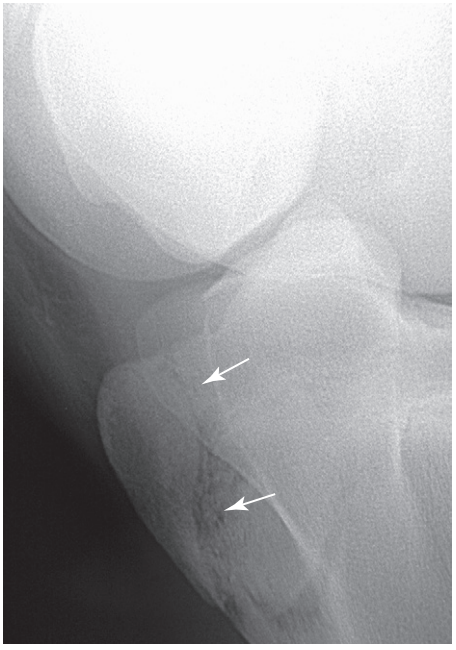

Figure 98-9. Lateromedial radiographic view of the most commonly encountered nondisplaced, nonarticular tibial crest fracture *(arrows).* This fracture was treated conservatively with stall rest.

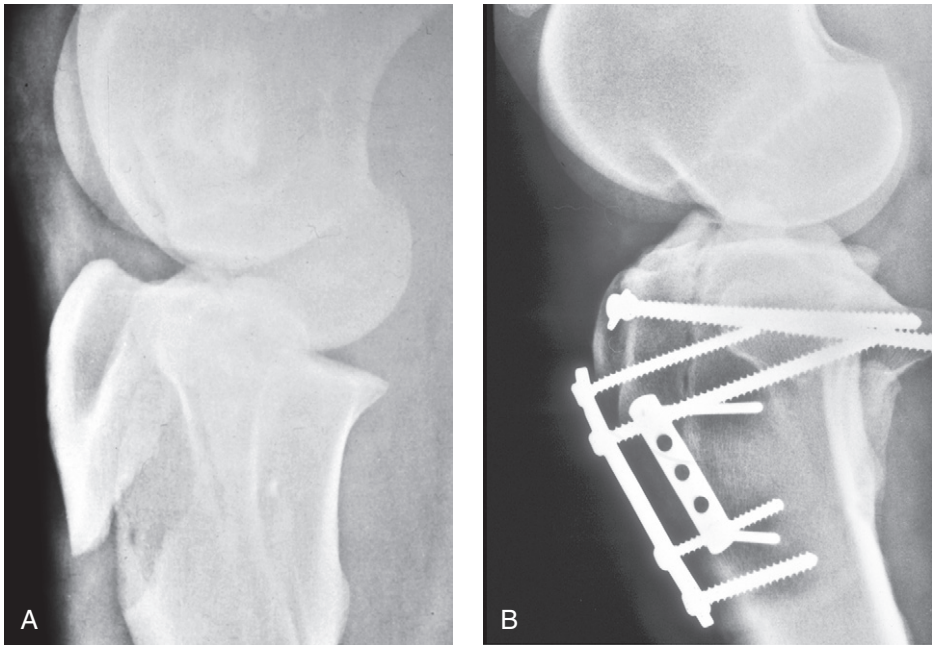

Figure 98-10. A, Lateromedial radiographic view of a displaced tibial crest fracture. **B,** The fracture was repaired proximally with two 5.5-mm cortex screws applied in lag fashion perpendicular to the fracture plane. A washer was used with one screw to avoid countersinking the head and weakening the bone. Additionally, a 5-hole narrow dynamic compression plate (DCP) was applied over the medial ridge as a tension band, and a 5-hole 2.7-mm cuttable small animal plate with two 3.5-mm cortex screws was applied over the lateral aspect of the fracture to provide additional stability. The fracture healed without complications. (Courtesy J. Auer, University of Zurich.)

fiberglass cast alone or in conjunction with selective screw placement. The architecture of the undulating articular surface of the distal tibia and the corresponding physis makes implant placement into the epiphysis especially difficult.

Tibial Crest Fractures

Tibial crest fractures are generally the result of direct trauma, often during attempts to jump a fence or from contact with other solid objects.[12] The most common tibial crest fracture is the nondisplaced nonarticular fracture (Figure 98-9). These fractures require no fixation because the patellar ligaments provide adequate stability for healing. Stall confinement for 60 days with follow-up radiographs at that time usually show healing adequate for gradual re-introduction to exercise.

If the tibial crest fracture is displaced or has an articular component (Figure 98-10), the fracture is stabilized in much the same fashion as a proximal tibial physeal fracture with fixation and stabilization employing a tension band cranially and implants sufficient to stabilize the fracture and reconstruct the joint surface.[12] Because of the anatomic configuration of the tibial crest, it is best to orient the plate obliquely along the tibial crest. The screws should be directed mediad and laterad in an alternating pattern to avoid aligning them in a straight line, which may predispose the underlying bone to fracture.

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CHAPTER 99

Stifle

Jennifer G. Fowlie, John A. Stick, and Frank A. Nickels

The stifle joint represents an important source of hindlimb lameness for equine athletes because it is the largest and most complex joint in the horse.[1] The clinical presentation of stifle disorders is highly variable, and the degree of stifle joint effusion, peri-articular swelling, and lameness differs depending on the underlying disorder. The degree of lameness may vary from virtually occult to non–weight-bearing, and when viewed from the rear, asymmetry of the gait is similar to hindlimb lameness from other causes.

The equine stifle consists of three compartments; the medial compartment of the femorotibial joint (MFT), the lateral compartment of the femorotibial joint (LFT) and the femoropatellar joint (FPT). There is a complex arrangement of soft tissues in the stifle (12 ligaments, 2 menisci, and numerous musculotendinous support structures) that provide functional stability, force transduction, and locomotion. Historically bony lesions (particularly osteochondrosis) have been cited to have a higher incidence than soft tissue lesions[2]; however, recognition of soft tissue injuries is increasing with recent access to large-bore magentic resonance imaging, improved ultrasonographic imaging, and more extensive application of traditional and novel arthroscopic techniques.

This chapter reviews the functional anatomy, general diagnostic techniques, and the diagnosis, treatment, and prognosis of specific disorders of the equine stifle joint.

FUNCTIONAL ANATOMY

The stifle consists of two articulations: the FPT and the femorotibial joints (MFT and LFT) (Figures 99-1 and 99-2).[3] The FPT has a large suprapatellar pouch, and the joint capsule inserts abaxially on the trochlear ridges, forming a lateral and a large medial cul-de-sac on each side. Communication between the MFT and LFT is not present under normal conditions,[3-6] and the femorotibial compartments are divided into a cranial pouch and a caudal pouch by the femoral condyles. The volume in the medial and lateral compartments of the femorotibial joint is 41.67 ± 5.77 mL and 61.67 ± 2.89 mL, respectively. The standing femorotibial joint angle has been reported as approximately 150 degrees, with flexion limited only by the contact of the

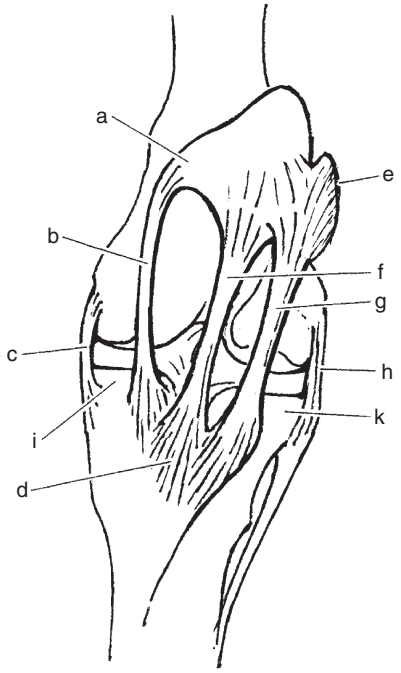


Figure 99-1. Anatomy of the equine stifle, cranial view, showing: *a,* parapatellar fibrocartilage; *b,* medial patellar ligament; *c,* medial collateral ligament; *d,* tibial tuberosity; *e,* cut stump of the biceps femoris muscle; *f,* middle patellar ligament; *g,* lateral patellar ligament; *h,* lateral collateral ligament; *i* and *k,* medial and lateral condyles of tibia.

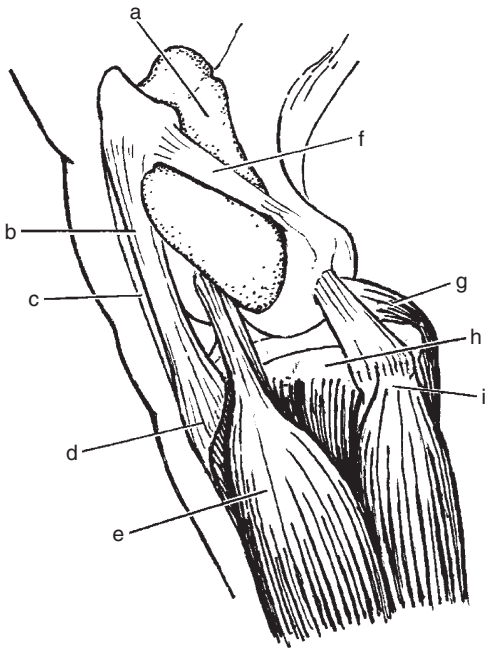


Figure 99-2. Anatomy of the equine stifle, lateral view, showing: *a,* femoropatellar joint (FPT); *b,* middle patellar ligament; *c,* medial patellar ligament; *d,* cut stump of the lateral patellar ligament; *e,* extensor digitorum longus muscle; *f,* lateral femoropatellar ligament; *g,* popliteus muscle; *h,* lateral tibial condyle; *i,* extensor digitorum lateralis muscle.

caudal leg musculature.[3] The "screw home mechanism," as reported in other species, is also seen in the equine stifle, such that the tibia undergoes slight external rotation on full extension and inward rotation on full flexion.[3] This occurs as a result of the interactions of the cruciate ligaments, the collateral ligaments, and the disparate size of the femoral condyles.

Experimental studies show a communication between the FPT and the MFT in 60% to 65% of horses, with communication between the FPT and the LFT occuring in only 1% to 25% of horses.[3,4,7] Variation in communication rates is seen with different materials (i.e., latex vs. contrast arthrography). Diffusion of mepivacaine anesthetic in cadaveric stifles from the FPT to the MFT, from the FPT to the LFT, and between the MFT and LFT (in both directions) occurred in 100% of limbs.[8] As well, mepivacane diffusion occurred from the LFT to the FPT in 90% of the limbs and the MFT to the FPT in 85%. Interestingly, communication between the FPT and the MFT was only noted in 3 of 16 (19%) clinical cases using computed tomographic contrast arthrography,[9] indicating that disease may obstruct these communications.

DIAGNOSTIC TECHNIQUES

Inspection

Lameness evaluation of the stifle includes observation of the horse at rest and during locomotion, palpation of the synovial compartments and the patellar ligaments, and additional diagnostic aids, such as manipulative tests and intra-articular anesthesia. Clinical signs depend on the severity and chronicity of the injury. Severe injuries, such as fractures, sepsis, or instability of the joint (cruciate or collateral ligament damage), generally produce the most severe lameness. Chronic lameness of a rear limb results in atrophy of the gluteal muscles, causing an asymmetric appearance of the croup when viewed from the rear. The lame horse frequently bears weight on the affected limb for a shorter time during movement, resulting in a shortened anterior phase of the stride when viewed from the side. When viewed from the rear at the trot, there is a greater excursion (vertical displacement) of the tuber coxae on the affected side compared to the contralateral side[10], which is typical of most sources of hindlimb lamenesses. Wearing of the toe on the affected side may be seen because of decreased flexion of the hock/stifle during the swing phase of the gait. There are no pathognomonic gait abnormalities that can be used to definitively localize hind limb lameness to the stifle.

Synovial distention of the FPT may be visually obvious (Figure 99-3) or quite subtle. Subtle changes can be detected by careful palpation between the patellar ligaments and cranial to the distal patella and by comparing the two stifle joints. Synovial distention of the femorotibial compartments (particularly the lateral) can be difficult to detect but can be palpated immediately cranial or caudal to the collateral ligaments. Palpation of swelling or crepitation of the patella may indicate a patellar fracture. Upward fixation of the patella should be suspected if delayed unlocking of the patella is palpated as the horse is walking, or if manipulation of the patella proximad and laterad results in pain or the inability to flex the stifle.

Manipulation

Manipulative tests may exacerbate the lameness, aiding in localizing the lesion to the stifle. These tests include the tarsus/stifle flexion test, the patellar test, the cruciate test, and collateral ligament stress tests.

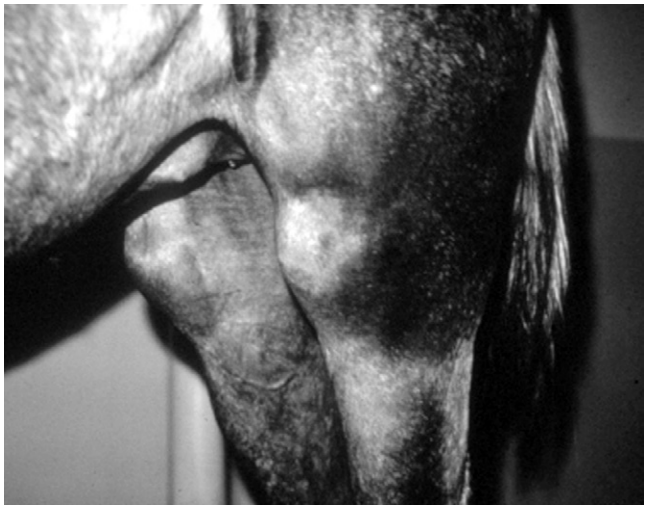


Figure 99-3. Appearance of bilateral FPT effusion. Note the enlargement over both patellar regions.

Figure 99-4. Cruciate ligament test used to detect rupture. The shoulder is placed against the back of the limb and the tibia is pulled caudad and released.

Tarsus/stifle flexion is performed by grasping the distal metatarsus and holding the tarsus/stifle joints in maximal flexion for 1 to 2 minutes. The metacarpo/metatarsophalangeal and phalangeal joints are only passively flexed by this method. A positive response to this test (an increase in lameness) may indicate tarsal or stifle lameness, thus one must use additional diagnostic aids to further localize the lameness.

Two manipulative tests have been described for evaluating the cruciate ligaments. These tests are designed to elicit pain and lameness because of the abnormal movement created between the femur and the tibia if there is damage to the cruciate ligaments. The first test is only safe in the most cooperative of horses. To perform this test, the examiner stands behind the affected limb and firmly grasps the proximal tibia with both hands (Figure 99-4). If any movement or crepitation is detected when the examiner gives a quick pull backward, the test is said to be positive.[11] The second test is carried out with the examiner standing directly in front of the affected limb, placing the inside hand on the cranial aspect of the proximal tibia, and grasping and pulling the tail to load the limb while rapidly pushing and releasing the force on the tibia 20 to 25 times. The horse is then trotted away to evaluate for increased lameness.

The medial collateral ligaments can be evaluated by elevating the limb off the ground and placing pressure on the lateral aspect of the stifle joint while abducting the distal limb. The lateral collateral ligament can be evaluated by elevating the limb off the ground and pulling the stifle joint laterally while adducting the distal limb.

Intra-Articular Anesthesia

Intra-articular anesthesia is an important diagnostic aid in determining articular involvement. Although local anesthesia diffuses between all stifle compartments about 85% to 100% of the time, anesthetic should be deposited in all three synovial compartments to ensure complete anesthesia of the stifle.[4,5,8]

Injection of the FPT can be performed through either a cranial or a lateral approach. The cranial approach is performed by inserting a 7.5-cm (3-inch) spinal needle between the medial and middle patellar ligaments or between the middle and lateral patellar ligaments and directing it proximad into the trochlear groove under the patella. The lateral approach is performed by inserting a 3.75-cm 18-gauge needle into the lateral cul-de-sac of the FPT just caudal to the lateral patellar ligament and approximately 6 cm proximal to the lateral tibial condyle.[12] This approach is best performed with the limb unloaded and is commonly the best approach for obtaining a fluid sample from the FPT.

The approach to the LFT is performed by inserting a 3.75-cm 18-gauge needle between the lateral patellar ligament and the long digital extensor tendon or between the previous tendon and the lateral collateral ligament above the proximal edge of the meniscus into the joint. The approach to the MFT is performed by inserting a 3.75-cm 18-gauge needle between the medial patellar ligament and the medial collateral ligament above the proximal edge of the meniscus into the joint. The use of an 18-gauge needle and a bleb of local anesthetic in the skin at the insertion point reduces the risk of limb flexion during the procedure, decreasing the chance of needle bending and breakage. An alternative approach to the MFT, which may decrease the risk of iatrogenic damage and increase the chance of fluid retrieval, is needle insertion 1.5 cm proximal to the tibial plateau in the depression between the medial patellar ligament and the tendon of insertion of the sartorius muscle.[13]

Injection of all three joint compartments with only one skin penetration may be achieved with an 8.9-cm 18-gauge spinal needle at a site just caudal to the lateral patellar ligament and approximately 2 cm proximal to the tibial plateau. The needle is directed to gently contact the medial and then lateral femoral condyles and then proximally under the patella, depositing anesthetic in all three joint compartments.

DIAGNOSTIC IMAGING

Radiography

Five radiographic views are necessary for a comprehensive evaluation of the stifle: lateromedial, flexed lateromedial,

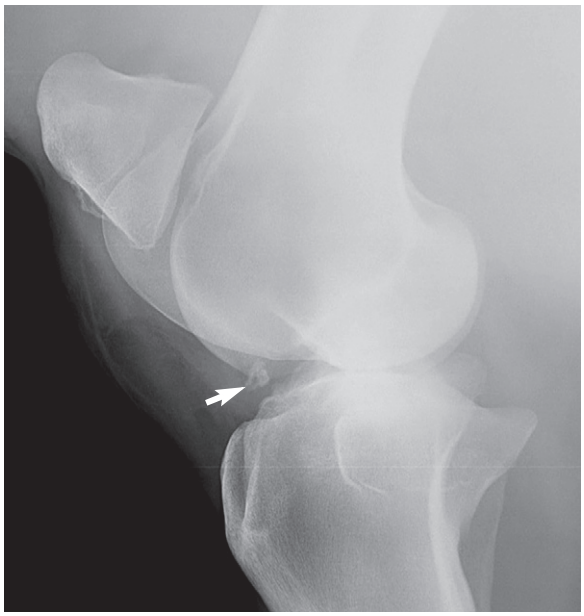


Figure 99-5. An avulsion fracture *(arrow)* of the cranial cruciate ligament is evident on this lateromedial stifle radiograph. The avulsed osseous fragment is evident near the distal insertion of the cranial cruciate ligament on the medial intercondylar eminence of the tibia.

caudocranial, caudal 30-degree lateral craniomedial oblique, and flexed cranioproximal craniodistal oblique view (skyline). X-ray machines need a minimum output of 90 kV and 20 mAs to obtain quality films of this large joint.

The lateromedial and flexed lateromedial views highlight osseous irregularities near the insertion of the cranial meniscotibial ligaments and the cranial cruciate ligament (Figure 99-5). A flexed lateromedial view of the stifle, with the limb lifted and retracted caudally, permits patellar and condylar lesions to be seen more easily. The caudal 30-degree lateral craniomedial oblique view decreases superimposition of the trochlear ridges and the condyles of the femur (Figure 99-6). The x-ray beam should be aligned 10 to 15 degrees proximodistally and 30 degrees caudolateral to craniomedial. Lateral trochlear ridge lesions are most clearly seen with this view.

The caudocranial view is best obtained by angling the x-ray beam 10 to 20 degrees proximodistally and centered on a line bisecting the limb between the semimembranosus and semitendinosus muscles at the level of the distal third of these muscles. The horse should be standing squarely with the leg underneath the body. This view is best for demonstrating medial condylar cysts (Figure 99-7) and osteoarthritis of the stifle. Osteophyte formation on the abaxial aspect of the medial tibial plateau is commonly the first radiographic sign of osteoarthritis of the stifle.

The cranioproximal craniodistal oblique view (skyline) (Figure 99-8) is used primarily to evaluate the patella (particularly for fractures), the intertrochanteric groove of the distal femur, and the trochlear ridges. In standing horses, the limb should be held semiflexed, drawn caudad and externally rotated to expose the stifle region. The cassette is held horizontally with its caudal edge against the cranial aspect of the tibia. The x-ray beam should be directed perpendicular to the cassette as much as possible.

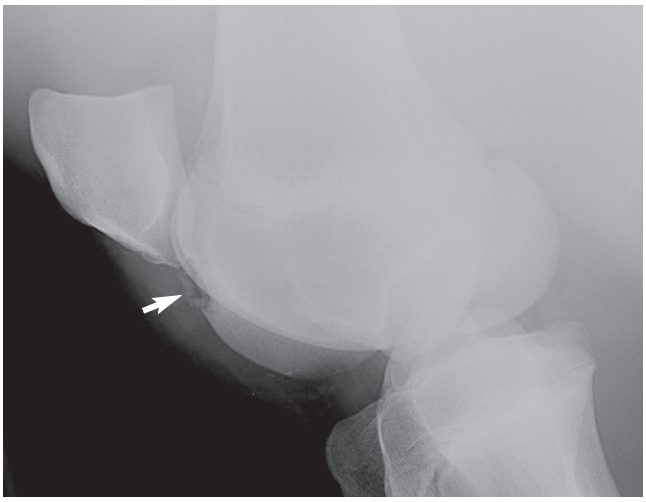


Figure 99-6. A caudal 30-degree lateral craniomedial oblique radiographic view of the stifle highlights an osteochondrosis lesion *(arrow)* of the lateral trochlear ridge of the femur.

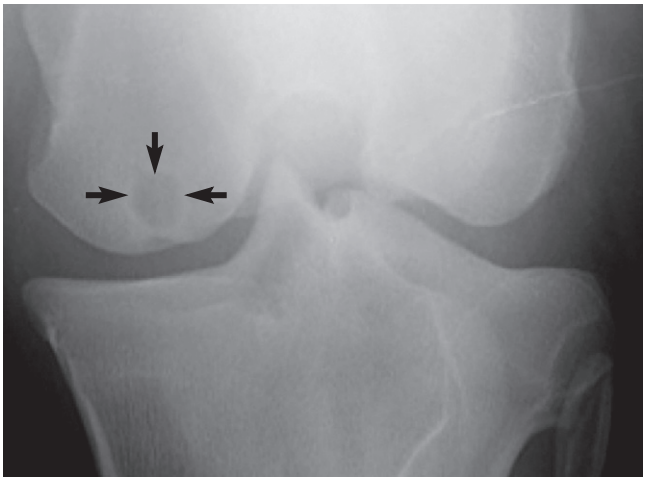


Figure 99-7. A caudocranial view of the stifle demonstrating a large subchondral bone cyst *(arrows)* in the medial femoral condyle.

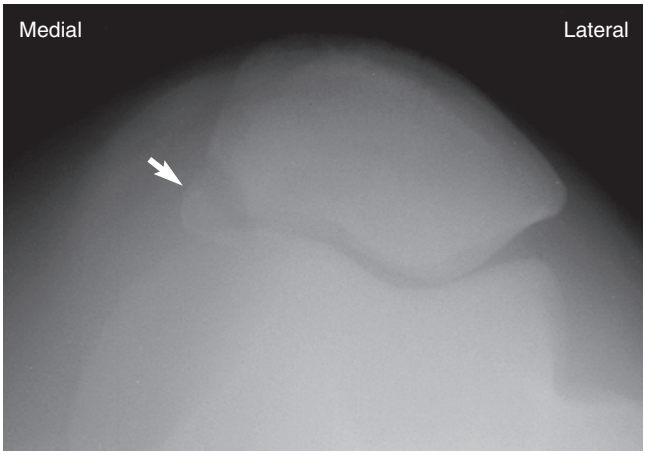


Figure 99-8. Cranioproximal craniodistal oblique radiographic view (skyline) of the equine stifle, which is used to evaluate the patella, intertrochanteric groove of the femur, and trochlear ridges. Note the sagittal fracture of the medial aspect of the patella *(arrow)*.

Radiographic mapping of the origins and insertions of the soft tissues in the stifle region has been completed and is useful in determining potential soft tissue injury if there is an osseous component, such as avulsion fracture or enthesiophyte formation.[14]

The stifle of the horse has six centers of ossification at birth. This makes radiographic assessment of the foal somewhat challenging. A summary of closure of growth plates and ossification sites is as follows. The patella is fully ossified by 4 months of age. The proximal portions of the trochlear ridges show irregular contours and granular subchondral bone opacity, which gradually become a smooth, regular subchondral bone outline between 3 and 5 months of age. The distal femoral physis closes at 24 to 30 months of age. The femoral condyles are smoothly outlined, and any irregularity except in young foals should be interpreted as pathologic. At birth, the center of ossification of the tibial tuberosity (apophysis) is separated from the proximal tibial epiphysis as well as from the proximal tibial metaphysis. The apophyseal epiphyseal physis closes at 9 to 12 months of age, and the apophyseal metaphyseal physis closes at 30 to 36 months. The proximal tibial physis closes at 24 to 30 months of age. The fibula has multiple ossification centers that can remain incomplete throughout the life of the horse.

In adult horses, the trochlear ridges are smoothly curved in outline, but the medial trochlear ridge has a small, flattened area at its junction with the metaphysis of the femur. This creates a plateau at its proximal aspect that articulates with the patella, thus functioning in the passive stay apparatus of the hind limb. On a caudocranial view in the adult horse, the patella is seen superimposed over the lateral cortex of the diaphysis of the femur. The medial femoral condyle is more round than the lateral condyle, and the lateral joint space of the femorotibial joint is narrower than the medial joint space. On the cranioproximal to craniodistal oblique view, the medial trochlear ridge is larger than the lateral ridge and the patella is approximately triangular with its medial angle blunter than the lateral angle.

Ultrasonography

Ultrasonography permits a noninvasive, relatively inexpensive examination of the soft tissue and bony structures of the stifle.[15-20] Ultrasonography is particularly beneficial in evaluating regions of the stifle that are obscured from arthroscopic examination,[21] and techniques for optimal ultrasonographic imaging of the stifle are well documented.[22] A 7- to 14-MHz linear transducer is recommended for evaluating the patellar and collateral ligaments, the menisci, and the femoropatellar and femorotibial joint capsules with the horse bearing weight. A 4- to 6-MHz curvilinear transducer is recommended for the caudal stifle region (during weight bearing) and the cranial aspect of the stifle (with the limb held in 90 degree flexion). The flexed cranial view is used for evaluating the cranial horn of the menisci, cranial meniscotibial ligaments, and tibial and femoral attachments of the cranial and caudal cruciate ligaments. Optimal visualization of the cruciate ligaments is challenging because of their deep position and oblique orientation. Visualization of the caudal aspect of the stifle is also difficult because a lower-frequency transducer is needed to penetrate the thick caudal musculature, resulting in a decreased resolution of the images. Thus, considerable practice is necessary to obtain and interpret diagnostic images.

Because of the complex shape and fiber orientation of the soft tissue structures of the stifle, all lesions should be confirmed in two perpendicular planes. Familiarization with the normal anatomy and comparison with the sound limb is necessary to avoid misdiagnoses. For example, a hypoechoic artifact is frequently created in the cranial medial meniscus and proximal medial collateral ligament with a linear transducer because of a loss of perpendicular alignment of the fibers with the ultrasound beam. These artifacts are not seen with a curvilinear probe, emphasizing that experience and careful interpretation are required for accurate diagnoses.[22]

Multiple lesions can be identified with ultrasonography, such as intra-articular bone fragments, cartilage lesions, thickening of the synovial membrane, joint effusion, desmitis or core ligamentous lesions, and meniscal tears and protrusion. Meniscal lesions may be more apparent when the limb is non–weight-bearing. Lesions that cannot be definitevly diagnosed on radiographs may be more clearly identified ultrasonographically, such as avulsion of the popliteal tendon origin.[23] Ultrasonography has a sensitivity and specificity of 79% and 56%, respectively for identifying meniscal injuries when compared to arthroscopic findings[24]; however, the high false-positive rate in this study may partially be caused by the inability to arthroscopically visualize regions of the joint that can be visualized ultrasonographically. Another study involving 29 cases of meniscal tears identified three horizontal abaxial tears that could not be seen arthroscopically and 10 cases where the tears were only seen arthroscopically,[25] indicating a high rate of false negatives. Horizontal tears of the medial meniscus were the most frequent soft tissue injury identified in another study.[26] Preliminary evaluation indicates that three-dimensional ultrasonography has increased diagnostic capabilities.[27] Ultrasonography will continue to assume a more prominent role in the diagnosis of stifle lesions in the horse, but ultimately multimodal diagnostics will provide the optimal evaluation of the stifle joint.

Nuclear Imaging

Nuclear scintigraphy identifies lesions based on their physiologic characteristics (osteoblastic activity and blood flow). However, nuclear scintigraphy has been inconsistent in imaging and localizing stifle joint pathologies, particularly in cases of subchondral bone cysts.[28,29] The scintigraphic appearance of normal stifles is established,[30] and multiple lesions have been recognized on scintigraphic examinations, such as anterior cruciate ligament avulsion,[31] osteoarthritis, and nonspecific increased radiopharmaceutical uptake (IRU) associated with intra-articular lesions (i.e., a meniscal tear with cartilage and subchondral bone changes).[28] Relatively late growth plate activity in the region of the stifle may mask lesions, and a lack of definitive radionucleotide uptake cannot be used to rule out a stifle lameness.

Computed Tomography

Recent access to advanced imaging such as computed tomography (CT) has permitted more extensive evaluation of the stifle and detection of lesions that would be inaccessible or incompletely identified with more conventional diagnostics (including arthroscopy).[9,32,33] CT arthrography is available at select referral institutes and has identified and further delineated intra-articular stifle lesions. Anatomic studies have used CT and

contrast techniques under fluoroscopic guidance (Figures 99-9 and 99-10).[6] CT arthrography is capable of identifying cruciate ligament pathologies, cartilage lesions, subchondral bone cysts, semitendinosis tendon and deep digital flexor muscle enthesiopathies, and enthesis/subchondral sclerosis of the tibia at the cranial medial meniscotibial ligament attachment that is not evident on radiographs.[9] An ambulatory pQCT (peripheral quantitative computerized tomography) scanner may be used to identify and localize bone fragments in the stifle for surgical removal.[34]

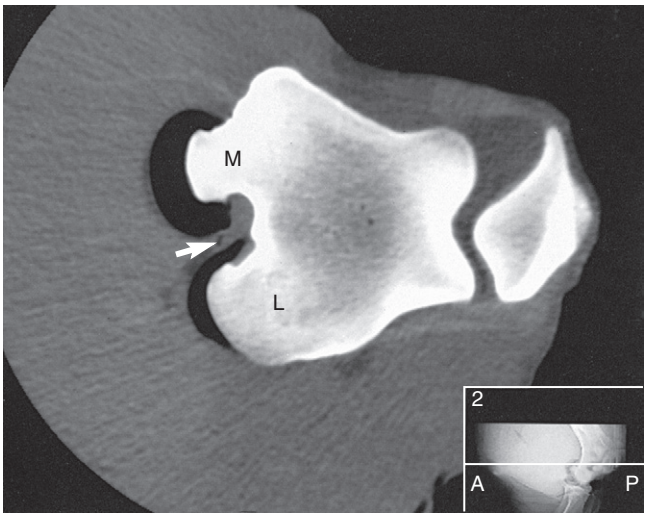


Figure 99-9. Computed tomographic (CT) image of a transverse section of an equine cadaver right stifle at the level of the proximal aspect of the medial and lateral femoral condyles. Injection of the caudal pouches of the femorotibial joints with shaving cream prior to imaging shows the extent of distention of these pouches. The popliteal artery and vein *(arrow)* run in the septum separating the caudal pouches. *M,* Medial femoral condyle; *L,* lateral femoral condyle. (From Trumble TN, Stick JA, Arnoczky SP, et al: Consideration of anatomic and radiographic features of the caudal pouches of the femorotibial joints of horses for the purpose of arthroscopy. Am J Vet Res 55:1682, 1994.)

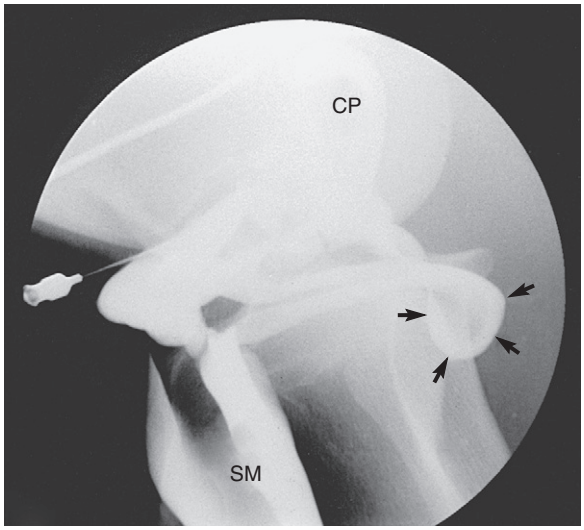


Figure 99-10. Fluoroscopic image of the LFT of an equine cadaver stifle after injection of positive contrast medium into the caudal pouch of the LFT showing distention of the proximal pocket of the caudal pouch *(CP)* of the LFT, the sulcus muscularis *(SM),* and the distal pocket of the caudal pouch of the LFT *(arrows).*

Magnetic Resonance Imaging

Recent access to wide-bore MRI has allowed imaging of clinical cases of equine stifle joint lameness. MRI of the human knee is a well-established diagnostic technique for evaluating soft tissue lesions; however, arthroscopy is still considered the gold standard given that the human knee can be distracted for excellent intra-articular visualization.[35] MRI of equine cadaveric limbs was first completed in 1995 to document the normal anatomy.[36,37] MRI of clinical cases was not possible until recent access to wide/short–bore magnets that can accommodate the average sized equine stifle.[32] Despite the wide (70 cm) and ultra short (125 cm) bore of the Siemens Magnetom Espree (Siemens Medical Solutions USA) 1.5 T magnet, imaging is still limited in adult horses to those with relatively long legs and narrow hips. Stifle MRI is completed under general anesthesia and is generally limited to imaging of one stifle within an anesthetic period because of time constraints and a recumbency change that would be required to complete the contralateral stifle. MRI has been invaluable for idenitifying meniscal and meniscal ligament tears, bone edema, osteochondral fragmentation, cartilage pathologies and cruciate, patellar and collateral ligament desmitis, and many clinical cases of stifle lameness that have multiple pathologies have been identified. MRI may also reveal lesions not identifiable with other diagnostic techniques (Figure 99-11).[32]

ARTHROSCOPY

Diagnostic and therapeutic arthroscopy of the equine stifle is well established. Arthroscopy is most commonly performed to treat osteochondrosis of this joint, and most of the approaches have been developed based on the characteristic locations of this disorder. Arthroscopy is also routinely used to establish a diagnosis and prognosis after traumatic stifle injury or chronic lameness that may involve damage to the menisci, meniscal ligaments, cruciate ligaments, and articular cartilage. Despite

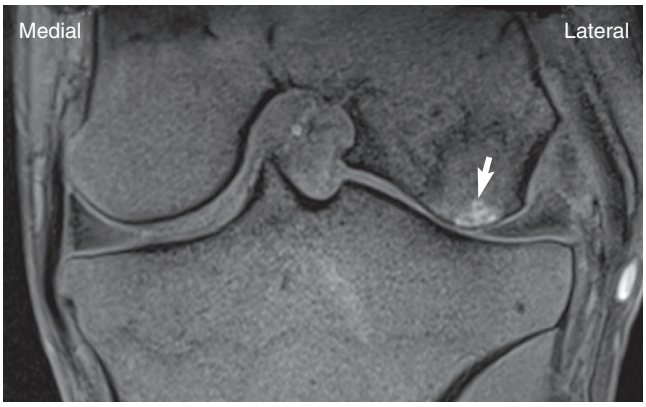


Figure 99-11. A T1-weighted VIBE magnetic resonance image of the stifle in the dorsal plane revealed a large area of bone edema, high signal (with contrast enhancement) in the subchondral bone, and extensive cartilage injury in the lateral femoral condyle *(arrow)* that was not evident on routine radiographs. (Courtesy C.E. Judy, Alamo Pintado Equine Medical Center.)

multiple approaches to the femorotibial joints, regions of the joint (particularly the tibial condyles and abaxial aspect of the menisci) remain inaccessible because distraction of the equine stifle joint is not possible unless collateral ligament injury is present.

Significant postoperative pain is intermittently seen with stifle arthroscopy, particularly if there is extensive débridement of a subchondral cystic lesion of the medial femoral condyle. It can be difficult to predict which cases will have severe postoperative pain. A preoperative epidural with morphine and detomidine has been shown to result in decreased lameness and heart rates after stifle arthroscopy.[38] We also recommend light hand-walking for 5 to 10 minutes three to four times per day beginning 24 hours after surgery to decrease the degree of postoperative discomfort.

Complete arthroscopic examination of the stifle necessitates exploration of six separate synovial compartments. However, this is rarely completed because joint compartments are usually explored based on clinical and radiographic findings that localize lesions to one or more aspects of the stifle. The horse is placed in dorsal recumbency for all approaches, and ideally the distal limb is supported by a hoist, such that flexion of the limb can be adjusted as needed. For all approaches, the stifle is placed at 90 to 100 degrees of flexion, with the exception of the femoropatellar joint, which is best completed at 120 to 130 degrees of flexion.

Each of the two femorotibial joint compartments can be divided into cranial and caudal pouches. The caudal pouch of the LFT is divided into proximal and distal pockets by the popliteal tendon. The following compartments can be explored:

- The FPT
- The cranial pouch of the MFT
- The caudal pouch of the MFT
- The cranial pouch of the LFT
- The proximal pocket of the caudal pouch of the LFT
- The distal pocket of the caudal pouch of the LFT

Femoropatellar Joint

Arthroscopy of the FPT may be performed via a cranial approach (Figure 99-12).[39,40] The horse is anesthetized and placed in dorsal recumbency with the stifle joint maintained at 120 to 130 degrees of flexion. An 8 mm long skin incision is made 2 cm medial to the middle patellar ligament and 2 cm distal to the distal aspect of the patella. The incision is extended through the superficial and deep fascia. Without prior joint distention, an arthroscopic cannula containing a blunt trocar is advanced through the incision distad and caudad, pinning the joint capsule between the trocar and the femur. A rapid but gentle advancement of the trocar with the cannula laterad and distad (or alternatively proximad into the trochlear groove) will advance it through the joint capsule. The trocar is replaced by the arthroscope, and after intra-articular location is verified, the joint is distended through the arthroscopic cannula. Sequential examination of the FPT includes evaluation along the entire length of both the medial and lateral trochlear ridges, the articulating surface of the patella, the attachments of both the medial and lateral patellar ligaments onto the patella, and the suprapatellar pouch. Instrument portal placement is completed with spinal needle guidance to obtain an optimal location for débridement of the specific lesion encountered.

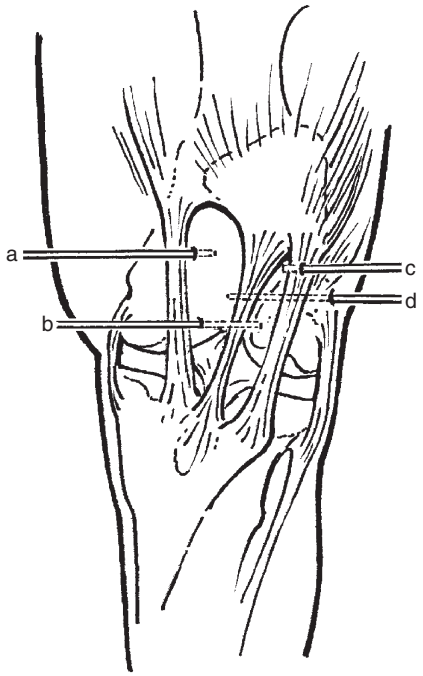


Figure 99-12. A schematic representation of the arthroscope portals used to explore the cranial pouches of the MFT, LFT and the FPT. *a,* The portal used to position the arthroscope in the FPT; *b,* the portal used to position the arthroscope in the LFT (medial approach); *c,* the portal used either to position the arthroscope in the FPT for closer examination of the lateral trochlear ridge, or as an instrument portal to débride the lateral trochlear ridge; *d,* the portal used to position the arthroscope in the MFT (lateral approach).

Arthroscope portal placement 2 cm distal to the patella allows the surgeon to subsequently enter the MFT or LFT via the cranial approach[39-41] and thus is our preferred approach. However, placement of the arthroscope portal in the FPT may be adapted based on the predicted pathology of the joint. For example, placement of the portal further distad—that is, halfway between the tibial crest and patella—facilitates improved access to the distal patella.[42] The most common reason to perform arthroscopy of the FPT is for the treatment of osteochondrosis dissecans of the lateral trochlear ridge, and portal placement is generally best between the medial and middle patellar ligaments, at a level approximately half of the distance between the tibial crest and patella. Portal placement has also been reported through a patellar ligament,[39] although this is likely unnecessary. If the instrument portal is made lateral to the lateral patellar ligament, it may be difficult to manipulate the instrument to operate effectively on lesions in this area.[43]

The suprapatellar pouch can be evaluated by passing the scope between the patella and intertrochlear groove with the limb in maximal extension. In larger horses, a longer scope and instruments are needed to adequately evaluate the suprapatellar pouch and remove debris from this compartment, or alternatively an instrument portal may be placed in the suprapatellar pouch with spinal needle (8.9 cm 18 gauge) guidance. A direct arthroscopic approach to the suprapatellar pouch has been evaluated in cadaveric limbs and clinical cases of sepsis and proximal OCD lesions and for removing debris.[44] Optimal portal placement is 2 cm proximal to the patellar base, and approximately 10 cm lateral to the longitudinal patellar axis in

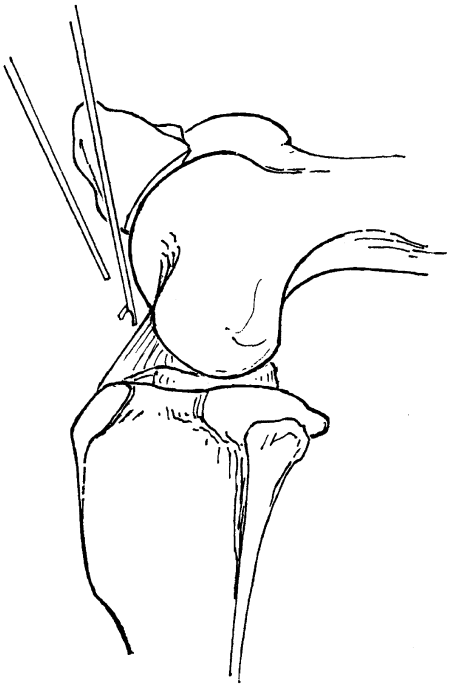


Figure 99-13. The cranial arthroscopic approach to the LFT. The arthroscope and hook scissors are drawn in the appropriate intraoperative positions, with the skin portals placed close to the distal border of the patella.

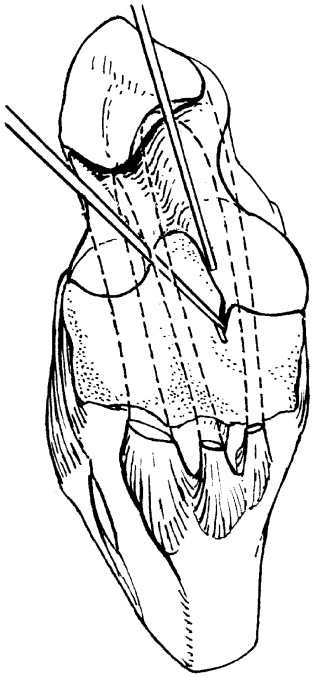


Figure 99-14. The cranial arthroscopic approach to the LFT. The arthroscope and hook scissors bisecting the synovium are drawn in the appropriate intraoperative positions. The *dotted lines* represent the medial, middle, and lateral patellar ligaments.

the intermuscular septum between the biceps femoris and vastus lateralis muscles. A lateral instrument portal can be made cranioproximally to the previous portal or cranially along the longitudinal axis of the patella in the most proximal aspect of the pouch. Medial portals are not recommended because of decreased maneuverability from abdominal interferance and thick musculature at the site and the risk of damage to major nerves and vessels.

Medial Femorotibial Compartment

Arthroscopic approaches to the cranial MFT are well established.[39,45-48] The two most common approaches are the cranial approach[40] and the lateral approach.[47]

The cranial approach involves placing the arthroscope in the FPT, with secondary entry into the cranial pouch of the MFT (and/or LFT), using arthroscopic hook scissors to incise the synovial fold at the distal end of the trochlear ridges (Figures 99-13 and 99-14).[40] Specifically, with the stifle joint at 90 to 100 degrees of flexion, the FPT is entered as described earlier, with the arthroscope portal placed 2 cm distal to the distal aspect of the patella and 2 cm medial to the middle patellar ligament. Following examination of the FPT, an instrument portal is created in the FPT 2 cm lateral to the middle patellar ligament, at the same level as the first arthroscopic portal. Arthroscopic hook scissors (Figure 99-15) are introduced through this portal and are used to elevate and cut (Figure 99-16) the slitlike synovial membrane identified overlying the cranial and distal aspects of the medial femoral trochlear ridge. The arthroscope is then advanced through the slit into the MFT (Figure 99-17). In cases of chronic joint disease, where the synovial membrane is thickened, the hook scissors may not be effective, and in such cases a scalpel blade must be used to transect the fibrous synovial membranes.

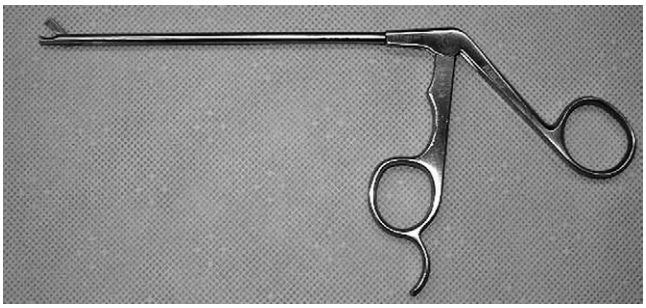


Figure 99-15. Hook scissors used in the cranial arthroscopic approach to the stifle joint.

We have identified multiple benefits to the cranial approach. It permits a controlled and atraumatic entry into the MFT and LFT, thereby decreasing the reported risk of inadvertent meniscal damage from blind trocar insertion. Extravasation of fluid into the periarticular tissues, which renders the surgical approach difficult, appears to be less common with the cranial approach. The cranial approach provides equivalent visualization of the femoral and tibial condyles and the cranial aspects of the menisci relative to the other approaches, and it provides an excellent view of the caudal cruciate ligament, which significantly enhances evaluation and débridement of this structure.

A unique feature of the cranial arthroscopic approach is that a large communication is created between the femoropatellar and femorotibial joints. Thus, when curetting medial femoral condylar cysts using this approach, it is important to

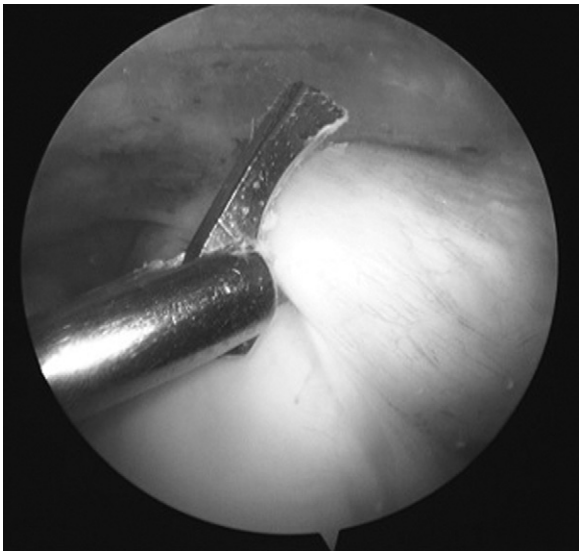


Figure 99-16. Arthroscopic view of the hook scissors engaging the synovium over the cranial and distal aspect of the medial femoral trochlea.

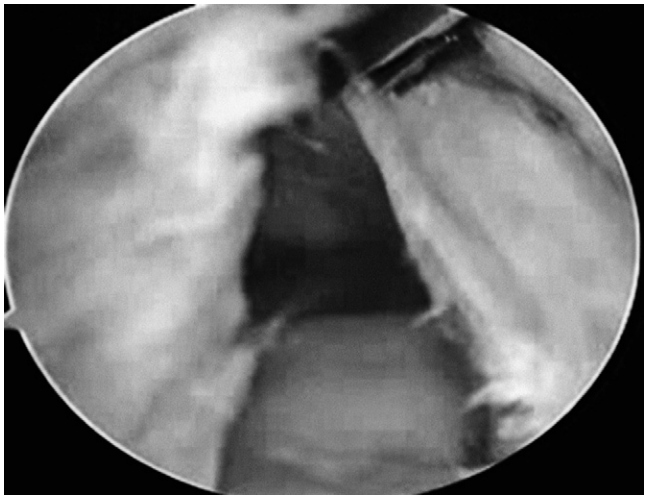
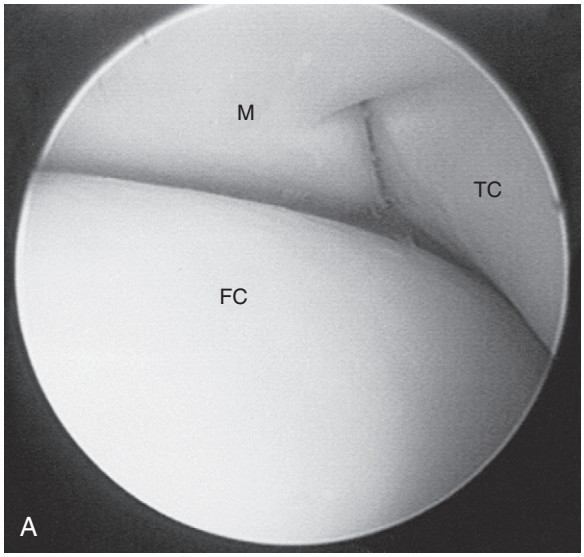


Figure 99-17. Arthroscopic view of the transected synovium located over the cranial and distal aspect of the medial femoral trochlea and the proximal medial femoral condyle. The telescope is advanced through the opening to enter the MFT joint. Limb flexion is also used to enhance the view of the condyle.



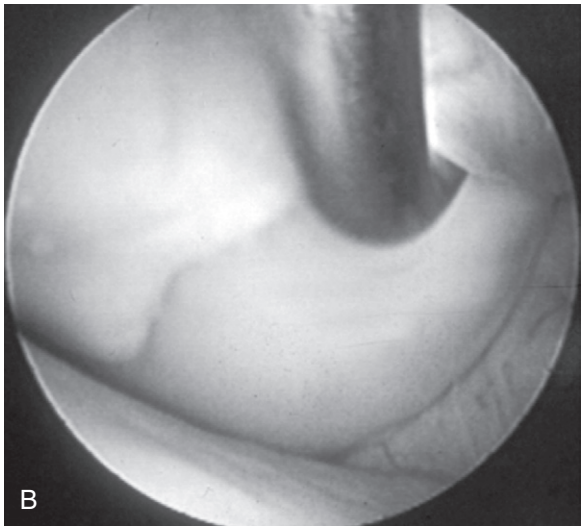


Figure 99-18. A, Arthroscopic view of the cranial pouch of the MFT joint as viewed from the lateral approach showing the medial femoral condyle *(FC)*, the medial meniscus *(M)*, and the tibial condyle *(TC)*. **B,** A probe can be used to elevate the cranial pole of the meniscus and meniscotibial ligament, exposing the medial tibial condyle.

remember to take care to prevent a significant amount of debris from falling into the FPT. It is therefore important to explore the suprapatellar pouch before exiting the joint with the arthroscope.

In cases of synovial sepsis, the cranial approach eliminates the natural synovial barriers between compartments, and care should be taken to avoid contaminating compartments that are not already affected. However, as compartment communication rates are highly variable, each case must be considered individually, and the cranial approach is particularly beneficial for sepsis of the LFT, as access to the sulcus muscularis, a common location of fibrin and debris accumulation, is improved.

The lateral approach is used by many surgeons to evaluate the cranial MFT. The arthroscope portal is located caudal to the lateral patellar ligament, cranial to the long digital extensor tendon, and 2 cm proximal to the tibial crest. With a No. 11 blade, the portal is made through the skin and the deep fascia. Using a blunt trocar, the arthroscopic cannula is directed mediad to penetrate the synovial membrane in the lateral aspect of the MFT (see Figure 99-12). Using this approach, the instrument portal is placed using needle guidance approximately halfway between the tibial crest and the patella and midway between the medial and middle patellar ligaments. This allows examination of the cranial meniscotibial ligament, the cranial aspects of the medial meniscus and tibial condyle, and the weight-bearing surface of the femoral condyle (Figure 99-18). The caudal cruciate ligament can be seen under the synovial membrane (Figure 99-19). Occasionally, the tibial attachment of the cranial cruciate ligament is seen if the synovial membrane of the septum is disrupted.

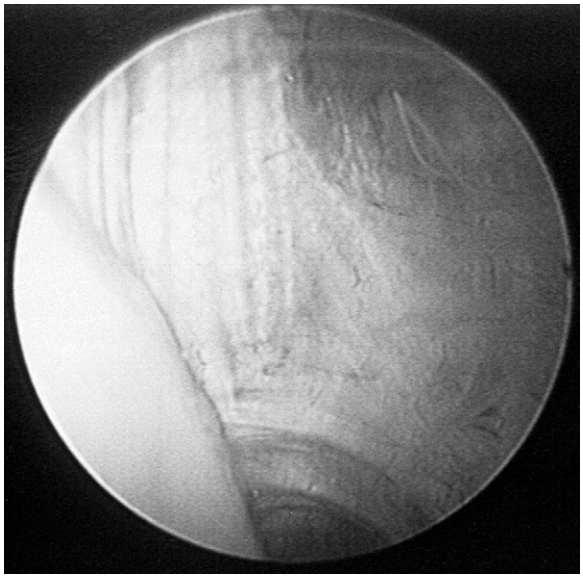


Figure 99-19. Arthroscopic view of the tibial attachment of the caudal cruciate ligament observed through the synovial membrane *(center).*

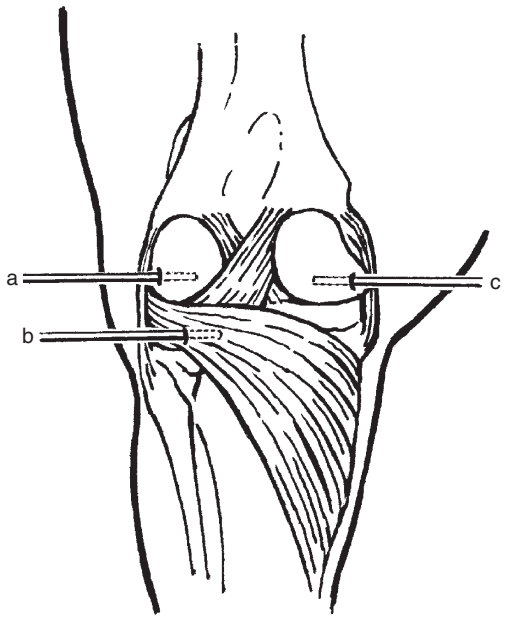


Figure 99-20. Schematic representation of the arthroscopic portals used to explore the caudal pouches of the MFT and LFT. *a,* The portal to the proximal pocket of the lateral caudal pouch is located at the level of the lateral femoral condyle, 2.5 cm proximal to the distal level of the lateral meniscus and 3 cm caudal to the lateral collateral ligaments; *b,* the portal to the distal pocket of the lateral caudal pouch is located at the level of the lateral meniscus, through the popliteal tendon, 1.5 cm caudal to the lateral collateral ligament; *c,* the medial portal is located at the level of the medial femoral condyle, 1 cm proximal to the distal level of the medial meniscus and 3 cm caudal to the medial collateral ligament.

Examination of the caudal pouch of the MFT is best accomplished by locating the arthroscope portal 1 cm proximal and parallel to a line drawn from the tibial crest to the medial condyle of the tibia and 3 cm caudal to the medial collateral ligament[6] (Figure 99-20). Spinal needle placement and distention of the joint to confirm adequate portal location and trajectory for canula/trocar placement is beneficial. The entire caudal aspect of the medial femoral condyle and the proximal aspect of the medial meniscus can be examined (Figure 99-21). The caudal cruciate ligament is located extrasynovially and can be located with a probe through the synovial lining. A similar but more caudal approach, 6 to 8 cm caudal to the medial collateral ligament (1 to 2 cm cranial to the medial saphenous vein, and cranioproximal to the palpable gracilis muscle) allows greater visualization of the axial aspect of the medial meniscus and caudal cruciate ligament.[49]

A cranial intercondylar approach is described for examination of the axial caudal MFT. This approach may be useful when increased visualization of the caudal axial aspect of the medial femoral condyle, the caudal cruciate ligament, and the caudal medial meniscotibial ligament is required.[50,51] With the stifle flexed at 90 to 100 degrees, the scope is inserted with the portal placed between the medial and middle patellar ligaments.[48] The location of the second portal is determined by visualization of a spinal needle advancing into the intercondylar space between the caudal cruciate ligament and medial femoral condyle from just proximal and caudal to the intercondylar eminence. The site of the portal is generally just lateral to the middle patellar ligament, 50% to 60% of the distance from the tibial crest to the patella. A smooth shafted conical obturator is visually guided as described earlier for the spinal needle, and the arthroscope canula is placed over the obturator. Puncture of the caudal joint capsule, scoring of the medial femoral condyle in the intercondylar space, and periarticular fluid accumulation are frequently encountered though of little apparent clincal significance. A caudomedial instrument portal can be created as described earlier for débridement of lesions.

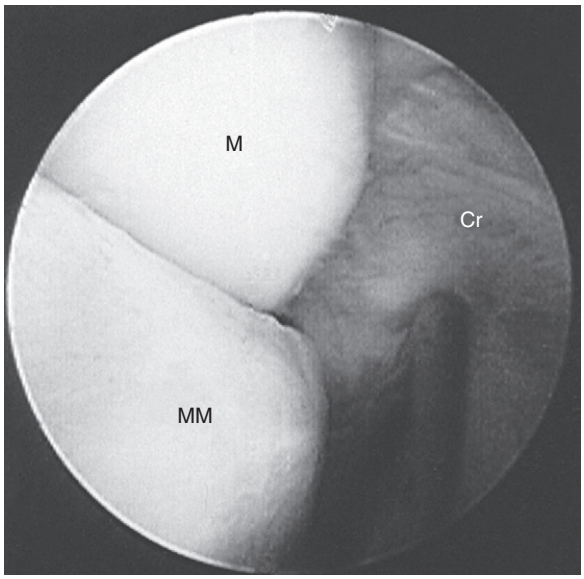


Figure 99-21. Arthroscopic view of the caudal pouch of the MFT joint of the stifle showing the medial femoral condyle *(M),* the medial meniscus *(MM),* and a probe placed on the caudal cruciate ligament *(Cr)* located behind the synovial membrane.

Lateral Femorotibial Compartment

Arthroscopy of the cranial pouch of the LFT is generally performed via a cranial or medial approach. The cranial approach, as described earlier for the MFT, may be used to enter the LFT at the distal end of the lateral trochlear ridge. Arthroscope portals in the FPT may be placed as described earlier, because the arthroscope does not need to be removed from its medial position to be driven into the LFT after the incision is made with the hook scissors at the distal end of the lateral trochlear ridge. Improved exploration of the sulcus muscularis containing the long digital extensor tendon and peroneus tertius is achieved with the cranial approach.

The arthroscope portal for the medial approach to the cranial pouch of the LFT is best located midway between the patella and the tibial crest and halfway between the medial and middle patellar ligaments. The arthroscope is directed caudolaterally to enter the LFT. The cranial cruciate ligament is seen from this approach lying in the medium septum. The lateral femoral condyle, the lateral meniscus, the cranial lateral meniscotibial ligament, and the lateral tibial condyle can be visualized, but this pouch is smaller than the cranial pouch of the MFT, and thus manipulation of the arthroscope is more limited. The opening into the distal synovial diverticulum (the sulcus muscularis) of the long digital extensor tendon and peroneus tertius can be seen; however, exploration of this compartment distad along the tendon is only accomplished through a cranial approach. Collection of fibrin and debris in this diverticulum with synovial sepsis is common, and thus its débridement and lavage is important.

The arthroscope portal that permits the best examination of the proximal pocket of the caudal pouch of the LFT is located 2.5 cm proximal and parallel to a line drawn from the tibial crest to the lateral condyle of the tibia and 3 cm caudal to the lateral collateral ligament (see Figure 99-20, *A*).[6] Spinal needle placement and distention of the joint to confirm adequate portal location and trajectory for canula/trocar placement is beneficial, as for all caudal approaches to the stifle. Most of the caudal aspect of the lateral femoral condyle and tendon of the popliteus muscle can be examined intrasynovially. Rotation of the tarsus laterally (clockwise rotation of the right hindlimb) loosens the popliteal tendon and creates a tunnel. Insertion of the arthroscope into the tunnel still does not permit visualization of the lateral meniscus or of the tibial condyle.

The arthroscope portal that permits examination of the distal pocket of the caudal pouch of the LFT is located on a line drawn from the tibial crest to the lateral condyle of the tibia and 1.5 cm caudal to the lateral collateral ligament (see Figure 99-20, *B*). Entrance at this level is directly through the popliteal tendon, permitting examination of the caudal lateral meniscus, part of the caudal aspects of the lateral femoral and tibial condyles, and the intra-articular portion of the popliteal tendon (Figure 99-22). Care should be taken when approaching the caudal pouch of the LFT to avoid damage to the common peroneal nerve, which is located less than 7 cm caudal to the lateral collateral ligament. Additionally, the popliteal artery and vein are situated directly between the medial and lateral femoral condyles on the caudal aspect of this joint (see Figure 99-9). Therefore, it is not safe to pass from one pouch to the other across the caudal aspect of the joint without risk of injury to this vasculature, which is the major deep blood supply to the stifle.[52]

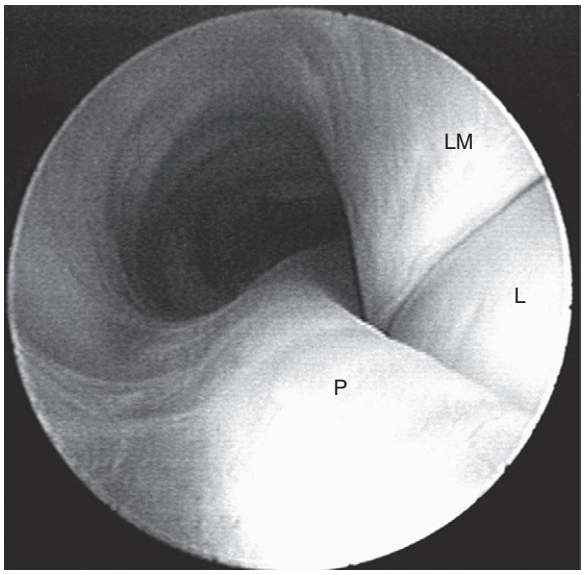


Figure 99-22. Arthroscopic appearance of the distal pocket of the caudal pouch of the LFT, which permits examination of the caudal aspect of the lateral meniscus *(LM)*, part of the caudal aspect of the lateral femoral condyle *(L)*, and the intra-articular portion of the popliteal tendon *(P)*.

DISORDERS OF THE STIFLE

Many stifle disorders present significant sources of acute or chronic lameness for equine athletes. Developmental or traumatic etiologies for these lesions are commonly suspected or identified. The most commonly identified stifle disorder, osteochondrosis, traditionally classified as a developmental condition, is believed to have an element of trauma in its pathogenesis. Soft tissue lesions are a significant source of lameness in other species, and recognition of these lesions in horses is increasing. Osteoarthritis commonly develops secondary to stifle joint pathologies or injuries that lead to joint instability or cartilage trauma and inflammatory mediator release. However, primary development of stifle joint osteoarthritis may be seen without identification of underlying pathologies.

Osteoarthritis

Early signs of osteoarthritis in the equine stifle are most commonly recognized as cartilage damage over the medial femoral condyle, which occurs secondary to joint injury or trauma.[53] These lesions produce lameness in the horse, and diagnostic arthroscopy may be necessary to make an accurate diagnosis.[54] Therefore, horses with lameness localized to the stifle through diagnostic anesthesia or scintigraphy that had no significant radiographic and ultrasonographic findings are good candidates for arthroscopic exploration of the joint. Débridement of focal cartilage lesions allows some horses to successfully resume performance activities.

Numerous small horizontal clefts in the cartilage over the weight-bearing surface of the medial femoral condyle represent pathologic lesions recognized as a source of lameness and poor performance, particularly in Quarter Horses (Figure 99-23). Lesions are generally only identified on arthroscopic examination. Débridement of the lesions would require extensive removal of articular cartilage and thus is generally not

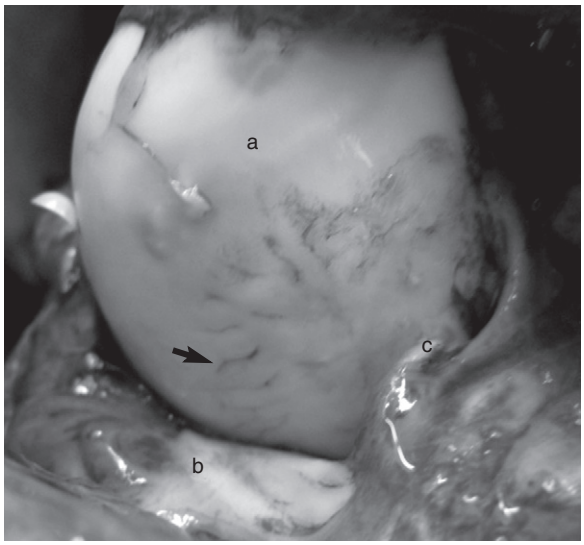


Figure 99-23. Postmortem dissection showing horizontal clefts *(arrow)* in the articular cartilage on the weight-bearing surface of the medial femoral condyle *(a)*. *b,* Cranial horn of the medial meniscus; *c,* medial intercondylar eminence of the tibia.

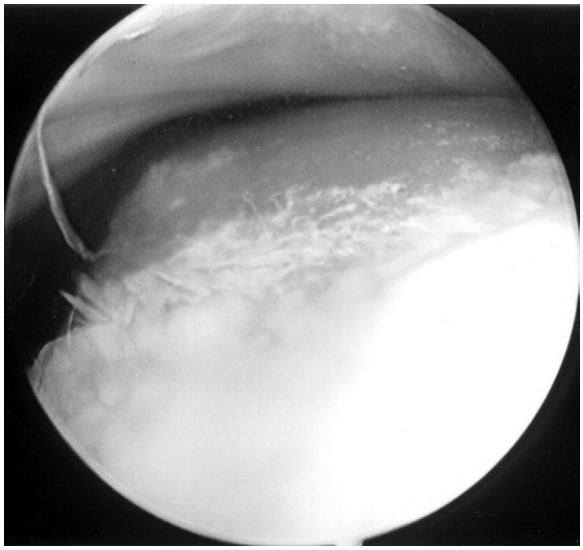


Figure 99-25. Arthroscopic appearance of advanced osteoarthritis of the medial femoral condyle. Articular cartilage débridement was accomplished using a motorized resector.

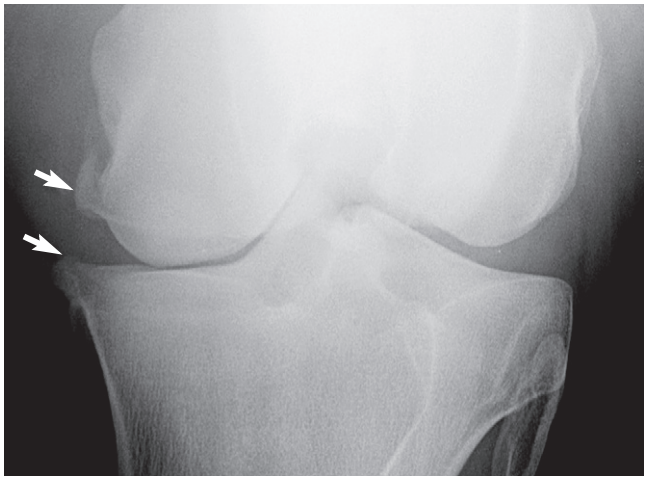


Figure 99-24. A caudocranial radiographic view of a stifle with advanced osteoarthritis showing narrowing of the MFT joint space and large enthesiophytes on the distal femur and proximal tibia *(arrows).*

completed. If recognized early, the lesions may improve with prolonged rest (i.e., 4 to 6 months), although the prognosis for long-term soundness is poor. In our opinion, continued training (with corticosteroid joint injection) is contraindicated because irreversible rapid progression of the cartilage degeneration is likely to occur.

Osteoarthritis of the stifle in the later stages is best detected on a craniocaudal radiograph when the FPT is involved and manifests as articular osteophyte formation, flattening of the articular surfaces, sclerosis of the subchondral bone, or lucent zones in the subchondral bone. Lipping (osteophyte formation) of the medial tibial plateau is most commonly the first recognizable radiographic sign of femorotibial osteoarthritis. Narrowing of the femorotibial joint space is seen in advanced cases (Figure 99-24). Positioning is important when making this determination; the horse should be bearing weight, with the limb centered under the body during this view. The lateromedial view best demonstrates FPT osteoarthritis, which is seen as periarticular osteophyte formation and changes along the distal aspect of the patella. Treatment for osteoarthritis of the stifle involves evaluation for and potentially treatment of any underlying pathologies (i.e., meniscal tears), followed by palliative treatment of the arthritis. As with all other arthritic conditions, treatment is directed at decreasing pain/inflammation and chondroprotection with intra-articular and systemic medications. Because of the size of these synovial compartments (with volumes between 40 and 62 mL), intra-articular medications must be of sufficient quantity to be effective.

Osteoarthritis is commonly established in the femorotibial joint secondary to meniscal or ligamentous injury; however, advanced cases of osteoarthritis with complete erosion of articular cartilage from the medial femoral condyle can be seen (particularly in aged racing Standardbreds) without evidence of other soft tissue injury (Figure 99-25). Treatment of this condition by arthroscopy may include articular cartilage débridement and micropick techniques (Figure 99-26). Microfracture has been shown to improve healing in full-thickness chondral defects of the medial femoral condyles.[55] Significant research into cartilage healing and resurfacing techniques has been completed including evaluation of autologous chondrocyte/cartilage fragment transplantation via a collagen membrane or polydioxanone scaffold,[56,57] interleukin-1 receptor antagonist/insulin-like growth factor-1 gene therapy,[58] and arthroscopic mesenchymal stem cell implantation (see Chapter 80).[59] Positive results from these studies indicate that greater availabilty and application of these techniques may be valuable.

Cartilage lesions on the articular surface of the patella are referred to as *chondromalacia* (Figure 99-27). These changes may be seen as a part of diffuse degenerative joint disease occuring within the stifle. The pathogenesis and significance of this condition in the horse are uncertain, but arthroscopic débridement of cartilage fragmentation using minimally traumatizing motorized cartilaginous débriders generally results in clinical improvement.

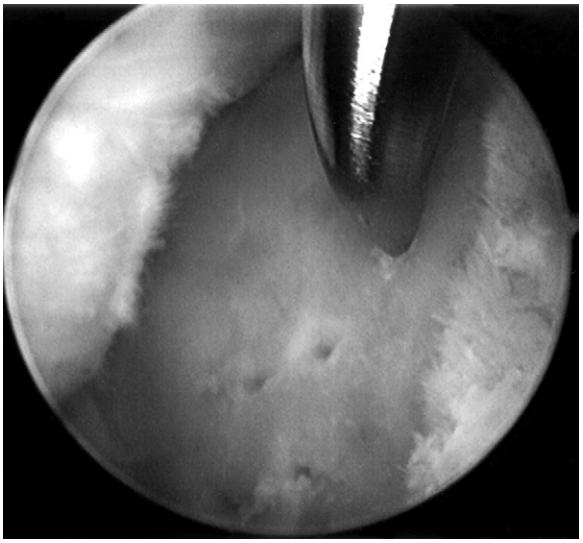


Figure 99-26. Arthroscopic appearance of the medial femoral condyle showing micropick technique.

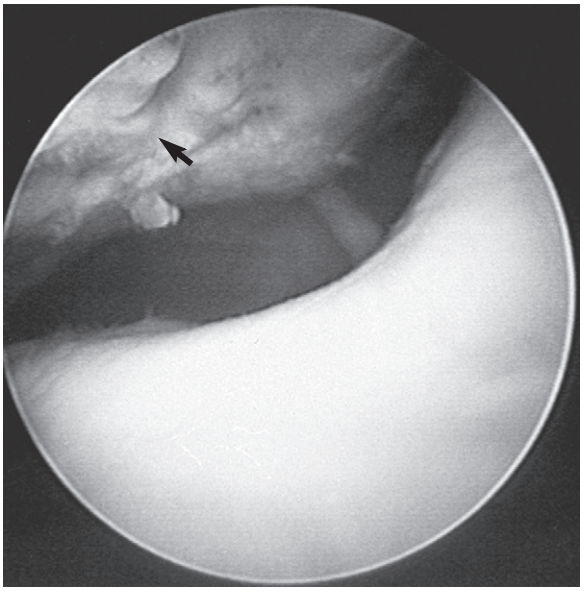


Figure 99-27. Arthroscopic appearance of chondromalacia of the patella *(arrow)*. The intertrochanteric fossa of the femur is seen ventrally.

Soft Tissue Disorders

In addition to collateral ligaments common to most joints, the stifle of the horse includes five ligaments associated with the menisci, cranial and caudal cruciate ligaments, and three patellar ligaments. All the soft tissues of the stifle are subject to sprains and tears.

Collateral Ligament Injury

The femorotibial joint has two large collateral ligaments at its medial and lateral aspect. They orignate on the femoral epicondyles and insert on a roughened area just distal to the margin of the tibial condyle (medial) and the head of the fibula (lateral). Both ligaments consist of a single bundle of fibers, in comparison to a deep and superficial portions as seen in the medial collateral ligament of humans. In my experience, the medial collateral ligament (MCL) has fibers from its cranial aspect insert into the adjacent abaxial aspect of the medial meniscus in approximately 15% of horses, whereas the lateral collateral ligament (LCL) has no attachment to the lateral meniscus. The popliteal tendon runs under the LCL with a bursa interposed between the two. A second bursa is present between the LCL and the tibial plateau.

Collateral ligaments of the femorotibial joint may undergo partial or complete tearing.[60-62] Diagnosis may be evident in acute cases with abnormal range of motion and/or ultrasonographic changes. Radiographs may be obtained with the joint under lateral or medial stress to confirm unilateral widening of the femorotibial space when complete rupture is suspected. Sprain of these ligaments can result in enthesiophyte formation at their attachments on the distal femur and proximal tibia.

Treatment for this condition involves anti-inflammatory medication and rest. Healing should be monitored by periodic ultrasonographic examinations. Surgical repair of a complete MCL rupture has been attempted with unsuccessful long-term results, and thus euthanasia is often recommended.[60,62] The prognosis for horses undergoing partial tearing is guarded to favorable, depending on the degree of tearing and instability of the joint.[63]

Concurrent injury of the menisci or cruciate ligaments commonly occurs in horses with severe injuries or complete rupture of a collateral ligament. The MCL and medial menisci are more commonly involved than the lateral aspect of the stifle. When concurrent structures, such as the cruciate ligaments or menisci, are involved, the prognosis is unfavorable for return to athletic use.

Patellar Ligament Injury

The lateral, middle, and medial patellar ligaments transmit the pull of the quadriceps muscle to the tibia through their origin on the distal patella and insertion on the tibial tuberosity. Patellar ligament disease has been recognized as a primary injury, with jumping horses appearing to be over-represented.[64] Injury to the patellar ligaments should be kept in mind for hindlimb lameness that cannot be blocked out (or shows only mild improvement with intra-articular stifle anesthesia), and the patellar ligaments should be carefully palpated with the limb loaded and unloaded. Ultrasonography is the best diagnostic tool for evaluating the patellar ligaments. Nuclear scintigraphy may localize a lameness to this region; however, false positives with scintigraphy have been recognized.[64] Based on a small number of horses with patellar ligament injury, the prognosis for complete recovery with conservative management is guarded.[64] Evaluation of the patellar ligaments in relation to upward fixation of the patella is discussed later in this chapter.

Cruciate Ligament Injury

The cranial cruciate ligament arises from the lateral wall of the intercondyloid fossa of the femur and runs craniad and distad ending on the central fossa of the tibial spine. The caudal cruciate ligament runs medial to the cranial cruciate ligament, and it arises in the cranial aspect of the femoral intercondyloid fossa, inserting on an eminence at the popliteal notch of the tibia. Both cruciate ligaments are extrasynovial between the MFT and

LFT. The cruciate ligaments may undergo partial or complete tearing and represent a recognized, although relatively uncommon, condition of the equine stifle. The cruciate test may be attempted in cooperative horses, but it is rarely diagnostic because of guarding of the limb. Complete rupture of the cranial cruciate ligament, or less commonly the caudal cruciate ligament, carries an unfavorable prognosis for the horse's return to athletic function.[65-68] Lameness from severe tears may improve over time, but often extensive osteoarthritis prohibits even salvage of the horse as a breeding animal.

Partial tearing of the cranial cruciate ligament occurs most commonly at the mid-body segment, but partial detachment of its insertions can occur. The cranial cruciate ligament is under tension when the stifle joint is in extension, and subsequently hyperextension and/or rotation of the stifle joint are suspected to be involved in its injury. Radiographs may identify enthesiophyte formation or occasionally an avulsion fracture of the medial intercondylar eminence of the tibia (MICET) or, more rarely, the femoral attachment (see Figure 99-5). Fractures of the MICET may be seen with relatively little disruption of the cruciate ligament, and thus some of these fractures may be caused by a traumatic lateral force from the medial femoral condyle as compared to the more typical pathogenesis of avulsion fractures. Proliferative bone changes at the MICET (best seen on a flexed lateromedial radiograph) may be present in chronic cases and was seen in 7% of cruciate injuries in one study.[62] Subchondral cystic lesions can develop at the sites of origin and insertion of the cruciate ligaments over time.

A definitive diagnosis is made after arthroscopic examination of the femorotibial joint. Owners should be warned prior to surgery of the risk that a partial cruciate tear may progress to a complete tear upon recovery from anesthesia, and a smooth, assisted recovery should be executed. A lateral or cranial approach to the MFT may be used, and the septum between the two cranial compartments typically have been ruptured with the traumatic injury, allowing examination of the typically extrasynovial cruciate ligaments through this approach (Figure 99-28). Treatment involves débridement of the damaged ligamentous tissue and bone fragments. Successful repair or replacement of complete tears has not been reported in horses.

Secondary cartilage disease was seen in 61% of cases in one study, and recovery rates for grade I, II, and III lesions were 46%, 59%, and 33%, respectively.[62] With regard to MICET fractures, removal and débridement of a large MICET fragment and sprain of about 25% of the tibial insertion of the cranial cruciate ligament in one horse[69] and internal fixation of a MICET fracture in another horse[70] have been reported with successful outcomes. In our experience, a majority of horses with minor superficial cranial cruciate tearing (Figure 99-29) can return to previous degrees of activity. However, when the damage becomes moderate and approaches disruption of greater than 50% of the ligament, or when there are multiple lesions in the joint (i.e., a meniscal or collateral ligament tear) the prognosis is unfavorable.

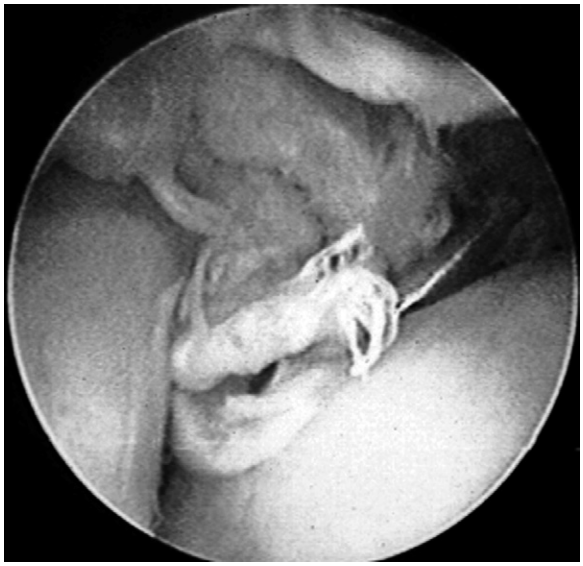


Figure 99-28. Endoscopic view of a torn cranial cruciate ligament as viewed from the cranial approach to the MFT. A probe easily separates the fibers of this ligament.

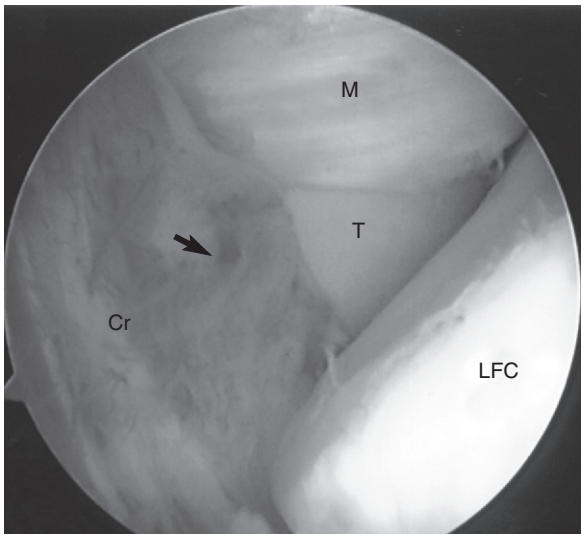


Figure 99-29. Arthroscopic appearance of a minor tear *(arrow)* in the cranial cruciate ligament *(Cr)* as seen in the LFT. The lateral femoral condyle *(LFC)* and the meniscotibial ligament *(M)* and tibial condyle *(T)* are visible.

Meniscal Injury

The menisci are paired semilunar fibrocartilaginous discs interposed between the convex femoral and relatively flat tibial condyles. Four meniscotibial ligaments at the axial aspects of the cranial and caudal horns are the sites of attachment to the tibia and an additional meniscofemoral ligament attaches the caudal horn of the lateral meniscus to the femur. One report documented meniscal tears as the most common soft tissue lesion in the stifle, as they were diagnosed in 68% of soft tissue stifle cases.[24] History may include trauma or a fall, but an insidious onset is common. Lameness is generally more severe initially and mild to moderate on average at the time of presentation.[62] Only about 39% of cases show joint effusion and only 66% show increased lameness with high flexion tests, and thus intra-articular anesthesia is frequently necessary to definitively localize the lameness to the stifle. Severe traumatic injuries may additionally result in injury to other major structures, such as the collateral ligaments and/or the cruciate ligaments, although primary meniscal injuries are more common.[62,68,71] Approximately half of all cases show radiographic changes including new bone at the medial

intercondylar eminence of the tibia (29%), generalized osteoarthritic changes (23%), and mineralization of the meniscus (8%).[72] With severe disruption of the meniscus, collapse of the femorotibial joint space may be evident on caudocranial radiographs. Ultrasonography is beneficial for recognition of meniscal tears, but the specificity and sensitivity of this technique are less than optimal.[24] Large bore MRI's have proven to be valuable in determining the extent of meniscal injury and assessing concurrent lesions.[32] (See "Diagnostic Imaging," earlier.) Arthroscopy (see Figure 99-28) allows direct visualization and assessment of the lesion, but horizontal tears and tears in a large portion of the abaxial menisci cannot be visualized.

Clinically, an isolated lesion of the cranial horn of the medial meniscus and its associated meniscotibial ligament is the most commonly arthroscopically identified site of meniscal lesions in horses.[62,72] The medial meniscus is involved in 79% of cases, and unlike meniscal tears in dogs and humans, only 14% of equine meniscal tears were associated with cranial cruciate ligament injury.[62] Horses with severe trauma and multiple soft tissue structure injury may be more likely to be euthanized prior to arthroscopy and definitive diagnosis, and thus may be underrepresented in case series studies. A grading system for cranial meniscal tears has been established[72]:

Grade I: Tears extending longitudinally down the cranial meniscotibial ligament into the cranial horn of the meniscus with minimal separation of tissues (Figure 99-30).
Grade II: Tears of similar orientation to grade I tears but with further separation of tissue, where the extent of the injury remains fully visible on arthroscopic exam.
Grade III: Severe tears that extend beneath the femoral condyle and cannot be fully visualized arthroscopically (Figures 99-31 and 99-32).

Diffuse degenerative changes to the menisci, particularly the axial aspect of the medial meniscus, have been identified with concurrent osteoarthritis of the femorotibial joint in older horses on postmortem examination and may be more common than currently diagnosed.

Treatment for equine meniscal tears consists of arthroscopic débridement (partial meniscectomy using motorized meniscal resectors) via a lateral, medial or cranial approach. Suturing of meniscal tears has been documented as difficult and

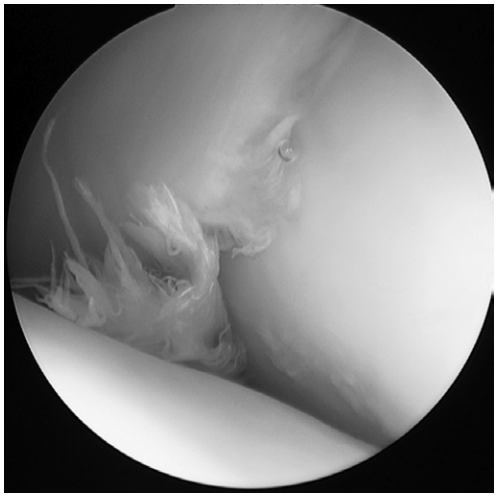


Figure 99-30. Example of a grade I meniscal tear that can be resected.

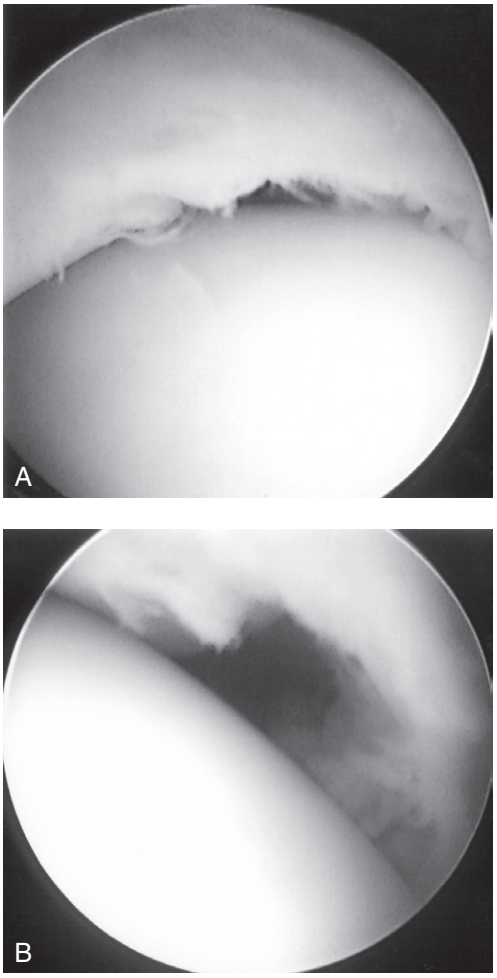


Figure 99-31. The arthroscopic appearance of a chronic medial meniscal tear before **(A)** and after **(B)** meniscal débridement.

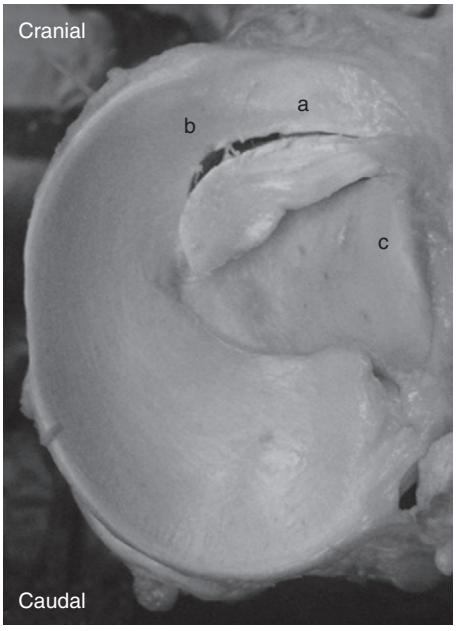


Figure 99-32. Proximal to distal view of the medial meniscus from the right stifle of a cadaveric specimen used to model a grade III meniscal tear. The tear extends longitudinally through the cranial meniscotibial ligament *(a)* into the cranial horn of the medial meniscus *(b)*. *c,* Medial intercondylar eminence of the tibia.

unsuccessful, although successful outcomes have been reported.[48,73] Typical postoperative recovery consists of 6 weeks of stall rest with hand-walking, and at least 6 months of small paddock rest, depending on the severity of injury.

Return to previous athletic function was seen in 63% of horses with grade I tears, 56% with grade II, and 6% with grade III cranial horn meniscal tears.[72] Articular cartilage disease was seen in 71% of cases at the time of diagnosis and had a negative effect on long-term prognosis. Loss of the equitable load transmission function of the meniscus may lead to secondary cartilage injury, particularly in the central portion of the medial femoral condyle. Radiographic changes, such as dystrophic mineralization of the meniscus, also appear to lower the prognosis of return to athletic function. Severe injuries involving multiple structures generally have a poor prognosis. Of 19 horses with meniscal tears and subchondral bone cysts diagnosed concurrently or sequentially, only 4 had a successful outcome.[74]

The pathogenesis of primary meniscal tears has not been clearly delineated, but research indicates that hyperextension leading to significant compression and cranial displacement of the cranial horn of the medial meniscus may place this region at greater risk of injury.[75] Potential etiologies for meniscal injury in association with medial femoral condyle subchondral cystic lesions include a single traumatic incident resulting in both lesions, or alterations in femoral condyle geometry and/or the resultant rim of bone at the débrided defect, resulting in meniscal trauma.[74]

Fractures

Patella

The patella is the largest sesamoid bone in the body and an integral portion of the quadriceps apparatus. Fractures of the patella are most commonly the result of direct trauma to the stifle from jumping over a fixed object or a kick from another horse. Patellar fractures result in variable lameness, soft tissue swelling, and effusion, depending on the amount and configuration of the fracture. Horses with patellar fractures commonly hold the limb in partial flexion, resting the limb with the toe touching the ground, and walk with a careful but weight-bearing gait. Radiographic evaluation should include not only the craniocaudal, lateromedial, flexed lateromedial, and caudal 30-degree lateral craniomedial oblique views but also the craniproximal craniodistal (skyline) view of the patella to thoroughly assess the fracture. Patellar fracture morphology is highly variable; however, sagittal fractures are most common and usually involve the medial aspect of the patella (see Figure 99-8). Fragmentation of the distal aspect of the patella is most commonly associated with medial patellar ligament desmotomy.

Techniques for partial patellectomy and internal fixation have been described; however, fractures located at the base of the patella commonly do not require surgical intervention (Figure 99-33).[76-78] Surgery should be attempted only if there is complete disruption of the quadriceps apparatus (i.e., inability to extend the stifle), the fracture gap is greater than 5 mm on radiographs, there is obvious malalignment of an articular fragment, or the fragments can be palpated under the skin. One should allow the swelling to decrease before surgical intervention is attempted. Medical management of a patella fracture should include administration of nonsteroidal anti-inflammatory drugs for 2 weeks and stall confinement for 2 to 3 months.

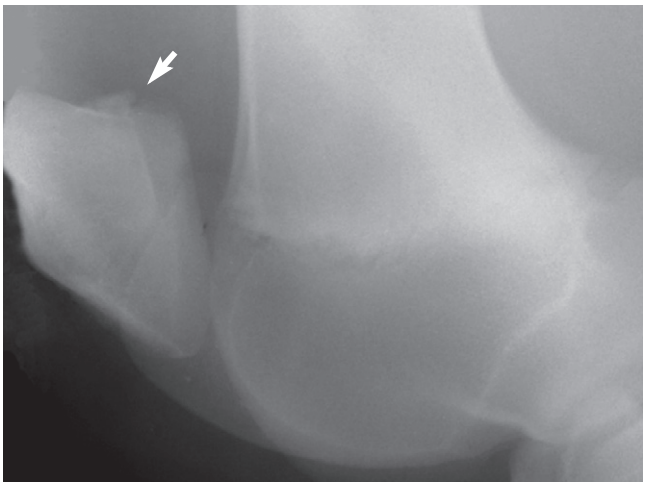


Figure 99-33. Lateromedial radiographic view of the stifle showing a fracture at the base of the patella *(arrow)*.

When there is disruption of the quadriceps apparatus or a very large displaced articular fragment, internal fixation is indicated (Figure 99-34). Screw fixation using lag technique with 5.5 mm cortex screws and/or application of small DCP or LCP plates may be performed via arthrotomy or arthroscopy of the FPT. Partial patellectomy is indicated in sagittal medial articular fractures.[79,80] This can be carried out by removing up to one third of the patella arthroscopically. Portal placement should be about one third to one half of the distance between the distal aspect of the patella and the proximal tibia; portal placement too close to the patella makes it difficult to view fractures on its distal aspect. A mechanical resector and an arthroscopic scalpel are essential tools to separate the fragment from the parent bone. With an osteotome, the fragment is divided into smaller fragments, which subsequently are removed with Ferris-Smith rongeurs. Although it is undesirable, it may be necessary to perform an arthrotomy, to dissect the fragment from its extensive patellar ligament attachments. Concurrent direct trauma to the lateral trochlear ridge is common, and should be evaluated and débrided arthroscopically. The prognosis after partial patellectomy for sagittal transverse fractures is favorable, with reports of an 83% to 100% return to full athletic function in cases with no pre-existing stifle arthritic changes.[80,81] When the quadriceps apparatus has been disrupted, the prognosis is guarded to unfavorable, depending on the success of the repair.

Femoral Trochlear Ridges

Fractures of the femoral trochlear ridges usually result from external trauma to the stifle and resultant fragmentation of the medial or lataral trochlear ridge.[82] They may occur in concert with patellar fractures. Clinical signs include a sudden onset of moderate to severe lameness and effusion of the FPT. Crepitus may be present on flexion of the joint. The fracture site might not be obvious unless a wound is present; however, the potential for joint sepsis must be investigated when a skin wound is present.

Fracture fragments should be removed, which is generally possible with arthroscopy. Trochlear ridge fractures have a good prognosis after fracture removal, if there is no significant damage

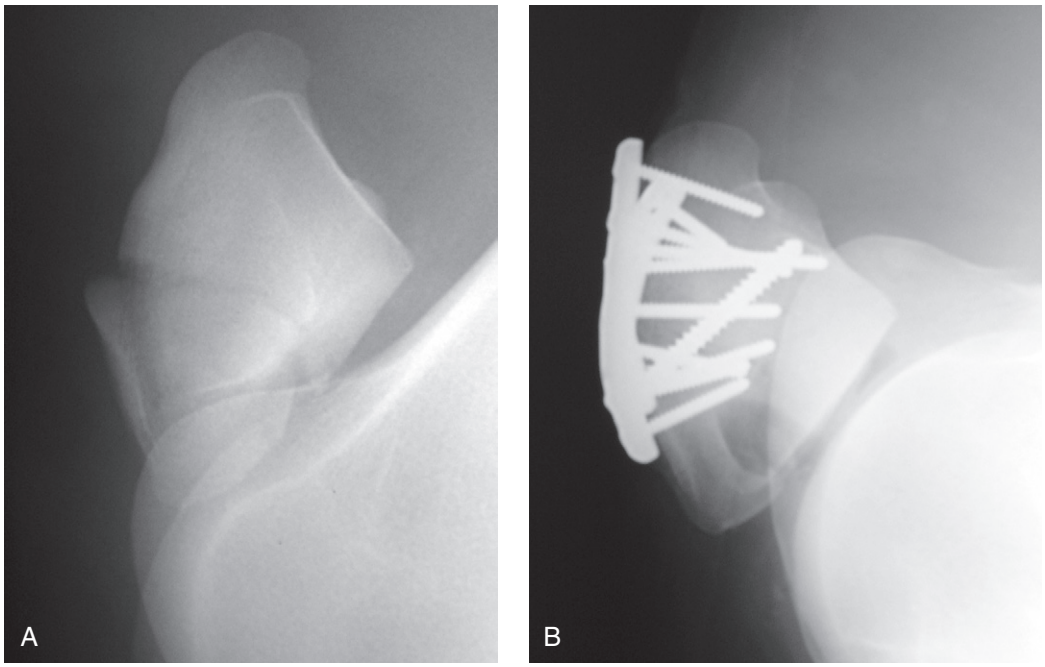


Figure 99-34. A, A lateromedial radiographic view of a patella fracture that resulted in complete disruption of the quadriceps apparatus. **B,** One 5.5-mm cortex screw used in lag fashion and two dynamic compression plates (DCPs) were used in the repair.

to deeper structures or extensive loss of the trochlear ridge. Because it is a non–weight-bearing articular surface where this usually occurs, horses can usually return to complete athletic activity.

Avulsion of the Origin of the Peroneus Tertius and Long Digital Extensor Tendon

The peroneus tertius and long digital extendor tendon arise from a common origin in the extensor sulcus just lateral to the distal aspect of the lateral trochlear ridge. Avulsion of the origin is commonly caused by a sudden forceful hyperextension of the hindlimb, particularly when the distal limb is entrapped. Foals are most likely to suffer avulsion fractures of the peroneus tertius origin, whereas tears of the body or insertion site in the tarsal region is more common in adult horses.[83]

Clinical signs are similar to those found with rupture of the peroneus tertius (see Chapters 83 and 97). However, distention of the femorotibial and femoropatellar joints is a cardinal sign of a proximal avulsion fracture. The condition can be diagnosed radiographically (Figure 99-35).

Treatment is usually initiated by arthroscopic evaluation, which offers the opportunity to examine the stifle for concurrent injury. Arthroscopy of the LFT can be completed via a cranial approach. The subchondral bone in the area of the extensor fossa and origin of the long digital extensor tendon can be débrided and bone fragments removed. Prognosis following this injury is guarded because many horses continue to have some degree of lameness following this injury.[83,84]

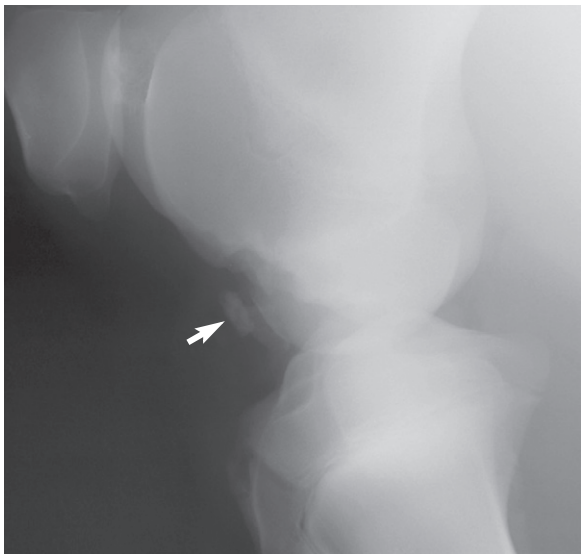


Figure 99-35. Lateromedial view of the stifle in a foal showing avulsion *(arrow)* of the femoral origin of the peroneus tertius and long digital extensor tendon.

Osteochondrosis

Osteochondrosis (OC) is a term used to describe the developmental cartilaginous disease where failure of endochondral ossification results in thickening of the metaphyseal growth plate or the articular cartilage.[85,86] The etiopathogenesis of osteochondrosis and subchondral cystic lesions are discussed in detail in Chapters 88 and 89. For the purpose of discussion in this chapter, OC lesions have been divided into two categories: osteochondritis dissecans (osteochondral fragments) and subchondral cystic lesions (bone cysts).

Osteochondritis Dissecans

Osteochondritis dissecans (OCD) is the most common cause of lameness in the stifle, and the FPT is one of the principal sites

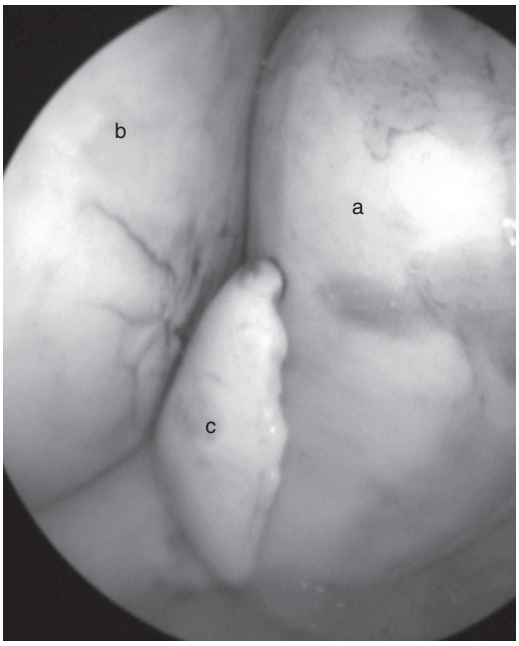


Figure 99-36. Arthroscopic appearance of osteochondritis dissecans of the lateral trochlear ridge *(a)* demonstrating an osteochondral flap *(c)* and an adjacent kissing lesion at the lateral aspect of the patella *(b)*.

of OCD in the horse.[87] The primary area involved is the lateral trochlear ridge of the femur (Figure 99-36). The medial trochlear ridge, the patella, and the trochlear groove are involved less often.[87] Bilateral involvement is common. OCD has been reported in many breeds, but the Thoroughbreds, Quarter Horses, Arabians, and Warmbloods are over-represented. Most cases of OCD are diagnosed in horses between 1 and 2 years of age.

Radiographic examination of Thoroughbreds at yearling sales allowed the prevalence of osteochondrosis of the stifle to be determined in this group.[88] The most common osteochondrosis lesion observed is flattening or fragmentation of the lateral trochlear ridge of the femur (6% of 660 horses).[89] This lesion has a significantly detrimental effect on future performance in racing Thoroughbreds.

Affected horses may present with acute, severe lameness or with a subtle lameness that becomes more obvious with increasing exercise.[85,90] The degree of synovial effusion and lameness varies with the lesion's severity. Young horses and horses that show clinical signs before training usually have more severe lesions from OCD than do those that do not show clinical signs until after training is initiated. In very young horses, the disease can be so severe as to cause difficulty in rising. Flexion test of the stifle usually causes increased lameness.

Radiographs revealing flattening or fragmentation of the trochlear ridge confirm the diagnosis. Foals younger than 5 months of age commonly have irregularity of subchondral ossification of the trochlear ridges that should not be confused with an OCD lesion.[91] Smooth subchondral bone concavities, subchondral bone lysis, and osteochondral flaps and fragments are other lesions that may be seen.[92] Radiographs usually underestimate the severity and the extent of the lesions, especially in foals younger than 9 months of age.[93] Therefore, if signs of stifle OCD are present (FPT distention and lameness), arthroscopic treatment might need to be delayed until radiographic signs are obvious. Generally, lesions are monitored until the foal is older than 9 months of age before surgical intervention is recommended, as many radiographic lesions progress or resolve in this time (see Chapter 88). Ultrasonography of the FPT may identify lesions that cannot be seen radiographically, particularly lesions of the medial trochlear ridge.[94]

The treatment of choice for OCD is arthroscopic removal of osteochondral fragments and undermined cartilage, and débridement of the subchondral bone lesion.[87,90,95] Surgical success rates of 64%[90] to 89%[93] have been reported, without regard to sex, location of the lesions, or bilateral or unilateral involvement. Increased size of the lesion is inversely related to the success rate for return to intended use, as lesions less than 2 cm, 2 to 4 cm, and more than 4 cm had success rates of 78%, 63%, and 54%, respectively.[90] Conservative management of OCD has yielded poor results but might be appropriate under certain circumstances.[96] In one study, early detection and rest allowed lesions to heal and approximately 50% of horses were able to race; however, these animals tended to have less-severe lesions and no joint fragmentation. Arthroscopic reattachment of relatively smooth OCD cartilage flaps in the FPT with resorbable polydioxanone pins has been reported to have a good success rate.[97]

Subchondral Bone Cysts

Subchondral bone cysts (SBCs) of the medial femoral condyle (MFC) are a common cause of hindlimb lameness. SBCs or cystlike lesions are also recognized, although rarely, in the lateral femoral condyle,[98] the proximal tibia,[99] and the patella.[100] There are two commonly proposed etiologies of SBC: developmental orthopedic disease in the form of osteochondrosis and articular cartilage/subchondral bone trauma.[101,102] SBCs have been induced by surgically creating a defect in the subchondral bone of the weight-bearing portion of the MFC.[102] For more information on SBCs, review Chapter 89.

Medial femoral condylar SBCs develop unilaterally in most cases but can be seen bilaterally. Affected horses can be from a few months of age to aged, mature horses, with most cases diagnosed in horses between 1 and 3 years of age. Lameness is the primary clinical sign and flexion tests of the stifle may increase the lameness, but the change is usually not dramatic. The condition is commonly observed as a stiffness in the limb or a reluctance to flex the limb at a walk or trot, and circling the horse in a trot toward the affected limb generally increases the lameness.

Synovial distention may be difficult to detect and the condition generally shows some, although variable, improvement with intra-articular anesthesia; thus radiographic examination provides a definitive diagnosis (see Figure 99-7).[47] Bilateral caudocranial and lateromedial projections are important to assess the nature of the SBC and to evaluate for bilateral lesions.[103] The size of the lesions on the radiographs can vary from a flattened condyle to a slight indentation in the subchondral bone surface to a round or oval lesion a few centimeters in height and width (grading systems are documented[104,105]). There is usually a communication of the cyst with the joint (a cloaca) that can range from being very narrow to quite broad.

The size of the lesion and the amount of articular cartilage involvement has been suggested to correlate with clinical signs and prognosis (70% returned to racing with less than 15 mm

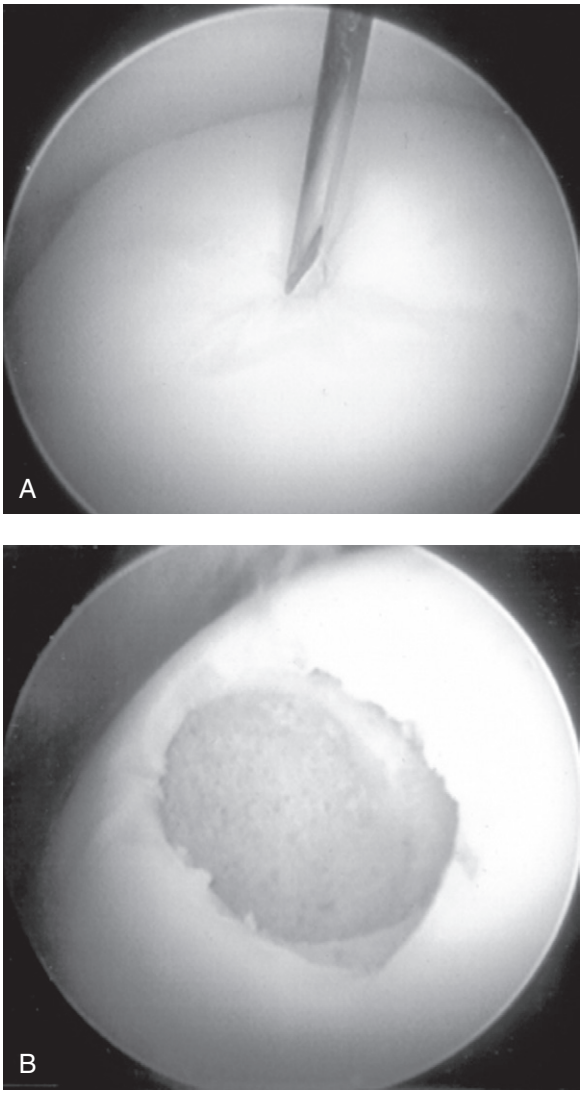


Figure 99-37. A, Arthroscopic appearance of a subchondral bone cyst of the medial femoral condyle showing the typical narrow opening at the articular surface. **B,** Appearance of the medial femoral condyle after débridement of the cyst.

surface débridement versus 30% with more than 15 mm[106]), but this finding has not been confirmed in all studies.[104,106]

Historically, the recommended treatment for this condition has been arthroscopic débridement of the lesion. Débridement of the cyst lining and removal of all debris from the joint is the goal (Figure 99-37). Surgical débridement of small condylar defects that are asymptomatic is contraindicated because it can provoke an enlargement of the cyst; intralesional corticosteroid injection (discussed later) may be beneficial in these cases. Horses with hindlimb lameness localized to the stifle that have subtle MFC radiographic changes (i.e., flattening of the condyle) warrant arthroscopic evaluation because cartilage and subchondral lesions were identified in 24 of 26 such limbs.[107] Subchondral bone drilling of débrided lesions is contraindicated because it may result in enlargement of the lesion.[104]

The success rates reported for return to previous function after surgical débridement of SBC ranges from 64% to 95%.[104,106,108] Following surgical débridement, 64% of Thoroughbreds raced, compared to 77% of siblings.[106] One study reported a 64% return to soundness in horses younger than 3 years of age, compared to 35% in horses older than 3 years of age.[109] More extensive cartilage damage was associated with a decreased odds of return to soundness and was more commonly identified in the group older than 3 years. Concurrent or sequential correlation of SBCs with meniscal tears is associated with a poor prognosis.[74]

Multiple grafting techniques have been developed and evaluated for their ability to accelerate and improve healing of the débrided SBC lesion. Compacted cancellous bone grafting did not result in an improvement in the outcome.[110,111] Mosaic arthroplasty (autologous osetochondral grafting)[112,113] in six horses was shown to result in successful graft incorporation, with 50% of horses returning to an equal or higher level of activity. Autogenous fibrin plugs containing allogeneic chondrocytes and insulin-like growth factor-I placed over cancellous bone grafts or tricalcium phosphate (TCP) granules packed into the débrided cysts resulted in success rates of about 75%.[114,115] Autologous patient-side grafting using bone marrow aspirate concentrate, platelet-rich plasma with TCP, showed positive results with regards to the rapidity of bone formation and thickness of the resultant subchondral plate.[116]

Intralesional or intra-articular injection of corticosteroids (triamcinolone) has been used at various time points perioperatively to depress the inflammatory mediators released into the joint.[117] Intralesional corticosteroid therapy has more recently been advocated as the primary and sole treatment of subchondral bone cysts. Injection can be completed with ultrasonographic or arthroscopic guidance,[105] with the latter technique having the additional benefit of examination and débridement of any significant cartilage lesions. Triamcinolone or methyprednisolone is deposited throughout the cyst lining, with the most effective technique involving multiple redirections of the needle to distribute the medication. Intralesional injection of corticosteroids resulted in success rates of 90% for unilateral cases and 67% for bilateral cases.[105]

Reports on conservative management of this condition, with or without the use of nonsteroidal anti-inflammatory medication, have reported success rates of 45% to 64%.[98,118] Interestingly, one study found no significant differences in racing results between yearlings with radiographically diagnosed SBCs in the MFC and the unaffected horses, leading to the prediction that some lesions spontaneously resolve without treatment.[119]

Postoperative care following surgical débridement of a cyst generally involves 1 to 2 months of stall rest followed by at least 4 months of small paddock rest, and horses should not be returned to work until they are sound at a trot. Postoperative convalescence prior to restoration of soundness may be prolonged (i.e., 4 to 18 months).

SBCs have been identified in the proximal cranial tibia of young and mature horses.[99] Successful arthroscopic débridement and return to athletic performance was achieved in 3 of 4 young horses in which an osteochondrosis etiology was suspected. Pre-exisiting osteoarthritis and other soft tissue injuries in the stifle of mature horses appeared to limit their future performance.

One report has documented an osseous cystlike lesion of the patella seen radiographically.[100] The lesion was seen on the medial articulating surface of the patella, corresponding to the most common location of patellar fractures.

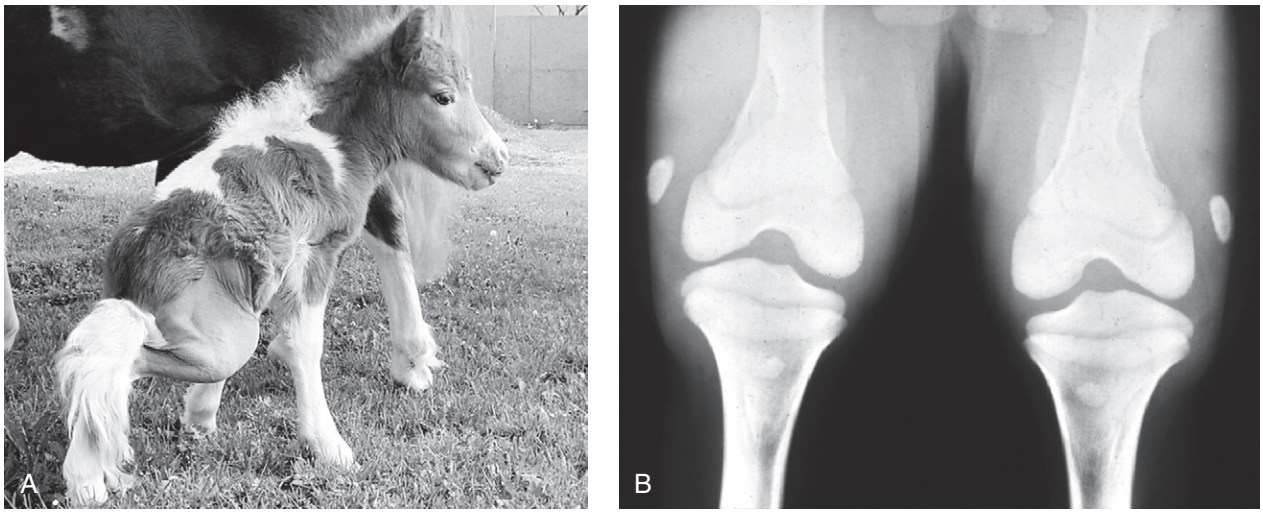


Figure 99-38. A, This 3-month-old Shetland pony foal shows the typical stance of an animal with complete lateral luxation of the patella. **B,** The craniocaudal radiographic views of both stifles show the patella located lateral to the distal femur. (Courtesy J. Auer, University of Zurich.)

Patellar Disorders

Lateral Luxation of the Patella

Lateral luxation of the patella is a rare condition that is most commonly seen congenitally in foals or Miniature Horses. The condition is believed to be heritable.[120-123] Luxation of the patella in mature horses is rare and generally traumatic in origin.

The condition can be unilateral or bilateral, and clinical signs depend on the severity of the displacement of the patella. Foals with complete luxation of the patellas are unable to stand (because the quadriceps acts as a flexor instead of an extensor of the joint), and they assume a classic crouched position (Figure 99-38, *A*). When the patellas are not completely luxated, clinical signs include limb stiffness or lameness at the walk and trot. The patellas can usually be reduced manually. The FPT is generally distended, and there may be crepitation on palpation of the patella.

Radiographic assessment of the stifle should include the degree of degeneration of the joint, the depth of the trochlear groove, the shape of the patella and trochlear ridges, and the amount of ossification of the trochlear ridges and the patella. Irregularity of subchondral ossification of the trochlear ridges in foals up to 5 months of age should not be mistaken for pathology. On craniocaudal radiographic views, the patella is located lateral to the femur (Figure 99-38, *B*); on the lateromedial view, it is projected over the femur or visible just caudal to it.

Surgical correction of patellar luxations includes lateral release incisions combined with reinforcement of the medial patellar support structures. In dorsal recumbency, under general anesthesia, surgical exposure is achieved with one large medially based curved incision extending from 6 cm proximal to the patella distad to the tibial crest. Alternatively, two curved incisions (craniomedial and craniolateral) can be used.

Lateral release of the patella is accomplished by incising the insertion of the biceps femoris, transecting the lateral femoropatellar ligament and the origin of the lateral patellar ligament without invading the joint.[124] Lateral release can sometimes be accomplished by partially transecting the tensor fascia and fibrous joint capsule while preserving the lateral femoropatellar ligament. Removal of excessive FPT synovial fluid with a needle and syringe may be beneficial prior to medial imbrication to decrease tension on the sutures. The medial joint capsule from the base to the apex of the patella is imbricated with No. 5 braided polyester suture. Alternatively, suturing the tendon of the sartorius muscle and the parapatellar fascia to the joint capsule and the medial patellar ligament may be necessary. We have used a mesh implant to reinforce this imbrication in a 370-pound yearling and in cases where the holding capacity of friable tissue is a concern.

A sulcoplasty has been advocated for lateral ridge hypoplasia or instability of the patella in the trochlear groove.[125] In small animals, two different techniques are described.[126] These techniques are performed on young animals that have a thick hyaline cartilage layer covering the ossifying center of the epiphysis. The skin incision as described earlier is used for the approach in both techniques. One technique involves elevation of a U-shaped cartilage flap within the trochlear groove and removal of the subchondral bone (Figure 99-39). The cartilage is incised at an angle (from superficial to deep toward the flap) and is carefully elevated with the help of a periosteal elevator. The soft bone is removed with rongeurs, and the surface is evened out with a curette. The cartilage flap is then repositioned and stabilized with sutures. If an abrupt change in trough angle on either side is noticed, additional cartilage can be removed with a scalpel to provide a smooth articular surface.

The other sulcoplasty technique involves the removal of a wedge from the trochlear groove (Figure 99-40) with a handheld saw. Each saw cut removes some bone, and thus by simply replacing the wedge, some deepening of the groove is achieved. If additional deepening is desired, a parallel saw cut is carried out on one side, and the wedge is replaced. The edges are smoothed out with a scalpel. If the wedge is properly seated, no fixation is required. The soft tissue release and imbrication techniques discussed earlier are also performed.

Both procedures have been used successfully on foals. Postoperative radiographs confirm correct positioning of the patella. Postoperative care includes the use of systemic antibiotics, nonsteroidal anti-inflammatory medication, and good nursing care. Seromas may occur but usually resolve without treatment.

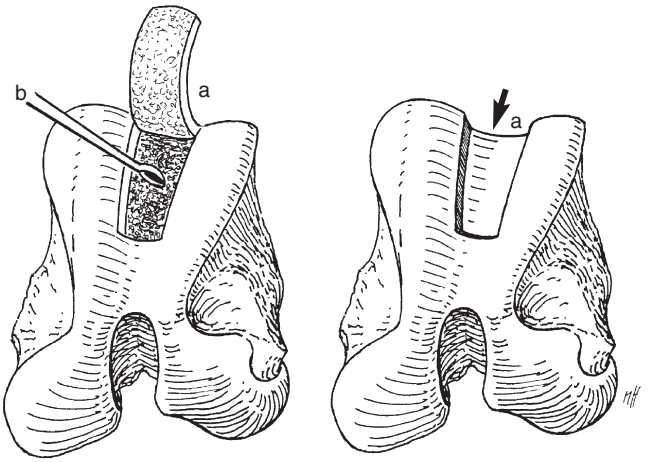


Figure 99-39. Sulcoplasty involving elevation of a U-shaped cartilage flap *(a)* and removal of the subchondral bone with rongeurs and a curet *(b)*. The flap is repositioned *(arrow)* and, if necessary, the edges are smoothed out. The flap can be secured with suture.

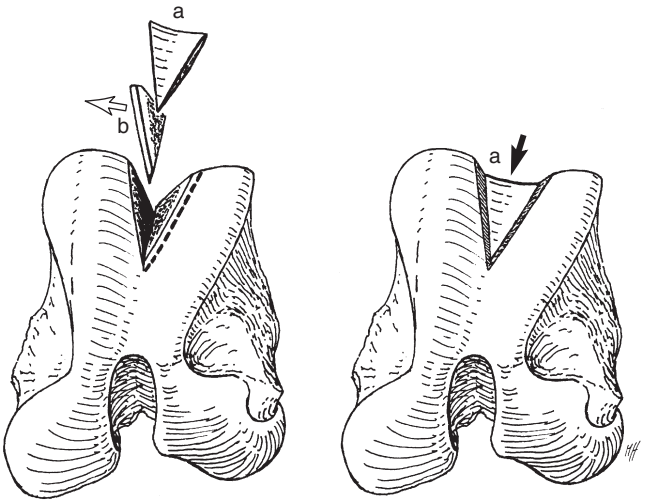


Figure 99-40. Sulcoplasty involving wedge *(a)* osteotomy of the intertrochlear groove. If necessary, an additional piece of bone *(b)* is removed *(arrow)* with an additional saw cut parallel to a previous one. The wedge *(a)* is replaced in the trough and pressed into position *(arrow)*, resulting in a functional intertrochlear groove.

Upward Fixation of the Patella

Upward fixation of the patella is a pathologic condition of the equine stifle that results from a difficulty or inability to release the passive stay apparatus mechanism of the stifle. This condition commonly occurs secondary to the initiation of training in young horses and is related to a straight hindlimb conformation. Severity can vary from a slight delay in flexion of the limb only observed by experienced riders and lameness clinicians to permanent fixation of the patella that cannot be manually corrected. Chronic intermittent upward fixation of the patella may result in excessive trauma to the FPT and secondary effusion and lameness. The treatment for this condition involves alterations in training to increase development of the quadriceps muscle, with surgical treatment reserved for severe cases (complete persistent inability to unlock the stifle) or those refractory to medical management.

Quadriceps development is accomplished by riding at an extended trot up and down hills in deep, sandy, or loamy soil. Working the horse in a circle in an arena (especially at a canter) should be avoided, as this merely contributes to the patella trauma and the horse's continued lack of quadriceps development because of lameness. Rather than box stall confinement, anti-inflammatory medication and turnout for the horse are recommended. Some acute severe cases, which are commonly traumatically induced, resolve with anti-inflammatories and rest, and thus delay of surgical intervention for a few days may be advantageous.

Injection of counterirritants into the middle and medial patellar ligament[127,128] has annecdotally shown some success in treatment of upward fixation of the patella.

MEDIAL PATELLAR LIGAMENT SPLITTING

Medial patellar ligament splitting has been advocated for treating upward fixation of the patella when conservative methods have failed.[129] We have noted numerous advantages over medial patellar desmotomy, particularly with regard to postoperative complications.

The procedure is carried out with standing sedation or under general anesthesia. Using ultrasonographic or palpation guidance, multiple longitudinal splitting incisions are made in the proximal third of the medial patellar ligament. This form of splitting results in a two- to threefold progressive increase in ligament diameter during the first 4 weeks after surgery. No anti-inflammatory medications are used, and hand-walking is initiated the day after surgery. Resolution of all clinical signs was noted in 7 of 7 cases within 12 days of surgery, with no recurrence of upward fixation of the patella.[130]

MEDIAL PATELLAR DESMOTOMY

If the appropriate conditioning and development of the quadriceps muscle tone with training and medial patellar ligament spliting does not resolve the condition, or if persistent inability to unlock the stifle is present, a medial patellar desmotomy (MPD) can be considered as a last resort. MPD has been associated with induction of chondromalacia of the patella, entheseophyte formation, osteolysis, and patellar fragmentation, particularly if horses were returned to work quickly (i.e., 3 weeks) after surgery.[76,131-133] These changes are believed to be induced by alteration in patella positioning and increased biomechanical strain on the remaining patellar ligaments (particularly the middle patellar ligament). These lesions were recognized as causing lameness in normal horses in which experimental MPD was performed. Arthroscopic débridement of these clinically significant lesions is indicated; if fragments develop, they should be removed because they will lead to osteoarthritis.

The surgical procedure of MPD is performed under standing sedation with the tail wrapped to avoid contamination of the surgical site. After preparation of the skin, local anesthetic is injected subcutaneously over the medial border of the distal aspect of the middle patellar ligament and around the distal aspect of the medial patellar ligament. A 1-cm vertical incision is made over the medial border of the middle patellar ligament close to the ligament's attachment to the tibial tuberosity; a subcutaneous fat pad is located in this area and thus the operator is less likely to enter the FPT. Curved Kelly forceps are forced through the fascia and passed behind the medial patellar ligament. A curved bistoury knife is inserted into this channel, the

cutting edge is turned outward, and the ligament is cut. The blade should be fully advanced to the other side of the medial patellar ligament before the ligament is severed. After the ligament has been severed, the tendon of the sartorius muscle feels like a tense band and might lead the inexperienced operator to believe that the medial patellar ligament has not been completely severed. The skin is closed routinely.

To decrease the risk of complications discussed earlier, a prolonged period of rest is recommended before the animal is turned out to allow some scarring to occur at the surgery site to improve realignment and stabilization of the patella in the intertrochanteric groove. However, such changes as new bone production on the tibial tuberosity and the middle patellar ligament attachment to the patella may be seen despite a stall rest period of 120 days after surgery.[134] A retrospective study of clinical cases of upward fixation of the patella found that incidence of gait abnormalities after medial patellar desmotomy may be lower with stall rest more than 3 months (25%) compared to less than 1 month (67%), but the only two horses that showed radiographic changes after MPD had been rested 3 months or more.[135]

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CHAPTER
100 Femur and Pelvis

Dean W. Richardson

Disorders of the femur and pelvis of the horse are not uncommon. Injuries ammenable to medical therapy are usually ligamentous in nature or involve the sacroiliac joint. Surgically treatable conditions of the femur and pelvis occur as fractures of either bone or coxofemoral luxations. These latter conditions comprise this chapter.

FRACTURES OF THE FEMUR

Fractures of the femur occur primarily in foals and weanlings, although they can be found in a horse at any age. Most fractures follow a severe traumatic event, such as a fall or a kick. Proximal femoral fractures in adults may occur in horses that fall down with their lower limb extended, such as while wearing a full limb cast or with an upwardly fixed ("locked") patella.

The diagnosis of a distal or diaphyseal fracture is not difficult to make in most cases because of the acute onset as well as the marked lameness and swelling that localize the problem. Although crepitus is usually palpable with manipulation of the limb, hemorrhage and muscle swelling can be severe enough to separate the bone ends and minimize this sign. More severely

comminuted fractures might not have crepitus, because motion between the individual fragments is limited. Less common presentations include shock caused by blood loss into the thigh musculature and dragging of the limb because of upward fixation of the patella.

Diagnostic radiography in larger horses (more than 300 kg) is usually performed under general anesthesia unless the fracture is located distally. There are techniques described for obtaining useful, but not entirely diagnostic, images in standing horses.[1] Ultrasonography can be used to confirm a diagnosis of a displaced fracture in a large horse.[2] It is best to sedate or anesthetize foals so that mediolateral and craniocaudal views can be obtained. For proximal fractures, a ventrodorsal view of the pelvis should be obtained.

Surgical treatment of mid-diaphyseal or proximal femoral fractures has generally been successful in foals and small ponies, although in larger weanlings and even yearlings it may be feasible if fracture configuration is suitable. Distal metaphyseal fractures can also be successfully repaired in yearlings with plates and screws. Locking intramedullary nails are an option for mid-diaphyseal fractures in foals and even larger yearlings,[3] but double plating is probably a superior technique.[4]

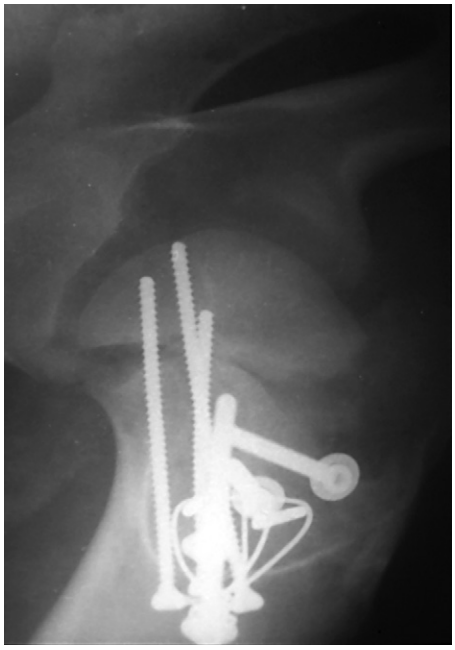

Figure 100-1. Postoperative radiographic view of the femoral head region of a foal. Three 4.5-mm cortex screws were inserted in lag fashion into the capital femoral epiphysis. The trochanteric osteotomy used for the surgical approach to the joint was repaired with screws and tension band wires. There also was radiolucent tension band placed under one washer.

Proximal (Head and Neck) Fractures

Proximal (head and neck) fractures are seen almost exclusively in foals and are the most difficult femoral fractures to diagnose without good-quality radiographs. Conservative treatment of such fractures is unlikely to result in a comfortable horse. Surgical repair is difficult to achieve, and the prognosis is guarded.

The favored surgical approach is craniodorsal, with a curved incision just cranial to the greater trochanter and that parallels the proximal shaft of the femur. Exposure is enhanced by either a tenotomy of the gluteal insertion or a trochanteric osteotomy. If an osteotomy is selected, the drill holes for later tension-band repair should be prepared before transecting the bone.

The bone can be cut with an oscillating saw or Gigli wire. The surgeon should review the anatomy carefully before making the trochanteric osteotomy. It is possible to cut too much and damage the caudal femoral head or too little and leave medial attachments that obscure the intended view. The approach and arthrotomy are extended to allow palpation of the head and neck of the femur. Additional fractures that are not evident radiographically are detected in this manner. It is very difficult to clearly see the fracture site except in small foals, but placing one's finger in the joint allows more accurate reduction of the fracture and alignment of the drill and drill bit.

Although use of multiple pins has been described, techniques involving screws provide more stability[5] (Figure 100-1). These techniques include screws applied in lag fashion, cannulated screws, or a dynamic hip screw plate.[6-8] Regardless of the specific implant selected, alignment is critical to prevent damage to the articular surface. Cannulated screws have a major technical advantage for this particular fracture, because reduction is maintained by the guide wire during the entire procedure and the direction and depth of the drill or screw can be monitored constantly (see Chapter 76). Unfortunately, cannulated screws are significantly weaker in bending than solid screws, so there is a higher risk of screw failure, especially in larger foals.

If a cortex screw and lag technique are used, the glide hole is drilled before reduction. This allows checking for central positioning of the bit before entering the epiphysis with the thread hole. The combination of a minimal length glide hole and a central location will maximize the number of threads engaged in the smaller fragment. If the screw is not centered in the epiphysis or the epiphyseal fragment is penetrated off center with the large bit used for the glide hole, stability of the repair will be significantly compromised.

Ease of reduction is variable and depends mainly on the degree of eburnation of the fracture surfaces. Smooth, eburnated fracture margins can make reduction very difficult. If the femoral shaft is held with forceps, leverage is provided when attempting reduction. However, reduction is maintained digitally because clamps cannot be applied. Predrilling the glide hole across the femoral shaft also minimizes the length of time that the fracture must be digitally maintained in reduction.

It is difficult to accurately estimate the ideal length of the screw from preoperative radiographs; therefore, intraoperative palpation and measurements are essential. To assess the depth and direction of drilling, a pin or drill bit of the same length is placed on the exterior of the bone in a parallel position as drilling is performed. This provides additional visual and tactile help for alignment as holes are drilled into the head of the femur. If at all possible, at least two screws should be used to ensure rotational stability.

Closure of the wound should be in layers with synthetic absorbable suture material. Pre-placing several sutures in the joint capsule before tying is recommended. If a trochanteric osteotomy was performed, it must be repaired with a tension-band technique. Either two screws or two smooth pins can be used to replace the trochanter, but both must be supported distally with 1.25- or 1.5-mm–diameter wire. The wire is passed through a drill hole across the femoral shaft made 5 to 7 mm distal to the osteotomy. In larger foals, two wires are usually

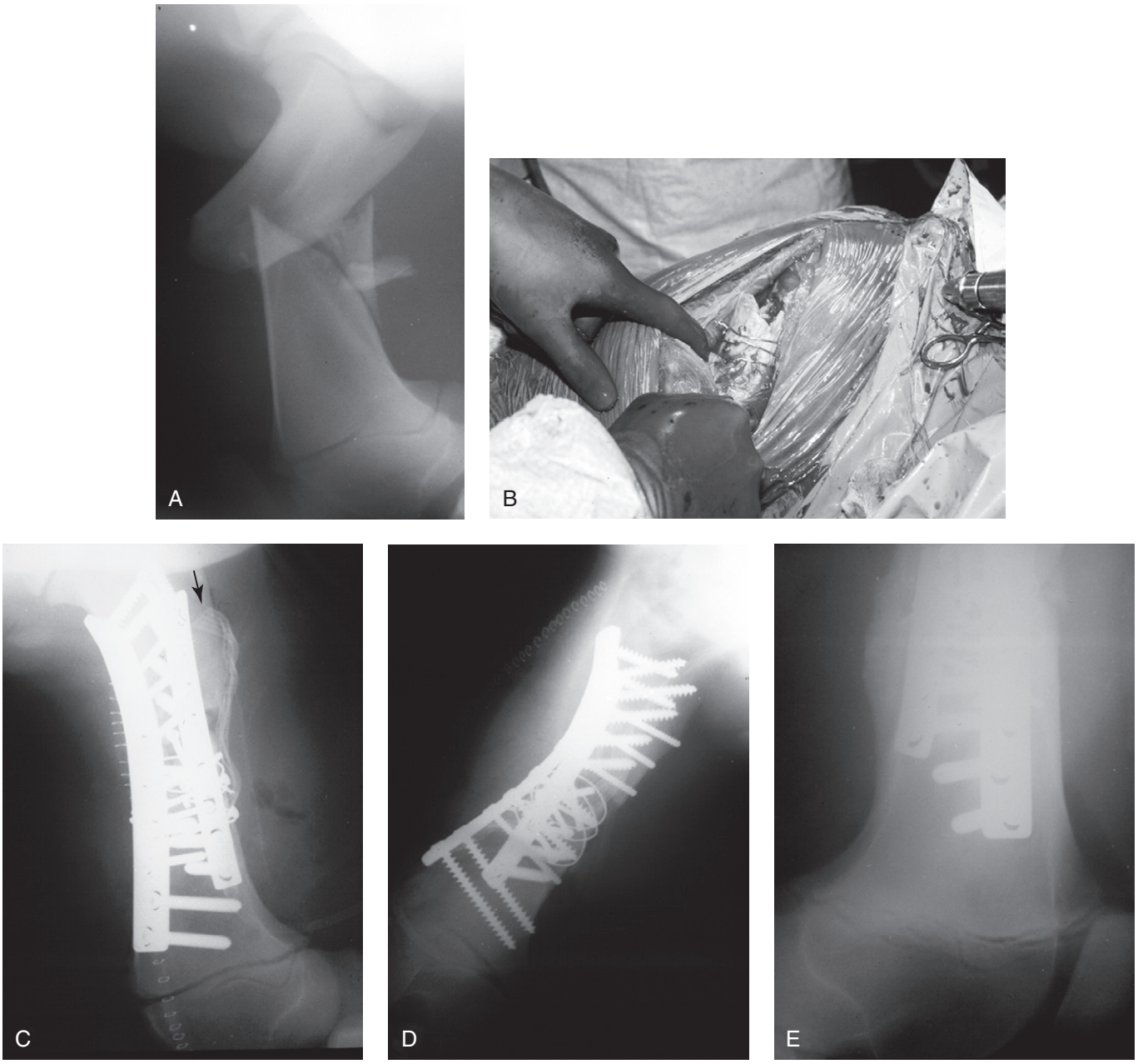

Figure 100-2. A, Lateromedial radiographic view of a multifragment femoral fracture in a foal. **B,** Intraoperative view showing the butterfly fragment, which was attached to the distal main fragment by means of two 3.5-mm cortex screws placed in lag fashion and multiple cerclage wires. **C** and **D,** Immediate postoperative radiographic views depicting the fracture repaired with two DCPs, one laterally and one cranially. Several fully threaded cancellous screws were used in the metaphyses. The *arrow* points toward the proximal end of the suction drain. **E,** Three-month follow-up radiographic view showing good fracture healing with substantial callus formation. A small fragment is visible at the cranial aspect of the bone.

used. If screws are used to replace the trochanter, it might help to feed the screws through washers so that the tension-band wire comes to lie between the washer and the bone, which secures the fixation.

Other types of proximal femur fractures involve the greater trochanter or, more commonly, the third trochanter. These fractures can be diagnosed with physical examination or scintigraphy. Ultrasonography can then be used to more specifically define the injury without general anesthesia. Unless the fractures are associated with a wound or become sequestrated, they should be treated conservatively with stall rest for several months.

Mid-Diaphyseal Fractures

Mid-diaphyseal fractures are the most common type encountered and are the fractures most amenable to successful surgical repair.[9] Most have a spiral or long oblique shape or are comminuted (Figures 100-2 and 100-3).

The skin incision is made laterally over the length of the bone between the greater trochanter and the lateral condyle. After the fascia lata is incised, it is easy to identify the demarcation between the vastus lateralis and biceps femoris. If a small incision is made there in the mid-diaphysis, the fracture hematoma can be digitally identified. The muscle bellies are easiest

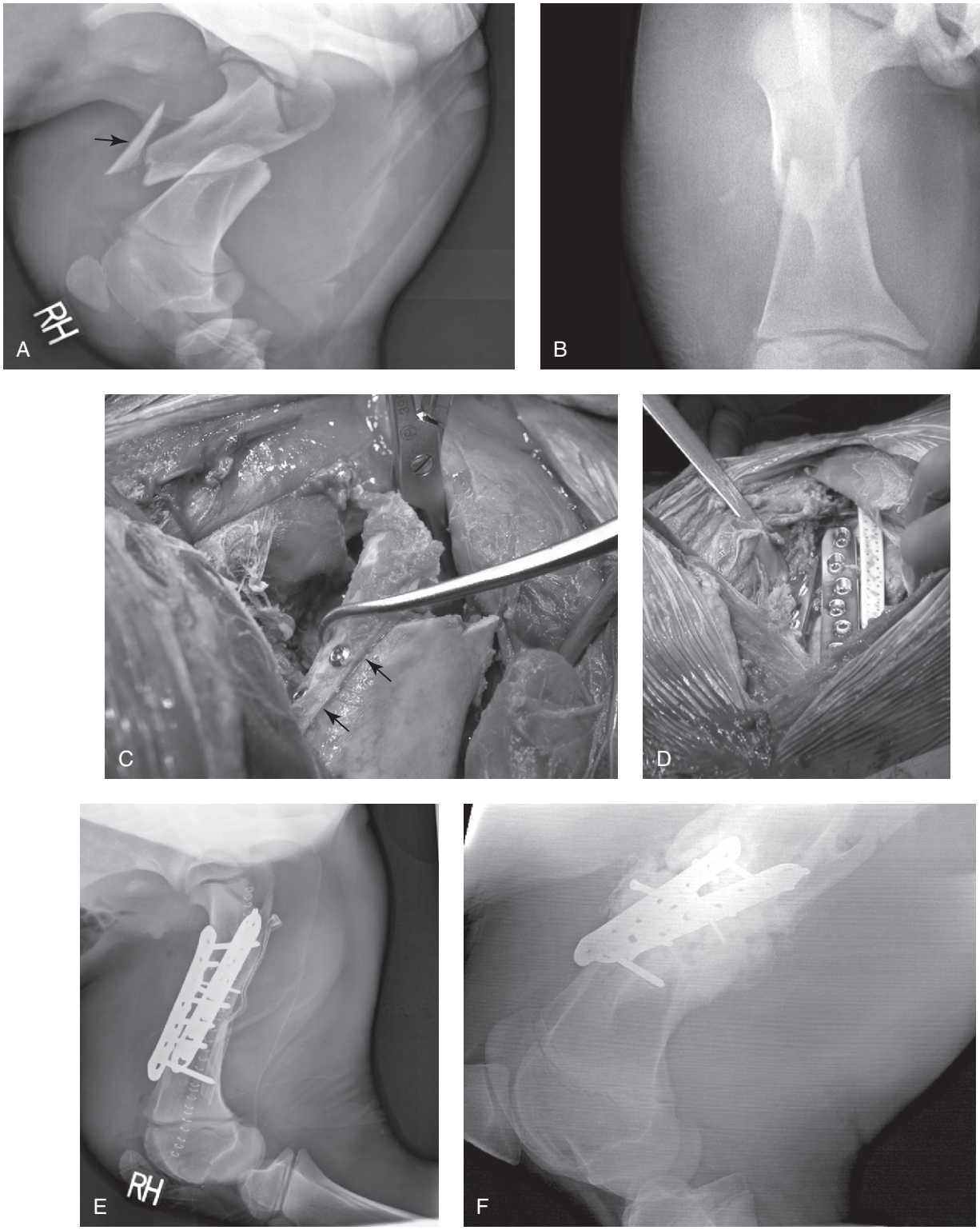

Figure 100-3. Mediolateral **(A)** and craniocaudal **(B)** radiographic views of a typical diaphyseal multifragment femoral fracture in a foal. **C,** A large butterfly fragment (*arrows* in **A** and **C**) was reconstructed with screws using lag technique to make a two-piece fracture. **D,** Intraoperative view of the fracture, repaired with two locking plates. The white suction drain is seen just caudal to the plates. The perpendicular placement of screws into the extremities of the cranial plate can be very difficult, even with vigorous muscle retraction. **E,** Immediate postoperative lateromedial radiographic view of the repaired fracture. **F,** Image at 2.5-month follow-up shows typical femoral fracture healing with large callus formation.

to separate when palpated from inside of the hematoma. The attachments to the third trochanter usually need to be severed to provide exposure at the proximal end of the bone.

Comminuted fragments should be independently reduced with screws inserted in positions outside of the planned locations of the plates until the fracture is reconstructed into two main pieces (see Figure 100-3, *C*). Comminution in some cases is severe and requires extensive reconstruction. It is often useful to place 3.5-mm cortex screws in lag fashion to reduce small fragments, countersinking their relatively small heads deep enough that the plates can be placed over them (see Figure 100-2, *B*). If there is a barrel stave configuration to the fracture extending down the diaphysis, cerclage wires or cables can be used to prevent splitting the bone (see Figure 100-2, *B*).

When the fracture is reconstructed to two pieces, reduction is usually possible by simply elevating the fragment ends out of the incision, aligning them properly, and folding the bone ends back into position. For long oblique fractures, it might be necessary to clamp the bone in an overriding position before sliding it incrementally by direct traction into reduction. Nylon cable ties or heavy cerclage wires can be pre-placed to help hold the reduction as it progresses. Large Lane bone forceps also may be useful. Vigorous traction might be necessary; therefore, it is essential that the foal's or pony's tail and body be tied securely with ropes and girths to the surgery table. It can also be helpful to engage a large (6.35 mm [¼ in]) Steinman pin down the proximal shaft of the femur and another pin across the distal fragments. With Jacobs' chucks attached to each, the pins can be used as "handles" to help manipulate the fragments.

After reduction is obtained, bone clamps are applied to maintain temporary alignment. One or two screws should be placed with lag technique across the oblique fracture in locations where they will not interfere with the lateral plate (see Figure 100-2, *B*). The bone-reduction forceps are removed, allowing application of the longest possible 4.5-mm locking compression plate (LCP) with 5.5-mm cortex screws and 5-mm locking head screws. Alternatively, the plate can be placed with a bone clamp maintaining the reduction, but this is much more difficult.

After applying the lateral plate, a shorter LCP is placed cranially. The LCP has major advantages[10-12] but it is important to note that it will prove quite difficult to place locking screws in all holes of a cranial LCP because of the quadriceps muscle. Hohmann retractors provide adequate exposure to position the plates and insert most screws, but locking screws at the ends of the plate often need to be placed via stab incisions through the musculature (see Figure 100-3, *C*). Lag technique is applied in plate screws crossing any reduced fracture plane for additional compression and stability. Interfragmentary compression increases the overall stability of the repair and helps ensure accurate reduction. The two plates must be staggered a half-hole apart, and the screws must be inserted perpendicular to the long axis of the plates to prevent interference of screws. All cortex defects are filled with autologous cancellous bone and the gaps in the plate holes next to the screws are typically filled with an antibiotic eluting material, such as polymethyl methacrylate.

The incision is closed in at least three layers: the fascial margins of the vastus lateralis and the biceps femoris, subcutaneous tissues, and skin. Interrupted sutures are recommended because wound problems are common and local drainage is sometimes necessary. Because of the considerable muscle trauma (original and surgical), compression bandaging is impossible; closed suction drainage is essential to minimize accumulation of serum (see Figures 100-2 and 100-3).

An alternative technique for simple diaphyseal fractures of the femur is insertion of an interlocking intramedullary nail (see Figure 76-44).[3] The axial position of the nail affords excellent bending stability, and the transfixing screws provide rotational stability. If possible, supplemental fixation in the form of lag screws, cerclage wires, or a locking plate with unicortical screws can be used in oblique or spiral fractures.

A routine incision is made as described earlier to reduce the fracture. A small incision is subsequently made over the medial aspect of the greater trochanter, and a 5.5-mm drill bit is used to prepare a hole across the bone into the medullary cavity. This hole and the medullary cavity are subsequently enlarged with reamers until a uniform-diameter path for the nail is created. The nail length is pre-selected based on measurements taken on radiographs of the contralateral intact bone. The nail is driven into the distal fragment with the targeting jig attached, seating it securely but being careful not to penetrate the distal end of the bone. After the nail is fully inserted, 5.5-mm cortex screws are placed through the bone and the nail. At least two screws are applied at either end of the nail, in most cases more.

One possible major complication of interlocking intramedullary nails is the development of a fracture through the screw holes. The nails are more applicable in mid-diaphyseal fractures than in more distal or proximal injuries, where it is difficult to adequately seat the nail in the shorter fragment.

Because of the massive surrounding muscles, even well-repaired femoral fractures tend to develop large calluses quickly (see Figures 100-2, *E*, and 100-3, *F*). This is valuable because even suboptimal repairs that develop some minor instability can successfully heal as the callus provides adequate stability relatively quickly, especially in younger individuals.

Distal Femoral Fractures

Distal femoral fractures are most commonly encountered in older weanlings and yearlings and are nearly always of the Salter-Harris type II. Most have the metaphyseal fragment positioned caudally. Salter-Harris types III and IV fractures have also been reported but are less common.[13-15] Any distal femoral fracture can be initially misdiagnosed as a stifle lesion because of the location of the swelling (Figure 100-4). Fractures of the distal femur tend to be quite unstable and therefore require surgical repair.

The fracture is approached through a craniolateral incision between the vastus lateralis and biceps femoris muscles. The incision is extended distad to the level of the tibial crest. The lateral femoropatellar ligament must be transected to allow a parapatellar arthrotomy and patellar luxation. Reduction may be quite difficult to achieve. Like many physeal fractures, exact anatomic alignment is difficult to assess. The metaphyseal part of the fracture is not readily visible and is covered by the extensive soft tissues on the distal caudal femur.

After reduction is achieved, a long screw can be placed through the exposed intercondylar notch and into the metaphysis to maintain alignment while the lateral plate is applied. Because the epiphyseal fragment is small, a specialized plate with some type of flared end is usually preferred (Figure 100-5). The strongest plate, which is best suited for larger yearlings, is a dynamic condylar screw (DCS) plate (Figure 100-6). Although the DCS plate is difficult to contour because of its large cross section, it can be bent to fit the distal femur.

Special care must be taken to fit the DCS plate accurately on the horse's curved distal femur, because the plate was designed

for the straight human femur. However, this plate provides superior stability in the distal fragment (see Figure 100-6). In horses weighing less than 175 kg, the condylar buttress plate with a flared end accommodating several screws is easier to apply and is strong enough despite its smaller cross section (see Figure 100-5).[16] LCPs with 5-mm locking screws also have the potential to work well in this location.

Regardless of the plate selected, the largest available screws (5-mm locking head, 5.5-mm cortex or 6.5-mm cancellous screws) should be used. The parapatellar fascial incision is closed with large tension-holding sutures (cruciate, near-far-far-near, or overlap); meticulous skin closure and a closed suction drain are all recommended. An adhesive or sutured stent bandage should be applied to keep the incision clean.

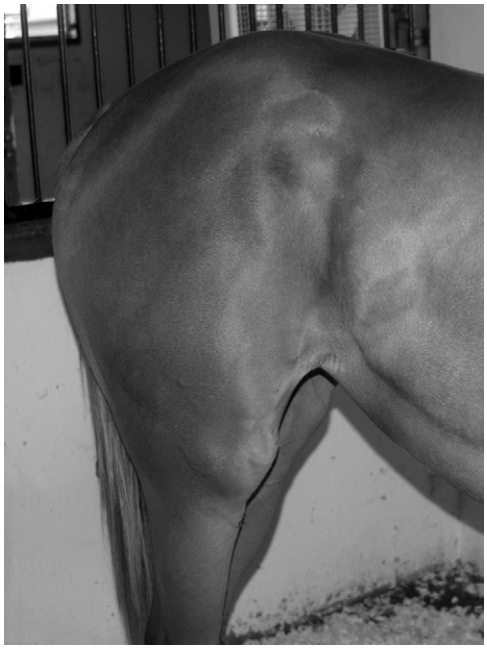

Figure 100-4. The swelling associated with a distal femoral fracture can be misinterpreted as a primary stifle joint problem. Typically, however, crepitus and instability are palpable.

Prognosis

The prognosis for fractures of the femur is guarded because of the high risk of complications.[9] As a result of muscle trauma and because a pressure bandage cannot be used on the area, surgical incisions are susceptible to extensive serum accumulations. Wound dehiscence and sepsis are common problems. If complications are avoided, however, healing of fractures of the femur is remarkably rapid in young animals because of excellent vascularity and muscle coverage. Callus is radiographically visible in foals within a few weeks. Even overtly infected femoral fractures have a reasonable chance to heal if long-term drainage, local antibiotics, and adequate stability are maintained.

Failure of plates and screws in adequately reduced fractures is uncommon, except when there is a bone defect in the caudal cortex. Such a defect leads to excessive cyclic loading of the implants and fatigue failure. If fracture healing is uncomplicated, it is not advisable to remove the implants; removal can be difficult and the repeat incision can still have complications. In distal physeal fractures, the plate might have to be removed if it is interfering with joint function or if there is significant remaining growth potential. Unfortunately, many physeal fractures of the distal femur result in permanent closure of the growth plate and consequently a shortened femur. This is acceptable for breeding soundness, because the animal can compensate somewhat by increasing its joint angles in the stifle and hock.

Although some fractures of the femur have been successfully managed conservatively,[17] younger animals often rapidly develop a serious varus deformity in the contralateral

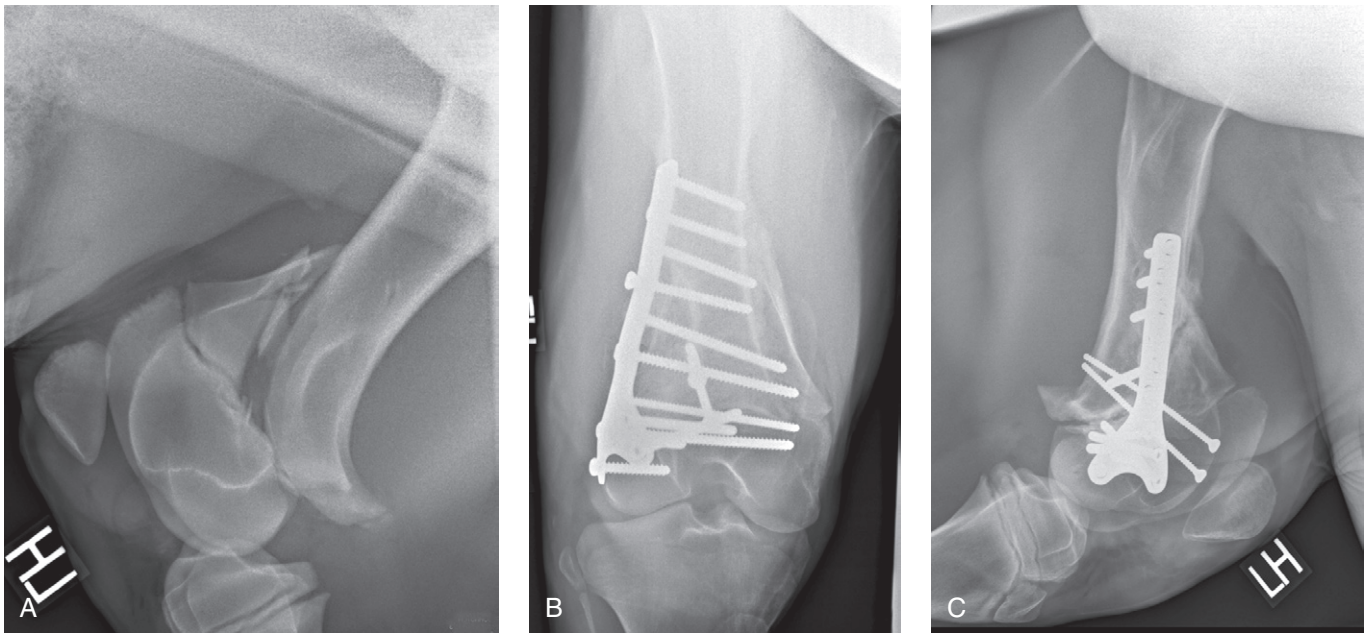

Figure 100-5. A, Salter-Harris type II fracture of the distal femur repaired with a condylar buttress plate having a flared end accommodating multiple screws. Two 5.5-mm screws were placed through the articular surface to help maintain reduction while the plate was applied. Caudocranial **(B)** and lateromedial **(C)** radiographic views of the fracture repair were taken 3 months postoperatively.

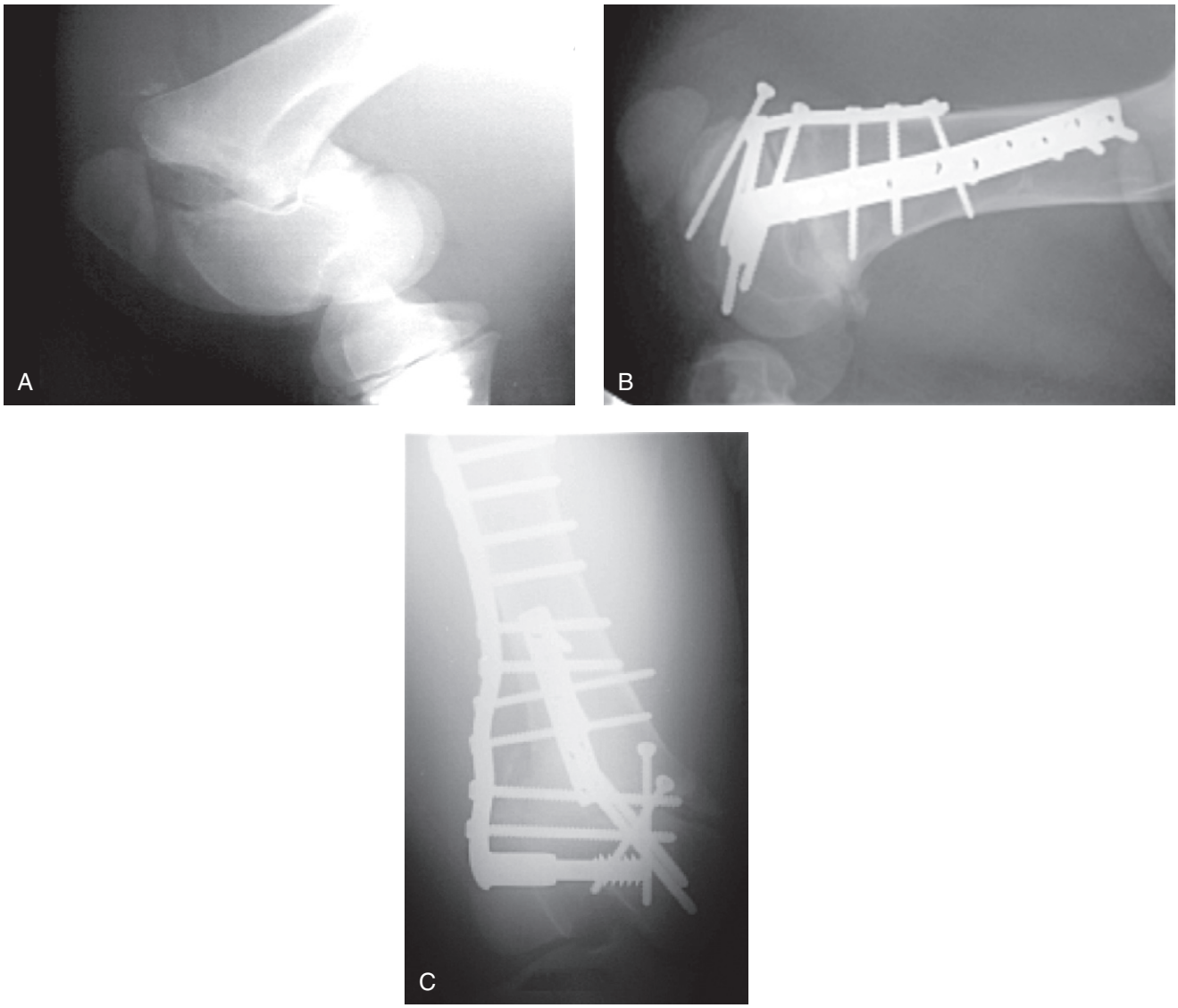

Figure 100-6. A, Salter-Harris type II fracture of the distal femur. Lateromedial **(B)** and caudocranial **(C)** radiographic views showing the repaired fracture. A dynamic compression screw (DCS) plate was applied laterally and augmented with a five-hole narrow dynamic compression plate (DCP) containing long screws penetrating the medial condyle. One single screw was buried in the articular surface to provide additional stability to the cranial aspect of the reconstruction. Longitudinally placed screws should augment the plate fixation. (Courtesy C. Lischer, University of Berlin.)

limb (Figure 100-7). Because these fractures can be repaired successfully in foals, the conservative approach should be reserved for older animals in which the surgical approach is too daunting or for cases where comminution is so severe that even locking plate fixation is not feasible.

FRACTURES OF THE PELVIS

Fractures of the pelvis occur in all animals of all ages but are disproportionately common in foals and yearlings.[18,19] Most follow a distinct traumatic event, such as a fall. In adult horses, pelvic fractures can occur during intense exercise without obvious trauma.

The most obvious clinical sign is overt lameness either of the swinging or supporting limb type. External swelling or asymmetry of palpable pelvic landmarks (e.g., the tuber coxae, tuber ischii, tuber sacrale, and greater trochanters) can be present. It is helpful to measure the distances between these landmarks and to compare the two sides. Pain can be elicited by manipulation of the limb or by direct pressure over the trochanter or other prominences. Examination *per rectum* can reveal obvious asymmetry, especially with acetabular fractures in which there is often a rounded, firm swelling over the fractured region.

Crepitus can be difficult to appreciate externally in horses with pelvic fractures, but it is often more obvious if the horse's weight is shifted or if the horse is walked a few steps during a rectal examination. Vigorous manipulation of the limb by an assistant can also be helpful in some cases, although care should be taken to test the horse's pain response to such manipulation before the rectal examination is undertaken. Crepitus is usually appreciated more easily on the first day or two after the injury.

In chronic cases, pelvic musculature is usually atrophied, although this is obviously not specific for a pelvic injury. The

medial thigh region should always be checked for asymmetry, because dependent swelling often develops in this area, especially with displaced acetabular and ileal fractures. The most dramatic presenting signs are seen with a laceration of a major artery (usually the internal iliac artery). This results in bleeding into the abdomen or thigh that is severe enough to cause shock or death.[19,20]

A definitive diagnosis of pelvic fractures is made with radiographs. This usually requires general anesthesia, although standing pelvic radiography has been described.[1,21] Even under general anesthesia, there are significant limitations in the sensitivity of radiographs of the pelvis in adult horses. Consequently, nuclear scintigraphy, either with an imaging or point-counting technique, is often used for screening.[22,23] Scintigraphy is the only technique that can identify subtle injuries, such as ileal stress fractures. If a displaced fracture is suspected, a simpler and less-expensive alternative is ultrasonography; it is possible to clearly identify discontinuities of a cortical surface in accessible locations.[2,24,25] Ultrasonography is particularly useful for diagnosing and defining tuber coxae fractures.

Figure 100-7. A 6-month-old weanling suffering from a femoral shaft fracture of 1 month's duration in the right hindlimb, which was treated conservatively. Note the marked varus deformity in the left hindlimb.

Treatment

Treatment of pelvic fractures is almost always conservative, although displaced iliac shaft fractures in foals can be repaired successfully with internal fixation (Figure 100-8). The ilium is approached by elevating the gluteal musculature sharply along its ventral attachments and forcefully retracting the musculature dorsally with Hohmann elevators. The fragments are reduced and secured with bone plates, preferably locking plates. In non-articular fractures, anatomic reduction is not mandatory for a good result. Surgical repair of pelvic fractures in foals is restricted to those with marked displacement and a need for athletic function.

There are no reported cases of the successful repair of acetabular fractures in horses of any age, although it is feasible to approach the dorsal rim in younger foals or Miniature Horses. Displaced tuber coxae fractures ("knocked-down hips") are usually not treated surgically either, although removal should be considered if they are open and draining.

In adult horses, stall rest for at least 3 to 4 months is combined with adequate analgesia, support bandaging, and frog support on the contralateral limb. Epidural analgesia can be used with caution, especially to manage the more intense pain in the initial period after the injury. Younger horses heal more quickly but also develop limb deformities more readily in the contralateral limb.

Prognosis

The prognosis for pelvic fractures depends primarily on the degree of displacement. Nondisplaced fractures of any portion of the pelvis, even those involving the acetabulum, heal without serious consequences, whereas displaced injuries that cause persistent pain lead to muscle wasting, contralateral limb and foot

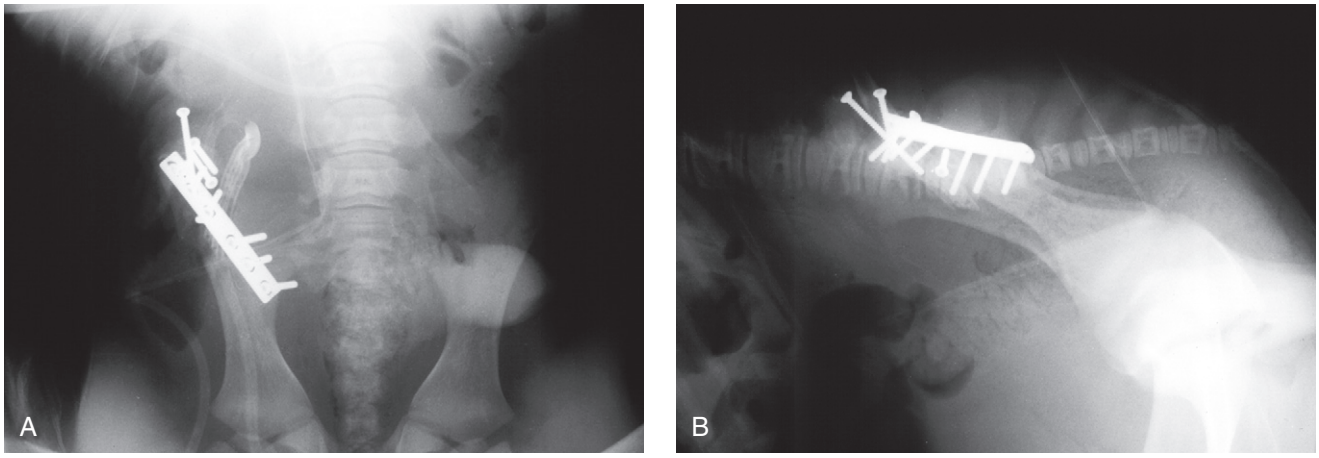

Figure 100-8. Ventrodorsal **(A)** and lateromedial **(B)** radiographic views of a 1-month-old foal with an iliac shaft fracture. The fracture was repaired using open reduction and internal fixation. Adequate exposure and reduction in larger individuals is enormously difficult. In this case, screws were used to hold the fragment in reduction as a single locking plate was applied to the iliac wing down onto the shaft.

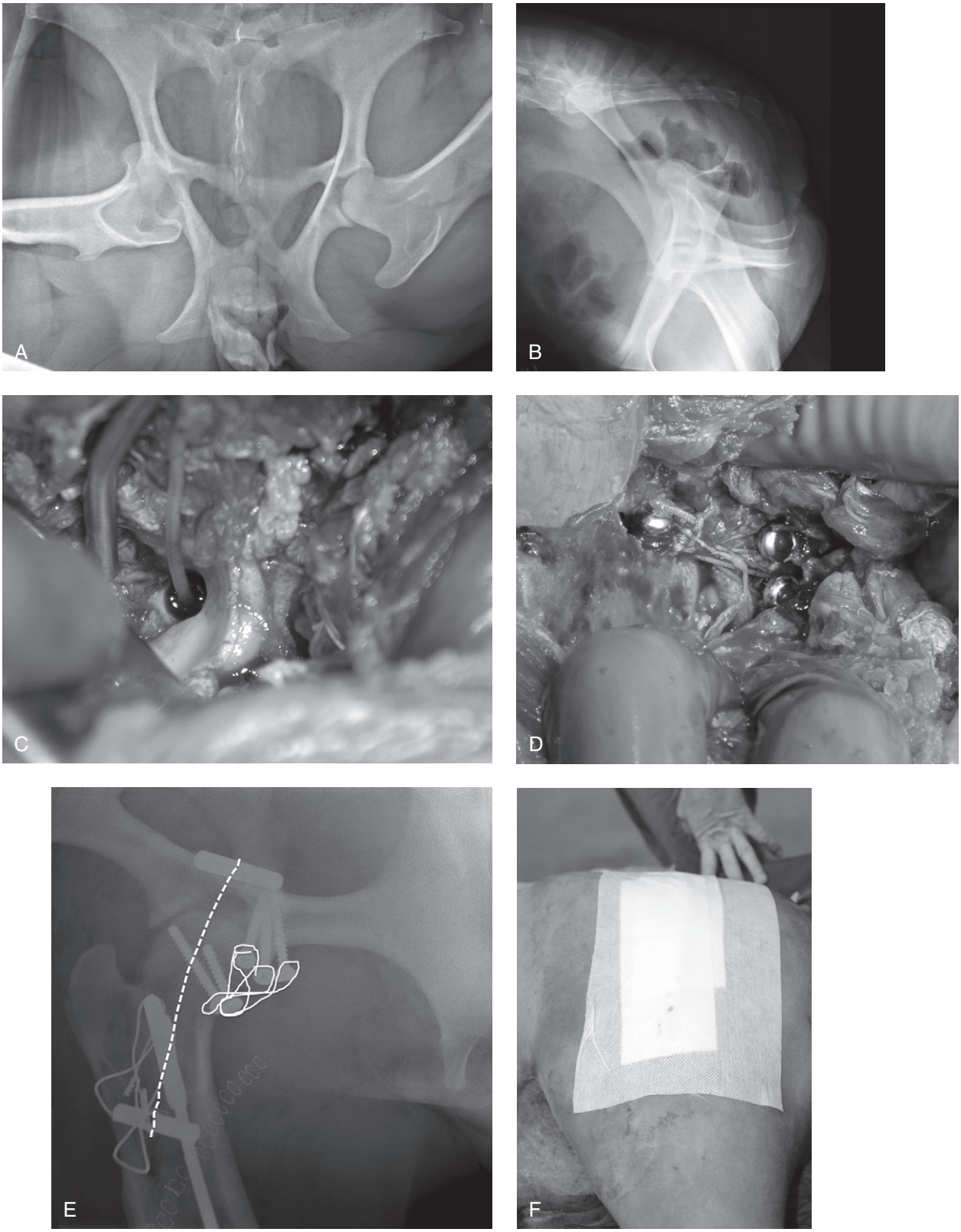

Figure 100-9. Ventrodorsal **(A)** and lateromedial **(B)** radiographic views showing a typical craniodorsal coxofemoral luxation in a Miniature Horse. **C,** Intraoperative view showing a hole made in the central acetabulum through which the toggle-pin is passed. **D,** Intraoperative view of the screws inserted along the acetabular rim and the proximal femur that are connected with multiple interlaced strands of braided suture or cable. **E,** The braided strand of suture or cable is passed through the femoral head and out the side of the femur, where it is secured to another toggle *(dotted line).* The braided wires between the screw heads in the acetabulum and femoral head are shown. **F,** An adhesive bandage is applied over the incision to keep it clean.

problems, and coxofemoral arthritis. In mares, markedly displaced fractures can compromise the birth canal and lead to dystocia. In one study, a successful outcome occurred in 77% of horses that were not euthanized immediately after the pelvic fracture was diagnosed.[19] With ultrasonographic diagnosis becoming more common, characterization of displaced fractures without general anesthesia may allow earlier, more accurate prognosis.[2] However, because surprisingly rapid improvement and healing can occur, precipitous decisions to euthanize horses with pelvic fractures should be avoided, unless it is absolutely clear that recovery is impossible or the economic pressures dictate against continued treatment.

COXOFEMORAL LUXATIONS

Coxofemoral luxations are rare in equids but are more common in ponies and Miniature Horses than in larger animals.[26] Most occur after falls, after attacks by larger horses, or while the animal is struggling to extract an entrapped limb or straighten an upwardly fixed patella.[27] Coxofemoral luxations and femoral head fractures can occur in horses and foals wearing full hindlimb casts.[28]

Clinical diagnosis of coxofemoral luxation is not difficult except to distinguish it from a femoral head fracture, which can be achieved through radiography or ultrasonography. The affected horse is very lame and holds the distal limb externally rotated. Because most luxations occur craniodorsally, the point of the tarsus is higher than the normal side when the horse is viewed from behind. Manipulation of the limb might or might not yield a soft, clicking crepitus. Upward fixation of the patella may be present.

Treatment

Treatment of coxofemoral luxation in Miniature Horses and ponies has been accomplished by reduction and primary repair or by excision arthroplasty.[27,29-31] Although it is possible that a closed reduction may succeed, open reduction and some form of stabilization is nearly always necessary.

Surgical repair involves a craniodorsal approach to the hip with or without greater trochanteric osteotomy. If a trochanteric osteotomy is elected, it is advisable to pre-drill one or two holes to facilitate tension-band repair during closure. Before the femoral head is reduced, the joint is cleaned of fibrin and any debris. Any remnants of the round ligament are removed. An effort should made to avoid any further injury to the dorsal labrum.

Reduction often requires mechanical assistance with either a calf jack or a pulley system rigged up in the operating room. A hip skid, which is a long levering instrument with a curved spoonlike end, can be helpful in reducing the luxation. In most cases, replacement alone is not successful because of continued instability. Therefore, toggle pinning or augmentation of the lateral joint capsule with synthetic sutures attached to screws might be necessary. The latter technique involves placing screws above the cranial acetabular margin and lateral femoral shaft just distal to the neck (Figure 100-9). A large braided suture is tied around the screw heads to help prevent the head from moving proximolaterally. Washers can help to minimize slippage of the suture material from under the screw heads. The success of this procedure is much higher in the smallest, lightest patients, that is Miniature Horses. In heavier Shetland type ponies or any larger individuals, failure of the fixation is fairly common.

The toggle pin technique involves drilling a hole from the lateral proximal femur aiming up through the femoral head precisely where the round ligament inserts. A toggle pin can be made by drilling a hole through the middle of a 2.5 to 3 cm piece of a large-diameter (5 to 6 mm) Steinman pin. The edges of the hole must be smoothed to avoid abrasion of the suture material. A 3- to 5-strand braid of the strongest available nonabsorbable braided suture material (e.g., polyester or, preferably, a polyester/polyethylene composite) is passed through the hole and doubled to a free end. A hole is then drilled in the acetabulum at the site of the round ligament origin. The toggle with braid attached is inserted vertically through the acetabular hole, and then tension is applied to seat it transversely dorsal to the acetabulum. The braid is then passed down the hole made at the round ligament insertion in the femoral head and retrieved from its exit point on the lateral femur. It is usually necessary to use a wire attached to the braid to pass it down the drill tract. The braid is then passed through a second toggle pin. The femur is abducted and the tension on the braid is adjusted until it is taut when the hip is allowed to adduct to a normal position. The braid can be tied over the toggle with routine knotting technique because the knot is tied with the limb abducted. The trochanteric osteotomy is repaired with a tension band technique (screws or pins and wire or cable [see earlier]). The surgeon must be cognizant of the path of the cable through the femoral head and proximal femur so that it is not damaged during the trochanteric repair.

Femoral head and neck excision does not usually result in acceptable long-term comfort except in small ponies and Miniature Horses. The surgical exposure is achieved through a craniolateral approach, and an oscillating saw, Gigli wire, or wide osteotome is used to transect the femoral neck as close to the shaft as possible. Postoperatively, the horse should be kept on optimal footing to prevent the animal from slipping. Use of soft rope hobbles may be helpful to prevent abduction if the horse will tolerate them. A medial patellar desmotomy is usually necessary to avoid persistent upward fixation of the patella.

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CHAPTER
101 Temporomandibular Joint Disorders

K. Josef Boening

The detailed anatomy of the temporomandibular joint (TMJ) and its direct relationship to adjacent structures is well documented in the literature.[1-4] This knowledge is a prerequisite for correct diagnosis of TMJ pathologies and enhances the clinician's ability to diagnose temporomandibular disorders.

ANATOMY

The TMJ has an incongruous shape formed ventrally by the mandibular condyle (condylar process) and dorsally by the zygomatic process of the temporal bone (Figure 101-1). Like all joints that are incongruous, a fibrocartilaginous disc (commonly referred to as a *meniscus*) is interposed to level out the incongruity. This meniscus has an elongated, roundish appearance, is approximately 5 mm thick at the border, and divides the joint into two separate compartments (Figure 101-2).[5,6] The dorsal discotemporal compartment is more spacious than the ventral discomandibular compartment. Two ligaments support the joint capsules in maintaining joint alignment (Figure 101-3).

The TMJ is covered by the parotid gland and is in close proximity to the more ventrally located transverse facial artery and vein and the transverse facial branch of the auricular-temporal nerve (Figure 101-4).

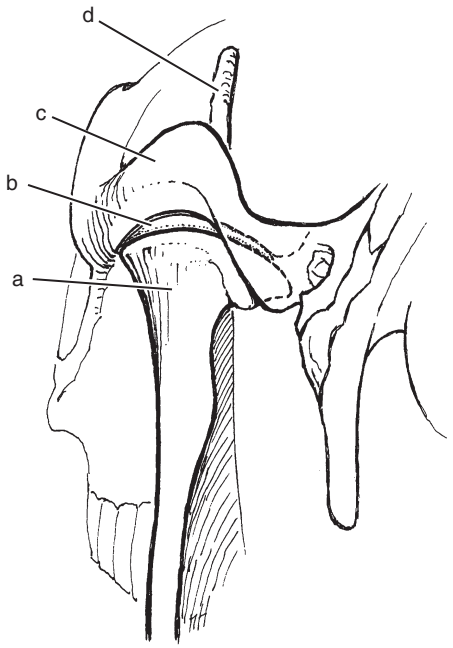

Figure 101-1. Caudocranial view of the temporomandibular joint. *a,* Vertical ramus of the mandible with the proximally located mandibular condyle; *b,* meniscus interspersed between the mandibular condyle and temporal bone; *c,* temporal bone; *d,* coronoid process of the mandible.

CLINICAL SIGNS

Clinical signs are variable and include reluctance to eat and asymmetry of the masseter region. Careful clinical examination

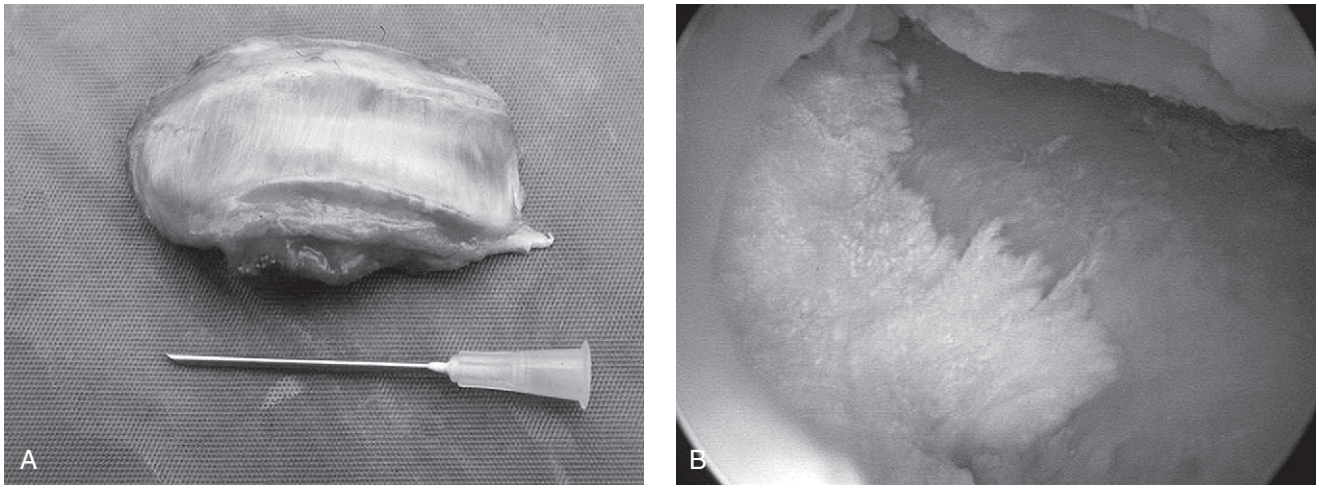

Figure 101-2. A, Normal size of the temporomandibular disc. **B,** End stage destruction of the disc associated with temporomandibular joint osteoarthritis.

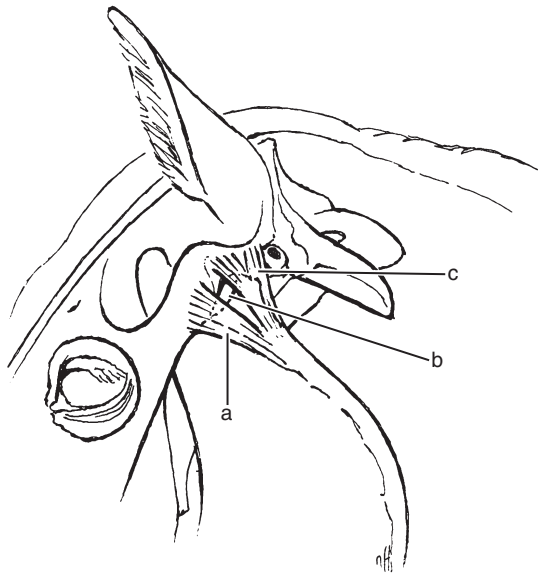

Figure 101-3. Side view of the temporomandibular joint. *a,* Lateral ligament; *b,* meniscus (the discotemporal- [proximal] and the discomandibular [distal] joint compartments are not shown); *c,* caudal ligament.

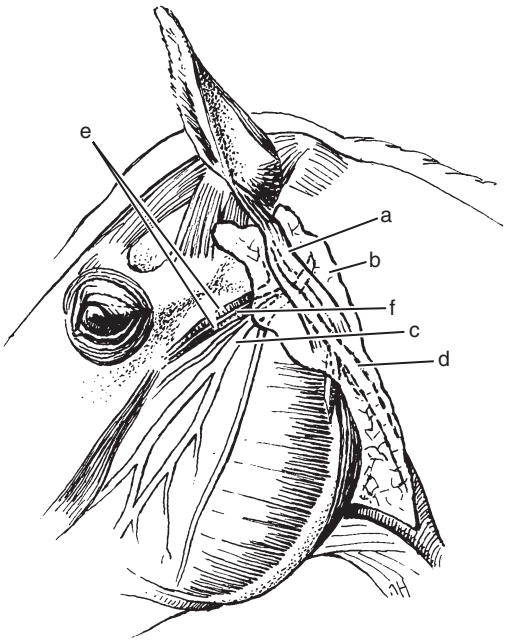

Figure 101-4. Anatomic landmarks for approaching the temporomandibular joint. *a,* Paratidoauricular muscle; *b,* parotid gland; *c,* facial nerve dividing into different branches; *d,* maxillary vein covered by the parotid gland; *e,* transverse artery and vein; *f,* transverse ramus of the facial nerve.

of the entire head, with special attention to the teeth, should be performed.[7-11] Asymmetry of the head can be caused by either muscle atrophy or a swelling on the affected side. It was only recently reported that head shaking, misbehavior, and head tilt were associated with TMJ disorders. Attitude changes, such as a lack of submissiveness, can also be attributed to TMJ disorders.[12]

DIAGNOSIS

In some cases, it is difficult to recognize which joint is affected without the help of diagnostic imaging techniques. Most information can be gained from standard lateral, oblique, and tangential radiographic projections.[13-16] Ultrasonography,[17-21] nuclear scintigraphy,[22] and, more recently, computed tomography (CT)[23-25] and magnetic resonance imaging (MRI) have become the preferred techniques for diagnosing TMJ disease in the horse.

TMJ sepsis[26] is rare in horses and most frequently is related to penetrating wounds.[27-29] However, standard diagnostic aids used to diagnose joint infections can be applied to the TMJ (see Chapter 85).

TREATMENT

As expertise in the diagnosis and management of TMJ disorders increases, invasive techniques are being developed to treat them.[30,31]

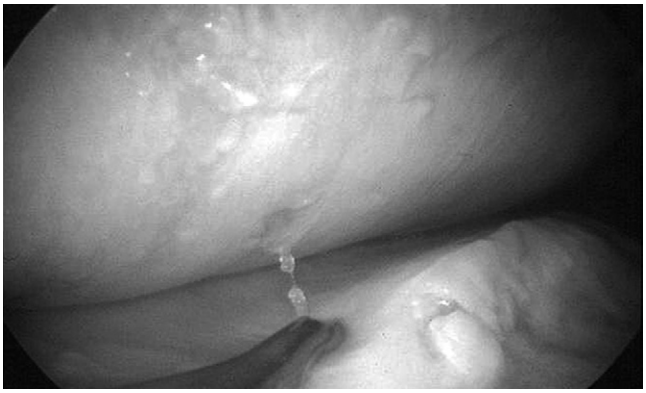

Figure 101-5. Arthroscopic view of the temporomandibular joint showing a focal cartilage defect. The disc is displaced ventrad with the help of a curette.

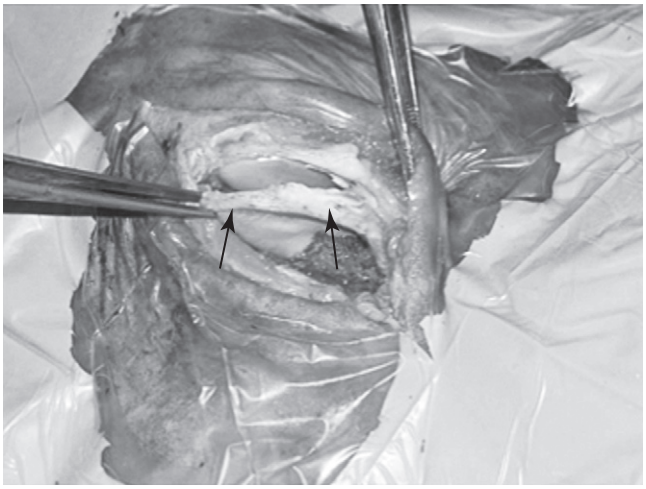

Figure 101-6. Arthrotomy into the TMJ for the purpose of mandibular condylectomy. The meniscus is shown *(arrows)*.

Arthroscopy

A first description of arthroscopic approaches to TMJ compartments was presented in 1986 as part of a clinical case report[12] describing a horse with submissiveness problems and a head tilt, which was associated with a transverse unilateral tear in the TMJ disc. The diagnosis was made after standing nuclear scintigraphy followed by intrasynovial anesthesia.

A cadaveric study revealed that a good portion of the TMJ can be evaluated arthroscopically.[32] The first retrospective clinical study in TMJ arthroscopy has been published and illustrates the advantage of this technique as both a diagnostic and a surgical treatment procedure.[33]

Approach

For unilateral arthroscopy of the TMJ,[34] the horse is anesthetized and placed in lateral recumbency on the surgery table. After routine aseptic preparation of the surgical site, the caudal recess of the proximally located discotemporal joint, which is usually easily palpable, is distended by injecting approximately 15 mL of Ringer's lactate solution through a 20-gauge needle.[35] A 5-mm skin incision is made with a No. 11 scalpel blade next to the needle, followed by penetration of the joint capsule in a rostromedial direction with the blunt trocar of the 4-mm 30-degree arthroscope. Care is taken not to injure the proximal aspect of the parotid gland that may extend into this region. An instrument portal is made adjacent to the arthroscope portal to assist in exploring the joint. Having an assistant performing sideway movements of the mandible facilitates exploration further. The arthroscopic portal to the rostral recess of the discomandibular joint compartment, is located immediately rostral to the mandibular head and ventral to the discotemporal joint space. Because this joint compartment is smaller than the proximal compartment, exploration of the discomandibular joint compartment is more difficult.

Either fluid or gas distention can be used in this joint, but the use of gas prevents the synovial villi from obscuring the surgeon's view.[36] Each arthroscopic procedure is completed by extensive fluid lavage and skin closure.

A clinical case series (Figure 101-5) described five Warmblood horses that underwent arthroscopic débridement of the affected TMJ lavage and in one case removal of a free-floating osteochondral fragment followed by débridement of the meniscal borders. All patients were also treated with intra-articular corticosteroid injections. Three horses became asymptomatic and could resume their athletic careers.[33] One case report described successful management of a septic TMJ with mechanized resection of synovium and fibrinous debris, copious lavage, and intra-articular and systemic antibiotics.[26] Eight months postoperatively, there was no clinical evidence of osteoarthritis or ankylosis of the TMJ.[26]

Condylectomy of the Mandible

Mandibular condylectomy is a radical procedure and is an option for treatment of fractures and chronic septic arthritis involving the TMJ that are not amenable to repair. Other indications include management of luxations and chronic painful osteoarthritis of the TMJ.[37,38] However, this technique should be viewed as a salvage procedure, and additional work is needed to find a practical solution for maintaining TMJs with osteoarthritis.

Surgical Procedure

A condylectomy is performed in lateral recumbency under general anesthesia.[37] An approximately 6-cm horizontal skin incision is made, centered over the TMJ joint and curving somewhat distad caudal to the mandibular condyle. The soft tissues are reflected ventrad to expose the joint capsule. The transverse facial artery/vein and the auricular temporalis nerve should be identified and avoided during the approach (see "Anatomy," earlier).

The TMJ is opened by a horizontal incision of the joint capsule (Figure 101-6). The periosteum on the condyle is incised vertically 2 cm distal from the joint space, and a periosteal elevator is used to reflect it both rostrad and caudad. An oscillating bone saw is used to create a 2-cm deep cut in the mandibular condyle 2.5 cm ventral to the articular surface.

A chisel is inserted into the osteotomy and the lateral portion of the condyle is pried dorsad until it fractures. It is necessary to remove the lateral portion of the condyle before continuing the cut through the rest of the mandible under the condyle in order to to provide room to introduce scissors to sever the

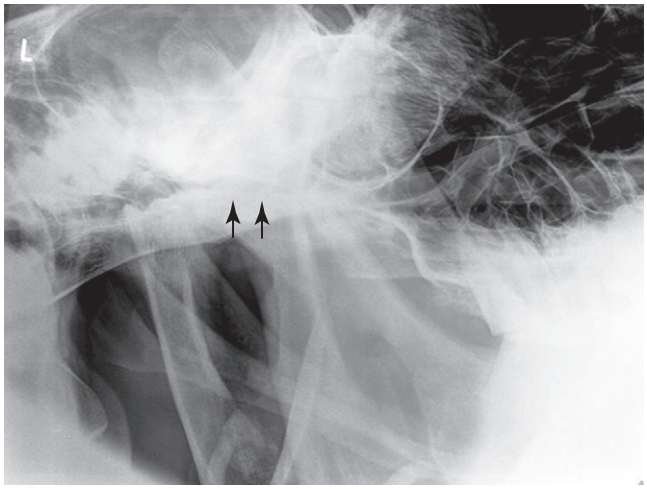

Figure 101-7. Radiographic appearance of the formation of a pseudocondyle after unilateral condylectomy *(arrows)*.

capsular attachments. The oscillating saw is used to continue the osteotomy through the remaining axial portion of the mandible below the condyle. The condyle is grasped with large forceps and removed following scissor transection of any remaining joint capsule attachments. The articular meniscus is removed after severing its attachments with scissors.[37,39] The bone edges are rounded with the help of a curette.

The incision is closed in three layers: the periosteum and joint capsule, the subcutaneous tissues, and the skin. Additional information on TMJ disorders is found in Chapter 30.

Complications and Prognosis

The results of mandibular condylectomy have been summarized for normal horses.[37]

Radiographic, gross, and histological evaluations show that the condylectomy sites undergo remodeling and bony proliferation to develop pseudocondyles (Figure 101-7).[37] Condylectomy has been performed in a case of septic arthritis and end-stage osteoarthritis.[37] Short-term complications in normal horses included masseter atrophy, malocclusion, weight loss, and difficulty in prehension and mastication.

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CHAPTER
102 Craniomaxillofacial Disorders

Jörg A. Auer

ANATOMICAL CONSIDERATIONS

The bones of the head consist of the skull, mandible, and hyoid bone (Figure 102-1). The skull can be considered to be composed of the cerebral cranium or neurocranium, which forms a solid shell around the brain, and the visceral- or splanchnocranium, which forms the basis for the face but also includes the mandible and the hyoid bone.[1] Alternatively, the skull can be considered to be made up of only the bones of the upper head (without mandible and hyoid bone).[2] In this chapter, the latter classification has been adopted. In contrast to humans, the visceral cranium of the horse is much larger than the cerebral cranium and lies rostral to rather than below it. The bones of the skull are usually flat and have a compact external and internal lamina separated by a cancellous/spongy layer. The different bones of the skull are joined by serrated suture lines.[1]

The *cerebral cranium* is divided into the roof and base of the skull and consists of the dorsally situated frontal bone, the interparietal bone and parietal bone, the laterally located temporal bone (composed of a petrous part, a tympanic part, which contains the inner and middle ear, and a squamous part attached to the zygomatic process), the ventrally situated sphenoid bone, the occipital bone, and the ethmoid bone.[2]

The bones of the *visceral cranium* form the ocular, nasal, sinus and oral cavities. The visceral cranium consists of the frontal bone and nasal bone dorsally and the lacrimal bone, zygomatic bone, the paired incisive bone, and the premaxilla laterally. The incisive bone consists of the alveolar part, which contains the alveoli of the upper incisors, the palatine process, which forms the rostral part of the hard palate, and the nasal process. Ventrally, the visceral cranium consists of the palatal bone, the vomer and pterygoid bone, and, caudally, the ethmoidal bone.[2]

The *mandible*, as a part of the visceral cranium, consists of the incisive part, interalveolar rim, molar part, mandibular ramus, condylar process and coronoid process. The anatomic terminology describing the mandible is not consistent. In this chapter, the tooth-bearing part is referred to as *body* or *corpus* and the remaining vertical part as *ramus* or *branch* of the mandible. The two mandibular branches are fused to form an intermandibular suture line via a synchondrosis, which ossifies during the second year of life.[2]

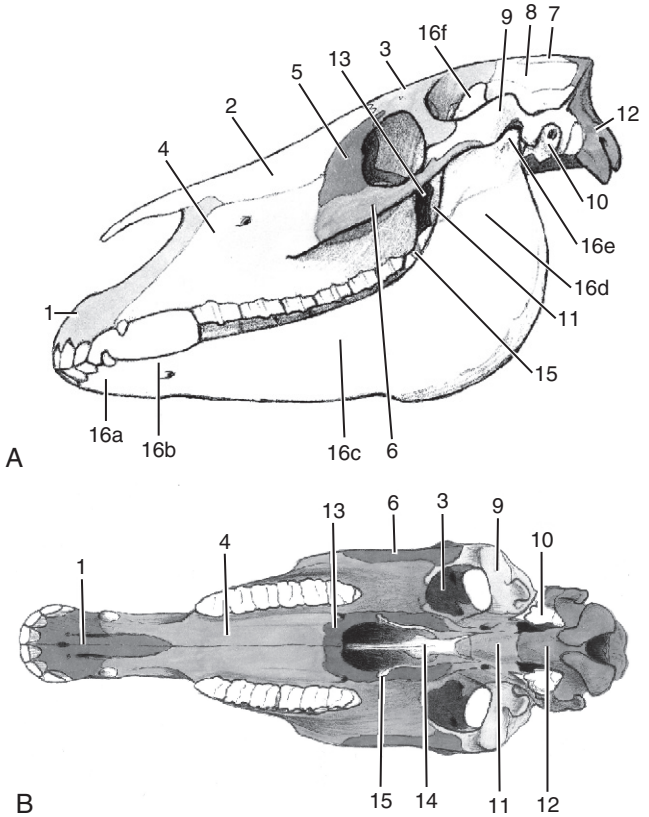


Figure 102-1. Graphic display of the bones of the skull. **A,** Lateral view. **B,** Ventral view. *1,* Os incisivum; *2,* os nasale; *3,* os frontale; *4,* maxilla; *5,* os lacrymale; *6,* os zygomaticum; *7,* os interparietale; *8,* os parietale; 9, os temporale; *10,* pars petrosa and pars tympanica of the os temporale; *11,* os sphenoidale; *12,* os occipitale; *13,* os palatinum; *14,* vomer; *15,* os pterygoideum; *16,* mandibula; *16a,* pars incisiva; *16b,* margo interalveolaris; *16c,* pars molaris; *16d,* ramus mandibulae; *16e,* processus condylaris; *16f,* processus coronoideus.

FRACTURES OF THE INCISORS, MANDIBLE, AND PREMAXILLA

Trauma to the head often results in fractures of the teeth, incisive bone, mandible, premaxilla, or maxilla. These injuries occur as blunt trauma from kicks, falls, and collisions or from being startled in the stall while cribbing on stall slats or mesh wire. Sudden jerking of the head while chewing on stationary objects may result in avulsion fractures of the incisors.

Whatever the cause, these fractures can be repaired with good cosmetic and functional results. Several factors make this possible. These bones have a good soft tissue covering and have an abundant vascular supply. Because the bones of the head are not subjected to the same loading forces as the bones of the extremities, the demands for fracture fixation are less challenging. The teeth serve as stabilizing aids. Implants of adequate strength are available, and the instrumentation required is in most cases inexpensive and readily available.

Diagnosis

A thorough physical examination is the basis for a correct diagnosis. One of the first signs of a fracture of the jaws is excessive drooling and lack of appetite. Soft tissue swelling, hemorrhage, instability, and malalignment of the teeth and bones are usually noticed on closer inspection. A fetid odor accompanies open fractures that are several days old.

Malocclusion of the incisors can be induced manually even in normal horses because of the normal side-to-side mobility of the temporomandibular joint. However, both mandibles move in the same direction with manipulation unless a fracture is present, in which case one side of the mandible can be manipulated independently of the other. Often, soft tissue

swelling obscures the detection of malalignment, especially if the fracture is located under the masseter muscles. Horses might object to manipulation and examination of the oral cavity because of pain. A protruding tongue should alert the clinician to suspect a bilateral fracture. Drooling occurs as a result of an inability to properly close the mouth.

The diagnosis is in most cases not difficult, but other structures might be injured concurrently; therefore, the nasal passages and cranial nerves should be examined. Horses can become significantly dehydrated after hemorrhage and salivary loss, which can be compounded by the patient's reluctance to drink. This is especially true if the fractures are not detected in a timely manner. The physical examination also should include cardiovascular parameters that would identify potential anesthetic risks. In the majority of the cases, repair is not an emergency procedure, allowing adequate time to arrive at a comprehensive surgical plan.

In most cases, radiographs are difficult to interpret because of the complexity of the bones in the head and the presence of the teeth, which often are superimposed over fracture lines.[3] Nevertheless, radiographs often demonstrate fractures even with minor malalignment and instability. Also, radiographs provide information on the precise location of the tooth roots in relation to the fracture(s), which helps in devising a surgery plan that avoids damage to the permanent teeth. Radiographs often aid in detecting potential sequestrum formation in chronic cases. Computed tomography greatly improves preoperative appreciation of the fracture configuration, especially when three-dimensional reconstruction of the region of the fracture is obtained (Figure 102-2).

Not all fractures require repair. Unilateral fractures of the mandible, maxilla, premaxilla, or incisive bone that are minimally displaced and do not result in significant malocclusion may be treated conservatively. Indications for repair include fractures that are unstable, result in malocclusion of the teeth, or are bilateral.[4] Other considerations that might favor surgical repair include improved cosmesis because of decreased callus formation, more rapid healing in the face of increased stability, and horses that are either unable or reluctant to eat because of pain.[4]

Preoperative Management

Simple fractures involving incisor teeth (when they do not require fixation devices that extend caudad to the canine teeth) can be managed in the standing, sedated horse with regional

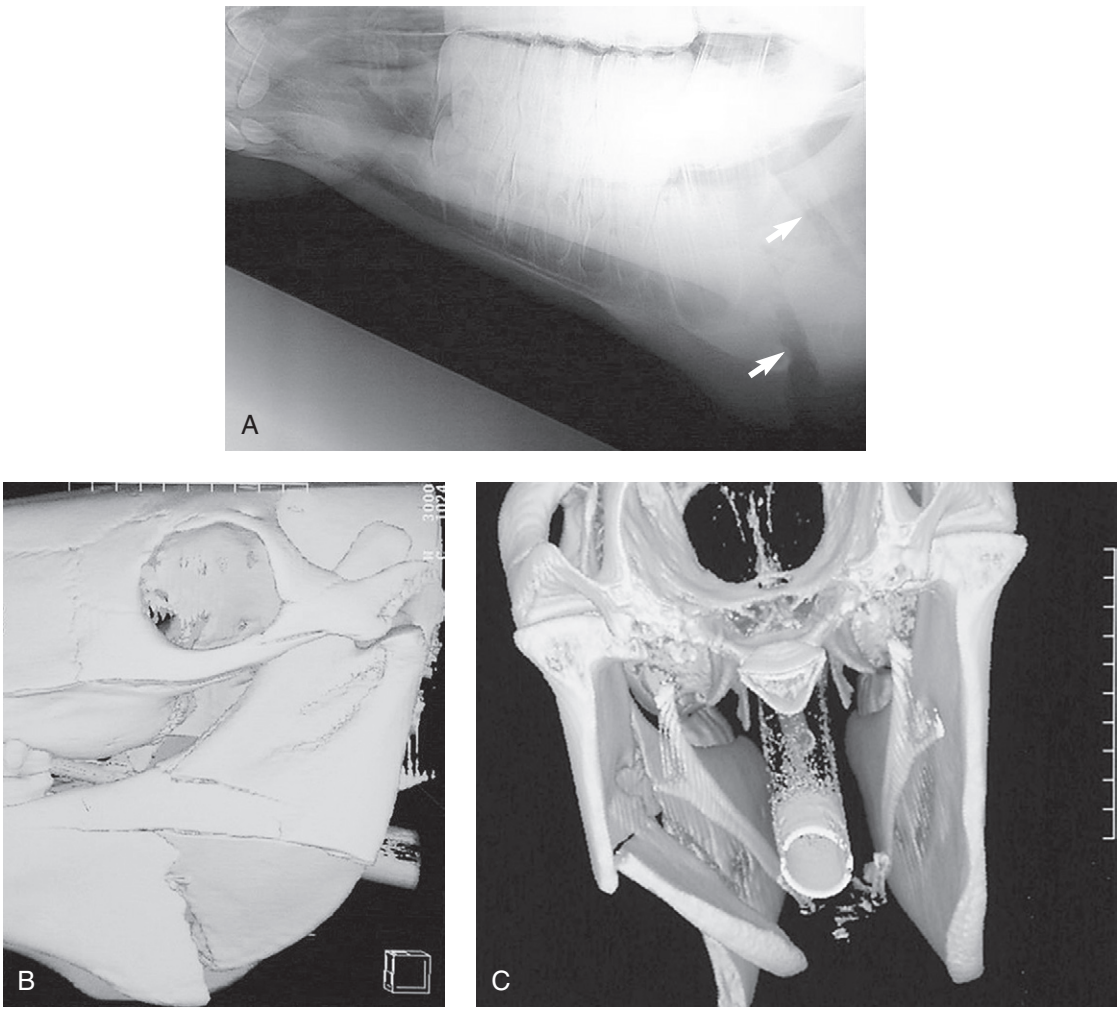


Figure 102-2. A, Lateromedial radiograph of a complicated mandible fracture in a foal. Two fracture lines are visible *(arrows).* Lateral view **(B)** and caudal cross-sectional view **(C)** of the three-dimensional reconstruction of the computed tomography study of this foal. The complexity of the fracture is easily visible.

anesthesia. Local anesthesia is easily accomplished by infiltrating 5 mL of lidocaine or mepivacaine around either the mental or infraorbital nerves at their exit point from their respective foramina.[5] Bilateral infiltration of the nerves is required in most cases.

The standing position facilitates observation of symmetry during surgical repair. However, general anesthesia is preferred because of the potential for injury to the surgeon by the patient in response to painful stimuli. Intravenous anesthetic regimens (see Chapter 19) are appropriate in most of the simple fractures, because patient and surgeon preparation and positioning requirements are minimal, allowing the repair to begin immediately after induction. When inhalation anesthesia is desired or indicated, nasotracheal intubation is preferred to allow unimpeded access to the fracture (see Chapter 18).

The location of the fracture, its complexity, and the personal preferences of the surgeon dictate the patient's positioning on the surgery table. Most uncomplicated unilateral fractures are repaired in lateral recumbency. Access to both sides of the mouth is required for bilateral fractures and is most easily accomplished with the horse in dorsal recumbency, with the poll flexed so that the surgeon can look down into the horse's mouth.[4]

Preoperative planning should include the probability of fracture reduction and determine which implants will provide the best stability. Additionally, any splint that might be required must be pre-shaped, ideally on a cadaveric specimen of approximately the same size as the patient to reduce anesthesia time.

Before repair, the mouth is rinsed with water to remove feed material; a dental pick is used to remove any feed material from fracture lines. The oral cavity is contaminated, and surgical scrubs are of dubious value if intraoral wiring alone is to be used.[4] Repair methods that use internal fixation devices deserve all of the precautions and considerations that internal fixation in other areas require because the implants are usually left in place. Prophylaxis against tetanus is indicated in all cases. Perioperative broad-spectrum antibiotics are strongly encouraged, because the surgery is usually performed in a contaminated field.

Surgical Considerations

The tension side of the jaw is the oral surface of both the mandible and the maxilla, and surgical repair should be directed accordingly when possible. Unilateral fractures of the mandible are partially supported by the intact contralateral side. This aids in the stability of the final repair. Avulsion fractures of the incisors, mandible, and premaxilla are usually simple fractures. The ends of the fractured bone usually interdigitate well and can be repaired using intraoral wiring.

Fractures of the incisors that result from kicks or collisions push the teeth caudad. These fractures are usually associated with a considerable amount of instability and some comminution of the bone caudal to the incisors. The extent of comminution can be assessed during physical examination by the stability of the fracture after it is reduced and by the resistance to collapse with manual manipulation.

Intraoral wiring alone tends to collapse comminuted fractures and results in malocclusion when the wires are tightened. In these cases, it is necessary to provide a buttress to maintain length and prevent collapse. This can be accomplished by intraoral splinting, external fixators, or bone plates applied in buttress fashion.[6]

Closed reduction is desirable to preserve blood supply and to leave soft tissues intact for support. Every effort should be made to prevent contamination of healthy tissues by exposure to the oral cavity.[4] Because accuracy of reduction is in most cases readily determined by visual inspection of the teeth, open reduction is often not necessary to ensure alignment of the fractures. The soft tissue attachments of the gingiva should not be disrupted. Small bone fragments devoid of their soft tissue attachments, and therefore their vascular supply, should be removed when the fracture is open. However, any bone fragment attached to the gingiva or periosteum should be left in place.

Loose teeth should *never* be removed until the fracture has healed and clear evidence is present that they are devitalized. Viability is not always easy to determine at the time of surgery, and many teeth will survive even if they are fractured or loose at the time of repair. More importantly, the teeth often serve a vital function as anchor sites in the thin flat bones of the jaws and quite often support the neighboring teeth when wires are used in the repair.

Intraoral wiring and external fixation methods have some advantages over internal fixation using plates and screws in fractures of the mandible and maxilla for several reasons. Intraoral wiring configurations are inexpensive and almost infinitely variable and adaptable to almost any fracture configuration. Bone in this area is soft, and screws can be easily stripped. The roots of both permanent and deciduous teeth occupy a large part of the mandible and maxilla and make placement of screws for plate fixation difficult to accomplish without damaging the teeth.[4] Radiographic control is usually necessary if internal fixation methods are used. Accuracy of screw placement, screw length, and proper bone engagement of the screws are not easily determined without intraoperative radiographs.

The oral cavity contains a large population of resident microflora. Fractures are usually open and contaminated so that internal fixation often results in infection, therefore necessitating eventual removal of the implant. Instrumentation for internal fixation requires more expertise, and the procedures are more time consuming. It is difficult to place bone plates on the tension surface. This does not preclude their use, and bone plates can result in satisfactory repair when placed on the lateral or ventrolateral aspects of the mandible because in this location the plates are loaded parallel to their width and not their thickness, which provides greater resistance against cyclic loading. The same holds true for the maxilla, where the plates should be applied laterally or slightly dorsolaterally.

When repairing fractures of the teeth, mandible, and premaxilla, the goals are anatomic alignment and stable fixation to restore the ability to masticate. Involved incisors are best ground down to prevent contact with the opposing tooth arcade during mastication, which interrupts fracture healing.

Many successful methods have been reported, and the simplest method of repair that achieves the goal of fracture stability should be applied to any situation. Techniques are often combined as necessary.

Surgical Techniques

Intraoral Wires

WIRING OF THE INCISORS

Fractures involving the incisors, incisive bone, symphysis of the mandible, and premaxilla are amenable to intraoral wiring. The wound is properly cleaned and the potential damage to tooth

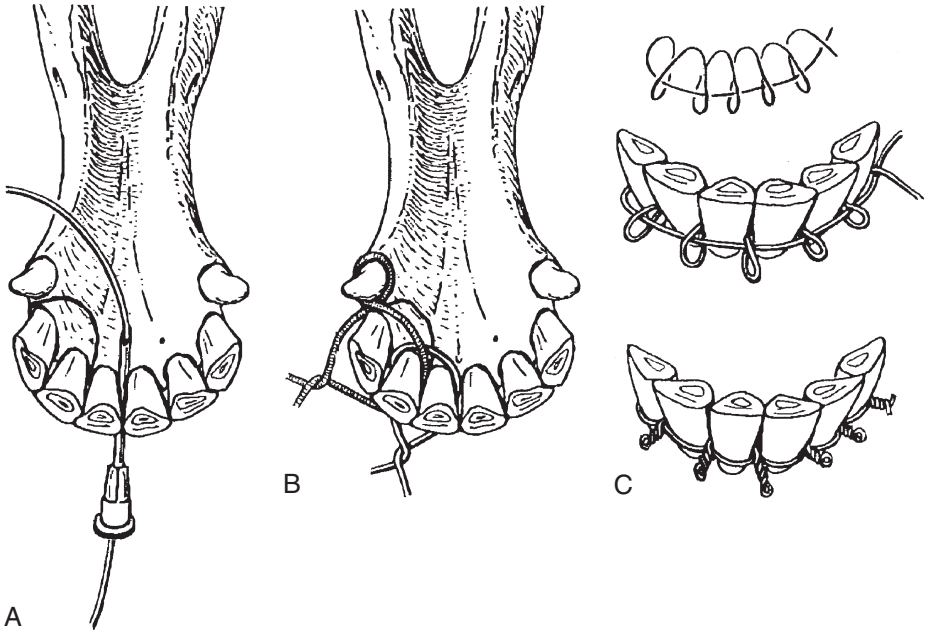


Figure 102-3. Cerclage wiring of a fracture involving 402 and 403. **A,** A 14-gauge needle is inserted between the teeth, and a 1.25-mm stainless steel wire is threaded into the needle as it is withdrawn. **B,** The fracture is treated with two cerclage wire loops, one around 401 and 402 and the other in figure-of-eight fashion around 402 and 403 and the adjacent healthy canine tooth. **C,** An alternate method involves exiting wire loops from the lingual side and the labial side, followed by feeding one end of the wire through each loop. After tightening the two wire ends, each wire loop is tightened as well, resulting in the most stable of all wire fixations. The twisted ends are directed toward the gingiva.

roots or deciduous tooth buds is assessed. Fracture reduction is accomplished by manual manipulation.

A 14-gauge needle is inserted between the teeth at the gum line, and 1.25-mm diameter stainless steel wire is threaded through the needle (Figure 102-3, *A*). Care is taken to prevent sharp bends in the wire from developing while weaving it around the teeth, because these bends prevent effective tightening of the wire. Additionally, cyclic loading of the wire during eating will straighten the wire and in doing so loosen the fixation. Sharp bends also predispose the wire to failure because of local weakening through prestressing. The wires are kept tight during application, and any slack in the wire is removed after each tooth is encircled. Fractured teeth are united to adjacent healthy ones with a figure-of-eight wire passed around all selected teeth and tightened at one end (Figure 102-3, *B*). Another technique involves multiple overlapping cerclage wires placed around the selected teeth in simple loops. Each wire fastens the unstable teeth and associated bone to the normal teeth until alignment and stability are achieved. The selection of the technique to be applied is at the discretion of the surgeon.

Wire fixation is most easily accomplished by simple interrupted wires encircling the teeth. One or two wires are usually sufficient to stabilize most simple fractures of the incisors. After each wire is tightened, the fracture is manipulated to ascertain the alignment and stability. Alternatively, a cable tie can be substituted for each wire, placed with the cut end on the oral side of the teeth to avoid laceration of the lip. Wire repair applying the figure-of-eight technique unites all teeth in one fixation, but because of the relatively long wire arranged in several circles around the involved teeth, tightening the wire is more difficult.[7] Repair is adequate if the incisors cannot be displaced by manual pressure.

A standard wiring technique applied in humans has been proposed.[8] Wire loops are inserted between each tooth from the oral to the labial side (Figure 102-3, *C*). One end of the wire is fed through each loop and is tightened with the other end. Each wire loop is subsequently tightened, resulting in a constricting wire loop around each tooth and therefore increased stability. This technique provides a rigid fixation that allows the patient to eat for months while the fracture heals (Figure 102-4).

Fractures that involve the corner incisors need some sort of anchoring farther caudally. In male horses the canine tooth may serve as such an anchor (Figure 102-5). It is usually necessary to create a notch in the canine tooth at the gum line with a small triangular file to prevent the wire from slipping off of the tooth. Alternatively, a caudal anchor can be created by inserting a 4.5-mm cortex screw into the interdental space and wrapping the wire around the screw head. Loosening of the screw with time is a complication generally encountered with this technique, especially in young horses. Still another technique is to drill a 2.5-mm hole across the lateral edge of the interdental space and feed the wire through it (see later). Tension-band wiring can also be achieved by feeding the wire around the second premolar (i.e., 406, see Chapter 30) and tightening the wire.

In rare cases, cerclage wires may be applied in a circular manner around oblique unilateral fractures of the interdental space (Figure 102-6). The intact opposite side of the mandible ensures maintenance of mandible length and some stability of the fracture ends, and the cerclage wires maintain the fragment ends in approximation and provide some compression across the fracture.

Hemicerclage wires are very useful in fractures of the mandible. Initially a K-wire of 2 to 2.5 mm is introduced across the fracture. The two ends of the pin are united across the surface of the bone in figure-of-eight fashion and solidly tightened,

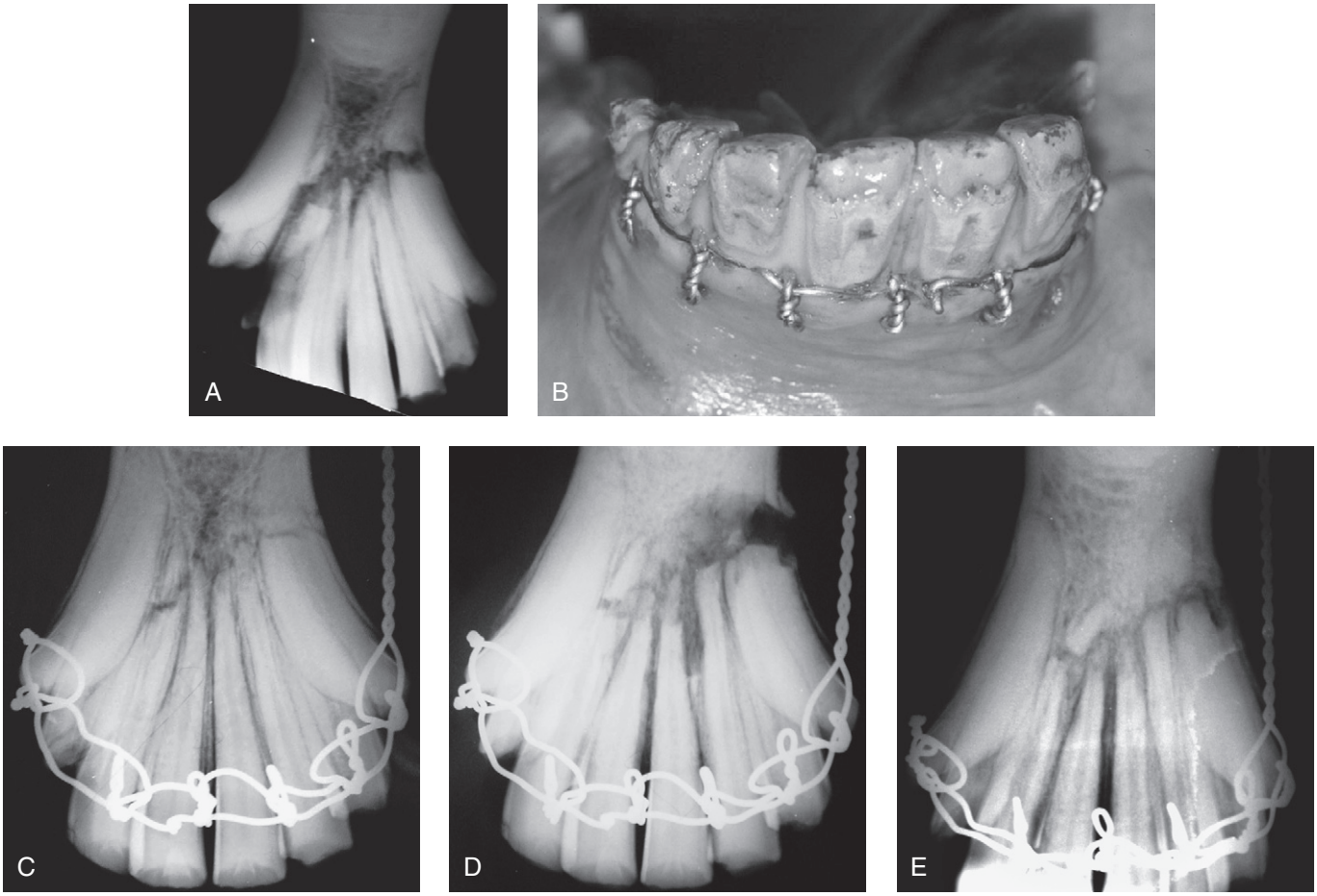


Figure 102-4. Intraoral wiring of a mandible fracture in an 11-year-old Arabian stallion. **A,** Preoperative intraoral radiograph of a fracture involving the base of the incisor teeth. **B,** The technique shown in Figure 102-3, C, was applied. Note the worn-down incisor teeth resulting from continuous crib biting. **C,** Postoperative intraoral radiograph of the fixation. Because the corner incisors were involved in the fracture as well, tension-band wiring was used. **D,** Four-month follow-up intraoral radiograph after curetting of an abscess near the tooth roots. **E,** Seven-month follow-up at the time of wire removal. The fracture has healed and the infection has resolved.

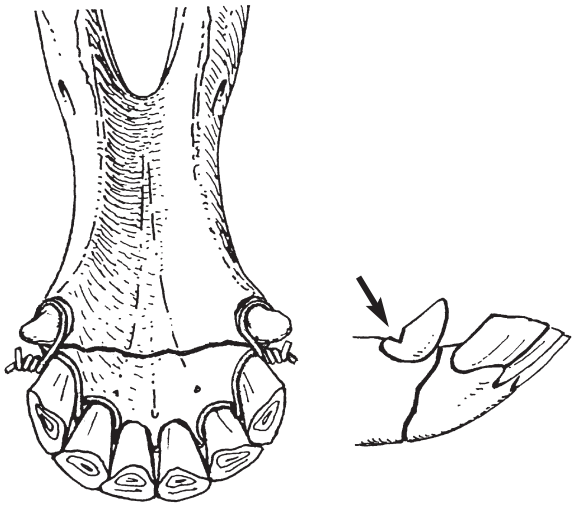


Figure 102-5. Tension-band wiring of incisors to the canine teeth used to repair a fracture involving the incisors. Note that an indentation *(arrow)* was created into the nuchal aspect of the canine teeth with a rasp to anchor the wire.

providing axial compression across the fracture (Figure 102-7). This technique is also very effective in fractures of the symphysis of the mandible in very young foals, where the bone has inadequate strength for screw fixation using lag technique (see later).

Interdental wiring becomes progressively more difficult as fractures happen more caudally on the mandible.[4] It is very difficult to wire one cheek tooth to another one with the limited room to work via an oral approach.

An alternative method of figure-of-eight wiring that does not use bone screws can be performed by anchoring the wire in the bone through holes drilled through the mandible on each side of the fracture (Figure 102-8). The surgical approach is the same. A 2-mm drill bit is used to prepare a hole from a lateral to a medial direction through the mandible near the ventral border approximately 2 cm from the fracture. Figure-of-eight wiring is performed using the drill hole as an anchor site. This technique is adequate for young foals but not for adult horses with fractures of the interdental space.

TENSION-BAND WIRING TO THE CHEEK TEETH

Fractures of the diastema of the mandible are good candidates for repair with intraoral wiring. Unilateral fractures can be realigned and stabilized with a wire placed around the second premolar (306 or 406) and secured to the incisors.

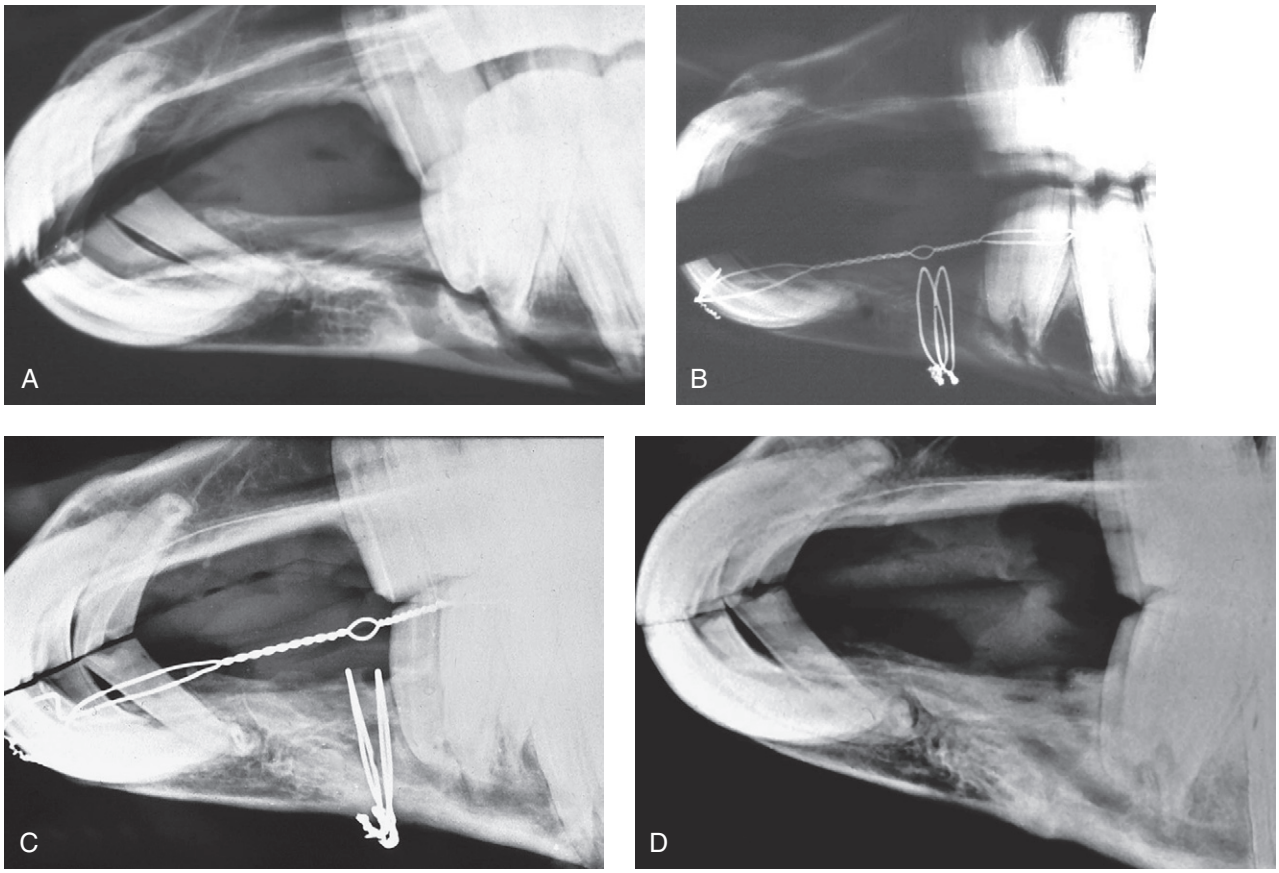


Figure 102-6. A, Preoperative radiograph of a long oblique fracture through the interdental space in a Shetland pony. **B,** Immediate postoperative radiograph showing the two circular wire loops in place, protected by a tension-band wire. **C,** Three-month follow-up radiograph showing the healed fracture. **D,** Radiograph after implant removal.

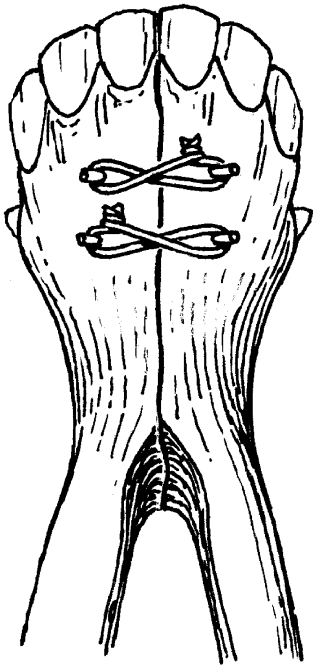


Figure 102-7. Hemicerclage fixation of a symphysis fracture. Two pins are placed across the symphysis. Figure-of-eight wires are placed around the protruding ends and solidly tightened to effectively stabilize the fracture. This type of fixation may also be applied to other locations.

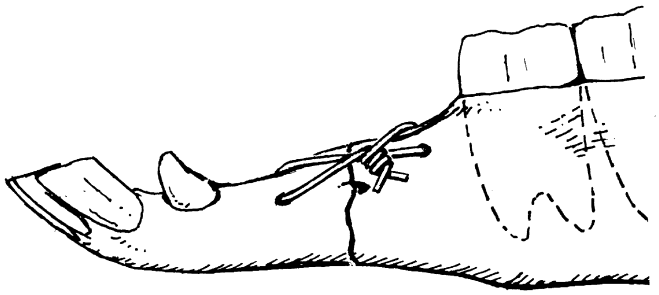


Figure 102-8. Figure-of-eight wiring of a vertical fracture of the mandible through the interdental space. The holes are created with a 2.5-mm drill bit or a Steinmann pin.

Under general anesthesia, the horse is placed in lateral recumbency on the surgery table with the fractured side toward the surgeon. With the help of hypodermic needles, the interdental space at the gingival level between 306 and 307 or 406 and 407 (see Chapter 30) is identified. Occasionally, the interdental space farther back is also used. Digital palpation facilitates identification as well.

When the space is identified, a stab incision is made through the cheek. A 2.5-mm–diameter drill sleeve is introduced through the stab incision and placed over the interdental space, and a 2.5-mm hole is created between the teeth at the level of the gingiva (Figure 102-9). A 1.25-mm diameter stainless steel wire

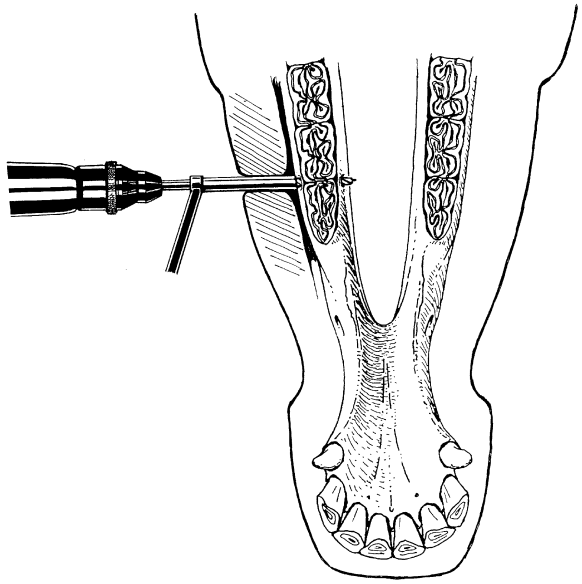


Figure 102-9. The drill guide is used to protect the cheek and gingiva as a drill bit creates a hole for passing the wire between the cheek teeth at the gum line. The wire is used to form a tension band across the interdental space.

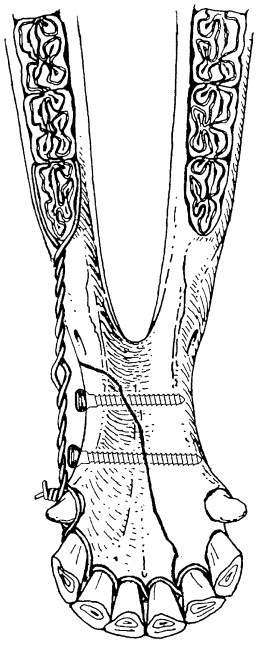


Figure 102-10. Screw repair using lag technique of a unilateral fracture of the mandible containing a large osseous fragment. The fixation is protected with tension-band wiring of the incisors to the first cheek tooth.

is threaded through the cheek and the hole that was drilled between the teeth. Both ends of the wire are pulled into the mouth and drawn rostrad out of the mouth. The wire is crossed and looped around the intermediate and corner incisors by threading the wire through a 14-gauge needle placed between the teeth. In young horses it is advisable to engage the central incisor of the opposite hemimandible to prevent inadvertent separation of the symphysis. The wire is twisted in the interdental space until an adequate tension is achieved (Figure 102-10). This results in a satisfactory repair because the wires compress the fractured bone ends, resulting in stability. Additional stability is provided by the contralateral intact hemimandible. Tension-band wiring to the premolars may be performed bilaterally, if indicated. This technique can be applied to the premaxilla, and in this location the other facial bones, the nasal septum, and the conchae add additional stability to the wiring fixation. This techniques also can serve as an adjunct fixation technique to plate fixation (see later).

Bilateral fractures are often comminuted. With comminution of the bone, tension-band wiring can lead to collapse of the fracture fragments and result in imperfect reduction. In these cases, additional support is required and can be provided by the methods described next.

ACRYLIC REINFORCEMENT OF INTRAORAL WIRING

As mentioned earlier, in comminuted fractures, where wiring causes collapse of the mandible or maxilla, some method of buttressing the bone is required to maintain its length. The simplest method uses acrylic reinforcement of the wires.

After the wires are loosely placed, an intraoral splint is prepared by molding dental acrylic around the interdental wires and contouring it to fit the mouth after anatomic reduction of the fracture (Figure 102-11). The acrylic conforms to either the roof or floor of the mouth from the caudal surface of the incisors to 306 or 406. The acrylic serves as a buttress to maintain length and alignment. Additional wires are placed as deemed necessary across the soft acrylic through drill holes in the bone and are tightened when the acrylic has set.

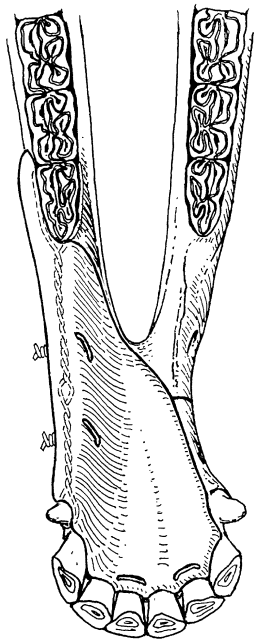


Figure 102-11. Intraoral splinting with dental acrylic (cold-curing type) formed around a tension-band wire between incisors and the first cheek tooth. Several additional wires are used to unite the splint with the mandible.

The surgeon should ensure that the acrylic splint is as form-fitting as possible and uses the least amount of material to serve the desired purpose. Excessive acrylic material in the mouth impedes eating by interfering with the horse's tongue. It is

impotrant to use dental acrylic because it sets with no appreciable exothermic reaction.

A more-precise fit of methyl methacrylate to the oral surface can be obtained by molding methyl methacrylate to the mouth as well as possible and allowing it to harden. The methyl methacrylate is removed after it starts to cure, and the shaping is finished with a rasp or dermal tool to remove excess material and round any sharp edges. After smoothing, holes are drilled in the splint for wiring to the mandible, premolars, and incisors as necessary. Wiring configurations vary with the individual fractures.[9,10]

U-Bar Brace

Bilateral fractures almost always require support in the form of an intraoral brace or external fixator in addition to tension band wiring to maintain axial alignment, limit side-to-side mobility, and prevent collapse (Figure 102-12).[4]

An intraoral splint is made from malleable brass or aluminum rods.[11,12] The pre-bent U-shaped brace is placed on the buccal surface of the incisors and spans the interdental space and the entire length of the dental arcade bilaterally. The brace is adaptable to fixation of fractures of the mandible, incisive bone, and premaxilla. The brace bar is fixed to the incisors and cheek teeth in multiple sites by cerclage wires encircling the teeth and bar. Wiring to the incisors is accomplished using individual cerclage wires, each encircling an incisor and the brace. Wiring to the cheek teeth is accomplished via stab incisions through the cheek and drilling between the teeth at the gum line.

This splint is very stable but is quite time consuming to apply (Figure 102-13). The splint can be aided by application of tension-band wiring as previously described.

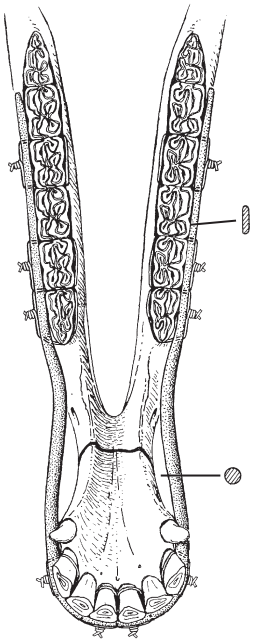


Figure 102-12. Intraoral splint made from an aluminum bar and molded around the contours of the mandible. The bar has a round cross-section in front and a flattened cross-section on each side. The aluminum bar is attached to selected teeth with cerclage wires.

Intramedullary Pins

Intramedullary pins can, in selected cases, be a useful adjunct in the repair of mandibular fractures.[13] Indications for intramedullary pinning are rare, because pin placement options are limited by the tooth roots. Intramedullary pins provide axial alignment, and protruding pin ends can serve as fixation points for wires. When used alone, intramedullary pins are likely to migrate. This type of fixation can be augmented by tension band wiring between the incisor and cheek teeth.

Screws

Interfragmentary screw fixation using lag technique is used to repair selected fractures of the mandible in the region of the diastema and symphysis.[7,14] This type of fixation is useful in this area, especially in adult horses, because the bone is strong enough for purchase of the screw threads, and the screws can be used under biomechanically favorable circumstances (see Figure 102-10). However, symphyseal fractures are rare in adult horses, so the opportunity to use screws is limited.

The fractures are reduced manually and held in reduction by pointed reduction forceps. A stab incision is made through the gingiva, and 3.5-mm, 4.5-mm, or 5.5-mm cortex screws are inserted in lag fashion across the fracture plane using routine technique. At least two screws should be placed to provide rotational stability. Interfragmentary screw fixation using lag technique is appropriate if the fracture configuration allows screw placement without damaging the teeth and there is adequate bone for purchase of the screws. Screws are not removed unless they become infected and are the source of persistent drainage or if they loosen.

A cortex screw may also be placed unicortically on each side of a fracture line so that the screw heads can be used as an anchor for figure-of-eight wiring. This provides a simple, minimally invasive method for tension-band wiring of the body of the mandible. This method also poses little risk to the tooth roots when the screws are placed on the ventral cortex.

A 5- to 7-cm longitudinal incision is made directly over the fracture on the ventral surface of the mandible. The fracture is reduced, and a 4.5- or 5.5-mm cortex screw is placed approximately 2 cm from the fracture line. Figure-of-eight wiring is completed with 1.25-mm stainless steel wire looped under each screw head and twisted until tight. Cortex screws can be inserted on the lateral surface of the mandible if preoperative or intraoperative radiographs are used to plan screw placement.

External Fixators

External fixators are good options for repairing fractures of the ramus or body of the mandible, premaxilla, and maxilla.[15,16] Advantages of external fixators include closed repair, reduced risk of sepsis, easy removal of implants, and good stability. Disadvantages are additional technical expertise and the expense of additional equipment, potential for traumatizing tooth roots, and, frequently, infection of pins associated with loosening. Intraoperative radiographic control is needed.

The indication for using type I external fixators is a unilateral fracture of the horizontal ramus of the mandible. With type I external fixators, the pins engage both cortices of one side of the mandible. A type I fixator may be applied with the horse positioned in either lateral or dorsal recumbency. Ideally, at

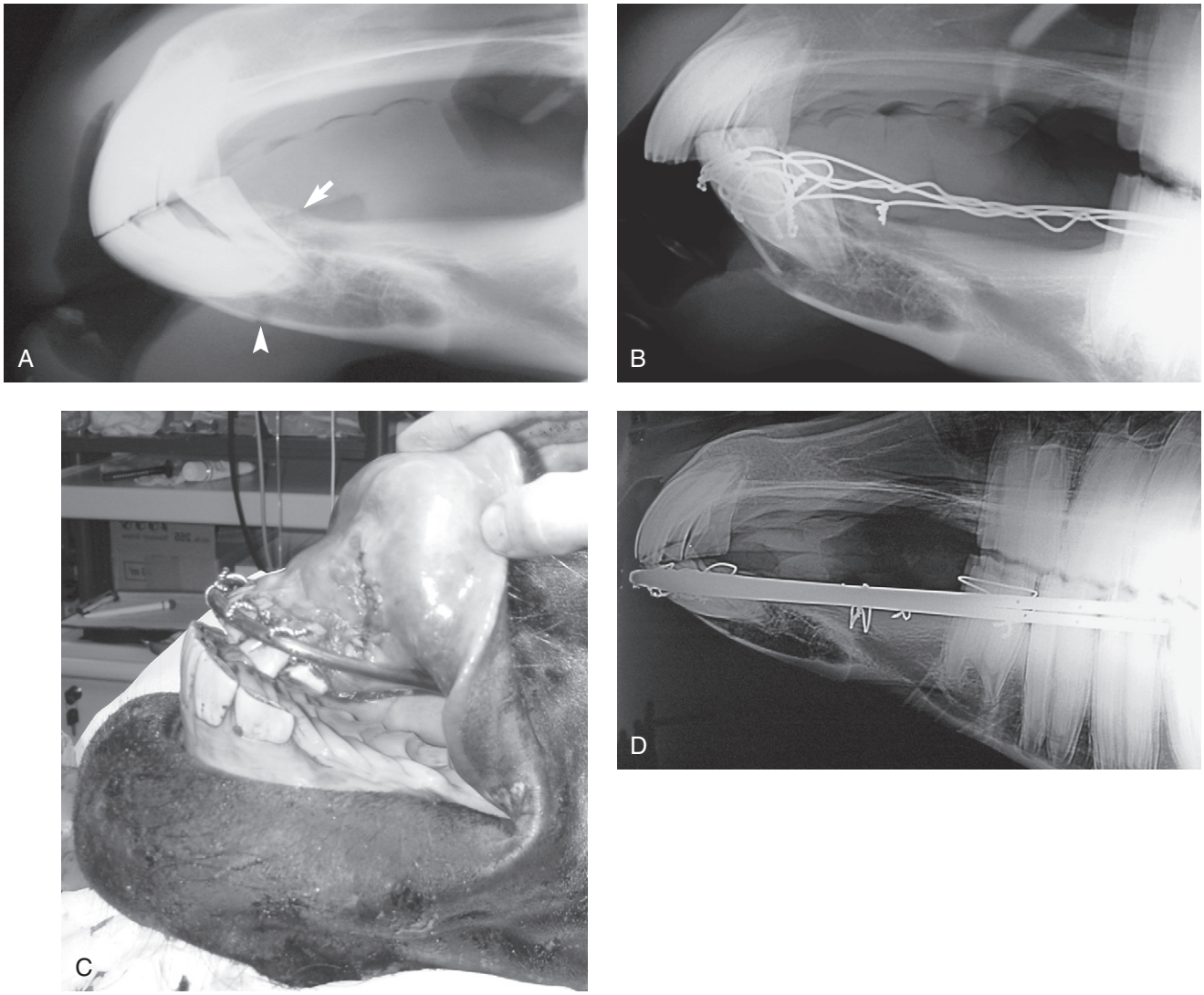


Figure 102-13. A, Preoperative lateromedial radiograph of a mandibular fracture. Good alignment is present and only slight disruptions at the cortex identify the fracture *(arrows).* **B,** Postoperative lateromedial radiograph after intraoral and tension-band wiring. Note the marked caudal displacement and upward rotation of the fragment caused by tightening the wires. **C,** The wires were removed and an intraoral brace was applied to fix the fracture and maintain the length of the mandible. **D,** Lateromedial radiographic view showing the brace in place.

least two pins should be placed rostral and caudal to the fracture.

Indications for a type II external fixator include bilateral fractures of the mandible or premaxilla or a highly unstable unilateral fracture (Figure 102-14). If a type II fixator is selected, Steinmann pins are used to penetrate the medial and lateral cortex of both mandibles, and a connecting bar is placed on each side. Type II fixators are best applied with the horse in dorsal recumbency.

The usual pin size is 4 mm in diameter, and a 6-mm diameter connecting bar is used. In type I fixators, positive profile *end-threaded* pins are preferred to standard smooth pins because of their increased holding power in bone and decreased risk of pin loosening. In type II fixators, positive profile *center-threaded* pins are preferred. The pin holes should be predrilled before inserting the pins. Holes are drilled starting with a 2.5-mm hole and sequentially enlarging it ideally to a size 0.1 mm smaller than the pin size, which provides perfect axial preload to ensure solid fixation within the bone.[17] During drilling, continuous flushing of the drill bit to reduce heat production prevents premature pin loosening caused by thermal necrosis of bone. High-torque, low-speed drills or hand drills are preferred to minimize thermal damage.

Steinmann pins are inserted through both cortices of either one or both mandibles via stab incisions in the skin. Radiographic assistance is used to place the pins either ventral to, or between, tooth roots. Pin placement perpendicular to the sagittal plane prevents the pin from angling in either a rostral or caudal direction and damaging a tooth on the opposite mandible. All pins should be inserted in the same horizontal plane to facilitate placement of the connecting clamps and rod.

An alternative technique that allows more variation in pin placement and is more forgiving of imperfect pin placement is to use an acrylic side bar. This obviates the need for all pins to be in the same plane. An acrylic side bar can be constructed with a piece of flexible rubber hose (25 mm inside diameter). The hose is skewered by the pins. The ends of the hose are occluded, and the lumen is filled with liquid

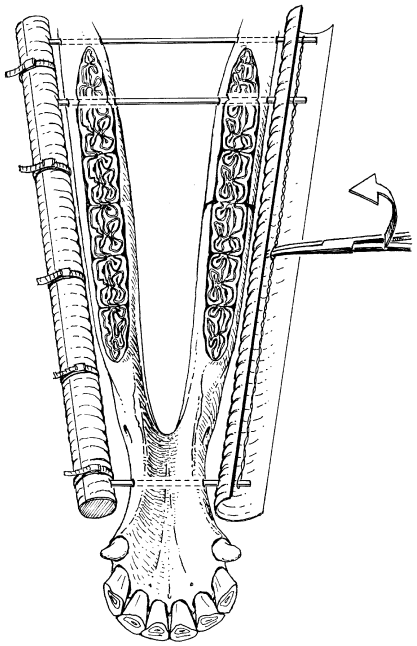


Figure 102-14. Type II external fixator is placed across both rami of the mandible. A tension-band wire is applied around the pins and tightened to achieve interfragmentary compression of the fracture. The open soft tubes are closed with parm bands, and the lumen is filled with epoxy material (e.g., polymethyl methacrylate, hoof acrylic).

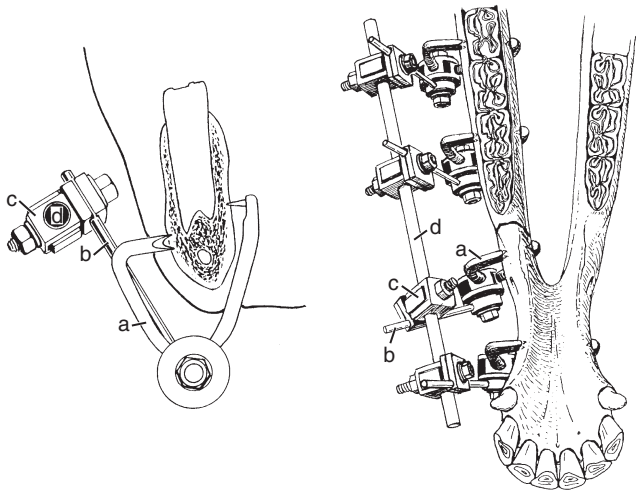


Figure 102-15. Pinless external fixator applied to a mandibular fracture. *a,* Asymmetric clamp attached to the ramus; *b,* small adjustable connecting rod; *c,* single external fixator clamp; d, tubular connecting bar, which fits into the clamp *(c).*

polymethyl methacrylate, which is allowed to harden. An acrylic side bar can be molded to fit closer to the horse's head, which decreases the likelihood of inadvertently catching the fixator on objects in the stall. However, an acrylic side bar cannot be adjusted should the need arise.

A variation of this method involves opening the hose over the whole length (see Figure 102-14). Wire is subsequently looped around the pins and tightened the same way as described for tension wiring of the cheek teeth. This compresses the fracture, resulting in greater stability. The hose is then closed with parm bands, and the hose is filled with acrylic, as described earlier.

PINLESS EXTERNAL FIXATOR

A pinless external fixator attaches to the mandible via clamps instead of conventional transcortical pins (Figure 102-15). Clamps are manufactured in a symmetric configuration with trocar-type points for attaching to the mandibular symphysis and an asymmetric clamp configuration with one trocar point and another bifurcated tip for fastening to the body of the mandible. Stab incisions are made in the soft tissues, and the tips of the bone clamp are embedded in the mandibular cortex by an applicator instrument. Clamp placement avoids soft tissue structures and tooth roots because the clamps attach only to the cortex. Connector rods, clamps, and a rigid side bar complete this type I external fixator configuration.

Use of the pinless external fixator has been described in the horse and in adult cattle.[7,18,19] It may be applied to add stability to a fracture treated with wires and/or a bone plate during the initial postoperative period (Figure 102-16). The benefits of this device include lack of pin tract infections, avoidance of traumatizing tooth roots, flexibility for selecting the application point, and the possibility to adjust the constuct during the healing period should the need arise. If a sequestum develops at the fracture site, it can be removed and the bed can be curetted without disturbing the construct. One disadvantage is the increased cost; however, these clamps can be reused several times, partially offsetting the purchase costs.

With either method of external fixation, daily pin tract care is required to prevent soft tissue infection. The pin–skin interface should be cleaned as necessary and antibiotic ointment and a bandage should be applied.

External fixators can inadvertently catch on objects in the horse's environment. This problem is minimized by fitting the fixator as close to the horse as possible, wrapping the fixator to cover areas prone to snagging, and stabling the horse in an area free of projections.

Plates

Bone plates are used for repair of fractures of the ramus and body of the mandible.[5,7,20] Plating has several disadvantages that limit its usefulness for repair of fractures of the horizontal ramus of the mandible. Plates must be placed on the ventral, ventrolateral, or lateral surface of the mandible. Plate application to the ventral aspect of the horizontal ramus is biomechanically inferior to placing it on the ventrolateral aspect, because the ventral aspect is the compression side of the bone. Exposure of the caudal ventrolateral mandible is complicated by the presence of the parotid salivary duct. Screw placement is difficult because of the tooth roots. There is little soft tissue to cover the plates on the ventrolateral aspect of the mandible. Also, most fractures of the mandible are open, communicate with the oral cavity, and result in contamination of the implants, which necessitates their eventual removal. Plate application to the maxilla also is very difficult except in the region of the interdental space.

Despite these drawbacks, there are indications for bone plates. Fractures of the horizontal ramus of the mandible involving the caudal cheek teeth and body of the mandible are difficult to repair with intraoral wiring procedures because of limited exposure. Severely comminuted fractures might benefit

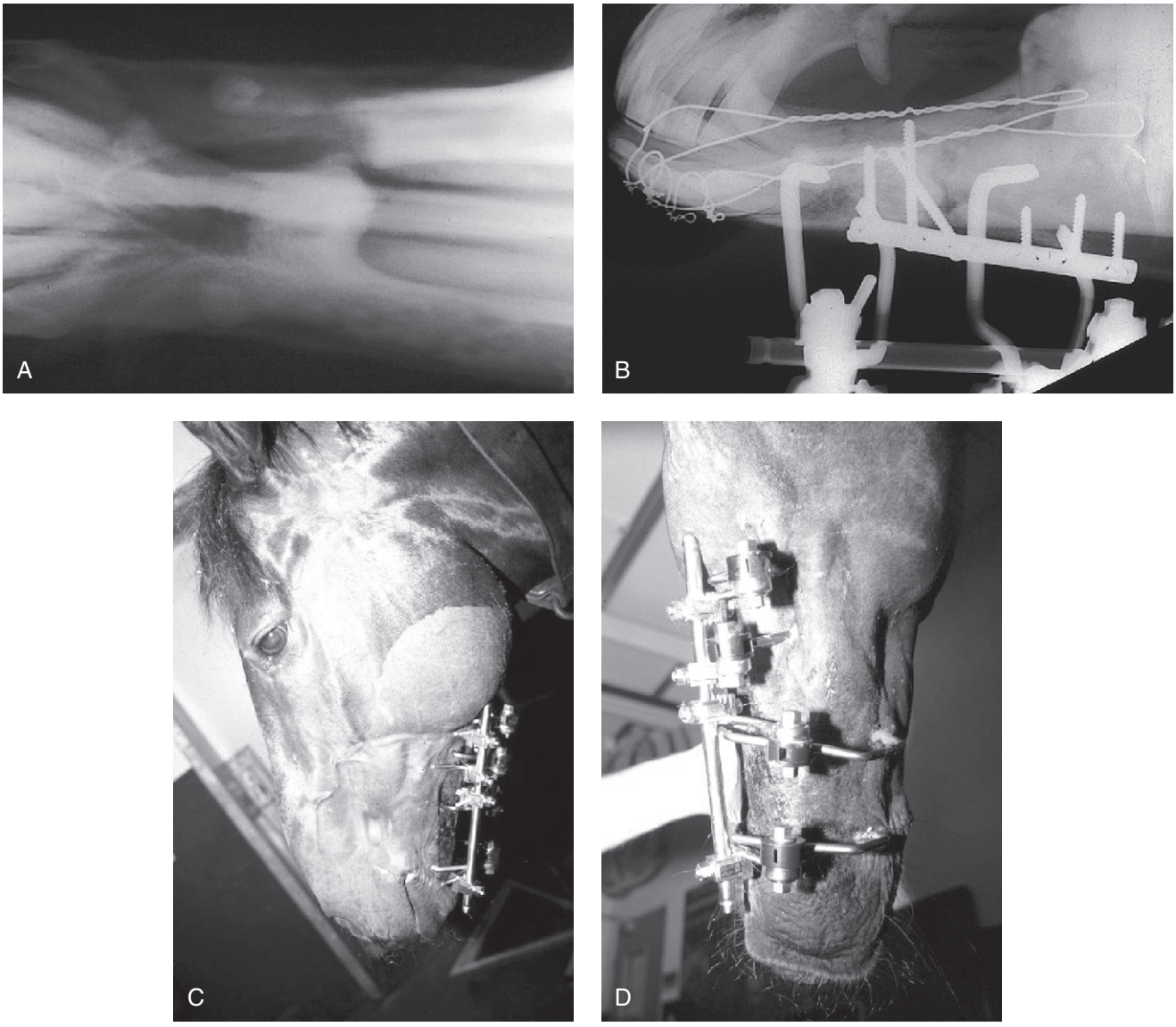


Figure 102-16. A, Ventrodorsal radiograph of a unilateral fracture of the horizontal ramus of the mandible with displacement. **B,** Postoperative oblique radiograph showing the plate implanted across the fracture supported by a tension-band wire. A pinless external fixator was used to provide additional support during the initial postoperative period. Lateral **(C)** and ventrodorsal **(D)** views of the head with the pinless external fixator applied.

from bone plates used in buttress fashion. Broad or narrow 3.5-mm limited-contact dynamic compression plates (LC-DCPs) or reconstruction plates or 4.5-mm LC-DCPs with at least three screws on each side of the fracture have been used. In selected cases, long plates have been used and screws inserted in selected holes along the plate.

As mentioned earlier, compression plating is an option for fractures of the horizontal ramus of the mandible in the interdental space (Figure 102-17). However, the most appropriate indication for the use of bone plates is closed fractures of the body and vertical ramus of the mandible. Horses demonstrate less pain and return to feed quicker when these fractures are stabilized. These fractures are usually closed and have adequate soft tissue covering. The tooth roots do not pose an obstacle to screw placement in this area.

Locking compression plates represent state-of-the-art technology and are currently the preferred choice for repair of craniomaxillofacial fractures.[21] Application technique is as described in Chapter 76. At least two locking-head screws are inserted on either side of the fracture plane to realize the advantage of locking technology. If the plate is applied to the ventral aspect of the mandible, the fixation needs to be augmented with tension-band wires between the incisors and the cheek teeth (Figure 102-18).

Plates are also excellent choices at the caudal aspect of the horizontal ramus. Conversely, the lateral aspect of the vertical ramus is extremely thin and does not accept 4.5-mm screws. Smaller screws may be used, but the implants are not strong enough to withstand the cyclic loading that occurs during chewing. Additionally, the facial nerve and the transverse facial artery and vein are located superficially on the lateral aspect of the ramus.

To place a plate on the caudal aspect of the horizontal ramus, a skin incision is made along the caudal edge of the mandible

that parallels its caudal border. In that location, the masseter muscle can be avoided and there is abundant bone to allow solid insertion of the screws. A large periosteal elevator is used to expose the caudal rim of the vertical ramus.[22] A plate spanning the fracture and providing three to five screw holes on either side is selected (Figure 102-19). Selecting a longer plate allows the surgeon to choose which plate holes to use. In selected cases, wire sutures may be used to appose fracture lines on the lateral surface of the ramus through small incisions (see Figure 102-19). The plate is contoured to the surface of the bone and applied using routine technique. The subcutaneous tissues and skin are reapposed over the plate, with sutures in two separate layers.

A study evaluating multiple fixation techniques of an osteotomy in the diastema of the mandible compared dynamic compression plate (DCP) fixation with an external fixator and

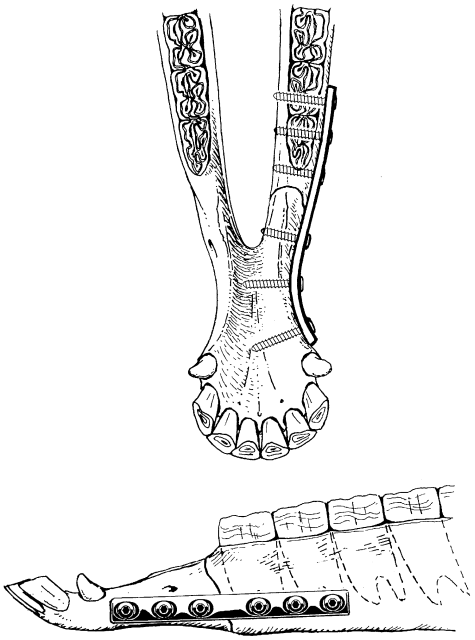


Figure 102-17. A six-hole 4.5-mm narrow dynamic compression plate is applied to the horizontal ramus of the mandible. The screws are inserted between the tooth roots.

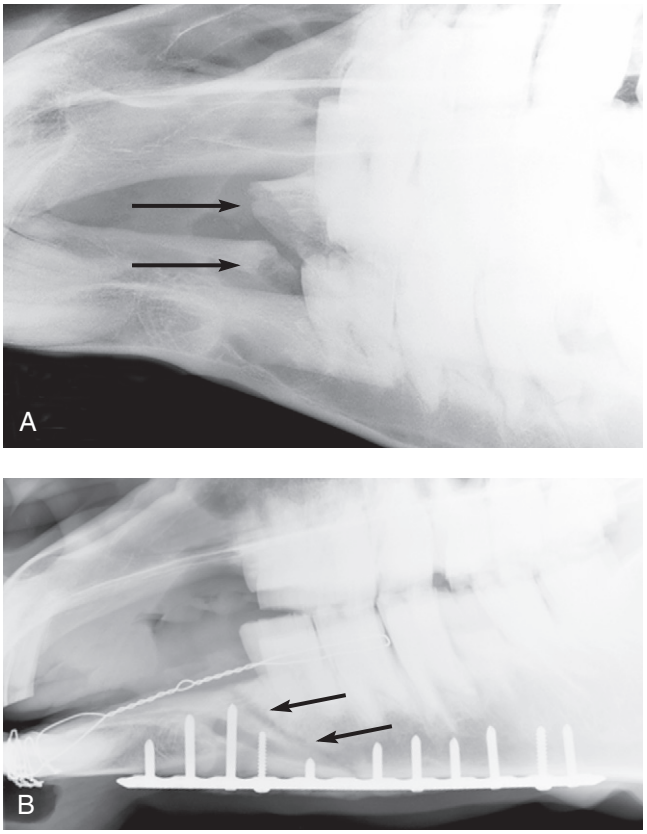


Figure 102-18. A, Oblique radiographic view of unilateral mandibular fracture *(arrows)* in the interdental space of an adult horse. **B,** Postoperative lateromedial radiographic view of the same fracture treated with a 12-hole narrow LCP and an interdental tension wire between the incisor- and cheek teeth. Good fracture reduction was achieved *(arrows)*.

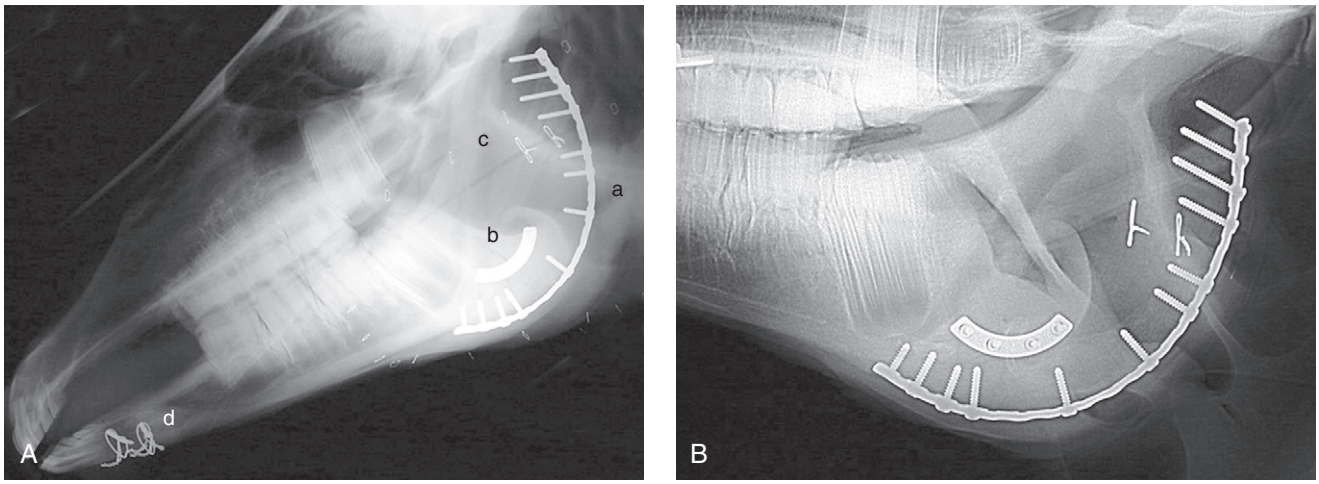


Figure 102-19. A, Immediate postoperative lateromedial radiographic view of the fractured mandible of the same foal as in Figure 102-2. The fractures were repaired with mulitple implants. *a,* A narrow 3.5-mm reconstruction plate was applied to the caudal edge of the ramus with 12 3.5-mm cortex screws placed in selected holes. Four screws were placed at each end; *b,* to provide additional stability to the triangular fragment, a four-hole small-animal 3.5-mm acetabular plate was implanted; *c,* the horizontal fracture was supported with two wire sutures; *d,* the fracture of the symphysis was repaired with two hemicerclage constructs. **B,** Four-month follow-up radiographs before implant removal. All fractures have healed.

interdental wiring and an intraoral splint and interdental wiring. This study revealed that DCP fixation had the greatest stiffness under monotonic bending to failure; however, the relatively low yield value might predispose it to earlier failure in fatigue testing without supplemental fixation.[23] The techniques using tension-band wiring in conjunction with an external fixator or an intraoral splint were similar to DCP constructs in yield, failure, and osteotomy displacement, whereas the external fixator constructs alone were biomechanically inferior to all others.

Therefore, plate fixation is probably the most stable form of fixation for comminuted fractures of interdental spaces.[21,23] However, for simple interdental space fractures, intraoral splinting together with interdental wire fixation should provide adequate stability with minimal invasiveness and decreased expense. Tension-band wiring significantly enhances the strength of type II external skeletal fixators and should be used to augment mandibular fracture repairs.[23]

Chronic mandibular fractures that become non-unions require special management. They usually present with a draining fistula that communicates with the fracture. If surgery is elected, the fracture site should not be disturbed. The skin at the lateral side, proximal to the draining fistula, is opened and the tissues are dissected until the fibrous capsule surrounding the fracture site is identified. The bone on either side of this capsule is exposed and a plate is selected that allows at least three screws to be inserted on either side of the fracture. The plate is contoured to fit over the capsule of the infected fracture. The screws are inserted using routine technique to apply compression across the fracture without inserting screws near the fracture itself (Figure 102-20). Locking compression plates are ideal for this purpose. The goal of this technique is to provide solid bridging fixation of the fracture without spreading the infection to the implants, which could lead to premature loosening of the screws and delayed fracture healing.

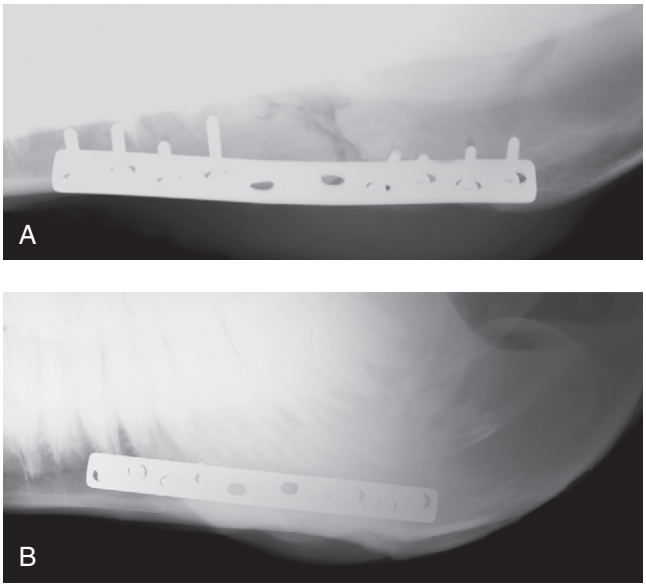


Figure 102-20. A, Immediate postoperative lateromedial radiographic view of a chronically infected mandible fracture in an adult horse repaired with a 10-hole DCP applied to the bone without opening the fracture site. **B,** Three-month postoperative radiographic view of the healed fracture immediately prior to plate removal.

Aftercare

Horses rarely demonstrate a reluctance to eat after mandibular fractures are stabilized, and they quickly return to routine feeding.[4] No attempt to modify the diet is made following repair of incisor fractures. Fractures involving the cheek teeth could give the horse more difficulty during mastication, and some dietary adjustment may be needed. Horses should be provided a palatable diet that can be easily chewed. They usually prefer customary foodstuffs to the gruels and mashes sometimes recommended during convalescence. Therefore, there is little need to modify the diet unless there is a clear indication that the horse is having difficulty in masticating.

All foodstuffs tend to accumulate around the intraoral wires, rods, and acrylic. Frequent garden hose lavage of the mouth to remove accumulated debris is beneficial if the horse will tolerate it.

Phenylbutazone is routinely administered at 4.4 mg/kg *per os* for 7 days. The decision to administer antibiotics is made on an individual basis. Most fractures are open and contaminated and benefit from administration of broad-spectrum antimicrobial agents.

Removal of the implant is usually carried out on the standing patient after healing has occurred, usually in 6 to 8 weeks. Bone plates and buried cerclage wires are not customarily removed unless the implants become infected.

Complications

Implants should be inspected regularly because wires tend to loosen and break (Figure 102-21). Loose wires may be tightened by additional twisting. Undetected loosening of implants can result in malocclusion and delayed healing because of instability. The decision to replace broken implants should be based on the timing of implant failure. The wires can be removed if the fracture is stable at the time of breakage. Failures that occur earlier in the postoperative period require replacement to maintain alignment and stability. Cable ties mentioned earlier serve as alternatives. Even 1 mm cables resist breakage much better than wires, and they should be considered if wires need to be replaced.

Plate breakage is a rare complication but can occur (Figure 102-22). If a plate breaks, it is an indication that there is a considerable amount of instability. The plate should be replaced and additional fixation should be added to the constuct to reduce the cyclic loading of the implants.

Drainage from the fracture is common following repair of open fractures, because infection always develops after exposure to the oral cavity. In most cases, the infection resolves with systemic antimicrobial therapy and effective drainage. Drainage that persists for more than 4 weeks should be investigated.

With closed repair of comminuted fractures, small fragments of avascular bone commonly become sequestra. Radiographs should be taken to ascertain whether there are sequestra if drainage persists. In many cases, the sequestra can be removed by curettage in the standing patient if the fragments are superficial.

Callus formation is to be expected with secondary bone healing of infected fractures and is more prominent with fractures that occur in the horizontal ramus of the mandible, because there is less soft tissue coverage. Factors that increase

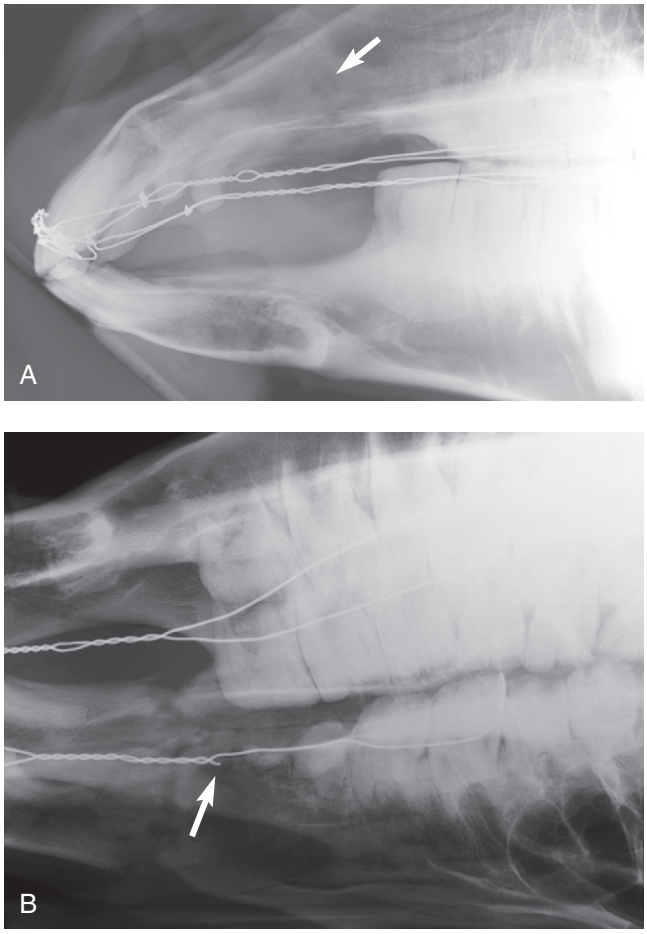


Figure 102-21. A, Lateromedial radiographic view of a unilateral maxilla facture *(arrow)* treated with bilateral tension band wires between the incisor and cheek teeth. **B,** One-month postoperative oblique radiographic view of the treated fracture. One wire is broken *(arrow)* and part of the loop around the cheek teeth is missing. The wire was replaced.

callus formation are comminution, infection, and instability in the repair. Efforts to attenuate these factors are rewarded with a more cosmetic outcome.

Prognosis

The prognosis for a serviceable outcome is favorable in most cases. Imperfect reduction of fractures leads to malocclusion. Fractures of the teeth themselves and fractures involving the alveolus sometimes result in tooth loss. Tooth loss (when it occurs) and irregular tooth wear because of malocclusion are managed effectively by regular dental care.

FRACTURES OF THE SKULL AND SINUSES

Fractures of the skull and sinuses are the result of blunt trauma from being kicked or from collisions. The most commonly injured bones include the nasal, frontal, and maxillary bones and the zygomatic process of the frontal bone. The trauma that produces fractures to these bones can cause damage to the brain, eye, and nasal passages, resulting in severe hemorrhage.[4] Depression fractures are often found.

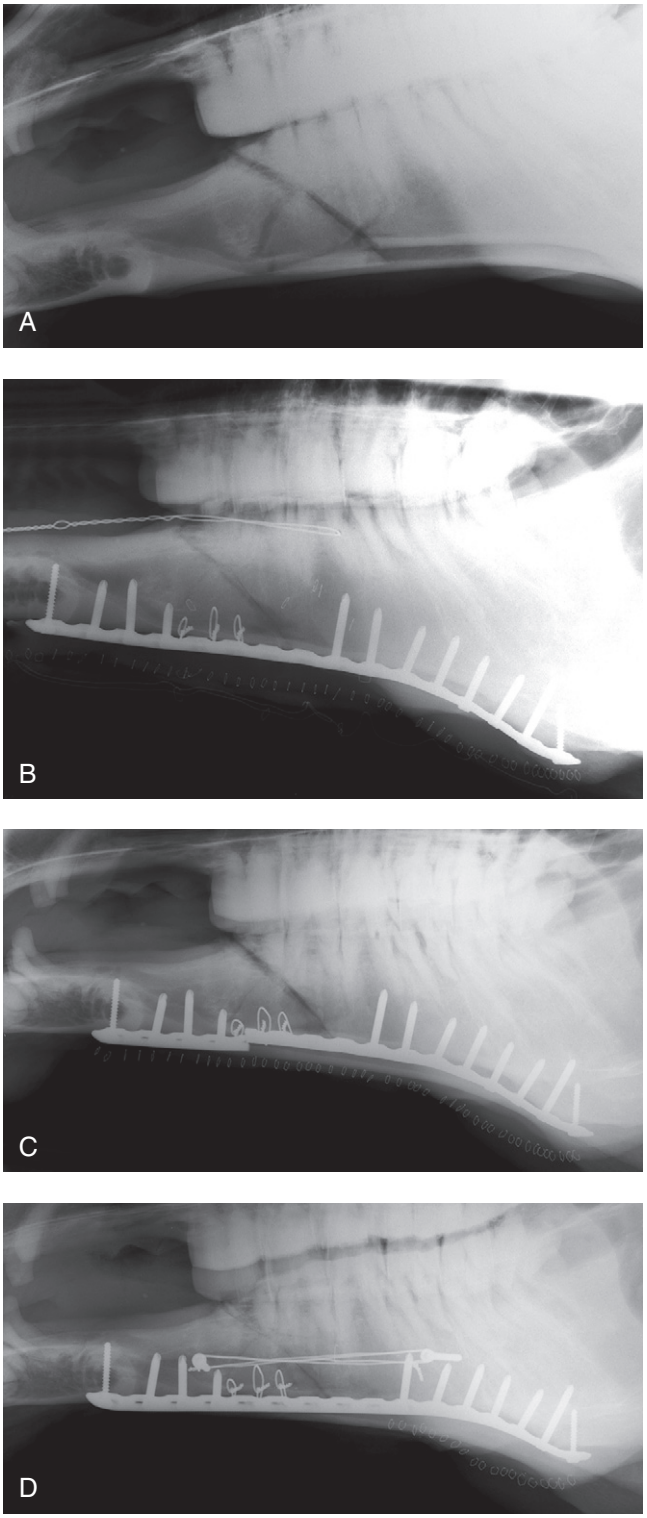


Figure 102-22. A, Lateromedial radiographic view of a multifragment (comminuted) mandibular fracture entering the interdental space. **B,** Postoperative lateromedial radiographic view of the fracture treated with a 16-hole narrow LCP augmented with a tension wire beween the incisor and cheek teeth. Wire sutures were used to unite some fragments. **C,** One-week postoperative lateromedial radiographic view of the fracture showing the broken plate. **D,** Lateromedial radiographic view of the fracture following revision of the repair. The plate was exchanged and the screws were inserted into the same holes. Lateral tension band wires were added to reduce interfragmentary instability.

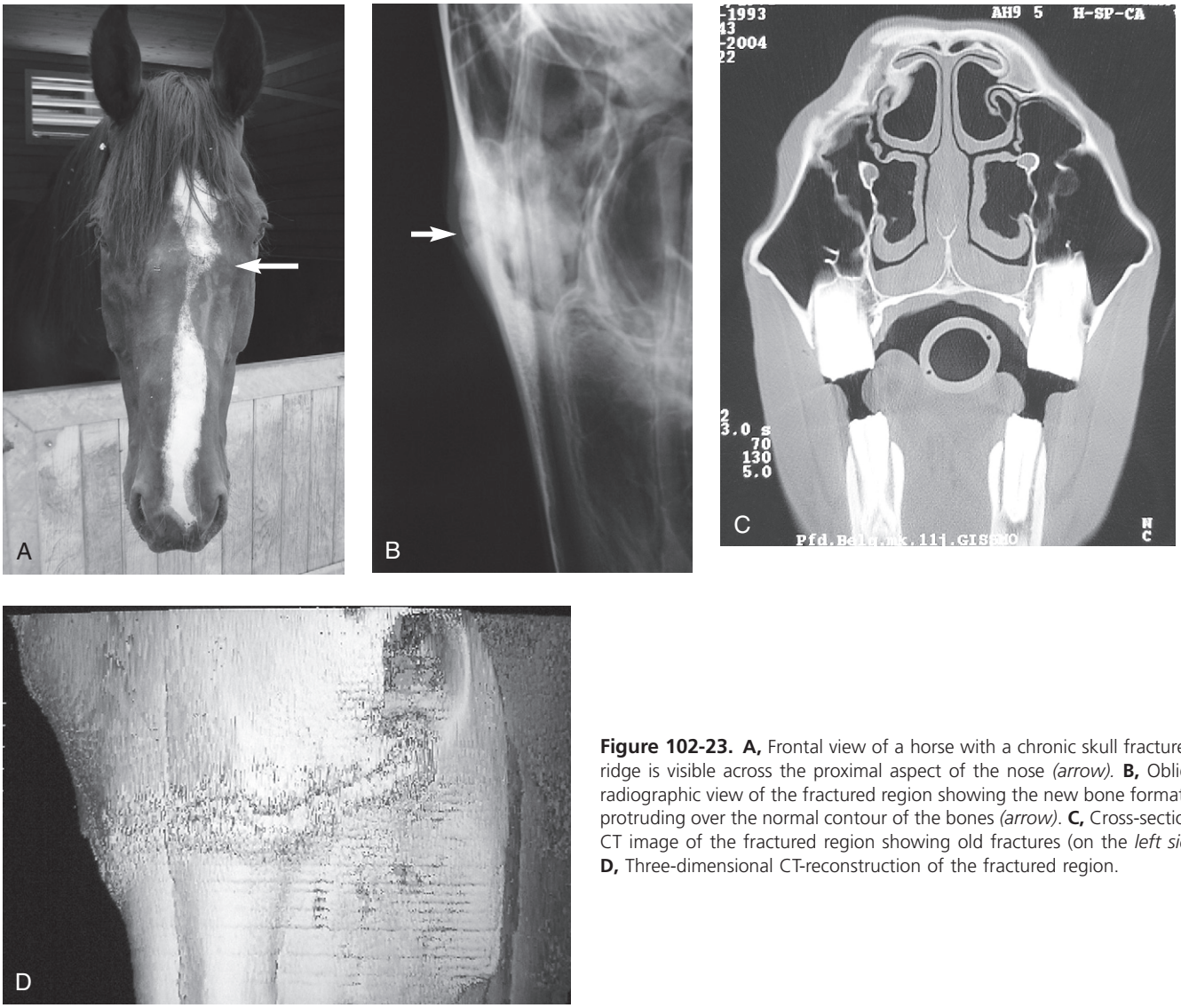


Figure 102-23. A, Frontal view of a horse with a chronic skull fracture. A ridge is visible across the proximal aspect of the nose *(arrow)*. **B,** Oblique radiographic view of the fractured region showing the new bone formation protruding over the normal contour of the bones *(arrow)*. **C,** Cross-sectional CT image of the fractured region showing old fractures (on the *left side*). **D,** Three-dimensional CT-reconstruction of the fractured region.

Diagnosis

Initially, a minimally displaced depression fracture can be difficult to detect on physical examination because of the soft tissue swelling that obscures the depression. Other signs include obvious bone displacement, epistaxis, and skin lacerations. Emphysema can be palpated in the soft tissues as crepitus when fractures communicate with the nasal passages or sinuses. Radiographs with multiple oblique views are necessary to demonstrate the fracture. Computed tomography greatly aids with the final diagnosis and preoperative planning of the repair (Figure 102-23). Physical examination should include endoscopic examination of the upper airways as well as neurologic and ocular examinations to assess for additional undetected trauma.

Treatment

Initial therapy should be directed toward stabilizing the patient, because in most cases there is no urgency in the repair.[4] In fact, fracture repair is often difficult if significant soft tissue swelling has developed, and therefore repair is best delayed until swelling resolves. Skin lacerations should be repaired immediately to prevent infection of the soft tissues, even if fracture repair is delayed. Hyperosmotic wound dressings applied under pressure wraps will dissipate the edema.[4] Phenylbutazone is administered to decrease inflammation, and antimicrobial therapy is indicated for any open fractures or fractures that communicate with the sinuses. Tetanus prophylaxis is indicated in all cases.

Trauma to the facial bones often results in hemorrhage into the sinuses, which shows up on a lateral radiograph as a fluid line within the sinus. Consideration should be given to standing sinus lavage for removal of accumulated blood when present, to prevent the development of empyema within the sinus. Lavage is performed via an ingress portal made with a 6-mm Steinmann pin inserted into the frontal sinus and polyionic fluid lavage with a pressurized fluid delivery system.

Repair should be performed as soon as the soft tissue swelling is resolved. Delay in repair beyond a few days can result in difficulty in achieving reduction. The goals of repair are to maintain a patent airway and nasolacrimal duct, prevent ocular damage, and obtain a cosmetic appearance. Cosmetic

considerations are the main purpose of repairing fractures that do not impact other body systems.

The typical configuration is a segmental depression fracture. Often, the skin and periosteal attachments are intact. Even so, the fractures usually communicate with the nasal passages or sinuses and will be contaminated, so that infection of soft tissues and of implants could develop.[24] Therefore, the surgical approaches should be as minimally invasive as possible. Many fractures can be reduced through stab incisions with no stabilization beyond that provided by the soft tissue attachments.[4]

Adequate reduction can often be achieved by elevating the fragments and wedging them back into place.[25] Whenever possible, closed reduction should be performed. The soft tissue attachments provide vascularity and the means for stabilizing the fractures after they are reduced. Adequacy of fracture reduction can be ascertained by direct inspection or evaluation of the facial contour; this is the reason for allowing the soft tissue swelling to resolve before attempting any repair.[4] Ultrasonography may be used to evaluate anatomic reduction of depression fractures. The depressed fragments can be elevated by drilling holes in the bone and inserting an elevator to pry the fragment back into place.[26] A disadvantage of this approach is that commercially made elevators typically have a broad, flattened shaft that requires large drill holes to insert into the bone.

A better way to elevate the fracture fragment involves drilling a hole into the depressed segment with either a 3.5-mm drill bit or a Steinmann pin on a Jacob chuck. A 3-mm diameter Steinmann pin can be bent and inserted into the hole to elevate and wedge the fragment back into place. The end of the pin is bent at a right angle so that when the pin is inserted into the hole drilled in the fracture segment it has a broad area of contact and added leverage for elevating the fragment (Figure 102-24). The length of the bent end should approximately match the width of the depressed bone segment. Another right angle bend is made in the opposite end of the pin to make a handle.[4] Axial traction is applied to pull the fragment back into place.

The skin over the drill holes is closed with a single suture. If necessary, multiple drill holes can be made with little or no disruption of the soft tissue attachments. In most cases the fragments can be wedged into place with no additional stabilization required because the fractures are not load bearing. If the fractures do require stabilization to maintain reduction, wire sutures can be applied in selected locations around the fragment. A small skin incision is made to expose the fracture line. A Steinmann pin (1.6 mm) and hand chuck or 2-mm drill bit is used to pre-drill holes for 0.8- to 1-mm cerclage wire sutures (see Figure 102-24). Skin closure is routine.

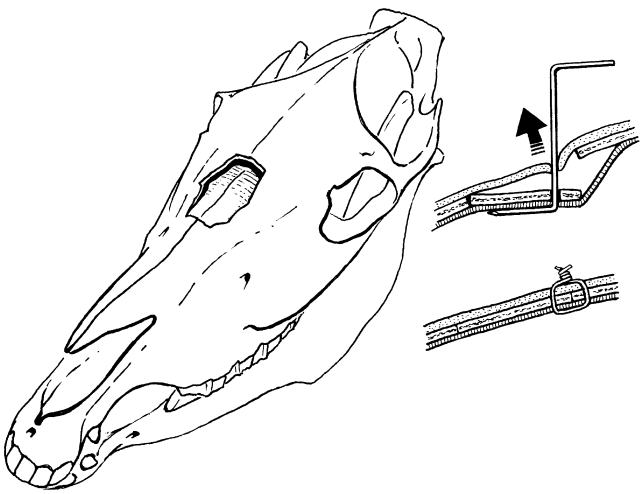


Figure 102-24. Insertion of a Steinmann pin through a small drill hole for elevation of a depressed bone fragment. If necessary, the fragment can be fixed with cerclage wire.

The FlapFix system has been developed for fragment fixation of flat bones. The system is designed for fast, easy, and stable fixation of bone flaps following a craniotomy or the treatment of craniomaxillofacial fractures in humans. The implant consists of a round, smooth, or toothed titanium plate that is centrally connected to a thin orthogonally oriented titanium tube (Figure 102-25, *A*). The plate is slightly cupped to ensure that only the outer rim makes initial contact with the underside of the bone. A smooth, also slightly cupped, cloverleaf-shaped plate is fed over the titanium tube (see Figure 102-25, *A*). The implants are available in three different sizes depending upon the shape and type of fracture or bone flap that needs to be repaired or fixed.

In a clinical case, the round plate with the perpendicular tube is placed underneath the fractured bone and the tube exits through the fracture line or the osteotomy cut (Figure 102-25, *B*). The cloverleaf plate is then fed over the vertical tube and slid down onto the bone. An alternative is to slide the cloverleaf plate over the tube, grab it with the Flap-Fix clamp, and maneuver the lower plate through the osteotomy cut/ fracture line underneath the bones to be united (see Figure 102-25, *B*) The Flap-Fix clamp is then moved down over the tube and pushed up against the second plate. By squeezing the main handles together, the proximal plate is pressed firmly onto the bone surface (Figure 102-25, *C*). When the desired clamping force is reached a second handle is squeezed to cut the tube flush with the upper plate (Figure 102-25, *D* through *F*).

These implants are manufactured from titanium, which renders them tissue-friendly, and no adverse reactions are usually encountered. The limited applications of this implant in horses have all been successful and none of the implants have had to be removed.[27]

The Flap-Fix represents an alternative to wire loops. Its advantages include the rapid speed of application, the increased stability of the fixation, the increased contact area of the implant with the bone, the inertness of the implant material, and the greater resistance to failure.[27] The major disadvantage is the high cost of the implant.

Occasional indications for the use of reconstruction plates arise with severely comminuted or unstable fractures. These plates are made in a variety of shapes and sizes and are easily contoured to fit specific fracture configurations.

Aftercare

Postoperatively, a pressure bandage is applied, and phenylbutazone is administered to decrease postoperative swelling and discomfort. Broad-spectrum antimicrobial agents are administered as necessary to treat soft tissue and sinus infections and prevent implant infections.

Complications

Inadequate reduction results in an inferior cosmetic outcome. Secondary bone healing results in callus formation, which causes an obvious blemish when it occurs over areas with no muscle. This is, however, only of temporary duration, because

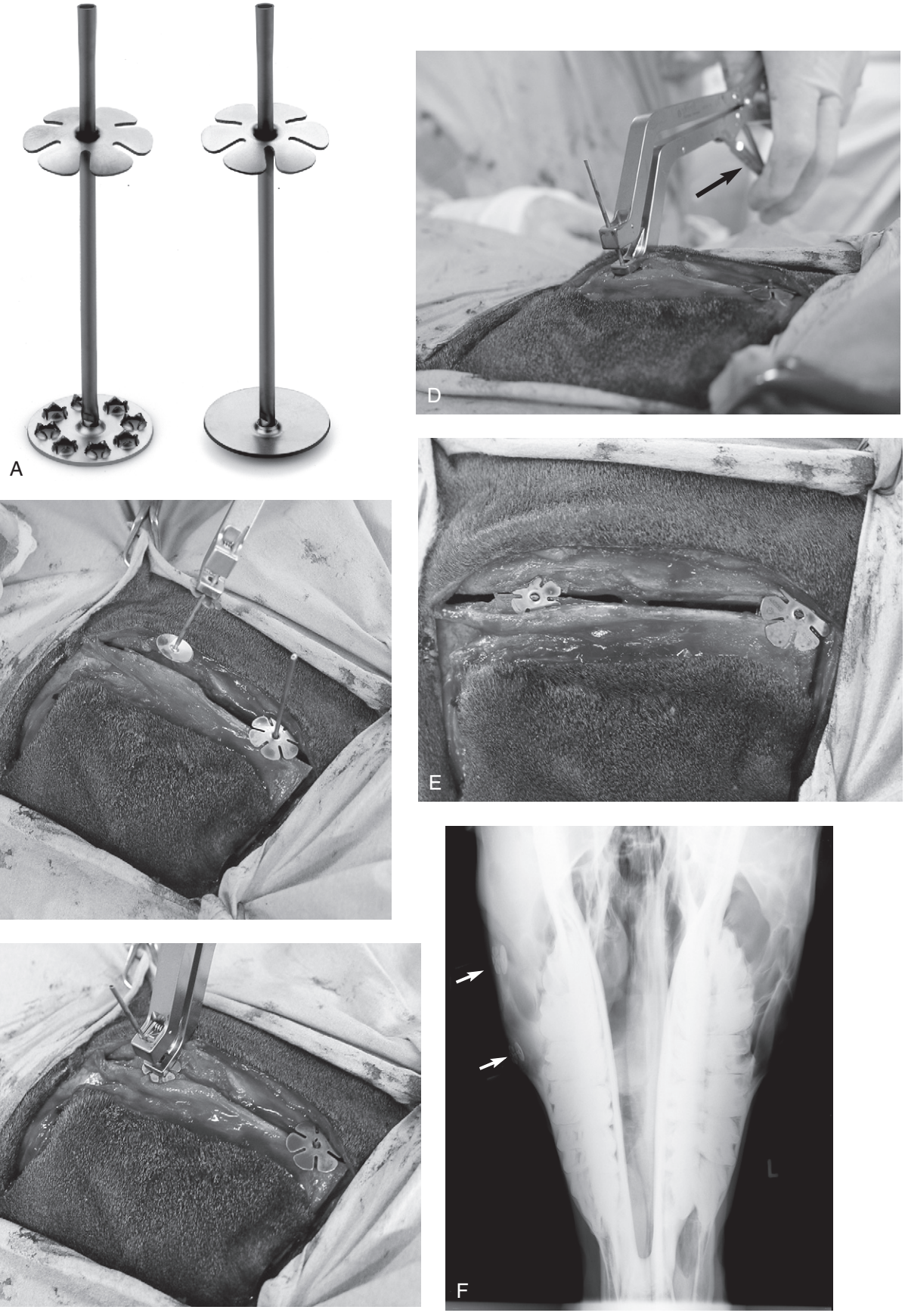


Figure 102-25. A, The two versions of the Flap-Fix assembled but not tightened (toothed type on the *left* and the smooth plate on the *right*). **B,** A large Flap-Fix is in place at one corner of a sinus bone flap, while a second, slightly smaller assembled Flap-Fix is manipulated under the other corner with the help of the Flap-Fix clamp. **C,** The first Flap-Fix has been tightened, while the smaller one is being tightened with the Flap-Fix clamp by squeezing the main handles. **D,** When appropriately tightened, the protruding vertical titanium tube is cut flush with cloverleaf plate by squeezing the secondary handle *(arrow).* **E,** Both Flap-Fix implants are in place and tightened. The vertical tube was cut flush on top of the cloverleaf plate. **F,** Ventrodorsal postoperative radiographic view of the skull of the horse shown in the previous figures. The Flap-Fix implants are easily recognized on the left side of the head *(arrows).*

after the fracture is healed remodeling of the callus occurs, normalizing the contours of the skull. Hemorrhage into the paranasal sinuses can develop into a sinus empyema if untreated. Stenosis of the nasal passages can occur with severe injuries; however, this is rare. If the fragment is denuded of its blood supply through a loss of the periosteum, sequestration can occur, but it is less frequent than with mandibular fractures.

When a sunken appearance is evident after healing because of inadequate reduction of depressed fragments or in cases that were left untreated (Figure 102-26), refracture and proper alignment is not easily accomplished. The facial contour can be restored by implantation of prostheses of either a silicone or a fluorocarbon polymer and carbon fiber combination.[28,29] General anesthesia and strict asepsis are required.

With either technique, a slightly curved incision is made over the defect to expose the sunken bone. The implant material is sculpted to fill the defect and restore the normal facial contour. The silicone prosthetic material is sutured in place between the periosteum and the skin. A subperiosteal dissection is performed for insertion of the fluorocarbon polymer–carbon fiber prosthesis between the bone and periosteum. Skin closure is routine.

Prognosis

The prognosis for return to previous level of performance is usually good. The cosmetic outcome depends on the reduction achieved at surgery and is usually acceptable and often excellent depending on the severity of the injury.

PERIORBITAL FRACTURES

Fractures of the orbit are common in horses. The orbit serves as a protective housing for the eye and associated adnexa.[4] The orbit consists of the lacrimal, frontal, and zygomatic processes of the frontal and temporal bones. Fractures are the result of direct trauma and are usually depression fractures. Concurrent injury can be sustained to the central nervous system, eye, lacrimal duct, other skull bones, and the paranasal sinuses. These structures warrant examination as part of the physical examination prior to repair.

Figure 102-26. An Arabian horse with a depression fracture *(black arrow).* There is some blood at the left nostril *(white arrow).*

Diagnosis

Diagnosis is made by physical examination. Abnormal facial contour, soft tissue swelling, crepitus, and hemorrhage are usually present.[4] Acute soft tissue swelling can obscure the fracture and make detection difficult by physical examination alone. Pain can preclude a thorough physical examination in the unsedated patient.

Radiographs are indicated to identify trauma to the orbit but are also useful to identify concurrent injury and problems such as fluid in the maxillary sinuses. Fractures of the zygomatic arch are visible on the appropriate views; however, trauma to other parts of the orbit are difficult to recognize radiographically.[4] Oblique views at different, sometimes acute, angles are often necessary to highlight the fractures. Ultrasonography might allow diagnosis of fractures and of tissue injuries lying deeper in the orbit. Computed tomography is the best imaging techique to achieve an accurate diagnosis.

The extent of ocular trauma is variable and ranges from chemosis to rupture of the eye. A thorough ocular examination should be performed as soon as possible following the injury, including an assessment of the patency of the lacrimal duct when fractures of the lacrimal bone are present.

Treatment

Fractures will heal without surgery, but in most cases they are associated with a facial deformity. Surgical repair is indicated to relieve impingement on and pressure to the eye. Injury to the eye is treated as circumstances warrant (see Chapter 55). Repair is best attempted as soon as the patient is stabilized. The goal of reconstruction is to relieve impingement on the globe and adnexa and restore the normal anatomic contours to achieve a cosmetic outcome.[4] There are no muscle attachments to the orbit, except the small palpebral muscles that are inconsequential to any repair undertaken. Fractures often need only to be realigned to heal properly.[24] It is important to palpate the inner surface of the orbit to identify and remove any bone fragments that can damage the eye. These fragments are very difficult to identify radiographically.

Fractures of the orbit that involve the zygomatic, lacrimal, or frontal bones are usually depression fractures and may be treated by elevation of the fragments via drill holes in the depressed segment as described in the previous section.

Zygomatic process fractures may be reduced noninvasively by use of a bone hook.[25] The horse is prepared for surgery with the affected side up. A finger is inserted into the orbit in the conjunctival space to palpate the fracture and guide the placement of a bone hook onto the ventral surface of the displaced fragment. Dorsal traction is used to elevate and wedge the fragment back into anatomic alignment (Figure 102-27).[21] Fractures of more than 48 hours in duration are more difficult to reduce.

Most fractures are sufficiently stable so that no other form of fixation is required. Fractures that do not maintain alignment

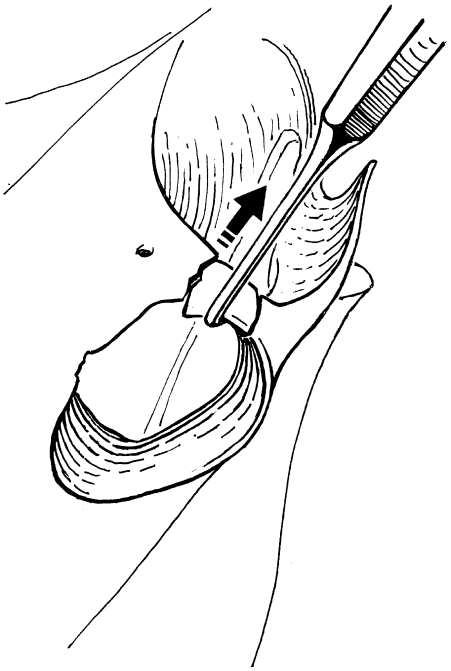


Figure 102-27. Use of a bone hook to reduce a depressed zygomatic arch fracture. The *arrow* shows the direction of the pulling force.

after reduction require some form of stabilization in addition to that provided by the soft tissue attachments. These fractures are usually comminuted or involve portions of the orbital rim other than the zygomatic arch. Open reduction and interfragmentary wiring is the easiest but not the sturdiest method to provide additional stability when needed.

An incision is made directly over the fragments to be wired. Holes are drilled into the bone with a 2-mm drill bit. Cerclage wire of 0.8 mm to 1 mm diameter is pre-placed and twisted to tighten.[25]

Reconstruction plates can be used to repair severely comminuted fractures of the zygomatic arch (Figure 102-28). These plates have the advantage of being easily contoured. Bone plates should be reserved for cases in which easier methods will not achieve a satisfactory result.

Aftercare

Phenylbutazone is administered postoperatively to decrease discomfort and soft tissue swelling. Antimicrobial agents are not required if the fracture is reduced noninvasively. Systemic antibiotic agents should be administered if the fracture is open or communicates with the maxillary sinus, or if implants are used. Pressure bandages are not used because they may cause problems with the eye. Selection of any topical medication, if needed, should be based on safety of the substance for use in the eye.

Complications

Periorbital fractures are often associated with trauma to the eye. Rupture of the eye, proptosis, corneal ulceration and laceration, chemosis, and damage to the nasolacrimal duct are reported complications.[24,29] Empyema of the maxillary sinus can occur secondary to hemorrhage into the sinus.

Prognosis

Healing of the fractures usually proceeds uneventfully. The cosmetic appearance depends on the accuracy of surgical reduction and the amount of callus but is usually favorable. A prognosis for associated ocular trauma depends on the nature and severity of the injury.[4]

CRANIOFACIAL DEFECTS (SINUS FISTULA)

Craniofacial defects can develop as a result of external trauma to the facial bones over the sinuses. If the fragments over a sinus are denuded of their blood supply, they can undergo necrosis, leading to the formation of sinus fistulas of various sizes (Figure 102-29, *A* and *B*).[30] Rarely, congenital anomalies are also associated with bone defects in the skull.

Suturing skin over a bone defect in the skull usually results in failure of the repair. Underneath the skin flap is an air-filled cavity, which dehydrates the skin and results in necrosis. Periosteal and occasionally muscle flaps have been used to correct the problem.[30,31] Periosteal flaps are dissected free from the area adjacent to the bone defect with a base at the edge of the bone defect. The vascularized periosteal flap is flipped over the bone defect and sutured to a similar flap taken from the opposite side (see Figure 102-29, *B*). A cancellous bone graft or biodegradable bone cement is applied over the periosteum. To facilitate closure of the skin over the repaired defect, relief incisions or transportation flaps, or both, might have to be performed.

The surgery site should be protected from trauma during the recovery phase and the immediate postoperative period. Also, the animal should be kept from rubbing the area to prevent destroying the surgical repair. Drainage of the flaps should be aided through insertion of drains, with early removal after drainage has ceased. Anti-inflammatory drugs and antibiotics are indicated to control inflammation and prevent postoperative infection. Sutures should be removed 2 to 3 weeks postoperatively.

Complications include complete or partial breakdown of the repair, usually because of trauma, which can lead to disfigurement. Postoperative infection is always a risk because of the contaminated nature of the surgical site. Complications can require another surgical revision.

RESECTION OF THE ROSTRAL MANDIBLE, PREMAXILLA, AND MAXILLA

Ossifying fibromas of the rostral mandible are the most common indications for a rostral mandibulectomy (Figure 102-30, *A* and *B*).[32] Other neoplasms, such as adamantinomas, and fungal and parasitic diseases can also be indications for the procedure. Inadequate local excision of these lesions results in a recurrence; therefore, block excision with wide nonaffected margins is required. This procedure was developed in an attempt to completely excise the lesions in one surgical intervention.

Surgical Procedure

The animal is anesthetized and placed in dorsal recumbency on the surgery table. The endotracheal tube is placed either through the nasal passages or via a temporary tracheotomy, allowing unimpeded access to the mouth. The oral cavity is washed, making sure that the cuff of the endotracheal tube is tight and

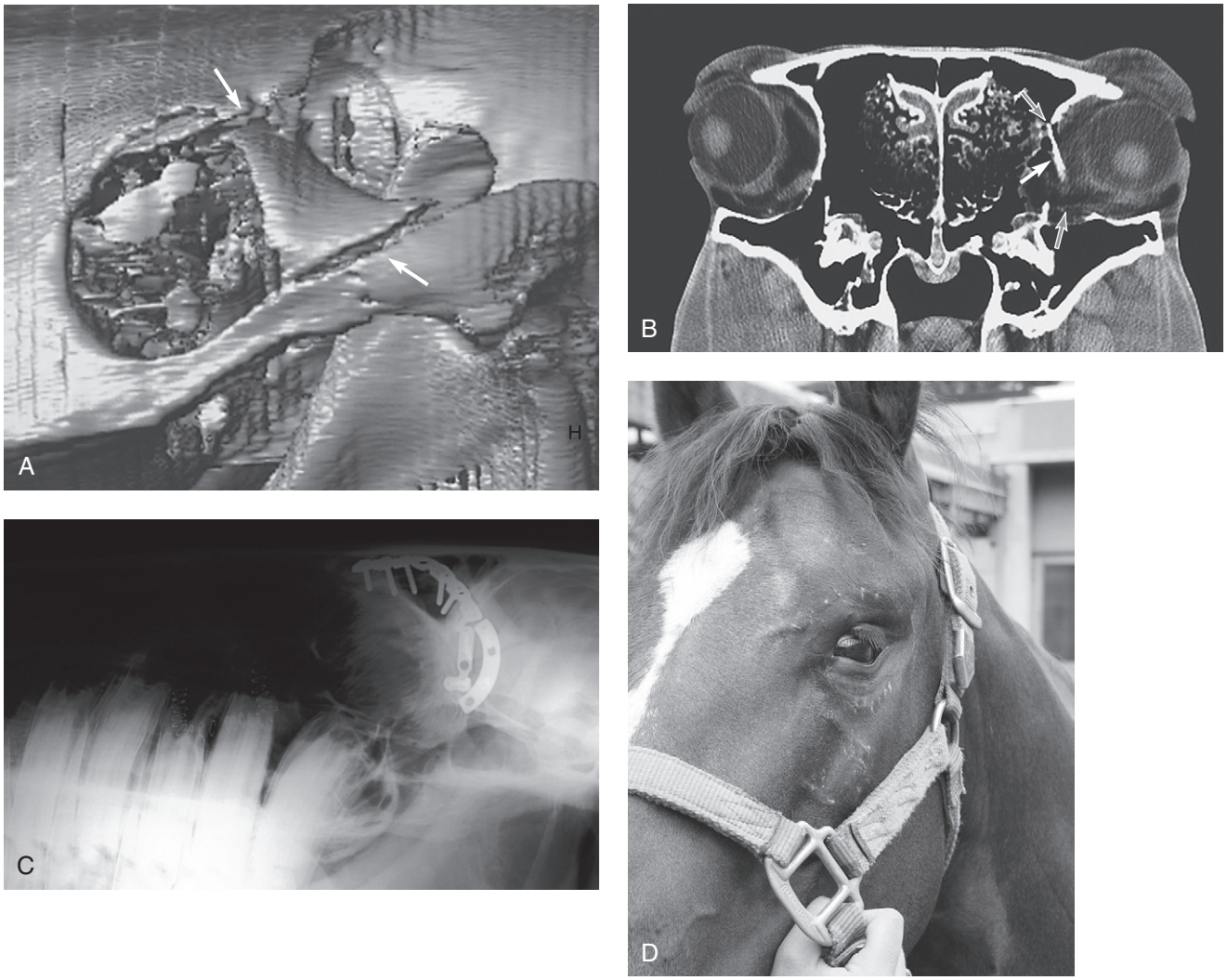


Figure 102-28. A, Three-dimensional CT reconstruction of the zygomatic arch fracture in an adult horse *(arrows).* **B,** Crossectional CT image of the fractured region showing additional fractures of the orbital floor. Proximomedial fracture line of the orbital floor *(upper arrow),* orbital floor fragment *(white arrow),* and periocular tissues protruding through a defect in the orbital floor *(lower arrow).* **C,** Oblique postoperative radiographic view of the repaired fracture. The repair was accomplished with the help of a 3.5-mm acetabular plate, a 6-hole 3.5-mm reconstruction plate, and a 2.7-mm L-plate. **D,** View of the horse after healing of the fractures. The eye has a slightly sunken appearance.

prevents fluids from entering the lungs. The jaws are spread and maintained in that position by carefully placing a speculum between the cheek teeth. The dorsal aspect of the oral cavity and the upper lip is draped.

A transverse incision is made in the oral mucosa on the lingual side of the incisors. The gingiva is incised on the labial surface of the incisors ventral to the lesion, and this incision is continued laterally and dorsally on both sides to connect with the initial incision. Both incisions are thus connected and continued down to the bone on the mandibular symphysis.

The soft tissues are elevated and reflected caudad with a periosteal elevator on the mandibular symphysis to expose normal bone. The soft tissues are preserved to allow coverage of the exposed mandibular stump.

The mandibulectomy procedure involves removing the incisive bone and as much of the symphysis of the mandible as needed, taking into account that part of the symphysis should be saved to ensure rostral continuity of the mandible. An oscillating bone saw or Gigli wire is used to transect the mandible at the selected level. After the bone edges are contoured to remove sharp edges, the soft tissue on the lingual surface of the mandible is folded rostrad over the exposed mandible and trimmed to fit the mucosal flap from the labial surface (see Figure 102-30, *B*). A Penrose drain is placed between the bone and the soft tissues.

Closure is performed to prevent the sutures from being subjected to trauma during mastication. An acceptable cosmetic appearance is retained as well as the ability to prehend feed despite significant shortening of the mandible (Figure 102-31).

A similar procedure for excision of the premaxilla and maxilla caudal to the canine teeth has been described.[33]

Complications and Prognosis

For both procedures, a recurrence of the lesions can only be prevented by completely resecting them. The cosmetic outcome is acceptable to many owners.

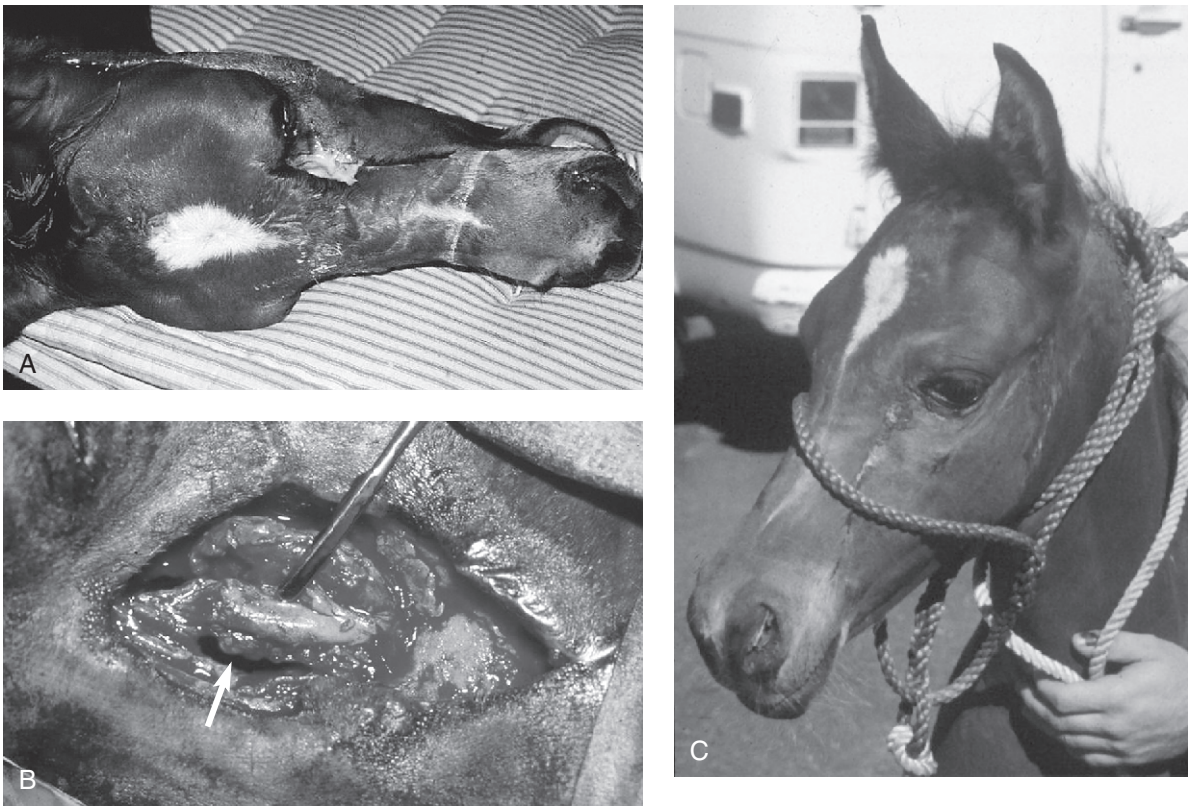


Figure 102-29. A, A 2-month-old foal with a sinus fistula just below the eye. The foal was kicked by a mare and subsequently sloughed a large bone sequestrum, which led to this fistula. **B,** Intraoperative picture showing the fistula *(arrow)* and the periosteal flap (under the elevator) that was sutured over the defect. A cancellous bone graft was placed over the flap (see Figure 77-1). The skin was closed and healed without complication. **C,** Two-month follow-up of the foal with a cosmetically acceptable result.

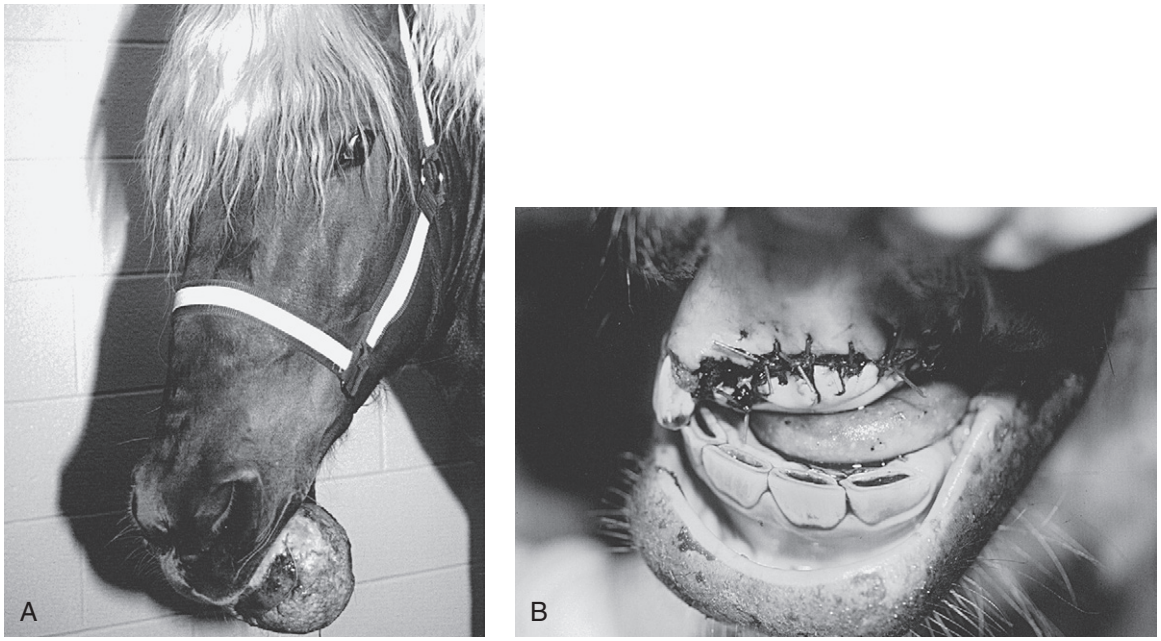


Figure 102-30. A, Preoperative picture of a 9-year-old Belgian horse with an ossifying fibroma of the mandible. **B,** Postoperative appearance after a partial rostral mandibulectomy. Note that the mucosa was sutured over the cut surfaces of the mandible.

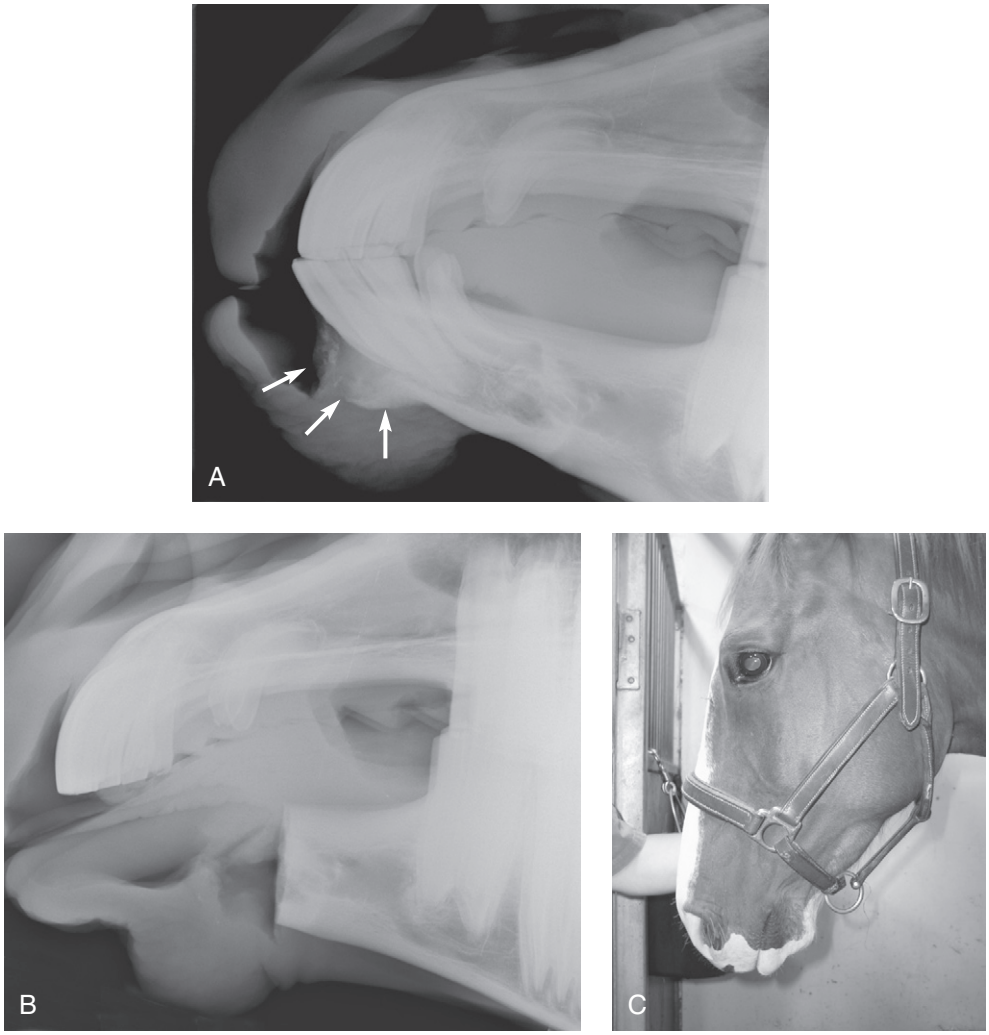


Figure 102-31. A, Lateromedial radiographic view of an adamantinoma at the rostral end of the mandible *(arrows).* **B,** Postoperative radiograph showing the missing rostral aspect of the mandible. **C,** Despite the mandibulectomy, the horse maintained an acceptable cosmetic appearance. (Courtesy P. Brink and A. Martensson, Malmö Equine ATG Clinic, Sweden.)

Flaccidity of the lower lip may occur with rostral mandibular resection. Horses have no difficulty with prehension of hay and grain; however, they may have difficulty with prehension of short grass. Protrusion of the tongue may occur.[34]

WRY NOSE (CAMPYLORRHINUS LATERALIS)

Wry nose is present at birth and is assumed to be a congenital disorder. In Arabian horses, it is possibly genetically linked, because of its frequency in this breed.[35] Intrauterine positioning as an etiology of this deformity is highly unlikely.

A computed tomography study of a foal with a wry nose revealed a potential etiology of this deformity. The proximal end of the incisive bone was fused with the nasal bone on the short side (Figure 102-32, *A*) but was not fused on the long side (Figure 102-32, *B*). This observation led to the theory that during the intrauterine development the incisive bone and the nasal bone fused on the short side. This stopped growth on this side, while growth continued on the other side, pushing the premaxilla into a wry position.

Usually a foal is presented within the first few days to weeks after birth because of the cosmetic appearance. Clinically, a varying degree of premaxillar deviation is noticeable upon inspection of the foal's head; the deviation usually results in malocclusion, with no contact between the incisors of the mandible and maxilla (Figure 102-33). Often there is also a certain rotational component in the deviation. Close observation also reveals an enhanced concavity of the hard palate in the interdental space.

An incongruity of the nostrils and nasal septum are additional anomalies. Some foals experience breathing difficulties or have increased respiratory sounds.

This condition will not self-correct, and a decision should be made to treat the foal using reconstructive techniques or to euthanize it to prevent it from suffering.

Surgical Procedure

Surgical correction should be delayed until the foal is 2 to 3 months old to allow the maxilla to gain enough strength to support implants. The foal is anesthetized and positioned in dorsal or sternal recumbency on the surgery table. Special padding is necessary if sternal recumbency is selected. The endotracheal tube is inserted ideally through a tracheostomy

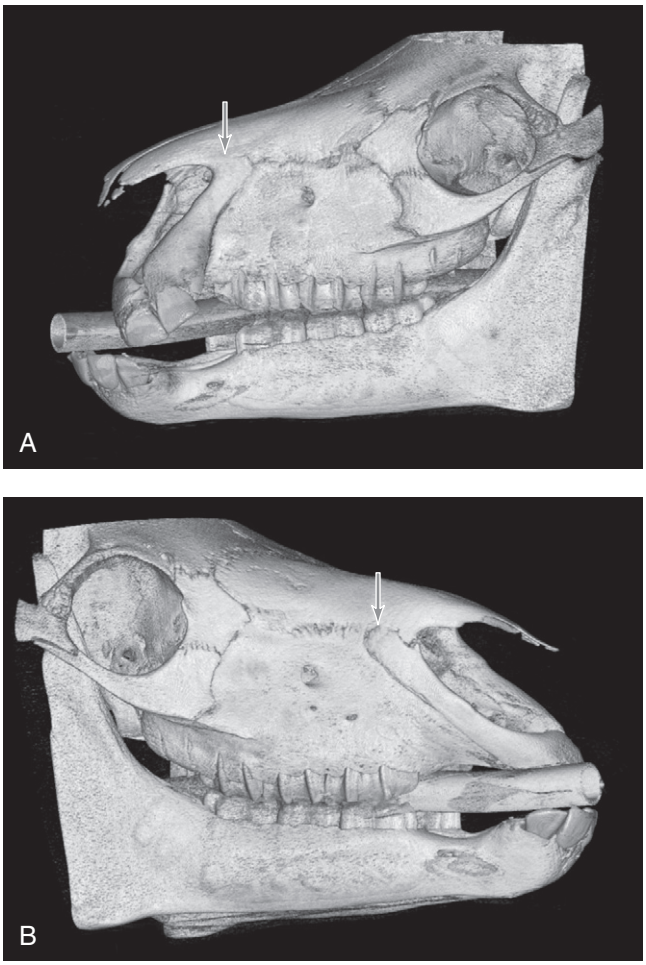


Figure 102-32. A, Three-dimensional CT reconstruction of the left side of a foal head with a wry nose. The incisive bone is fused to the nasal bone at its proximal aspect *(arrow).* **B,** Three-dimensional CT reconstruction of the right side of the same foal showing the open suture line *(arrow)* between the incisive and nasal bones allowing normal growth.

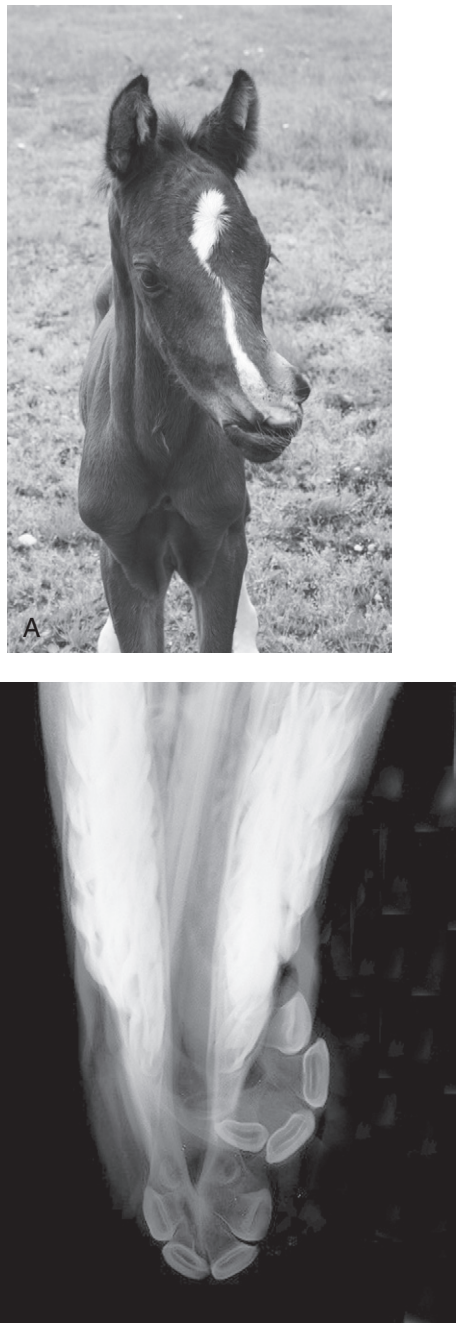


Figure 102-33. A, A photograph of a 1-month-old Warmblood foal with a wry nose. **B,** A ventrodorsal radiographic view showing the mandible and the incisor teeth that are oriented at a 90-degree angle relative to each other.

approach, the cuff is inflated, and the mouth is thoroughly washed.

Because the nasal septum is involved in the deviation, it is best to resect a 6-cm length of the rostral nasal septum at the onset of the surgery. The secion to be removed is centered on the midde of the bend in the nasal septum. The rostalmost aspect of the nasal septum should be left in place to prevent collapse of nares. The partial resection of the septum eliminates resistance to correction after the bilateral osteotomy of the maxilla. The nasal passages are packed with gauze sponges soaked in antiseptic solution to control hemorhage. A surgical approach to the interdental space is made on both sides of the head, and the maxillary periosteum is elevated.

The dorsal aspect of the nose is approached through a curved incision along the midline of the nose. Care is taken not to incise the narrow ligament that connects the nasal bones. The periosteum is left intact. The nasal bones are identified and subsequently transected perpendicular to their long axis, at the point of maximum curvature, with an oscillating saw. Care is taken not to penetrate the underlying parietal cartilage of the nasal septum. The deviation is corrected and stabilized with the help of 2.4-mm Unilock plates. These plates are applied over the periosteum. The gap created at the concave aspect of the nasal bones can be filled with a small portion of a rib graft harvested to fill the gap in the premaxilla.

With an oscillating saw, both rami of the maxilla are transected. The transected rostral portion of the upper jaw is rotated toward the sagittal plane of the head until the premaxillary and mandibular incisors are properly aligned. To achieve symmetry of the upper and lower jaws and normal interdental contact, the incisors of the maxilla and mandible should be temporarily wired shut.[36] An individual wire brace is applied to the incisors of the mandible and maxilla (Figure 102-34). Prior to tightening

Figure 102-34. Interdental wiring of the teeth to align the maxilla and mandible during repair of the osteotomies of the hemimaxillae. The wiring can be accomplished with a circular wire loop around the base of the incisor teeth or through holes drilled through the corresponding incisor teeth as shown here.

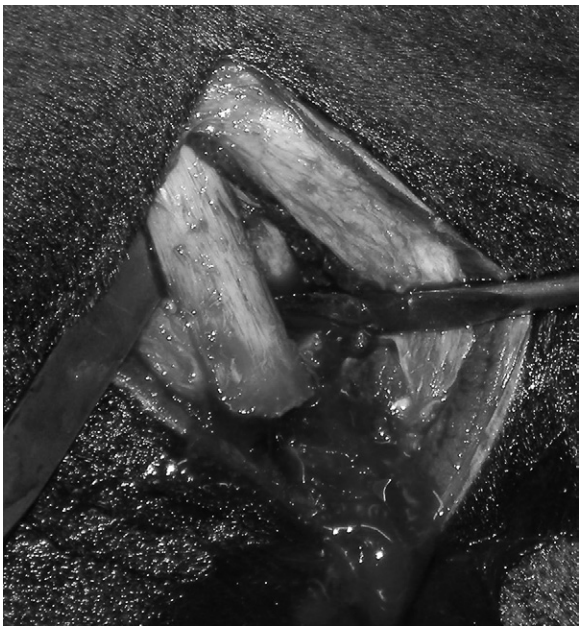


Figure 102-35. The rib graft is dissected free and bluntly separated at the osteochondral junction. The rib is slightly elevated to facilitate separation at the proximal end with an oscillating saw.

of the wire loops around each tooth, a double strand of wires is alternately pulled in a simple continuous fashion between the mandible and maxilla. The individual wires in the mandible are now tightened, followed by the wire loops in the maxilla. Last, the ends of the double-stranded wire suture between the incisors of the mandible and maxilla are connected and solidly tightened, effectively preventing opening of the mouth.

The application of an autogenous cortical bone graft in the form of a rib (Figure 102-35) has been described in the literature.[37-39] The rib graft is cut to fit the gap in the concave side of the premaxilla and wedged between the bone ends. In the next step, 3.2-mm Steinmann pins are inserted from the rostral aspect of the mandible in a caudal direction, crossing the osteotomy sites, providing stability to the maxilla.[35-39] The pin crossing the concave aspect of the mandible also penetrates the medullary cavity of the rib graft before entering the caudal fragment of the mandible.[38]

The wiring of the tooth arcades is left in place to ensure anatomic reconstruction and can be maintained until the osteotomies have partially healed and can provide some stability. During this period, enteral nutrition is provided for the foal, often by milking the mare and administering the milk through a stomach tube.

An alternative technique is to apply bone plates, ideally LCPs (Figure 102-36) to both osteotomies. The rib graft is incorporated into the plate fixation as well. The surgical incisions are closed in two layers using routine technique and covered by a stent bandage consisting of rolled gauze sponges sutured over the skin incisions. When the osteotomies are stabilized, the interdental wiring between the premaxilla and the mandible is removed. The surgical incisions are closed in three layers using routine technique.

Some asymmetry of the rostral part of the nose will persist. Postoperatively the foal is allowed to nurse but should be prevented from eating hay and grain.

Distraction osteogenesis has been described in the literature as a method to treat wry nose.[40] A type II external fixation device was applied to the maxilla and premaxilla with a double connnecting bar on the convex side and a distraction bar on the concave side of the nose. Over the next couple of weeks, the concave side was gradually distracted, eventually ending in a cosmetically acceptable result.[40]

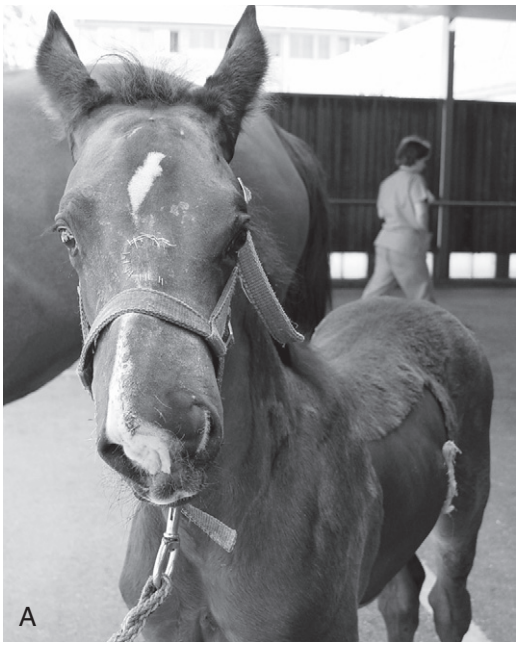
Aftercare

The tracheostomy tube is maintained, changing it with a clean tube every day. Broad-spectrum antimicrobial therapy and nonsteroidal, anti-inflammatory drugs are administered for 5 or more days. Half of the packing of the nasal passages is removed one day postoperatively and the second half one day later. At this time, the tracheostomy tube can be removed. Removing the packing may be difficult if the packing becomes impaled on the point of a pin or wire that inadvertently entered the nasal cavity.[39] The stent bandages are removed at 4 or 5 days, and staples or sutures are removed at 10, 12, or 14 days after surgery. The foal can usually be discharged from the hospital at 2 to 3 weeks after surgery.

Steinmann pins and Kirschner wires used to stabilize the premaxillae/maxillae are removed between 4 and 5 weeks, but most become loosened and are spontaneously shed at about this time. Plates used to stabilize the nasal bones are removed at about 3 months postoperatively.

Complications

Postoperative infection of the surgery sites and breakdown of the fixation are the most serious complications. Depending on the severity and duration of the problem, euthanasia of the patient has to be considered. Partial collapse of the nasal passages as a result of nasal septum removal may lead to respiratory problems. Surgical resection of the alar folds may at least partially correct this problem.[41]



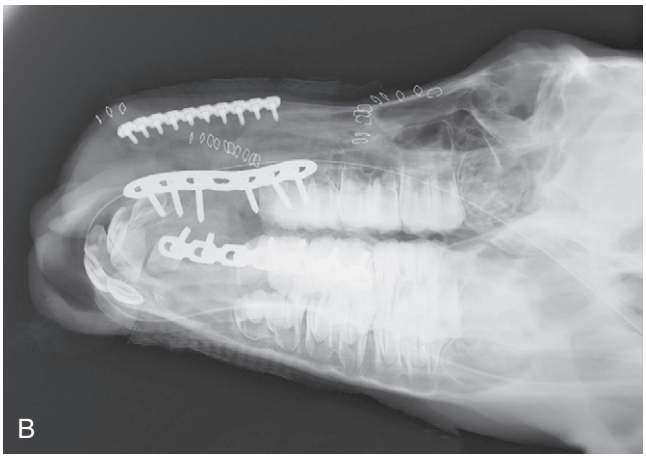
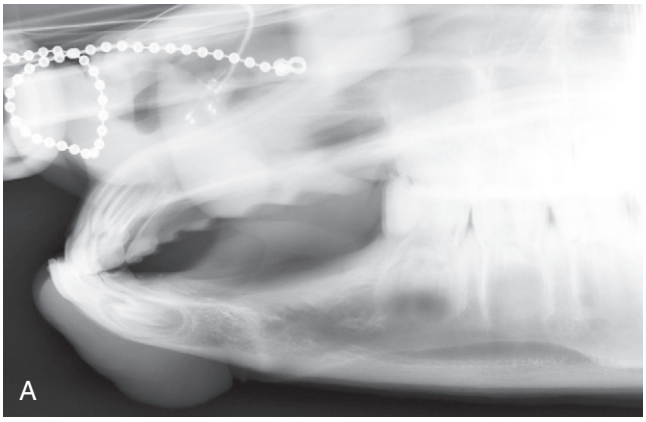
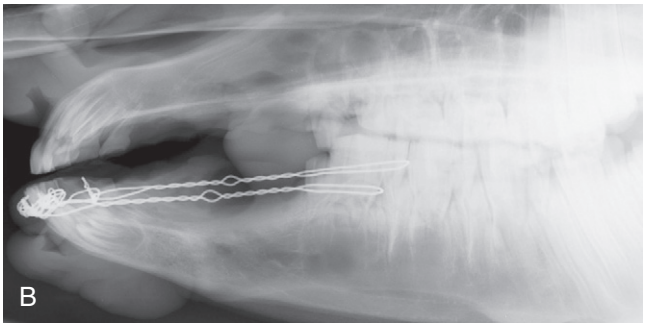


Figure 102-36. A, 1-week postoperative picture of the same foal as Figure 102-33. The sutures are still in place. The C-shaped suture line represents the site where the caudal cut was made for partial removal of the nasal septum. The white marking of the nose accentuates the lopsided appearance of the nose. **B,** Oblique postoperative radiographic view of the head showing the three plates used in the correction of the abnormality.





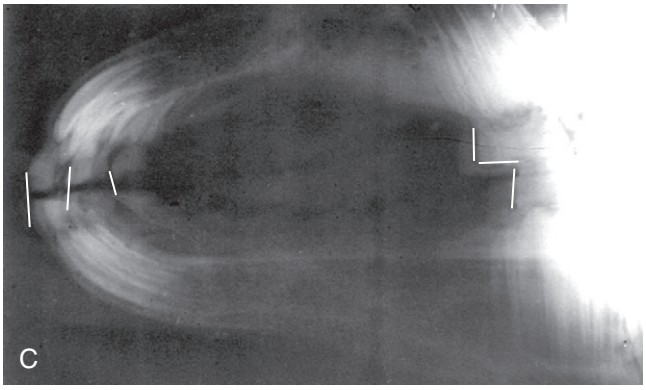


Figure 102-37. A, Lateromedial radiographic view of a 2-year-old Arabian horse suffering from prognathia inferior. The horse is intubated through the nose. **B,** Immediate postoperative lateromadial radiographic view of the horse with the tension wires in place. **C,** Follow-up lateromadial radiographic view of the horse 3 months later. The tension wires have been removed from the mandible and good alignment of the upper and lower arcade is appreciated. Note the mandible shows an increased bend in the region of the incisor arch.

CORRECTION OF PROGNATHIA SUPERIOR (PARROT MOUTH) AND PROGNATHIA INFERIOR

Prognatia superior (or brachygnatia inferior) refers to an assymmetry between the length of the maxilla (and premaxilla) and the mandible, with the mandible being shorter. This condition, called *parrot mouth*, is the most common congenital oral malformation in horses and may be associated with other congenital problems.[41] Varying degrees of parrot mouth may exist, ranging from minor malocclusion to complete lack of contact between the upper and lower incisor teeth. The result is abnormal wear of the incisor teeth, eventually leading to abnormal wear of the premolar and molar teeth. The condition is primarily of cosmetic concern to the horse's owner. On rare occasions, the opposite problem is encountered where mandible is longer than the maxilla and premaxilla, and is called *prognathia inferior* (Figure 102-37, *A*).

Treatment

The teeth should be examined at regular intervals and the teeth floated as necessary to prevent hooks on the rostral and caudal cheek teeth and to keep the incisors from wearing abnormally.

The treatment for both malformations is the same; temporarily slowing down growth is performed on the longer componenet of the malformation, either at the maxilla in prognathia

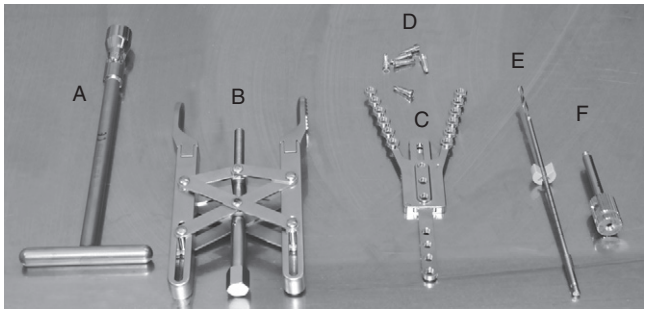


Figure 102-38. RachetDis instrumentation. *A,* Socket wrench for distraction; *B,* distractor placed between the incisors and cheek teeth used to distract the implant 1 mm at a time; *C,* RachetDis implant consisting of a narrow bar containing four round holes for the insertion of 3.5-mm locking head screws, a wider body surrounding the narrow bar and containing three similar holes, and two easily bendable sidearms containing holes for locking head screws to increase stability; *D,* several 3.5-mm locking head screws; *E,* 2.8-mm drill bit; *F,* insert drill sleeve for the drill bit.

superior or at the mandible in proganthia inferior. Extension is performed on the shorter componenet.

In foals with severe malocclusion that impairs their ability to graze and eat, growth of the maxilla or mandible can be temporarily slowed by applying tension wires between the incisor teeth and the cheek teeth (see Figure 102-37, *B*). After the difference in jaw length has evened out, the wires are removed (see Figure 102-37, *C*). In some foals corrected for prognathia superior, the maxilla assumes a dorsal bend over the nose during the time longitudinal growth is halted, rendering a shorter and more stocky head.

An alternative technique involves a surgical implantation of a RachetDis (Figure 102-38) on both hemimandibles or maxillae. The RachetDis allows a distraction of up to 2 cm and is completetly implanted. Distraction is achieved indirectly via distraction of the incisor and the premolar teeth.

Both rami of the mandible are approached through the ventrolateral 12-cm skin incision. The periosteum is separated from the mandible over the entire length. A RachetDis is contoured to each mandible and attached to it through four locking head screws in the rostal part and at least three 3.5-mm locking head screws in the caudal part. The future osteotomy location is marked on the bone and the RachetDis devices are removed. A vertical osteotomy is performed through both interdental spaces followed by reapplication of the RatchetDis implants (Figure 102-39, *A*). If needed, additional screws can be implanted through one or both sidearms of the implant. The periosteum is apposed if possible and the rest of the incision is closed in two layers using routine technique. A stent bandage is applied over the skin incisions.

To improve the conditions for the application of the distractor it is advisable to reinforce the mandibular incisor arcade with polymethyl methacrylate (Technovit). Prior loose application of cerclage wires around the incisors provides additional stability to the construct (Figure 102-39, *B*). While the horse is still under ansethesia, the distraction device is applied between the incisor teeth and the premolar teeth through the interdental space, and with the socket wrench traction is applied until a click is heard, representing a distraction of 1 mm. This action is repeated twice on the same side, followed by a 3-mm distraction on the other side. The horse is allowed to recover from anesthesia.

Figure 102-39. A, The RachetDis implanted to one side of the mandible in a 2-year-old horse with a prognathia superior. The oral cavitiy was not invaded during the approach. The osteotomy is clearly visible. **B,** The prognathia superior shown immediately postoperatively. The cerclage wires were implanted to provide stability to Technovit support of the mandibular incisor arcade needed as a thrust-bearing device for the distractor. **C,** Lateromedial radiographic view of the head of the at the end of the distraction. The RachetDis implants of both hemimandibles are superimposed on each other. The incisor arcades of the maxialla and mandible are evenly aligned and the gap at the osteotomy site is easily recognized. The implants stay in place until the gap is healed in with bone. (Courtesy C. Lischer, Berlin, Germany.)

Aftercare

Routine postoperative management is applied consisting of broad-spectrum antimicrobial and anti-inflammatory therapy for 5 days, stent bandage removal after 5 days, and skin suture or staple removal after 10 to 12 days. Starting on the fifth postoperative day, the RachetDis is distracted daily for 1 mm with the foal under heavy sedation. After perfect occlusion is achieved, distraction is stopped (see Figure 102-39, *C*). The implants are removed 3 months later.

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CHAPTER 103

Postoperative Physiotherapy for the Orthopedic Patient

Andris J. Kaneps

Physical treatment and rehabilitation of horses is often a major adjunct to surgical or medical therapy. It may also be the primary therapy under which circumstances a horse may be competing under *Federation Equestre Intrenationale (FEI)* or other competition regulations that prohibit the use of medications. Physical treatment and rehabilitation can be organized into five catagories: thermal therapy, manipulative therapy, exercise, extracorporeal shock wave therapy, and hyperbaric oxygen therapy.

THERMAL THERAPY

One of the most accessible and time-tested methods of physical treatment is thermal therapy. Heat or cold may be administered to horses using many modalities and can range from simply applying water from a garden hose to using deep-heating ultrasound technologies.

Cold Therapy

Physiologic Effects

The major physiologic benefits of cold therapy are decreased local circulation, decreased pain, and reduced tissue swelling.[1-5] These benefits are most effective very early in the period following injury or surgery (Table 103-1).

The primary effect of local cold application is to constrict blood vessels. Local reflexes and central nervous system responses mediate this *vasoconstriction*. The reduced blood flow to tissues may help reduce edema, hemorrhage, and extravasation of inflammatory cells. The consequential *reduced tissue metabolism* may inhibit the effect of inflammatory mediators and slow down enzyme systems.

Cyclical *rebound vasodilatation* is another response to cold therapy.[6] Following a minimum of 15 minutes of cold therapy that results in tissue temperatures that range from 10° to 15° C, cycles of vasoconstriction and vasodilatation occur. An example of this effect is the warm sensation in your fingers after exposure to cold. Rebound vasodilatation associated with cold therapy may help further resolve tissue edema.

Analgesia follows cold therapy. Explanations for the pain relief induced by thermal agents include the following: (1) the stimulus from cold (or heat) receptors closes the neural gate, which modulates the perception of pain via blocking afferent stimuli from peripheral pain receptors (gate-control theory), and (2) cold or heat may act as a counter-stimulus to pain, making the afferent impulses of pain less noticeable centrally.[1-3] Small myelinated nerve fibers are more sensitive to cold therapy and, as they are cooled, conduction is reduced earlier than in similar-sized unmyelinated fibers. For example, application of an ice pack over the ulnar nerve in cats for 20 minutes reduced nerve conduction velocity by 29%.[5]

The viscoelastic properties (i.e., extensibility) of soft tissues are reduced with cold therapy, which is an undesirable effect of this thermal therapy.[1-3]

Indications

Cold therapy is indicated in acute musculoskeletal injuries and following surgical procedures to reduce edema, slow the inflammatory response, and reduce pain. It is particularly effective during the first 24 to 48 hours after injury or surgery.[7] Cold immersion of the distal limbs is also effective in reducing severity of laminitis by decreasing the activity of laminar matrix metalloproteinase (MMP) and causing laminar vasoconstriction when applied during the developmental phase.[8]

Methods of Application

Cold may be applied by ice water immersion, application of ice packs or cold packs, and ice water–charged circulating bandages or boots. The most beneficial therapeutic effects of cold occur at tissue temperatures between 15° and 19° C (59° to 66° F).[1-3] Tissue temperatures of 10° C and less may cause cold thermal damage. Average time of cold application is 15 to 20 minutes. Treatments are best repeated every 2 to 4 hours during the first 24 to 48 hours of injury or surgery if the goal is to reduce tissue inflammation and edema.

TABLE 103-1. Thermal Therapy

Therapy type	Indications	Methods of application	Responses to treatment
Cold	Acute injury (first 24-48 hours) Reduce pain Reduce swelling	Ice water immersion Ice surface application Cold packs	Restricts blood flow Reduces metabolism Reduces activity of inflammatory enzymes Reduces pain
Heat	Chronic injury (after 72 hours) Enhance tissue stretching Enhance healing response	Warm water from hose Hot packs Leg sweat Therapeutic ultrasound	Increases blood flow Increases metabolism Increases activity of tissue enzymes Relaxes muscle spasm Reduces pain Increase tissue extensibility

Cold therapy is effective from the skin to a depth of 1 to 4 cm depending on the amount of adipose tissue present in the area and the local blood supply.[1-3] Cold treatment is rapid and effective in the distal limbs of horses because they lack an adipose layer, and target tissues such as tendons, ligaments, and joint capsules are located superficially.

Direct contact of ice water with the skin is the most effective method of cold therapy. Buckets or turbulator boots may be used depending on the site. If immersion therapy is used immediately following surgery, the wound must be protected with a water-impervious barrier. In human physical therapy, ice baths are maintained at temperatures from 13° to 18° C (55° to 64° F).[1] Ice may be placed in a plastic bag or water may be frozen in a paper cup and applied to the site. To prevent laminitis, *continuous* cold therapy is applied to the distal limbs using plastic bags filled with ice, ice water immersion, or commercial cold therapy boots.

In a study of ice water immersion therapy applied to the metacarpal region in a horse, skin surface temperatures stabilized at 9° to 10° C approximately 10 minutes after therapy was started. Temperature stabilized at 17° to 19° C in subcutaneous tissue and at 20° to 24° C between the flexor tendons approximately 10 minutes after therapy was started. Tissue temperatures did not return to baseline by 10 minutes after therapy was stopped.[5] Ice water immersion of the equine digit for 30 minutes resulted in significant decreases in soft tissue perfusion and laminar temperatures. Vascular perfusion decreased, but not significantly.[9]

Malleable commercial cold packs are an effective means of cold therapy because they readily conform to the anatomic site (First + Ice cold packs. MacKinnon Inc.). Minimizing air gaps between the cold pack and treatment site improves tissue cooling.[10] Commercial products, such as cold leg wraps (Ice Tape cold therapy leg wrap), have the same gel substance as cold packs. The gel is formed as a tape that may be wrapped on a limb and held in place with a wet bandage.

Boots that connect to a cold source and circulate fluid through them are also very effective at chilling tissue. Systems are available with a variety of boot configurations for different portions of the limb, making effective cold therapy logistically very simple. Some of the systems also provide compression and may be used for cold (or heat) therapy (Figure 103-1).

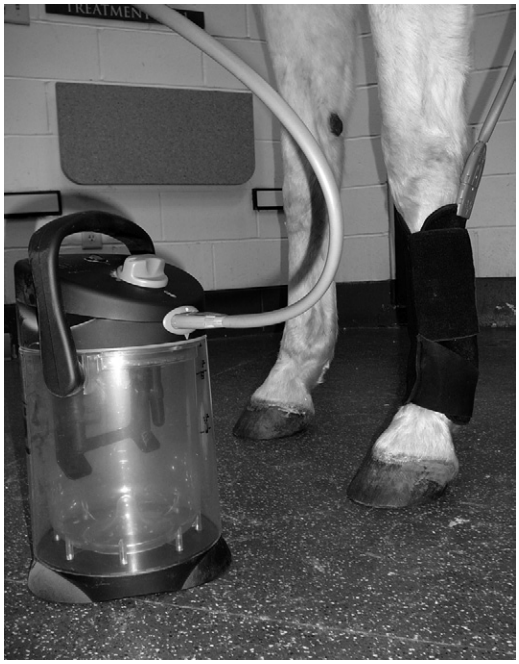

Figure 103-1. Cold and compression therapy using a system that circulates ice water or heated water through site-specific wraps (VitalWear Equine System).

Heat Therapy

Physiologic Effects

The major physiologic benefits of heat therapy are increased local circulation, muscle relaxation (and therefore, reduction of muscle spasms and associated pain), and increased tissue extensibility (see Table 103-1).[1-3]

Increased local blood flow helps mobilize tissue metabolites, increase tissue oxygenation, and increase metabolic rate of cells and enzyme systems. In general, metabolic rate increases two to three times for a tissue temperature increase of 10° C.[1] These responses to heat therapy are especially beneficial for wound healing.

Increased local blood flow may be initiated by three mechanisms: (1) by the axonal reflex, representing a direct response of the blood vessels to local increases in tissue temperature; (2) by local inflammatory mediators, such as histamine, prostaglandins (PGE_2 and prostacyclin), and bradykinin, which may also initiate vasodilatation and increase capillary permeability; and (3) by local spinal reflexes that are stimulated by afferent impulses from the thermoreceptors at the site of heating and result in decreased postganglionic sympathetic adrenergic nerve activity, which decreases smooth muscle tone of vessels, allowing them to dilate.[1]

Increased blood flow and vascular permeability may promote resorption of edema, which is a common reason for heat application in horses. However, vasodilatation of a dependent limb may actually lead to edema by the same mechanisms (increased blood flow and vascular permeability).

Heat application also decreases pain. Pain is relieved via the mechanisms cited earlier for cold therapy.

Muscle spindle firing decreases when muscle temperature increases, resulting in decreased muscle spasms. Decreased spindle firing helps to break the cycle of pain-spasm-pain. The same sort of cycle may be partially responsible for flexural contractures in foals with physitis, where severe physeal pain results in reflex increases in muscle spindle firing. The flexor muscles are the largest group of forelimb muscles in the foal, and continued spasm of the large flexor muscles may lead to flexural contracture (see Chapter 87).

Soft tissues may be stretched more effectively when they are warm. Heat decreases tissue viscosity and increases tissue elasticity. Low-load, prolonged stretching of tissues heated between 40° and 45° C (104° to 113° F) results in increased extensibility of tendons, joint capsules, and muscles.[1-3]

Indications

Heat is best applied after acute inflammation has subsided. It is useful for reducing muscle spasms and pain because of musculoskeletal injuries. Heat therapy can be used to increase joint and tendon mobility, particularly by their application before

active stretching. Heat may benefit recovery of localized soft tissue injuries by accelerating the healing response.

Methods of Application

Superficial and deep heat modalities are available. Superficial heat is most commonly applied using hot packs and hydrotherapy. These modalities provide heat penetration to approximately 1 cm below the level of the skin, which is often sufficient for many veterinary applications on distal limbs because of the lack of an insulating fatty subcutaneous tissue layer. Deep heat may be applied using therapeutic ultrasound.

The most profound physiologic effects of heat occur when tissue temperatures are raised to 40° to 45° C (104° to 113° F).[1,3,4] However, tissue temperatures above 45° C may result in pain and irreversible tissue damage. Skin and subcutaneous tissue temperature increases approximately 5° C after 6 minutes of heat application and maintains that temperature for up to 30 minutes following cessation of treatment.[10] For deeper tissues, such as tendon or muscle, 15 to 30 minutes is required to elevate tissue temperature to the therapeutic range.

Direct contact of the skin to temperatures over 45° C (115° F) may cause thermal injury and tissue damage. When using heat sources warmer than 45° C, the source must be wrapped in several layers of moist towels before application.[4] This is especially important if you are heating the site using hydrocollator packs (Hydrocollator moist heat pack), which are heated to 71° to 79° C or rechargeable hot packs (Sodium acetate rechargeable hot pack), which are heated to 54° C. Heat from these sources is usually applied for 20 to 30 minutes.

Warm water is probably the most accessible method of heat therapy. Methods of application include the use of a hose, wet towels, water immersion in a bucket, turbulator boot, and circulating treatment system. A general rule is that water as hot as your hand can comfortably stand has a temperature of 38° to 41° C (101° to 105° F). However, tissue heated by water at this temperature may only reach the lowest tissue therapeutic range. Therefore, the target temperature should be above this level, but horses commonly experience discomfort with water 45° C and warmer. An electric bucket heater may be used to warm water. To render warm hydrotherapy most effective, a thermometer should be used to determine the water temperature. An inexpensive indoor-outdoor home thermometer with a wire thermocouple is very useful to determine water temperature.

A study of hot water hose therapy applied to the metacarpal region in a horse revealed that water as warm as a human could comfortably stand resulted in skin surface temperatures on the horse of 39.5° to 41° C. Temperature of subcutaneous and deep tissues stabilized at 39° to 40° C approximately 9 minutes after therapy was initiated. Tissue temperatures returned to baseline approximately 15 minutes after therapy was ceased.[10] Heating the equine digit by standing in a 47° C water bath for 30 minutes resulted in significant increases in soft tissue perfusion and laminar temperatures. Vascular perfusion increased, but not significantly.[9]

Therapeutic ultrasound may be used for superficial and/or deep heating of tissues. Ultrasound selectively heats tissue with high protein/collagen content. Tendons, muscles, fibrous connective tissue and bone may be effectively heated to 45° C, but adipose tissue is relatively transparent to the effects of therapeutic ultrasound. The most intense heating occurs at tissue interfaces, much like a diagnostic ultrasound image has sharp delineations between tissue interfaces such as skin, tendon, and fluid. In dog thigh muscles, ultrasound treatment with a 3.3-mHz transducer at 1.5 W/cm^2 resulted in temperature increases of 4.6° C, 3.6° C, and 2.4° C at 1 cm, 2 cm, and 3 cm depths, respectively.[11] Soft tissue temperature has been shown to increase 0.2° C per minute with a 1-mHz transducer set at 1.5 W/cm^2.[12]

An additional benefit of therapeutic ultrasound is the deep massage of tissues caused by the sound waves, referred to as *cavitation* and *streaming*. These nonthermal effects result in compression and expansion of tissues and tissue fluids that may improve tissue healing.[13] Fibrous connective tissue scars may be more effectively stretched following heating with therapeutic ultrasound.[14] Treatment is usually performed once or twice daily for 10 to 14 days. Ultrasound coupling gel must be used to provide good contact between the transducer and the skin. In horses, treatment is usually conducted with a 1-mHz transducer for deepest penetration at 1.5 W/cm^2 for 10 minutes.

MANIPULATIVE THERAPY

Stretching and massage are true physical methods of treatment that may be applied without the need for sophisticated equipment. Stretching is useful for maintaining or increasing the range of motion of an injured or operated site and for increasing a horse's flexibility to improve performance.

To maintain or increase joint mobility, *stretching* should be initiated 24 hours after surgery or after the injury has been accurately evaluated and a diagnosis has been made. Stretching is done by flexing the joint just to the point of discomfort, holding at that level for 10 to 15 seconds, moving the joint to its limit of extension and repeating the sequence 12 times. Joint stretching may be repeated two or three times daily.

Massage therapy may primarily provide some level of warm-up to the muscles and may assist in soothing muscles after exercise. Experience with human athletes has shown that pre- and post-exercise massage makes athletes more comfortable and helps decrease stiffness following competition.[15]

There are variations in massage therapy techniques that focus on specific mechanisms of action. *Trigger point massage* focuses on relaxing tight bands of muscle or fascia that are sensitive to manipulation, thereby reducing sensitivity of the affected areas. This type of massage has been found to reduce heart rate and blood pressure and to significantly relax human patients.[16]

Massage also aims to decrease the sensitivity of active myofascial triggers that may cause discomfort in a distant area.[17] *Acupressure massage* results in stimulation of regions that are connected to Chinese meridians similar to acupuncture points.[18] Massage therapists use friction, vibration, deep friction, and a variety of stroking techniques to achieve the desired outcome.[17]

Massage therapy should be considered palliative, because few of the specific claimed advantages of the techniques have been scientifically tested and verified as being efficacious.[19] Using a trained therapist with a close professional relationship with the attending veterinarian is imperative for safe and effective treatment.

EXERCISE

Controlled exercise and exercise that minimizes concussion may be used during the rehabilitation period after injury or surgery. Controlled exercise protocols have been established

Figure 103-2. The underwater treadmill reduces concussion and encourages full range-of-motion limb use while conditioning muscles and the cardiorespiratory system (AquaPacer equine underwater treadmill). (Courtesy Dr. Steve Adair, University of Tennessee.)

for rehabilitation of tendon and ligament injuries.[20] Gradually increasing the time and intensity level of exercise is beneficial for healing of soft tissues and bone because both tissues become stronger with use than with rest, particularly in growing horses.[20-22] Commonly, the horse is maintained in stall confinement with controlled exercise via hand-walking, ponying, or by use of a mechanical exerciser. Harness race horses may readily enter a controlled exercise program by designating the number of jogging miles at a given pace for each exercise session.

Swimming or underwater treadmill exercise is an excellent method of providing controlled exercise with minimal concussion (Figure 103-2). These methods spare joints and bones from concussion but enhance muscle and cardiorespiratory fitness. Another benefit of swimming and underwater treadmill exercise is that the limbs are used through a full range of motion. Swimming should not replace conditioning for fitness under tack and under circumstances similar to competition. During swimming, the horse will not use the same muscle groups used during weight-bearing exercise and this activity may cause hyperextension of the back muscles.[17,23] Swimming must be combined with regular ring or track work to condition the muscles used in normal work and to strengthen the skeleton and joints.

Treadmill exercise represents a good intermediate step between exercise with minimal concussion (such as walking in hand, riding at a walk, or underwater treadmill exercise) and exercise in the horse's regular tack and training surface. Most treadmills have a rubber belt that reduces concussion during weight bearing. Speed and inclination of the treadmill is adjusted to the level of exercise intensity suited to the horse. The treadmill allows objective control of the exercise program that may be useful during and after rehabilitation is complete.[24] The horses in one study had a substantial amount of their base fitness established with treadmill exercise, followed by the gradual addition of regular track workouts. Eventually treadmill exercise was only 30% to 50% of the total exercise work. Horses trained in this fashion had fewer training and racing injuries and better race times than conventionally trained horses.[24]

Ultimately, horses must work under the same conditions they will encounter in competition. This means that riding or driving with a gradual increase in duration and intensity of exercise will be needed. The key to retraining a horse is to realize that cardiovascular fitness declines significantly after 4 to 6 weeks of rest[25] and that bone strength decreases significantly within 12 weeks of rest.[26] Retraining will result in noticeable improvement of cardiac measurements within 6 weeks, increased bone mineral density within 16½ weeks, and tendon dimensions within 16 weeks.[20,27,28] The studies on bone and tendon do not identify the earliest time that significant strength returns to these tissues so as to allow training or competition without reinjury. I assume that 3 to 4 months is the minimum time required to re-establish musculoskeletal tissue strength following a period of complete rest.

EXTRACORPOREAL SHOCK WAVE THERAPY

Extracorporeal shock wave therapy (ESWT) has few direct indications following surgery, but this modality is very useful for treatment of soft tissue and bone injuries. ESWT consists of short duration (5 μsec) high-pressure shock waves (up to 80 mega pascals) that stimulate tissue.[29]

Focused and non-focused ESWT devices are available for application in horses. Reports of positive treatment effects have been made for both types of ESWT.[29-31] Each treatment usually consists of 2000 pulses. Intensity of shock wave therapy is set in a range of 0.9 to 1.8 mJ/mm^2, or according to the atmospheric pressure at the output probe (2.5 to 4 bar), depending on the manufacturer's recommendations.[29,31] The horse must usually be sedated for treatment. Each treatment lasts 10 to 15 minutes and is repeated at 1- or 2-week intervals. Treatment protocols require one to four separate treatments depending on the nature of the injury and response to initial therapy.

Tissue compression and shear loads occur as the shock wave passes tissue interfaces, resulting in stimulation of bone and soft tissue healing. Bone healing and remodeling have been enhanced and an analgesic effect has been noted for 2 to 4 days following treatment.[29,32,33] ESWT treatment of arthritis of equine distal tarsal joints (bone spavin) resulted in improvement of lameness grade in 59 of 74 horses treated.[30] Chronic suspensory desmitis was successfully treated in 24 of 30 horses after three ESWT treatments.[31]

ESWT is indicated for treatment of insertional desmopathy (such as at the origin or insertion of the suspensory ligament) dorsal cortical stress fractures, incomplete fractures of the proximal sesamoid bone, arthritis, and navicular disease and has also been used for treatment of tendinitis.

HYPERBARIC OXYGEN THERAPY

The use of hyperbaric oxygen therapy in horses has gained some popularity, without much scientific evidence to support its claimed beneficial effects. For example, it has been used in healthy training horses supposedly to achieve quicker recovery after strenuous exercise.

Hyperbaric oxygen is administered in a walk-in pressurized chamber. Horses are treated with pressures of 2 to 3 atmospheres and breathe 100% oxygen for 60 to 90 minutes.[34]

Therapeutic effects of hyperbaric oxygen therapy on complex wounds in humans include reduction of tissue edema, improved tissue oxygenation, and enhanced antibiotic activity.[35] Suggested indications for other uses in horses include severe anemia, fungal pneumonia, thermal burns, cellulitis,

rhabdomyolisis, infected wounds, skin grafts, and nonunion fractures.[34] However, in the only controlled study of this therapy, full-thickness mesh skin grafts did not have an improved tissue response in horses that underwent hyperbaric oxygen therapy, compared to controls.[36]

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APPENDIX

A Drugs and Manufacturers

Drug Name	Generic Drug Names	Trade Names	North American Manufacturer Information	European Manufacturer Information	Chapter(s)
Acepromazine	acepromazine		Various U.S. manufacturers		19 to 22
		Prequillan		Arovet AG, Zollikon, Switzerland	19, 21, 22
		PromAce	Boehringer Ingelheim Vetmedica Inc, St. Joseph, MO		20
Alfaxalone		Alfaxan 10mg/ml	Not marketed in the U.S.	Vétoquinol GmbH, Ravensburg, Germany	19
Alfentanil		Rapifen, Alfenta	Taylor Pharmaceuticals, Marietta, GA	Janssen-Cilag AG, Baar, Switzerland	18
Atipamezole		Antisedan	Pfizer Animal Health, New York, NY	Pfizer AG, Zurich, Switzerland	20, 22
Atracurium besylate		Tracrium (no longer manufactured under this trade name)	Hospira, Lake Forest, IL	Glaxo Smith Kline, Middlesex, UK	58
Atropine	Atropine SA		Butler Animal Health Holding Company LLC, Dublin, OH; Vedco Inc, St. Joseph, MO		20
	Atropinsulfat 0.1%			Kantonsapotheke Zurich, Zurich, Switzerland	22
Avocado and Soya Unsaponifiables		Cosequin ASU	Nutramax Laboratories, Edgewood, MD		79
		Platinum Performance CJ	Platinum Performance, Inc, Buellton, CA		79
Benztropine mesylate		Cogentin	Merck & Co, Whitehouse Station, NJ		22
Buprenorphine		Temgesic		Essex Chemie AG, Lucerne, Switzerland	22

Drug Name	Generic Drug Names	Trade Names	North American Manufacturer Information	European Manufacturer Information	Chapter(s)
Butorphanol tartrate	Alvegesic 1% forte ad us. vet.			Virbac AG, Glattbrugg, Switzerland	18, 19, 22
		Torbugesic	Fort Dodge Animal Health, Fort Dodge, IO		20, 34, 48, 73
		Butrohic	Lloyd Inc, Shenandoah, IA		
		Dolorex	Intervet Inc, Millsboro, DE		
Hyoscine Butylbromide		Buscopan	Boehringer Ingelheim, Ridgefield, CT	Boehringer Ingelheim GmbH, Ingelheim, Germany	38, 59
Carprofen		Rimadyl	Pfizer Animal Health, New York, NY	Pfizer, Kent, UK	23, 79
Chlorpromazine HCl		Thorazine	Baxter Healthcare Corp, Deerfield, IL		22
Climazolam		Climasol ad us. vet.		Dr. E. Graeub AG, Bern, Switzerland	19, 20, 22
Dalteparin		Fragmin	Pfizer Inc, New York, NY	Pfizer is global.	4, 13, 40
Suxibuzone		Danilon Equidos		Janssen Animal Health, Buckinghamshire, UK	23
Dantrolene	Dantrolene i.v. "P & G"	Dantrolen		Vifor SA, Villars-sur-Glâne, Switzerland	21
		Dantrium	JHP Pharmaceuticals, LLC, Rochester, MI		
Desflurane		Suprane	Baxter Healthcare, Deerfield, IL	Baxter AG, Volketswil, Switzerland	18, 20, 21
Detomidine		Domosedan		Domosedan Gel Janssen Pharmaceuticals Beerse, Belgium	20
		Dormosedan	Pfizer Animal Health, New York, NY		20
	Equisedan ad us. vet.			Dr. E. Graeub AG, Bern, Switzerland	18, 19, 21, 22
Dexmedetomidine		Dexdomitor	Pfizer Animal Health, New York, NY	Orion Pharma, Espoo, Finland	20
		Precedex	Hospira Inc, Lake Forest, IL		20
Diazepam		Valium 5mg/mL		Roche Pharma Schweiz AG, Reinach, Switzerland	19, 20, 22
Diazepam injection	Diazepam		Abbott Animal Health, Abbott Park, IL		19, 20, 22
Diclofenac sodium		Surpass Topical Cream	Boehringer Ingelheim, St. Joseph, MO		7, 23, 82

Continued

Drug Name	Generic Drug Names	Trade Names	North American Manufacturer Information	European Manufacturer Information	Chapter(s)
Dimethyl sulfoxide (DMSO 20%)	DMSO			Streuli Pharma AG, Uznach, Switzerland	2, 15, 21, 37, 40, 44, 51, 54, 83, 85,
		Domoso	Fort Dodge Animal Health [Pfizer], Fort Dodge, IA		78, 82
Di-tri-octahedral smectite		Biosponge	Platinum Performance, Buellton, CA		2
Dobutamine Hydrochloride		Dobutrex	Eli Lilly and Company, Indianapolis, IN	Abbott Laboratories, Abbott Park, IL Teva Pharma AG, Aesch, Switzerland	20, 21
Dorzolamide hydrochloride		Trusopt	Merck & Co Inc, Whitehouse Station, NJ		58
Droperidol		Inapsine	Hospira Inc, Lake Forest, IL		22
Edrophonium		Enlon	Ohmeda PPD, Liberty Corner, NJ		58
		Reversol	Organon Pharmaceuticals, Allentown, PA		58
		Tensilon	Valeant Pharmaceuticals, Aliso Viejo, CA		58
	Elanone-V	Lenperone	A.H. Robins Co, Richmond, VA		22
Enoxaparin		Lovenox	Sanofi-Aventis U.S. LLC, Bridgewater, NJ		4
Epinephrine	Ephedrine		Akorn Pharmaceuticals Akron, OH		20
	Adrenalin Sintetica			Sintetica S.A., Mendrisio, Switzerland	22
	Adrenaline	Primatene Mist CFC Metered-Dose Inhaler	Cannot be manufactured or sold in the U.S. after December 2011		
		Marcaine	American Regent Inc, New York, NY		20
		Equiplas J	Plasvacc USA Inc, Templeton, CA		2
Etilefrine		Effortil		Boehringer Ingelheim Vetmedica GmbH, Ingelheim, Germany	32
Famotidine		Pepcid AC	Johnson & Johnson, Merck Consumer Pharmaceuticals Co, Fort Washington, PA		20

Drug Name	Generic Drug Names	Trade Names	North American Manufacturer Information	European Manufacturer Information	Chapter(s)
Fentanyl (transdermal)		Duragesic	JOM Pharmaceuticals, Raritan, NJ		20
		Durogesic		Janssen Pharmaceuticals, Beerse, Belgium	
Flumazenil		Anexate	Haorui Pharma-Chem Inc, Edison, NJ	Roche Pharma (Schweiz) AG, Reinach, Switzerland	22
		Romazicon	Hoffmann-La Roche Inc, Nutley, NJ; Various generic manufacturers	Roche Laboratories Inc, Basel, Switzerland	20
Flunixin	Flunixin meglumine		Various generic manufacturers		20, 79
		Banamine	Intervet/Schering-Plough Animal Health, Summit, NJ		
		Finadyne		Schering-Plough Animal Health	
Fluphenazine decanoate		pms-Fluphenazine decanoate	Pharmascience Inc, Montréal, Quebec, Canada		22
Guaifenesin		Guailaxin (not currently available)	Fort Dodge Animal Health, Fort Dodge, IO		20
		Gecolate (not currently available)	Summit Hill Laboratories, Navesink, NJ		20, 21
		Myolaxin 15% ad us. vet.		Vétoquinol AG, Ittigen, Switzerland	18, 19, 22
Halothane		Halothane B.P.		Arovet AG, Zollikon, Switzerland	18, 21
Hemoglobin glutamer-200 (bovine)		Oxyglobin	Biopure Corp, Boston, MA		3, 4
Hyaluronic acid		Acrivet Syn 2%		Acrivet, Inc, Hennigsdorf, Germany	58
		Hylartin V	Pfizer Animal Health, New York, NY (previously Pharmacia & Upjohn)		58
		Legend	Bayer Animal Health, Shawnee Mission, KS		79
		Conquer HA	HA Concepts, Inc, Lexington, KY		79
HydroxyEthyl Starch		HAES-steril 10%		Fresenius Kabi AG, Bad Homburg, Germany	21

Continued

Drug Name	Generic Drug Names	Trade Names	North American Manufacturer Information	European Manufacturer Information	Chapter(s)
Ibuprofen		Motrin	McNeil Consumer and Speciality Pharmaceuticals, Washington Park, PA		
		Advil	Pfizer Healthcare, Richmond, VA		
		Caldolor	Cumberland Pharmaceuticals, Nashville, TN		20
Isoflurane		Isoflo	Abbott Laboratories, Abbott Park, IL		18, 20
		IsoFlo ad us. vet.		Dr. E. Graeub AG, Bern, Switzerland	21
S (+)-ketamine		Keta-S ad us. vet.		Dr. E. Graeub AG, Bern, Switzerland	18, 19
Ketamine		Narketan 10 ad us. vet.		Vétoquinol AG, Ittingen, Switzerland	18, 19, 21, 22
		VetaKet	Lloyd Inc, Sheneadoah, IA		
		Ketaject	Teva Animal Health, St. Joseph, MO		
		Vetalar	Boehringer Ingelheim Vetmedica Inc, New York, NY		
		Ketaset	Fort Dodge Animal Health, Fort Dodge, IO		20
Ketoprofen		Ketofen	Fort Dodge Animal Health, Fort Dodge, IO		20, 79
Levallorphan		Lorfan	Hoffmann-La Roche Inc, Madison, WI	Hoffmann-La Roche, Inc, Genzach, Switzerland	20
Levomethadone		L-Polamivet ad us. vet.		Veterinaria AG, Pfäffikon, Switzerland	20, 22
Lidocaine	Lidocaine HCl 2% or 5%				18 (5%), 19 (2%), 22 (5%)
	Lidocaine HCl 2% injection		Various generic manufacturers		
		Xylocaine	American Pharmaceutical Partners (APP), Schaumburg, IL	Vedco Ltd, Berkshire, UK	20
		Xylocaine Jelly 2 %	American Pharmaceutical Partners (APP), Schaumburg, IL	Astra Zeneca, Vienna, Austria	20
Lorazepam		Ativan	Baxter Healthcare Corp, Deerfield, IL	Valeant Pharmaceuticals International, Zug, Switzerland	20

Drug Name	Generic Drug Names	Trade Names	North American Manufacturer Information	European Manufacturer Information	Chapter(s)
Medetomidine	Dorbene			Dr. E. Graeub AG, Bern, Switzerland	19, 21, 22
		Domitor (no longer sold or marketed in the U.S.; replaced by Dexdomitor)	Pfizer Animal Health, New York, NY		20
Meloxicam		Metacam	Boehringer Ingelheim Vetmedica Inc, St. Joseph, MO	Boehringer Ingelheim Ltd, Berkshire, UK	23
Methadone		Methadon Streuli		Streuli Pharma, AG, Uznach, Switzerland	22
Midazolam		Dormicum		Roche Pharma (Schweiz) AG, Reinach, Switzerland	18, 19, 20
		Versed	Abbott Laboratories, Abbott Park, IL; Numerous generic manufacturers		20
Mitomycin		Mutamycin	Bedford Laboratories, Bedford, OH		57
		MS CONTIN, Astramorph/PF, Avinza, DepoDur, Duramorph, Infumorph, Kadian, MS Contin, Oramorph SR, RMS, Roxanol	Professional Compounding Centers of America (PCCA), Houston, TX		16, 20
Morphine	Morphin HCl sintetica 10 mg			Sintetica S.A., Medrisio, Switzerland	18, 19, 21, 22
Naloxone		Naloxone		Bristol-Myers Squibb GmbH, Baar, Switzerland	18, 20, 22, 40
		Narcan	Various generic manufacturers (no longer marketed under this trade name in the U.S.)	Bristol-Myers Squibb GmbH, Baar, Switzerland	18, 22
Natamycin		Natacyn	Alcon, Fort Worth, TX		57
Norepinephrine		Levophed	Hospira Inc, Lake Forest, IL	Teva Parenteral Medicines, Inc, Lyon, France	20
Omeprazole		GastroGard, Ulcergard		Merial Ltd, Lyon, France	20
Pancuronium bromide		Pavulon	Organon Pharmaceuticals, Allentown, PA		58

Continued

Drug Name	Generic Drug Names	Trade Names	North American Manufacturer Information	European Manufacturer Information	Chapter(s)
Phenylbutazone		Butadion ad us. vet.	Not marketed in the U.S.	Streuli Pharma AG, Uznach, Switzerland	21
	Phenylbutazone USP		Various generic manufacturers	Vedco Ltd, Berkshire, UK	20
Phenylephrin		AK-Dilate, Lusonal, Mydfrin 2.5%, Neo-Synephrine, Phenoptic, Rhinall, Sudogest PE, Vicks Sinex, Sudafed PE		Laboratorium Dr. G. Bichsel AG, Interlaken, Switzerland	22
Phenylephrine		Quartuss		G. Streuli & Co AG, Uznach, Switzerland	21, 22
	Phenylephrine Hydrochloride		Baxter Healthcare Corp, Deerfield, IL		20
		Neo-Synephrine HCl, 10mg/mL	Winthrop Pharmaceuticals, New York, NY		37
Hyaluronic acid + chondroitin sulfate + glucosamine		Polyglycan	Arthrodynamic Technologies LLC, Versailles, KY		97
Polymyxin B		Neosporin	X-GEN Pharmaceuticals, Big Flats, NY		2, 4, 36, 37, 40, 57, 58, 85
Promazine HCl		Primazine, Prozine, "acepromazine"	Fort Dodge Animal Health, Fort Dodge, IO		22
		Sparine	Wyeth Laboratories, Philadelphia, PA		22
Propionylpromazine		Combelen		Bayer AG, Leverkusen, Germany	22
		Tranvet Injectable Solution	Fort Dodge Animal Health, Fort Dodge, IO		22
Propofol		PropoFlo, PropfFlo	Abbott Laboratories, Abbott Park, IL		20, 28
	Propofol 1% MCT Fresenius			Fresenius Kabi (Schweiz) AG, Stans, Switzerland	19, 21
Ranitidine		Zantac	GlaxoSmithKline, Research Triangle Park, NC	Boehringer Ingelheim, Ltd, Berkshire, UK	20
Reserpine	Reserpine	No brand name	Wedgewood Pharmacy, Swedesboro, NJ		22
Romifidine		Sedivet		Boehringer Ingelheim Ltd, Berkshire, UK	18, 19, 20, 21, 22, 48
Salmonella typhimurium antiserum		Endoserum	Immvac Inc, Columbia, MO		2
Sarmazenil		Sarmasol		Dr. E. Graeub AG, Bern, Switzerland	18, 19, 20, 22

Drug Name	Generic Drug Names	Trade Names	North American Manufacturer Information	European Manufacturer Information	Chapter(s)
Sevoflurane		Sevoflo	Abbott Laboratories, Abbott Park, IL		20
		Sevorane		Abbott AG, Baar, Switzerland	18, 21
Sodium chloride, Sodium acetate anhydrous, Sodium gluconate, Potassium chloride, and Magnesium chloride		Normosol-R	Abbott Animal Health, Abbott Park, IL		1, 3, 20, 40
Azaperone		Stresnil ad us. vet.	Janssen Pharmaceutials NV, Summit, NJ	Janssen Pharmaceuticals, Beerse, Belgium	22
Thiopental sodium		Pentothal	Hospira [Abbott], Lake Forrest, IL	Ospedalia AG, Hünenberg, Switzerland	22
Timolol maleate		Timoptic	Merck & Co Inc, Whitehouse Station, NJ		58
Tolazolin		Tolazolin HCl 120 mg ml^{-1}		Kantonsapotheke Zurich, Zurich, Switzerland	22
Vasopressin		Pitressin	American Regent Inc, New York, NY		20
Vedaprofen		Quadrisol		Pfizer Ltd, Kent, UK	23
Xylazine		Rompun	Bayer Corporation, Shawnee, MI		20, 48
	Xylazine		Various generic manufacturers		
	Xylazin Streuli ad us. vet.		Glenwood LLC, Englewood, NJ	Streuli Pharma SA, Uznach, Switzerland	18, 19, 21, 22
Yohimbine		Yocon			20
		Yobine Injection	Lloyd Inc, Shenandoah, IA		
	Actibine, Aphrodyne, Baron-X, Dayto Himbin, PMS-Yohimbine, Prohim, Thybine, Yocon, Yohimar, Yohimex, Yoman, Yovital		Sigma Chemicals Co, Saint Louis, MO		22, 40
Zolazepam		Zoletil ad us. vet		Virbac Schweiz AG, Glattbrugg, Switzerland	19, 22
Zolazepma/ Tiletamine		Telazol	Fort Dodge Animal Health [Pfizer], New York, NY		19, 22

APPENDIX

B Products and Manufacturers

Product Name	Manufacturer Information	Chapter(s)
10% povidone iodine solution	Betadine Purdue Pharma, Stamford, CT	5, 7, 9, 12, 56, 57
18-French equine enteral feeding tube	Mila International, Florence, KY	3, 37
2.7-mm reconstruction plate	Synthes, West Chester, PA	48, 93
20% acetylcysteine solution	Ben Venue Inc, Bedford, OH	37
3M Custom Support Foam	3M Animal Care Products, St. Paul, MN	17
4.0-mm cortex screws	Synthes Inc, West Chester, PA	90
45-cm–long Deschamps needle	Eickemeyer, Tuttlingen, Germany	16, 38
5 Dacron	Deknatel Inc, Fall River, MA	38
6 French venous thrombectomy catheter	Fogarty-Edwards Laboratories, distributed by American V. Mueller, Chicago, IL	46
6-F introducer system	Cook Medical Inc, Bloomington, IN	46
6-F single-end-hole nylon angiographic catheter	Cook Medical Inc, Bloomington, IN	46
90-degree hook radiofrequency probe	Stryker Endoscopy, San Jose, CA	91
Abcocide	Abco Dealers Inc, LaVergne, TN	9
Accel HLD 5	ViroxTechnologies, Oakville, Ontario, Canada	9
ACell	ACell Inc, Jessup, MD	27, 83
Acutrack Equine Screw	Acumed Veterinary, Hillsboro, OR	76
Adaptic	Johnson & Johnson Inc, Fort Dodge, IA	25
Adi-Zyme	STERIS Corp, Mentor, OH	9
AMD Foam	Covidien, Mansfield, MA	26
AMSCO Sterilization Container System	STERIS Corporation, Mentor, OH	9
anchor screws	Veterinary Instrumentation, Sheffield, UK, www.vetinst.com	97
Anioxyde 1000	Clinipak Medical Products, Bourne End, UK	9
AquaPacer equine underwater treadmill	Ferno Veterinary Systems, Wilmington, OH	103
Arcadis orbic 3D C-arm	Siemens AG, Munich, Germany	13, 97
Arnold-Bruning intracordal injection syringe, catalog No. 7754	Storz Instrument Company, St. Louis, MO	45
Arrow catheter	Arrow International, Reading, PA	6
Ascal	Dagra Pharma, Ax Diemen, Netherlands	13
Asepti-zyme	Ecolab Healthcare, Mississauga, Ontario, Canada	9
Avagardä	3M Animal Care Products, St. Paul, MN	7, 9, 10
Avitene	Davol Inc, Warwick, RI	4
Bailey-Gibbon rib contractor	Miltex Inc, York, PA	48
Bair Hugger	Arizant Healthcare Inc, Eden Prairie, MN	20
Baktolin Balm	Bode Chemie, Hamburg, Germany	10
Betadine surgical scrub	Purdue Frederick Co, Norwalk, CT	9, 10, 55, 56, 58, 61, 90
Bio-Oss	Geistlich, Wolhusen, Switzerland	77
Biopsy Punch Rongeurs	Sontec Instruments, Centennial, CO	91
Biopsy Suction Punch Rongeur	Dyonics, Smith & Nephew, Memphis, TN	91
Callicrate Bander	No-Bull Enterprises, St. Francis, KS	60
Canevasit	Amsler und Frei, Schinznach Dorf, Switzerland	76
Cardell	Minrad Inc, Orchard Park, NY	20
Cellulose sponges	Weck-Cel Medtronic, Jacksonville, FL	56
cervical fusion plate	Synthes Inc, West Chester, PA	94
ChloraPrep	Medi-Flex Inc, Leawood, KS; Cardinal Health, Dublin, OH, www.cardinal.com	9
Chlorhexidine diacetate 2%	Nolvasan Solution and Surgical Scrub, Fort Dodge Laboratories Inc, Fort Dodge, IA	5, 9

Product Name	Manufacturer Information	Chapter(s)
Chlorhexidine gluconate 4%	Hibiclens, Stuart Pharmaceuticals, Division of ICI America Inc, Wilmington, DE	7, 9, 46
ChronOs Inject	Synthes Inc, Solothurn, Switzerland	77, 89
CicaCare	Smith-Nephew Canada Inc, St-Laurent, Quebec, Canada	5
Cidex	Johnson & Johnson's Advanced Sterilization Products, Fort Dodge, IA	9
Cidex OPA		
CleantopWM-S	CBC Medical Device Group, Duesseldorf, Germany	9
Closed fenestrated drain system	International Win Ltd, Kennett Square, PA, www.internationalwin.com	40
CM Heal	CM Equine Products, Norco, CA, www.cmequineproducts.com	40
Coil sets	International Win Ltd, Kennett, Square, PA, www.internationalwin.com	3
COLLOSs E	OSSACUR AG, Oberstenfeld, Germany	77
Comply EO chemical integrators	3M, St. Paul, MN	9
Conray 400	Mallinckrodt Medical Inc, St. Louis, MO	46
Cosequin ASU	Nutramax Laboratories Inc, Edgewood, MD	79
crimping sleeve	Synthes Inc, West Chester, PA	81
CTFN 135	Yocan Medical, Thornhill, Ontario, Canada	46
Curasalt, 20% hypertonic saline on a Kerlix gauze	Tyco Healthcare/Kendall, Mansfield, MA	26
Dallmer cuff shoes	Nanric Inc, Versailles, KY	87
Davol-Simon skin graft dermatome	Davol Inc, Cranston, RI	25
diathermic snare	Olympus Optical Co, Irving, TX	46
Disposable Skin Stapler	Richard-Allen Medical, Richland, MI	36
Dura-Prep Surgical Solution	3M Health Care, St. Paul, MN	9
Dynacast Prelude	Smith & Nephew, Memphis, TN	87
Dynamap	Critikon, Tampa, FL	20
Dynamic Locking Head Screw (DLS)	Synthes Inc, Solothurn, Switzerland	76
Dynavet	Veterinary Dynamics, Templeton, CA	3
Dyonics	Smith & Nephew, Memphis, TN	91
Elasticon	Johnson & Johnson, Sommerville, NJ	39, 76, 86
electric dermatome	Zimmer, Warsaw, IN	25
electrolyzed weak acid water	Sterilox Technologies Inc, Radnor, PA	9
Embolization coils	Cook Medical Inc, Bloomington, IN	13, 46
Endo-GIA 30	United States Surgical Co, Norwalk, CT	59
Endopath Optiview surgical trocar	Ethicon Endo-Surgery Inc, Cincinnati, OH	34
Endostitch 10-mm Suturing Device	Covidien, Mansfield, MA	62
Endozyme	Ruhof, Mineola, NY, www.ruhof.com	9
Endozyme AW Plus		
Endurasplint 2	Carapace Inc, New Tazewell, TN	86
EnSeal SurgRx	Ethicon Endo-Surgery Inc, Cincinnati, OH	13, 32, 59
Enterotomy table	Kimzey Metal Products, Woodland, CA	37
Enzol	Johnson & Johnson, New Brunswick, NJ	9
Enzygnost	Dade-Behring Inc, Wilmington, DE	4
Equilox	Equilox International, Pine Island, MN	87
Equine Large Fragment Set	Synthes Inc, West Chester, PA	76
Equine laryngeal forceps	Karl Storz, Tuttlingen, Germany	44
ES-10	No-Bull Enterprises, St. Francis, KS	60
Exhalyzer V	Eco Medics, Dürnten, Switzerland	42
EZ Drape Adhesive	Clinipad Corp, Rocky Hill, CT	9
E-Z Pass Foal Enema Kit	Animal Reproduction Systems, Chino, CA, www.arssales.com	37
Fiberwire	Arthrex Inc, Naples, FL	16, 44
fine silica or aluminium powder	Harltons, Elmwood, WI	30
Finochietto retractor with long blades	Sontec Instruments, Centennial, CO	11, 61
First + Ice cold packs	MacKinnon Inc, San Diego, CA, www.mackinnonicehorse.com	103
FlapFix system	Synthes Inc, West Chester, PA	102

Continued

Product Name	Manufacturer Information	Chapter(s)
Fleet enemas	CB Fleet Co Inc, Lynchburg, VA	37
FloSeal	Baxter International Inc, Deerfield, IL	4
GC 8/95	Laboratories Nycomed S.A., Paris, France; Yocan Medical Systems, Thornhill, Ontario, Canada	46
Gelfoam	Pfizer, New York, NY	4, 55
GG-Free Horse Serum	Gibco Laboratories Inc, Grand Island, NY	25
GIA Premium 55	United States Surgical, Norwalk, CT	37
Gigasept FF	Schülke & Mayr UK Ltd, Meadowhall, Sheffield, UK	9
GmbH	Sterigenics, Waldems-Esch, Germany	9
GoldValve Balloon	Laboratories Nycomed, Zurich, Switzerland	46
Gomco Equipment	Chemetron Medical Products, Buffalo, NY	46
Guttural Pouch Catheter	Cook Veterinary Projects, Bloomington, IN Mila International, Florence, KY	46
GVB 17	Yocan Medical Systems, Thornhill, Ontario, Canada	46
Gzyme	Germiphene Corp, Brantford, Ontario, Canada	9
Headless, tapered screws	Acutrak, System Accumed, Hillsboro, OR	94
heavy-duty 75-cm forcep with Oschner-type jaws	Sontec Instruments, Centennial, CO	15
HemCon	HemCon Medical Technologies Inc, Portland, OR	4
Henderson Equine Castrating Instrument	Stone Manufacturing and Supply Co, Kansas City, MO	59
Heparin Leo	Leo Pharmaceutical Products, Parsippany, NJ	13
Herbert screw	Zimmer Orthopedics, Warsaw, IN	76
Hibisoft	Sumitomo Pharmaceutical Co, Osaka, Japan	10
Hibisol	Promed, Killorglin, Ireland	10
Hydrocollator moist heat pack	Chattanooga Group, Vista, CA	103
Hydrox	Virox Technologies, Mississauga, Ontario, Canada	9
I-Blade Technology	Ethicon Endo-Surgery Inc, Cincinnati, OH	13
Ice Tape cold therapy leg wrap	Dover Saddlery, Littleton, MA, www.doversaddlery.com	103
IDEXX VetStat	IDEXX Laboratories Inc, Westbrook, MN	20
Instat	Ethicon, Somerville, NJ	4
Interpulse	Stryker Corporation, Kalamazoo, MI	5
intra-osseous catheters	Cook Medical Inc, Bloomington, IN	7, 85
Ioban	Johnson & Johnson, Raritan, NJ	9
Ioban 2	3M Animal Care Products, St. Paul, MN	9, 10, 21, 76
Isopropyl alcohol, 74% w/v	3M Health Care, St. Paul, MN	10
i-STAT System	Abbott Point of Care Inc, Princeton, NJ	20
KCC	Wilson Tools and MFG, Spokane, WA	52
Keralit	Scheule GmbH, Kirchheim, Germany	90
Kerlix AMD	Tyco Healthcare/Kendall, Mansfield, MA	26
Kimberly-Clark InteguSeal Microbial Seal	Kimberly-Clark, Dallas, TX	10
Klenzyme	STERIS Corp, Mentor, OH	9
laparoscopic ligation instruments	Richard Wolf Company, Tuttlingen, Germany	13
large pointed reduction forceps	Synthes Inc, West Chester, PA	76, 91, 92, 95
large wire passer	Synthes Inc, West Chester, PA	81
LCP technology	Synthes Inc, West Chester, PA	48, 81, 94
LigaSure Atlas Laparoscopic Sealer/divider Instrument	Valleylab, Boulder, CO; Covidien, Mansfield, MA	13, 16, 32, 55, 59, 61, 62
Linatex	Four D Rubber Co Ltd, Heanor, Derbyshire, UK	21
Locking Compression Plate	Synthes Inc, West Chester, PA	52, 53, 76, 91
long-handled, right-angled alveolar curettes	Kruuse Worldwide, Marslev, Denmark	30
Marlex Mesh #1266	Dowd Inc, Providence, RI	36
McCoy's 5A Medium	Gibco Laboratories Inc, Grand Island, NY	25
Medical Adhesive	Hollister Inc, Libertyville, IL	9
memory-helical polyp retrieval basket	Cook Medical Inc, Bloomington, IN	46
Mesh Skin Graft Expander, No. Z-PD-170	Padgett Instruments Inc, Kansas City, MO	25
Metrizyme	Metrex Research Division of Sybron Canada Ltd, Morrisburg, Ontario, Canada	9
Mini-Tourquer	Laboratories Nycomed, Zurich, Switzerland	46
Monocryl	Ethicon, Somerville, NJ	44

Product Name	Manufacturer Information	Chapter(s)
morcellator	Lumenis Inc, Santa Clara, CA	59, 62
MTS Bionix 858	MTS Systems Corporation, Eden Prarie, MN	76
Multifire GIA-80 or ILA-100	United States Surgical Corp, Norwalk, CT	36
N-acetylcysteine	Sigma Chemical Company, St Louis, MO	37
No. 5 Ticron	Sherwood-Davis & Geck, St. Louis, MO	45
Nu Cidex 0.35%	Johnson and Johnson's, Fort Dodge, CA	9
Octenidine dihydrochloride	Schülke & Mayr GmbH, Norderstedt, Germany	9
Ocular conformers	Jardon Plastics Research Corporation, Southfield, MI Dallas Eye Prosthetics, Dallas, TX	55
Ometech OPTI CCA-TS blood gas analyzer	AVL Scientific Inc, Rosswell, GA	20
Omnicide 28	Baxter Healthcare Corp, Deerfield, IL	9
Omniguide	Omniguide, Cambridge, MA, www.omni-guide.com	15
Optim22	Virox Technologies, Mississauga, Ontario, Canada	9
parathyroid peptide (PTH1-34) -enriched fibrin hydrogel	Kuros Inc, Zurich, Switzerland	89
Pinless External Fixator	Synthes Inc, West Chester, PA	76
Pinnacle Introducer Sheath	Medi-Tech, Natick, MA	46
PIP arthrodesis plate	Synthes Inc, West Chester, PA	81
Plastazote	Smith & Nephew, Hull, UK	87
plastic bags and vacuum-collection glass bottles	Baxter Fenwal, Deerfield, IL MWI Veterinary Supply, Meridian, ID	4
Platinum Performance CJ	Platinum Performance Inc, Buelton, CA	79
Pleur-Evac Thoracic Catheter	Teleflex Medical, Research Triangle Park, NC; Genzyme Biosurgery, Fall River, MA	40, 48
positive-profile pins	IMEX Veterinary Inc, Longview, TX	76
Premade AIPMMA with tobramycin	Stryker, Mahwah, NJ	85
Quarpel	Natick Research Laboratories, Natick, MA	10
RachetDis	AO Foundation, Davos, Switzerland	102
Redden brace	International Equine Podiatry Center, Versailles, KY	86
resin-impregnated foam padding	3M Corporation, St. Paul, MN	76
Release	Johnson & Johnson Products Inc, New Brunswick, NY	25
retractable, swedged-on 23-gauge needle	Mill-Rose Laboratories. Inc, Mentor, OH	43
R-gel	Royer Animal Health, Frederick, MD	85
Rotating Y Adapter	Medi-Tech, Natick, MA	46
rotational thromboelastometry	ROTEM, Munich, Germany	4
SCCLRS	Securos Veterinary Orthopedics, Charlton, MA	48
Schirmer tear test strips	Schering-Plough Animal Health, Kenilworth, NJ	56
Screw Retrieval Set	Synthes Inc, West Chester, PA	76
Scrub Care Skin Prep Tray	Cardinal Health, Dublin, OH, www.cardinal.com	9
Securos Equine Tie-Back System	Securos, Chalton, ME	45
Sekusept Aktiv	Ecolab Center, St. Paul, MN	9
Seldrill Schanz Screw	Synthes Inc, West Chester, PA	76
Septi-Chek	Becton Dickinson, Sparks, MD	85
Siemens Magnetom Espree	Siemens Medical Solutions USA Inc, Malvern, PA	99
Sigafoos	Sound Horse Technologies Inc, Unionville, PA	76, 90
Silicone implant	Jardon Plastics Research Corporation, Southfield, MI	55
Siremobile ISO 3D	Siemens Medical Solution, Erlangen, Germany	93
Smart Screw	ConMed Linvatec Biomaterials Ltd, Tampere, Finland	86
Snyder Hemovac	Zimmer, Dover, OH	17, 40
Sonoclot analyzer	Sienco, Arvada, CO	4
Sporox	Reckitt & Colman, Montvale, NJ	9
StarFill 2B dual-cure composite	Danville Materials, San Ramon, CA	30
Sterilit Power Spray	Aeculap AG, Tuttlingen, Germany	9
Sterilization fleece	Salzmann Medico, St. Gallen, Switzerland	9
Sterillium	Bode-Chemie, Hammburg, Germany	7, 9, 10
STERIS 0.20%,	STERIS Corporation, Mentor, Ohio, and STERIS Limited, STERIS House, Basingstoke, Hampshire, UK	9
Steriset Containers	Wagner GmbH, Munich, Germany	11

Continued

Product Name	Manufacturer Information	Chapter(s)
Sterizyme	Anderson Products, Haw River, NC	9
Sterrad Sterilization System	Advanced Sterilization Products, a division of Johnson & Johnson Medical Inc, Irvine, CA	9
Stryker Electric Dermatome	Stryker Electro-Surgical Unit, Kalamazoo, MI	25
Superglue	Loctite Co, Cleveland, OH	25, 76
Surgical Simplex P-Stryker	Stryker, Kalamazoo, MI	85
Surgicel	Ethicon, Somerville, NJ	4, 32
Surgimed	Hospital Marketing Services Inc, Naugatuck, CT	39
TA 90, TA-90 Premium	United States Surgical Corp, Norwalk, CT	16, 37, 38, 62
Techni-care	Care-Tech Laboratories Inc, OTC Pharmaceuticals, St. Louis, MO	10
Technovit	Jorgensen Laborstories, Loveland, CO	102
Telfa Sterile Pads	Kendal Co, Hospital Products, Boston, MA	25, 86
TetraVisc viscous ophthalmic drops 0.5%	OCuSOFT, Richmond, TX	56
Throat Support Device	Vet-Aire Inc, Ithaca, NY, www.Vet-Aire.com	44
Thrombin-JMI	King Pharmaceutical Inc, Bristol, TN	4
Thromboelastography	Haemoscope, Niles, IL	4
TISSEEL	Baxter, Deerfield, IL	4
Titanium Corkscrew	Arthrex Medical Instrument Company GmbH, Karlsfeld, Germany	45
Tonopen	Medtronic, Jacksonville, FL	58
TonoVet	AcriVet, Salt Lake City, UT	58
TraumaDex	Medafor Inc, Minneapolis, MN	4
Tristel	Tristel Co Ltd, Snailwell, UK	9
Type U-10M, 25 kN, 250 Nm	HBM, Darmstadt, Germany	76
UFN	Synthes Inc, Solothurn, Switzerland	76
Ultra-high-molecular weight polyethylene (UHMWPE) cable	Securos, Charlton, MA	76
Unilock system	Synthes Inc, West Chester, PA	94, 102
uterine biopsy forceps	Richard Wolf Medical Instruments Corporation, Vernon Hills, IL	32, 44
Variable Angle LCP Distal Radius System	Synthes Inc, West Chester, PA	76
VersaTron 12 mm probe	High Medical Technologies, Lengwil, Switzerland	79
VetGATE	ARTORG Center for Biomedical Engineering Research, University of Bern, Bern, Switzerland	13, 90
Vettec	Vettec Hoof Care Products, Utrecht, Netherlands	90
Vicryl	Ethicon, Somerville, NJ	7, 16, 36, 37, 39, 44, 55, 56, 58, 66
Virkon	Antec International Ltd., Sudbury, Suffolk, UK	9
Vulketan gel	Jannsen Animal Health, Toronto, Ontario, Canada	5
Watson skin grafting knife	Padgett Instruments Inc, Kansas City, MO	25
Zimmer Meshgraft Dermatome	Zimmer, Warsaw, IN	25
Z-PD-100R	Padgett Instruments Inc, Kansas City, MO	25

INDEX

Page numbers followed by *f* indicate figures; *t*, tables; *b*, boxes.

M

N